Genetic Correlation Patterns Between Somatic Cell Score and Protein Yield in the Italian Holstein-Friesian Population

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ABSTRACT

Genetic parameters for somatic cell score (SCS) in the Italian Holstein-Friesian population were estimated addressing the pattern of genetic correlation with protein yield in different parities (first, second, and third) and on different days in milk within each parity. Three approaches for parameter estimation were applied using random samples of herds from the national database of the Italian Holstein Association. Genetic correlations for lactation measures (305-d protein yield and lactation SCS) were positive in the first parity (0.31) and close to zero in the second (0.01) and third (0.09) parities. These results indicated that larger values of SCS were genetically associated with increased production. The second and third sets of estimates were based on random regression test-day models, modeling the shape of lactation curve with the Wilmink function and fourth-order Legendre polynomials, respectively. Genetic correlations from both random regression models showed a specific pattern associated with days in milk within and across parities. Estimates varied from positive to negative in the first and second parity, and from null to negative in the third parity. Patterns were similar for both random regression models. The average overall correlation between SCS and protein yield was zero or slightly positive in the first lactation and ranged from zero to negative in later lactations. Correlation estimates differed by parity and stage of lactation. They also demonstrated the dubiousness of applying a single genetic correlation measure between SCS and protein in setting selection strategies. Differences in magnitude and the sign of genetic correlations between SCS and yields across and within parities should be accounted for in selection schemes.

Key words: somatic cell score, protein yield, genetic correlation pattern, Holstein-Friesian cow

INTRODUCTION

Mastitis is one of the most frequent and costly diseases in dairy herds. Direct selection for mastitis resistance is a current practice only in Nordic European countries (Denmark, Finland, Norway, and Sweden) where diseases are routinely registered in a national database. Heritability estimates for mastitis incidence are generally <0.06 (for a review, see Heringstad et al., 2000), making genetic improvement for this trait difficult.

Direct measurement of clinical mastitis incidence (Heringstad et al., 2000) is costly and difficult to implement in national recording schemes. Somatic cell count, generally log-transformed into SCS, is used for indirect selection for mastitis resistance in many formal dairy selection programs (Interbull, 2008). Heritability of SCS is greater than heritability of mastitis incidence, and genetic correlation between SCS and mastitis incidence is large (Mrode and Swanson, 1996; Heringstad et al., 2000). The estimates of genetic correlation between SCS and clinical mastitis were around 0.70 for all lactations (Carlén et al., 2004; Koivula et al., 2005).

Initially, EBV for SCS were based on lactation measures, but the implementation of test-day models (TDM) in national evaluations has allowed for an analysis of test-day SCS (Mrode and Swanson, 1996). Random regression TDM (RR-TDM) can be used to estimate genetic merit of animals for SCS for each parity and day of lactation (Jamrozik and Schaeffer, 1997).

With the application of multiple-trait RR-TDM, estimation of breeding values accounts for patterns of relationships between traits and along a time trajectory. On the other hand, the weighting of EBV in selection indices often assumes constant relationships among traits across time and does not account for possible patterns of genetic relationship among traits within and between parities. Somatic cell score is strongly affected by stage of lactation, parity, season, and breed, as well as udder infections and their causative pathogens, and

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Table 1. Number of records, SCS lactation mean (LSCS) and SD, and 305-d protein yield lactation mean (P_{305}) and SD, by parity

Parity	Records, n	LSCS (score)		P_{305} (kg)	
		Mean	SD	Mean	SD
First	13,746	3.09	1.23	269	47
Second	8,357	3.46	1.31	296	55
Third	4,428	3.78	1.39	300	57

has a clear pattern of changes in phenotypes along the lactation (de Haas et al., 2002).

This research was implemented from the analyses performed in different years during a doctoral study period on the subject (Samoré, 2003). The primary aim of this study was to analyze the pattern of genetic correlations between SCS and protein yield, the production trait with the largest emphasis in most selection programs in dairy breeds. Other objectives were to suggest a biological interpretation of the pattern of genetic correlations and to discuss how to account in a selection goal for the variation in genetic correlation between SCS and protein yield along the lactation and between parities.

MATERIALS AND METHODS

Data

Monthly test-day (TD) records for SCC and protein yield of Italian Holstein Friesian cows were provided by provincial milk recording agencies. Somatic cell counts were log-transformed to SCS: $SCS = \log_2 [(SCC/100,000) + 3]$. Records were removed when date of calving was not available or when the test date occurred before 5 or after 305 DIM. No restrictions were imposed on the number of TD records per lactation, but a TD record was required to include information on both traits. Three different data sets were used for variance component estimation according to the model of analysis. For each data set, the specific pedigree file was extracted from the Holstein national herd book by tracing back 3 generations.

Variance Component Estimation

The estimates of variance components were obtained with a lactation model (LACM) and 2 RR-TDM, modeling the shape of lactation curve with either the Wilmlink function (WF-TDM) or Legendre polynomials (LP-TDM).

Because of computing limitations, estimation of variance components using the complete data set was not feasible and small subsets of data were created by

selecting all records pertaining to randomly sampled herds. Sampling of herds was always made from the most complete national database available when the analysis was conducted. This included data from 1989 to 1998 for the LACM, from 1990 to 1998 for WF-TDM, and from 1992 to 2002 for the LP-TDM. The data sets were independently sampled and the sizes of data sets were targeted to computer facilities available at the time of each analysis. The analyses were performed during the study for a doctoral thesis on genetic aspects of SCC (Samoré, 2003) justifying, by consequence, the different time periods of the 3 data sets used for variance component estimations.

Lactation Model. A data set of 5,292,362 lactation records was available with complete information on SCS and protein yield. The SCC test-day records collected from 5 to 305 DIM were first log-transformed into SCS test-day records and the lactation SCS value (LSCS) was calculated as the mean of SCS test-day records. Only the first 3 parities were considered, with first-parity records required for all cows. Sampling of records produced a data set of 26,531 lactations from 13,746 cows (Table 1). Age at calving ranged from 20 to 58 mo and 4 seasons of calving were defined as January to March, April to June, July to September, and October to December. The model to estimate variance components included the fixed effects of herd-year-season and the interaction of age at calving in months by season of calving. The additive genetic and the residual were included as random effects.

Variance components across traits and parities were estimated in 11 separate multiple-trait runs: 2 trivariate runs within trait (LSCS or 305-d protein yield) and across parity, considering the phenotype in each parity as a different dependent variable, and 9 bivariate runs across traits, within and across parities. Genetic parameters and (co)variance components for LSCS and protein yield were estimated using ASREML (Gilmour et al., 1995).

Test-Day Models. The database of Italian Holstein-Friesian cattle included 10,955,039 TD records from the first 3 parities collected from 1984 to 2001. The random sampling yielded a data set of 82,368 observations from 5,675 cows. Herds included at least 1,000 TD records

Table 2. Heritabilities (on diagonal), genetic (above the diagonal), and permanent environmental (below) correlation coefficients for SCS lactation mean (LSCS) and 305-d protein yield (PY₃₀₅), in first (LSCS1, PY₃₀₅1), second (LSCS2, PY₃₀₅2), and third (LSCS3, PY₃₀₅3) parity (SE in parentheses)

	LSCS1	LSCS2	LSCS3	PY ₃₀₅ 1	PY ₃₀₅ 2	PY ₃₀₅ 3
LSCS1	0.18 (0.02)	0.93 (0.04)	0.87 (0.07)	0.31 (0.06)	0.06 (0.08)	0.15 (0.11)
LSCS2	0.25 (0.02)	0.21 (0.03)	0.92 (0.05)	0.20 (0.08)	0.01 (0.09)	0.10 (0.12)
LSCS3	0.18 (0.02)	0.36 (0.03)	0.21 (0.04)	0.26 (0.11)	0.10 (0.13)	0.09 (0.16)
PY ₃₀₅ 1	-0.16 (0.02)	0.00 (0.02)	0.05 (0.03)	0.30 (0.02)	0.90 (0.03)	0.82 (0.04)
PY ₃₀₅ 2	-0.07 (0.02)	0.18 (0.03)	0.02 (0.03)	0.43 (0.02)	0.31 (0.03)	0.92 (0.03)
PY ₃₀₅ 3	-0.02 (0.03)	0.10 (0.12)	0.21 (0.04)	0.32 (0.03)	0.59 (0.03)	0.35 (0.04)

from 1990 to 1998. All production traits (milk, fat, and protein yield) and SCS had to be available for each TD. For each parity, 2 classes of age by 4 seasons of calving were created. Thresholds for the 2 classes of age within parity were 28 mo for first, 41 mo for second, and 55 mo for third parity. Seasons were defined in the same way as for the LACM. The number of herd-test-day levels was 2,544 for first, 2,197 for second, and 1,725 for the third parity.

The model was a multiple trait RR-TDM animal model. The model for trait *i* (milk, fat, protein or SCS) in lactation *j* (1, 2, or 3) was:

$$y_{ijtlmno} = HTD_{ijl} + \sum_{k=1}^q b_{ijnk} z_{tk} + \sum_{k=1}^q a_{ijmk} z_{tk} + \sum_{k=1}^q p_{ijmk} z_{tk} + e_{ijtlmno},$$

where $y_{ijtlmno}$ was the record *o* on cow *m* made on day *t* within herd-test-day effect *l*, for a cow in the subclass *n* for season-age of calving, HTD_{ijl} was fixed herd-test-day effect, b_{ijnk} were fixed regression coefficients specific to subclass *n*, a_{ijmk} were random regression coefficients specific to cow *m*, p_{ijmk} were random permanent environmental coefficients specific for cow *m*, $e_{ijtlmno}$ was the residual effect for each observation, and z_{tk} were covariates assumed to be the same for both fixed and random regressions. For the WF-TDM, fixed and random regressions were Wilmink's functions (Wilmink, 1987) defined as:

$$W(t) = w_0 + w_1 t + w_2 \exp^{(-0.05t)},$$

with number of regression parameters (*q*) equal to 3.

Random residual effects on different DIM were considered uncorrelated within and between individuals. Residual covariances differed across the 4 stages in each lactation: 5 to 45 DIM, 46 to 115 DIM, 116 to 265 DIM, and 266 to 305 DIM. A Bayesian approach with Gibbs sampling, as described in Jamrozik et al. (1998), was used to obtain means of posterior distributions for all parameters of the model. Two chains of 55,000

samples were generated. The first 5,000 samples were discarded as the burn-in period in each chain. Estimates were averaged across the chains.

A second RR-TDM was applied, where fixed and random regressions were Legendre polynomials of order 4 (LP-TDM) with number of regression parameters (*q*) equal to 5. All other assumptions were the same as in WF-TDM. The data set for the LP-TDM included 141,696 records from 10,275 cows, collected from 1992 to 2002. Estimates of (co)variances were obtained with a Bayesian method using Gibbs sampling. A single chain with a length of 100,000 was generated discarding the first 5,000 samples (burn-in period).

Genetic (co)variances, heritabilities, and genetic correlations for protein yields and SCS were estimated for each day of lactation from 5 to 305 d (Jamrozik and Schaeffer, 1997). Only results related to SCS and protein yield will be presented in this paper.

RESULTS

Lactation Model

Heritability estimated from lactation records (Table 2) increased from the first to later parities, both for LSCS (0.18 to 0.21) and for protein yield (0.31 to 0.35). Within trait, strong genetic correlations between parities were found, with values greater than 0.90 for adjacent parities and smaller values (>0.80) between first and third parity.

The estimate of genetic correlation between protein and LSCS in the first parity was 0.31, indicating that an increase in lactation protein yield was associated with larger LSCS. The genetic correlation between LSCS and protein decreased to values close to zero in the second (0.01) and third (0.09) parity.

Test-Day Model with Wilmink Function

Average daily heritabilities (Table 3) varied between 0.15 and 0.25 for SCS and from 0.30 to 0.38 for protein yield. Daily heritabilities from 5 to 305 DIM (Figure

Table 3. Average daily heritabilities (on diagonal), genetic (above diagonal) and permanent environmental (below diagonal) correlations for SCS and protein yield (PY), in first (1), second (2), and third (3) parity from random regression test-day models with Wilmink function (WF-TDM) and with Legendre polynomials (LP-TDM)

Model	SCS1	SCS2	SCS3	PY1	PY2	PY3
WF-TDM						
SCS1	0.15	0.29	0.26	0.02	0.09	0.08
SCS2	0.28	0.19	0.4	0	0.14	0.12
SCS3	0.2	0.41	0.25	0.01	0.05	0.14
PY1	-0.11	0.04	0.03	0.3	0.52	0.45
PY2	0	0.19	0.09	0.33	0.35	0.6
PY3	0.01	0.1	0.24	0.18	0.33	0.38
LP-TDM						
SCS1	0.17	0.33	0.32	0.08	0.02	0.04
SCS2	0.29	0.21	0.38	0.08	0.03	0.01
SCS3	0.2	0.36	0.25	0.02	0.13	0.18
PY1	-0.14	0.01	0.01	0.28	0.68	0.57
PY2	-0.07	0.23	0.01	0.42	0.3	0.72
PY3	-0.05	0.15	0.2	0.34	0.45	0.33

1) ranged from 0.12 to 0.30 for SCS, and from 0.20 to 0.44 for protein yield (Figure 2). Estimates generally increased with parity for both traits.

Average genetic correlations (Table 3) for different parities and traits (SCS and protein yield) ranged from -0.14 to 0.60 while environmental correlations varied from -0.19 to 0.41. The values of genetic correlation between the same trait in different parities were moderate and positive (0.26 to 0.40 for SCS and 0.45 to 0.60 for protein yield). The genetic correlations between SCS and protein yield were null or negative and ranged from -0.14 to 0.01.

The average genetic correlation between SCS and protein yield on the same DIM increased in magnitude from -0.02 in first parity to -0.14 in second and third parities. Genetic correlations within parity indicated that greater protein yields were associated with smaller SCS. Average genetic correlations between SCS and protein across different parities were close to zero.

The trend of daily values of the genetic correlation between SCS and protein yield were also evaluated for the first 3 parities (Figure 3). Genetic correlations in the first parity were positive (<0.14) at the beginning of the lactation until DIM 157, when values became negative and reached a minimum of -0.17 in late lactation. This trend suggested that the null average association between SCS and protein yield in the first parity (Table 3) was the result of positive and negative correlations at the beginning and at the end of the lactation, respectively. In second and third parities, with an exception for the first 30 DIM of the second parity, all genetic correlations had negative values, with minima of -0.34 and -0.27 for second and third parity, respectively.

Average correlations for permanent environmental effects were positive across different parities for the

same trait and negative between the 2 traits (Table 3). Permanent environmental correlations between TD protein and SCS were generally negative in all lactations, decreasing with increased DIM (results not shown).

Test-Day Model with Legendre Polynomials

Estimates of average heritabilities from the LP-TDM increased with parity, from 0.17 to 0.25 for SCS, and from 0.28 to 0.33 for protein yield (Table 3). Daily heritabilities of protein yield were more uniform and with fewer differences between parities than the estimates from the WF-TDM (Figure 2).

Average within-parity genetic correlations between SCS and protein yield decreased from 0.08 to -0.18 from the first to the third parity (Table 3). Daily additive correlations differed both in pattern and in the sign between parities (Figure 3). In the first parity, the estimates were positive (<0.13) until DIM 280. In the second parity estimates showed an earlier and steeper decrease (>-0.28) with a null correlation at DIM 152. In the third parity, daily genetic correlations were all negative (from 0 to -0.24).

Average correlations for permanent environmental effects were positive between different parities for the same trait and null or negative between traits (Table 3).

DISCUSSION

Variance Component Estimates

Heritabilities. Estimates of heritability for SCS and protein yield from different models were similar

Figure 1. Daily heritabilities of SCS estimated with 2 different random regression test-day models using Wilink function (WF-TDM) or Legendre polynomials (LP-TDM), in first (WF-TDM1, LP-TDM1), second (WF-TDM2, LP-TDM2), and third parity (WF-TDM3, LP-TDM3), from 5 to 305 DIM.

Figure 2. Daily heritabilities of protein yield estimated with 2 different random regression test-day models using Wilink function (WF-TDM) or Legendre polynomials (LP-TDM), in first (WF-TDM1, LP-TDM1), second (WF-TDM2, LP-TDM2), and third parity (WF-TDM3, LP-TDM3), from 5 to 305 DIM.

Figure 3. Daily additive genetic correlations estimated with 2 different random regression test-day models using Wilink function (WF-TDM) or Legendre polynomial (LP-TDM), between SCS and protein yield on the same DIM in first (WF-TDM1, LP-TDM1), second (WF-TDM2, LP-TDM2), and third parity (WF-TDM3, LP-TDM3), from 5 to 305 DIM.

across parities. The largest between-parity variation in heritability for SCS and protein yield was found for the WF-TDM.

Estimates of heritability for SCS used worldwide in national genetic evaluation programs differ across countries and evaluation systems: from 0.11 to 0.43 for the LACM, and from 0.06 to 0.35 for TD models (Interbull, 2008). First-parity heritability for SCS estimated in the present study was larger than estimates reported for the same population using a repeatability TDM with either the Wilink function or Legendre polynomials for fixed regressions (Samoré et al., 2001). Heritabilities for SCS from RR-TDM for WF-TDM and LP-TDM were slightly smaller than those obtained with a random regression WF-TDM for the Canadian population, as reported in Samoré et al. (2002). For protein yield, estimates of heritability were comparable with those for the Italian Holsteins and other Holstein populations using a LACM (Interbull, 2008).

Mrode and Swanson (2003), using a RR-TDM with orthogonal polynomials of order 2, reported a pattern of daily heritabilities for SCS increasing in magnitude from the beginning to the end of lactation and an increase in heritabilities with parity. Daily heritabilities also increased with parity in the current study. The pattern along the lactation showed an initial downward slope followed by an increasing trend at the end of each lactation.

Genetic Correlations Between SCS and Protein Yield.

Correlation estimates from both WF-TDM and LP-TDM showed different values in different parities and different trends within lactations. Additive genetic correlations from TD models were mainly positive in the first parity, suggesting an antagonistic association between SCS and yield. In the second parity, correlation coefficients were equally distributed between positive and negative values along the lactation, resulting in a null average relationship. In contrast, estimates of daily additive genetic correlations for the third parity suggested a negative association between the 2 traits, with smaller values of SCS corresponding to greater protein yield.

Positive relationships were recently reported in the literature between SCS and protein yield in the first parity, with values ranging from 0.12 to 0.22 (Miglior et al., 2007; Muir et al., 2007). A positive genetic relationship of SCS and milk yield has also been reported in Finnish Ayrshire (Pösö and Mäntysaari, 1996), Holsteins (Rupp and Boichard, 1999; Carlén et al., 2004), and Jersey (Roman and Wilcox, 2000) dairy cows, whereas a negative relationship was described in later parities by Monardes and Hayes (1985), and Schutz et al. (1990). The change in sign of the correlation was explained by the culling practice in the first parity based simultaneously on mastitis incidence and milk production.

Pösö and Mäntysaari (1996) and Haile-Mariam et al. (2001) reported a trend of daily genetic correlations between SCS and protein yield over parity and within lactation for different DIM that was similar to the findings of our study. Large positive genetic correlations were observed at the beginning of the first lactation and correlations decreased to negative or zero values with increasing DIM. The same pattern was found for second and third lactations. Both TDM gave similar patterns of changes in genetic correlations between SCS and protein yield.

Biological Interpretation

The changes in value of additive correlations with parity and the pattern of genetic relationships within lactation will be considered to interpret the trend of additive association between SCS and protein yield on a biological basis.

Changes with Parity. The change in values of genetic correlation between SCS and protein yield with increasing parity number was explained by Haile-Mariam et al. (2001) as the result of the balance of 2 mechanisms. Cows with high milk production may be more susceptible to infection, resulting in a positive genetic correlation at the beginning of first parity. The occurrence of mastitis events may then cause damage to the texture of the udder and lead to decreased production, resulting in a negative correlation in later parities.

The genetic correlation between involuntary culling and level of SCS was positive (0.31) in Italian Holsteins (Samoré et al., 2003). Furthermore, the correlation between resistance to mastitis infections and risk of being culled was estimated to be -0.40 (Neerhof et al., 2000). Both values indicated that culling strategies based on production level and sensitivity to mastitis may affect the genetic correlation between production and mastitis in second and later parities. Moreover, in second parity, cows have already been exposed to pathogens causing mastitis and their reaction to mastitis can be different from that of first-parity cows. The different reaction could influence, in consequence, the value of genetic association between SCS and production traits. Finally, SCS in milk can be associated with different genes in first and later lactations. Nevertheless, it should be noted that, although these interpretations can explain the change in sign of correlations from first to second parity, the explanation of the further negative trend from second to third parity is more difficult.

Change Within Lactation. Concerning the pattern of daily correlations within parities, Haile-Mariam et al. (2001) suggested that the variation in SCS values at the beginning of lactation depends on spikes of increase in SCS level due to mastitis infection, whereas in the

latter part of the lactation, SCS levels are usually more consistent. This can also be associated with different predominant pathogens causing the intramammary infection at a specific stage. In the period after calving, when cows are metabolically stressed by the peak of production, mastitis is often due to infection by environmental pathogens, with a brief increase in SCS (Detilleux et al., 1997), whereas different species of pathogens may be predominant in later stages of production. It was also suggested that the mechanisms of reaction may differ according to pathogens causing the infection and the subsequent increase in SCS (de Haas et al., 2002) leading to different associations between SCS and production yield. Environmental pathogens are the main pathogens responsible for intramammary infections in cows that are metabolically stressed. These infections generally occur in the first few months after calving, and cause a brief but strong increase in SCS. *Staphylococcus aureus* infections also cause a considerable increase in SCS, but often lead to chronic mastitis with high SCS persisting for weeks to months after the initial lactation. Infections by streptococci are associated with a continuous increase in SCS until clinical symptoms are observed and SCS remains high afterwards (Detilleux et al., 1997; de Haas et al., 2002).

The pattern of genetic correlations can also be partly explained by the changes in specific protein content in milk after udder infection with an increase in gamma immunoglobulin and a degradation of α -, β -, and κ -caseins, and in whey, an increase of serum albumin content and a decrease of β -lactoglobulin and α -lactalbumin (Kostyra, 1990). This means that after a case of either clinical or subclinical mastitis, the level of SCS increases and the content of total milk protein increases or remains constant but with a composition different from the milk of healthy cows. The change in total protein content is the result of 2 factors: the presence of serum protein produced as a reaction to the infection (immunoglobulin or serum protein) and the degradation of caseins and other milk proteins produced in the udder. The altered balance between SCS and protein would, by consequence, influence the genetic correlations between the 2 traits.

Somatic Cells and Production

The genetic relationship between production and SCS at the beginning of lactation in first parity is positive but becomes negative later in first parity and in subsequent lactations. The environmental correlation, however, is negative from the beginning of first parity. It should be considered that only at the beginning of the first lactation the cow does not have a "disease

history.” Later on, the pattern of genetic correlations should consider the momentum: the genetic correlation between instant SCS and instant production. Unfortunately, it should also be emphasized that the model may not appropriately distinguish between instant SCS and SCS history, meaning that in the second stage of first lactation and in later parities, the genetic correlation is confounded with the environmental correlation of earlier records. Ødegård et al. (2003) proposed, as a possible alternative, the use of mixture models to analyze TD records for SCS as the result of effects of several possibly confounded factors and distributions. Mixture models would allow different treatment of records for infected and noninfected cows and may be able to distinguish mastitis caused by different pathogens. Heringstad et al. (2006) suggested also that SCS is, genetically, a heterogeneous trait that is not the same trait in infected and noninfected cows. Using simultaneous and recursive models, Wu et al. (2007) estimated that the direct effect of SCS on milk components was large and negative and, in contrast, the reciprocal effects from milk to SCS were small and positive in the first 60 d, and not different from zero in the second part of lactation. Furthermore, smaller direct causal pathway effects of SCS on production were estimated in the second period of lactation. The estimated effects seemed to be yield-dependent with a greater effect for high-producing cows (Wu et al., 2007). Finally, using a structural equation model, de los Campos et al. (2006) attributed the negative association between yield and SCS more likely to the result of the infection event on production than to the dilution of milk with high yield.

Somatic Cells and Selection Strategies

The evidence of a trend in additive correlations between SCS and protein yield should be taken into account when defining selection strategies. Usually, a single value of genetic correlation between SCS and protein yield is considered, ignoring the pattern of genetic correlations both with parities and within lactations. This would, consequently, influence the expectations of possible genetic progress.

Genetic improvement in dairy cattle populations depends largely on bull selection, and the choice of new young bulls is based on information from first-parity daughters for both production and SCS. The accumulation of daughter phenotypic data increases the reliability of EBV for SCS and production and it can change the EBV of a bull. Phenotypic information on later lactations will also contribute to the EBV. Nevertheless, the availability of data from later parities would occur later in life, when a bull is already being used as sire

in the population. Additionally, first-parity data of SCS are not influenced by the involuntary culling performed by farmers for udder infection. For these reasons, one can suggest that, if a single value of the genetic association between SCS and production should be defined, the most correct value would be the first-parity genetic correlation level or, based on the results of this study, a small positive association.

In contrast, when selection is effectively based on multiple-parity data, the use of the same value for the genetic correlation could be both incorrect and difficult. The utilization of different additive genetic correlation values for various stages of lactation and parity would, therefore, be the only possible choice to give the correct emphasis to SCS and production yield into a selection program. It might also be possible to think of the development of a selection index within parity to account for different correlation values between protein yield and SCS. In the second stage, an overall index could be produced by weighting information from each parity.

The choice of the correlation value should also consider the relationship between SCS and mastitis, which is more important in determining the weight on SCS in selection strategies than are the correlations between SCS and yield. Based on results from the present study, it could be reasonably argued that a zero correlation between SCS and yield should be assumed. An additional observation could be that the genetic correlation between SCS and clinical mastitis should also be studied across parities and DIM. It might be useful to compare rankings of bulls, based on different assumed correlation matrices, to demonstrate the importance of using the correct correlations.

CONCLUSIONS

Additive correlations between SCS and protein yield estimated with 2 RR-TDM differed with parity and with DIM within lactation. When setting up selection strategies, the choice of a global genetic correlation between the 2 traits is therefore difficult and may lead to incorrect prediction of genetic progress for each trait. A single value of first-parity additive genetic correlation can be used in selection programs assuming that genetic choices are mainly based on production of first-parity daughters. Alternatively, it can be suggested to account for the changes in genetic association between SCS and production with parities and stage of lactation by using multiple-trait RR-TDM. The interpretation of these relationships is complex and can be referred to both biological aspects and properties of RR-TDM. It would be useful to distinguish between instant SCS and SCS “history.” The use of models distinguishing between specific SCS distributions for the udder health

status and for the pathogen causing the infection would be of interest to account for each specific pattern of SCS increase following the infection. This would probably help to estimate the level and sign of the genetic relationship between SCS and protein yield along the cow's life.

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