



J. Dairy Sci. 95:3428–3434

<http://dx.doi.org/10.3168/jds.2011-4739>

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Genetic relationships among mastitis and alternative somatic cell count traits in the first 3 lactations of Swedish Holsteins

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ABSTRACT

The objectives of this study were to estimate heritabilities of, and genetic correlations among, clinical mastitis (CM), subclinical mastitis (SCM), and alternative somatic cell count (SCC) traits in the first 3 lactations of Swedish Holstein cows, and to estimate genetic correlations for the alternative traits across lactations. Data from cows having their first calving between 2002 and 2009 were used. The alternative SCC traits were based on information on CM and monthly test-day (TD) records of SCC traits of 178,613, 116,079, and 64,474 lactations in first, second, or third parity, respectively. Sires had an average of 230, 165, or 124 daughters in the data (parities 1, 2, or 3, respectively). Subclinical mastitis was defined as the number of periods with an SCC >150,000 cell/mL and without a treatment for CM. Average TD SCC between 5 and 150 d was used as a reference trait. The alternative SCC traits analyzed were 1) presence of at least 1 TD SCC between 41,000 and 80,000 cell/mL (TD41–80), 2) at least 1 TD SCC >500,000 cells/mL, 3) standard deviation of log SCC over the lactation, 4) number of infection peaks, and 5) average days diseased per peak. The same variables in different parities were treated as distinct traits. The statistical model considered the effects of herd-year, year, month, age at calving, animal, and residual. Heritability estimates were 0.07 to 0.08 for CM, 0.12 to 0.17 for SCM, and 0.14 for SCC150. For the alternative traits, heritability estimates were 0.12 to 0.17 for standard deviation of log SCC, TD SCC >500,000 cells/mL, and average days diseased per peak, and 0.06 to 0.10 for TD41–80 and number of infection peaks. Genetic correlations between CM with SCM were 0.62 to 0.74, and correlations for these traits with the alternative SCC traits were positive and very high (0.67 to 0.82 for CM, and 0.94 to 0.99 for SCM). Trait TD41–80 was the only alternative trait that showed negative, favorable, genetic correlations with CM (–0.22 to –0.50) and SCM

(–0.48 to –0.85) because it is associated with healthy cows. Genetic correlations among the alternative traits in all 3 parities were high (0.93 to 0.99, 0.92 to 0.98, and 0.78 to 0.99, respectively). The only exception was TD41–80, which showed moderate to strong negative correlations with the rest of the traits. Genetic correlations of the same trait across parities were in general positive and very high (0.83 to 0.99). In conclusion, these alternative SCC traits could be used in practical breeding programs aiming to improve udder health in dairy cattle.

Key words: dairy cattle, somatic cell count, mastitis, genetic parameter

INTRODUCTION

Mastitis is a common and costly disease in dairy cattle, associated with decreased milk yield, discarded milk, reduction in milk price due to high SCC, veterinary and treatment costs, increased labor, and increased culling rate (Nielsen, 2009). It is generally accepted that undesirable genetic relationships exist between production and mastitis (e.g., Emanuelson et al., 1988; Heringstad et al., 2000; Hansen et al., 2002). The heritability of clinical mastitis (CM) using threshold models has been shown to be low: 0.07 to 0.10 in different populations (Heringstad et al., 2005; Zwald et al., 2006; Pérez-Cabal et al., 2009; Hinrichs et al., 2011). Lactation average log SCC has often been used as an indicator of CM (e.g., Heringstad et al., 2000).

Recently, other SCC-derived traits have been proposed, to improve breeding for udder health (de Haas et al., 2008; Windig et al., 2010). In these studies, SCC traits were defined on the basis of lactation stage, occurrence of excessive SCC, and other SCC traits based on patterns in peaks of SCC. Urioste et al. (2010) focused on genetic variability of novel traits that could be derived from information present in the test-day (TD) SCC records, using a small amount of research data with weekly observations, and explored the feasibility of applying those findings into monthly records of SCC.

Mastitis frequency (Carlén et al., 2004) and level of SCC (Da et al., 1992; Reents et al., 1995) increase

Received July 17, 2011.

Accepted January 24, 2012.

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with increasing parity. The resistance against mastitis may also differ, depending on the infection history of the cow. Consequently, resistance against mastitis might not be exactly the same trait in different parities (Carlén et al., 2004). Additionally, subclinical mastitis (SCM) is of concern. Unlike CM, no visible clinical signs occur and, therefore, diagnoses are based on a preassumed threshold level of SCC. The trait has been defined, at udder level, when a California Mastitis Test score of 3 to 5 is found on at least 1 sampling occasion (e.g., Thorberg et al., 2009), or simply having any quarter with SCC >100,000 cells/mL (Urech et al., 1999). In this research, another definition is proposed and its genetic properties investigated.

Compared with de Haas et al. (2008) and Windig et al. (2010), the present study assesses a combination of slightly different SCC traits and both clinical and subclinical mastitis are defined differently. Additionally, a trait using the standard deviation of TD SCC measures has not been genetically analyzed in field data, and the use of normal SCC levels (e.g., TD SCC between 40,000 and 80,000 cells/mL) to detect healthy animals has not been reported before.

The objectives were to estimate heritabilities of, and genetic correlations between, CM, SCM, and alternative SCC traits in a large field data set including the first 3 lactations of Swedish Holstein cows, and to estimate genetic correlations for the alternative traits across lactations.

MATERIALS AND METHODS

Swedish Animal Care and Use Committee approval was not needed for this study because the data were extracted from an existing database.

Data

Data on clinical mastitis (327,071 observations) and TD data with SCC records (more than 14 million

observations) were extracted from the Swedish milk recording scheme, and were edited to include records from the first 3 lactations of Swedish Holstein cows having their first calving between 2002 and 2009. Only TD records between 5 and 366 DIM were included.

The CM data were merged with the TD data. The diagnosis date was assigned to a given lactation, if it was between a preceding calving date (−10 d before calving) and the following calving date −10 d. Defined minimum and maximum ages for first, second, and third calving were 19 to 38, 31 to 52, and 42 to 66 mo, respectively. Cows were grouped, within each parity, in 6 age group classes of 3 mo, on average. Cows belonging to a herd-year class with fewer than 5 observations, and from sires with fewer than 40, 30, or 20 daughters in the data (lactations 1, 2, or 3, respectively) were excluded from the analyses. A minimum of 5 TD per lactation was also required.

From an initial pedigree file containing 974,439 animals, specific files were constructed, using pedigree information of the cows with phenotypic data back 3 generations. Because cows were not the same, due to editing criteria applied, the number of animals considered in the pedigree varied among the data sets. The numbers of observations after editing for analyzed traits in the 3 parities, and the general structure of the data and genealogy are given in Table 1.

Traits

Clinical mastitis was defined as the presence of a veterinary-treated clinical case from 10 d before calving to 10 d before the following calving; it was scored as present (1) in a given lactation, if at least 1 case of veterinary treatment was recorded; otherwise, it was scored as absent (0). It was assumed that CM, as defined above, was the same trait along the lactation. Taking advantage of the recording of CM in Sweden, SCM was defined as the number of periods (TD ± 15 d.) from DIM >45 with a SCC >150,000 cells/mL and without

Table 1. General structure of the data and pedigree in the first 3 parities of Swedish Holsteins

Item	Parity 1	Parity 2	Parity 3
Data			
No. of records	178,613	116,079	64,474
Average no. of test days per lactation (range)	9.74 (5–13)	9.49 (5–13)	9.36 (5–13)
Calving years	2002–2009	2003–2009	2004–2009
No. of herd-year groups	6,941	6,502	6,901
Mean herd-year group size (range)	26 (5–224)	18 (5–143)	9 (5–68)
Mean age (mo) at calving (range)	27.6 (19–38)	40.8 (31–53)	54.0 (42–66)
Pedigree			
No. of animals in pedigree	611,105	586,873	522,521
No. of sires	778	702	521
Mean daughter group size (range)	230 (40–7,559)	165 (30–5,422)	124 (20–2,949)

a treatment for CM. It has been shown (Vazquez et al., 2009) that linear, threshold, and Poisson models have a similar predictive performance for mastitis traits, so it was decided to treat this trait as linear. Test-day SCC were averaged over the early lactation period (5–150 d; **SCC150D**) and used as a reference trait, to maintain coherence with previous work (de Haas et al., 2008; Urioste et al., 2010).

Based on conclusions from Urioste et al. (2010), 5 alternative SCC-derived traits were used in this study, as potential indicator traits for CM and SCM, capturing different aspects of mastitis:

- Binary traits were defined as the presence of at least 1 TD SCC between 41,000 and 80,000 cells/mL (**TD41–80**), or at least 1 TD SCC >500,000 cells/mL (**TD >500**).
- An infection peak was defined as a period of increased SCC (>150,000 cells/mL) between 2 low ($\leq 150,000$ cells/mL) TD observations. The number of peaks (**NPeak**) was considered as a trait.
- A practical approximation of persistency of disease was recorded as the number of days between the start and the end of an infection peak (TD with SCC <150,000 cells/mL and having at least a TD >150,000 cells/mL in between). Average days diseased per peak (**ADSick**) was defined as the total number of days diseased divided by the total number of peaks, trying to distinguish between short and long durations, the latter often associated with contagious pathogens. The variable was then log transformed to improve parameter estimation.
- Standard deviation of log TD SCC during lactation (**SCCSD**), as proposed by Green et al. (2004), was also used.

Statistical Analysis

The following linear animal model was used for all traits:

$$y_{ijklm} = hy_i + year_j + month_k + age_l + a_m + e_{ijklm},$$

where y_{ijklm} denotes the response trait; hy_i is the effect of the i th herd by year of calving; $year_j$ is the effect of the j th year of calving; $month_k$ is the effect of the k th month of calving; age_l is the effect of the l th age at calving, with 6 classes per lactation; a_m is the effect of the m th animal; and e_{ijklm} is the residual effect.

Herd-year and animal effects were assumed to have zero means and the covariance structure for the bivariate analyses was

$$V = \begin{bmatrix} \mathbf{I}\sigma_{hy_1}^2 & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ & \mathbf{I}\sigma_{hy_2}^2 & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ & & \mathbf{A}\sigma_{a_1}^2 & \mathbf{A}\sigma_{a_{12}} & \mathbf{0} & \mathbf{0} \\ & & & \mathbf{A}\sigma_{a_2}^2 & \mathbf{0} & \mathbf{0} \\ & & & & \mathbf{I}\sigma_{e_1}^2 & \mathbf{I}\sigma_{e_{12}} \\ & & & & & \mathbf{I}\sigma_{e_1}^2 \end{bmatrix},$$

where \mathbf{A} is the additive relationship matrix, \mathbf{I} is the identity matrix, σ_{hy}^2 , σ_a^2 , and σ_e^2 are the variances of the herd-year, additive effect, and residual effect, respectively, and the indices represent the 2 traits in the bivariate analysis. An initial attempt of using the herd-year effect as fixed (proper uniform priors) did not have good convergence properties; therefore, it was decided to treat them as uncorrelated random effects, to avoid extreme category problems associated with observations in a given herd-year class falling all in the same category (0 or 1).

A threshold liability approach (e.g., Gianola and Foulley, 1983) was used for traits expressed as a discrete (0/1) response (CM, TD41–80, and TD >500). Threshold-linear analyses were made according to Foulley et al. (1983). Herd-year, additive genetic, and residual (co)variances were drawn from their respective posterior distributions, in a Bayesian approach, using flat priors and Gibbs sampling, as implemented for threshold and linear trait analyses in the program Thrgibbs1f90 (Misztal et al., 2002). Based on visual inspection of trace plots in earlier runs (a binary and a continuous variable were tested with chains of 50,000, 100,000, and 250,000, thinning intervals of 25 and 50 samples, 25 or 50% of burn-in, and found to converge to the same values), a chain of 125,000 iterations was run for each trait combination, including a burn-in of 25,000 iterations, keeping every twenty-fifth sample for inference of posterior features (4,000 effective samples).

Estimates of genetic correlations between traits and between lactations for the same trait were obtained from bivariate analyses; heritabilities (h^2) were averaged over the bivariate runs. The heritability was computed as the ratio between the additive genetic and the sum of additive and residual variances. Genetic correlations were calculated as the ratio between the additive genetic covariance and the products of the involved traits' additive standard deviation. Post-Gibbs analysis (pos-

terior means, medians, percentiles, and distributions) was done with SAS software (SAS Institute, 2005).

RESULTS AND DISCUSSION

Basic Statistics

The overall means for the traits in the 3 parities can be seen in Table 2. The general trend along parities was an increase in all traits, with the exception of TD41–80, whose decline was linked to poorer health as well. Compared with earlier data (1995–2000) of Carlén et al. (2004) on the same population, the incidence of CM slightly increased in all parities. In a Canadian study by Olde Riekerink et al. (2008), the mean incidence rate of mastitis was 23%. Levels of CM reported by Windig et al. (2010) from Dutch herds were slightly lower than those obtained in this study. Those authors also reported a lower mean of the total number of peaks (both environmental and contagious) per lactation, of 0.11 to 0.13. Levels of SCC reported by Haile-Mariam et al. (2001) for Australian dairy cattle in first, second, and third parity were similar to those presented here.

Genetic Parameters for Mastitis Traits

Heritability for CM and SCM and their genetic correlations with alternative SCC traits are presented in Table 3. Heritability estimates for CM (0.07–0.08) were well in accordance with what is known from the literature when using a threshold model approach (e.g., Heringstad et al., 2005; Zwald et al., 2006; Pérez-Cabal et al., 2009) and a previous study based on data from a research herd (Urioste et al., 2010). This low value indicates that the use of traits genetically correlated with CM would be beneficial for selection purposes.

Heritability of SCM (0.13 to 0.17), as defined in this paper, has not been reported before, and was twice the genetic variability found for CM. One explanation

could be a scale effect: CM is binary and SCM is not. Genetic variability seems to decrease in the third parity. No definition is agreed upon regarding SCM when the information stems from TD records. For example, Halasa et al. (2009) defined a case of SCM, in a given cow and lactation, if a TD SCC >100,000 cells/mL was preceded by a TD SCC <50,000 cells/mL. de Haas et al. (2008) and Windig et al. (2010) treated SCM as a binary trait and defined its presence as at least 1 TD SCC above a certain cut-off value, which varied for heifers and multiparous cows. de Haas et al. (2008) found very low heritabilities (0.02–0.03) for SCM, defined as a 0/1 trait and analyzed with a linear model. In this respect, SCM as defined in this research shows more promising features for selection purposes.

Posterior mean (SD) of genetic correlations between CM and SCM were 0.74 (0.04) in parity 1, 0.72 (0.03) in parity 2, and 0.62 (0.08) in parity 3. This decrease in genetic correlation with parity number agrees with the trend observed by Windig et al. (2010), who obtained estimates of 0.58, 0.55, and 0.26 in parities 1, 2, and 3, respectively, although definition of SCM was different. Two conclusions can be drawn: that CM and SCM are distinct traits, and that selection against one of them will bring genetic improvement to the other.

Genetic correlations of CM and SCM with the alternative SCC traits were positive and high (0.67 to 0.82 for CM, and 0.94 to 0.99 for SCM; Table 3). This was expected, because SCM is directly derived from TD SCC and CM is not. Although SCCSD and TD >500 show similar correlations to CM as SCC150D, they are probably capturing more of the biological background. They are phenotypically more associated with CM (Urioste et al., 2010) because they describe the effect of clinical IMI on SCC. As demonstrated by Green et al. (2004), the impact of infection on a TD with >500,000 cells/mL is likely to be greater than that on mean SCC, because the mean is influenced by all SCC readings during lactation. Additionally, this measure could be

Table 2. Overall means for the traits analyzed in this study for parities 1, 2, and 3

Trait ¹	Parity 1	Parity 2	Parity 3
CM (%)	12.5	15.2	19.4
SCM (no. of periods)	1.7	2.7	3.3
SCC150D (cells/mL)	148,948	231,502	301,731
SCCSD (cells/mL)	205,856	301,283	378,516
TD41–80 (%)	79.9	74.0	68.6
TD >500 (%)	28.6	41.1	49.6
NPeak	0.95	1.21	1.34
ADSick (d)	56.9	82.9	99.8

¹CM = clinical mastitis; SCM = subclinical mastitis; SCC150D = average SCC in early lactation (5–150 d); SCCSD = SD of SCC; TD41–80 = at least 1 test day (TD) between 41,000 and 80,000 cells/mL; TD >500 = at least 1 TD >500,000 cells/mL; NPeak = number of peaks; ADSick = average days diseased.

Table 3. Posterior mean (SD) of heritability (h^2) estimates of liability to clinical (CM) and subclinical (SCM) mastitis and of genetic correlations with alternative SCC traits in the first 3 parities¹

Item	Parity 1		Parity 2		Parity 3	
	CM	SCM	CM	SCM	CM	SCM
h^2	0.08 (0.01)	0.17 (0.01)	0.08 (0.01)	0.16 (0.01)	0.07 (0.01)	0.13 (0.01)
SCC trait ²						
SCC150D	0.82 (0.02)	0.95 (0.01)	0.81 (0.02)	0.96 (0.00)	0.76 (0.04)	0.94 (0.01)
SCCSD	0.82 (0.02)	0.97 (0.00)	0.79 (0.03)	0.97 (0.00)	0.74 (0.04)	0.94 (0.01)
TD >500	0.80 (0.03)	0.94 (0.01)	0.87 (0.02)	0.94 (0.01)	0.78 (0.04)	0.92 (0.01)
TD41–80	–0.22 (0.05)	–0.48 (0.04)	–0.50 (0.06)	–0.81 (0.02)	–0.26 (0.08)	–0.85 (0.02)
NPeak	0.79 (0.03)	0.96 (0.00)	0.74 (0.04)	0.94 (0.01)	0.67 (0.05)	0.84 (0.03)
ADSick	0.81 (0.03)	0.99 (0.00)	0.76 (0.03)	0.99 (0.00)	0.66 (0.06)	0.98 (0.00)

¹Posterior means and SD are averages of 7 bivariate analyses.

²CM = clinical mastitis; SCM = subclinical mastitis; SCC150D = average SCC in early lactation (5–150 d); SCCSD = SD of SCC; TD41–80 = at least 1 test day (TD) between 41,000 and 80,000 cells/mL; TD >500 = at least 1 TD >500,000 cells/mL; NPeak = number of peaks; ADSick = average days diseased.

associated with environmental pathogens, both for CM and SCM (de Haas et al., 2008).

A trend seemed to exist for lower genetic correlations with CM with increasing parity, whereas correlations with SCM were more stable. Results obtained here were similar to those obtained by Windig et al. (2010) in Dutch dairy herds, the only comparable study. Genetic correlation estimates between mastitis and SCS obtained by Carlén et al. (2004), in the same Swedish population, ranged between 0.66 and 0.77.

In Urioste et al. (2010), TD41–80 was a trait positively associated with clusters of healthy cows when measurements were recorded monthly (recall that a TD41–80 = 0 identifies a cow that never has had a TD with SCC between 41,000 and 80,000 counts/mL). A recent review (Schukken et al., 2003) reported that uninfected quarters have a mean SCC of approximately 70,000 cells/mL, with some variation around this mean, which calls for a closer look to a trait reflecting such features. The genetic properties of such a trait have not been described before using large data sets. Here, it was the only alternative trait that showed favorable, negative genetic correlations with CM and SCM; correlations were weak to moderate for CM (–0.22 to –0.50), and moderate to strong (–0.48 to –0.85) with SCM.

Genetic Parameters for the Alternative SCC Traits

Traits of interest for further analysis in large data sets, according to Urioste et al. (2010), were SCCSD, NPeak, ADSick, TD >500, and TD41–80; the latter trait is more often expressed in healthy than in mastitic

cows. Table 4 depicts heritabilities and genetic correlations among the reference SCC150D and the 5 alternative SCC traits for first and second parity. Estimates for third parity are not shown but were similar to those from second parity.

Heritabilities were well in accordance with previous estimates (Urioste et al., 2010) using a data set of monthly SCC records. Heritability of SCC150D varied between 0.13 and 0.16. For the same trait, de Haas et al. (2008) reported values of 0.08 to 0.10. The heritability estimates of Koeck et al. (2011) on alternative SCC traits for Canadian Holsteins were lower and varied between 0.01 and 0.07.

Two levels of heritability existed for the alternative traits: 0.12 to 0.17 for SCCSD, TD >500, and ADSick, and 0.06 to 0.10 for TD41–80 and NPeak (and ADSick in third parity). The genetic variability in the first group is then at the same level as SCC150D, which was also anticipated from our previous study. Our findings are consistent with those of de Haas et al. (2008), who found heritabilities of traits describing the dynamics of SCC to be between 0.03 and 0.11, and 0.01 to 0.05 for patterns of peaks.

Genetic correlations among traits in each parity were very high (0.93–0.99 in first parity, 0.92–0.98 in second parity, and 0.78–0.99 in third parity), and similar to the results of Windig et al. (2010) and Koeck et al. (2011), whose estimates often were above 0.95. The only exception was TD41–80, which showed moderate to strong negative correlations with the rest of the traits. The high positive correlations suggest that any of the new traits with heritability similar to SCC150D could be

Table 4. Posterior mean (SD)¹ of heritability and genetic correlations among SCC traits² within parity (first parity below, second parity above diagonal)

Item	h ²	SCC150D	SCCSD	TD >500	TD41-80	NPeak	ADSick
h ²		0.16 (0.01)	0.16 (0.01)	0.17 (0.01)	0.09 (0.01)	0.08 (0.01)	0.15 (0.01)
SCC trait							
SCC150D	0.14 (0.01)		0.98 (0.00)	0.97 (0.01)	-0.81 (0.02)	0.93 (0.01)	0.97 (0.00)
SCCSD	0.14 (0.01)	0.97 (0.00)		0.99 (0.00)	-0.79 (0.03)	0.92 (0.01)	0.98 (0.00)
TD >500	0.12 (0.01)	0.94 (0.01)	0.94 (0.01)		-0.80 (0.02)	0.89 (0.01)	0.97 (0.00)
TD41-80	0.05 (0.00)	-0.37 (0.05)	-0.46 (0.04)	-0.49 (0.04)		-0.65 (0.04)	-0.79 (0.02)
NPeak	0.10 (0.01)	0.96 (0.01)	0.96 (0.00)	0.93 (0.01)	-0.43 (0.05)		0.95 (0.01)
ADSick	0.14 (0.01)	0.97 (0.00)	0.99 (0.00)	0.97 (0.00)	-0.50 (0.04)	0.98 (0.00)	

¹Posterior means and SD are averages of 7 bivariate analyses.

²CM = clinical mastitis; SCM = subclinical mastitis; SCC150D = average SCC in early lactation (5–150 d); SCCSD = SD of SCC; TD41-80: at least 1 test day (TD) between 41,000 and 80,000 cells/mL; TD >500 = at least 1 TD >500,000 cells/mL; NPeak = number of peaks; ADSick = average days diseased.

used in its place, but they do not add very much information. This is probably a partial effect of autocorrelation; all traits are built from the same information. The only trait that seems to add new useful information is TD41-80, because it identifies the healthy and not the sick cow and, therefore, could be useful for use in a selection index.

Genetic Correlations Across Parities

The genetic correlations of the same trait across parities (Table 5) were positive and very high (0.83 to 0.99), suggesting that they could, in practice, be considered as the same trait. The only exception is the genetic correlation for TD41-80 in parities 1 and 3, which was moderately positive (0.69). Carlén et al. (2004) obtained correlation estimates of CM across lactation above 0.7 and of SCS above 0.8, whereas in the study of Windig et al. (2010), correlations among alternative SCC traits in different lactations ranged

between 0.54 and 0.99. These results suggest 1) that the use of a simpler repeatability model could be used for traits with several parities, and 2) that selection decisions can be made already in the first parity. The results have particular value for less-developed recording conditions, because not all countries have records on mastitis cases, but standard BLUP methodology applied on any of the studied traits may help in obtaining genetic evaluations that can be used to improve udder health regarding both CM and SCM.

CONCLUSIONS

This research, performed with the largest field data set published to date and very large progeny groups, has shown that CM and SCM are distinct, albeit correlated, traits. Furthermore, alternative SCC traits show genetic variability and are closely associated with both CM and SCM, confirming their potential use as biologically valuable indicator traits. Although most traits

Table 5. Posterior mean (SD) of genetic correlations within trait across parities

Trait ¹	Parities 1 and 2	Parities 1 and 3	Parities 2 and 3
CM	0.89 (0.01)	0.84 (0.02)	0.91 (0.01)
SCM	0.98 (0.00)	0.95 (0.01)	0.99 (0.00)
SCC150D	0.97 (0.00)	0.92 (0.01)	0.98 (0.00)
SCCSD	0.98 (0.00)	0.94 (0.01)	0.97 (0.00)
TD >500	0.98 (0.00)	0.93 (0.01)	0.97 (0.00)
TD41-80	0.90 (0.01)	0.69 (0.04)	0.97 (0.01)
NPeak	0.95 (0.01)	0.83 (0.02)	0.95 (0.01)
ADSick	0.98 (0.00)	0.93 (0.01)	0.99 (0.00)

¹CM = clinical mastitis; SCM = subclinical mastitis; SCC150D = average SCC in early lactation (5–150 d); SCCSD = SD of SCC; TD41-80: at least 1 test day (TD) between 41,000 and 80,000 cells/mL; TD >500 = at least 1 TD >500,000 cells/mL; NPeak = number of peaks; ADSick = average days diseased.

are positively associated with CM and SCM, TD41–80 is a measure identifying healthier cows, thus showing negative genetic correlations with mastitis and SCC traits. Estimated genetic parameters could be useful for testing alternative indices and developing genetic evaluations for more robust dairy cows.

ACKNOWLEDGMENTS

This work was carried out as part of the RobustMilk Project, which is financially supported by the European Commission under the Seventh Research Framework Programme, Grant Agreement KBBE-211708. The content of this paper is the sole responsibility of the authors, and it does not necessarily represent the views of the Commission or its services. The authors are grateful to the Swedish Dairy Association (Stockholm, Sweden) and farmers for providing the data. The first author greatly acknowledges H. Naya (Institut Pasteur Montevideo, Montevideo, Uruguay) for facilitating the numerous and tedious runs done in Uruguay.

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