Genetic Association of Clinical Mastitis with Test-Day Somatic Cell Score and Milk Yield During First Lactation of Finnish Ayrshire Cows

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ABSTRACT

In this study the genetic association during lactation of 2 clinical mastitis (CM) traits: CM1 (7 d before to 30 d after calving) and CM2 (31 to 300 d after calving) with test-day somatic cell score (SCS) and milk yield (MY) was assessed using multitrait random regression sire models. The data analyzed were from 27,557 first-lactation Finnish Ayrshire cows. Random regressions on second- and third-order Legendre polynomials were used to model the daily genetic and permanent environmental variances of test-day SCS and MY, respectively, while only the intercept term was fitted for CM. Results showed that genetic correlations between CM and the test-day traits varied during lactation. Genetic correlations between CM1 and CM2 and test-day SCS during lactation varied from 0.41 to 0.77 and from 0.34 to 0.71, respectively. Genetic correlations of test-day MY with CM1 and CM2 ranged from 0.13 to 0.51 and from 0.49 to 0.66, respectively. Correlations between CM1 and SCS were strongest during early lactation, whereas correlations between CM2 and SCS were strongest in late lactation. Genetic correlations lower than unity indicate that CM and SCS measure different aspects of the trait mastitis. Milk yield in early lactation was more strongly correlated with both CM1 and CM2 than milk yield in later lactation. This suggests that selection for higher lactation MY through selection on increased milk yield in early lactation will have a more deleterious effect on genetic resistance to mastitis than selection for higher yield in late lactation. The approach used in this study for the estimation of the genetic associations between test-day and CM traits could be used to combine information from traits with different data structures, such as test-day SCS and CM traits in a multitrait random regression model for the genetic evaluation of udder health.

Key words: dairy cattle, udder health, genetic correlation, random regression model

INTRODUCTION

Selection for increased milk yield without simultaneous selection for mastitis resistance will lead to increased susceptibility to mastitis (Rupp and Boichard, 1999). This is costly to the farmer and uncomfortable for the cows. When selection against mastitis is included in a total merit index, the genetic level of mastitis may be kept constant or may even be improved (Heringstad et al., 2003).

Estimates of genetic correlations between clinical mastitis (CM) and milk production traits vary in the literature. In general, correlations between CM and production traits have been unfavorable (e.g., Hansen et al., 2002; Carlén et al., 2004; Negussie et al., 2006c). Estimates of genetic correlation between susceptibility to mastitis and milk yield (MY) based on Nordic lactation-average data ranged from 0.24 to 0.55 (Heringstad et al., 2000).

The efficiency of SCS as a selection criterion to reduce frequency of CM depends on the genetic association between the 2 traits, and estimates ranging from 0.34 to 0.72 have been cited in the literature (Pösö and Mäntysaari, 1996; Rupp and Boichard, 1999; Carlén et al., 2004; Negussie et al., 2006a). A review of studies using Nordic field data (Heringstad et al., 2000) reported that genetic correlations between CM and SCC range from 0.3 to 0.8, with an average of 0.6.

So far most estimates of genetic correlations between CM and MY or SCS have been from lactation-average models. A genetic analysis based on lactation-average models does not utilize all information in the data, because it does not allow simultaneous estimation of stage of lactation effects (Ødegård et al., 2003; Negussie et al., 2006b). Because SCS and milk production traits vary with stage of lactation, the genetic association between CM and test-day traits may also vary during lactation.

Clinical mastitis evaluation may benefit from multitrait analyses with correlated traits such as SCS, udder conformation, or production traits (Negussie et al., 2006a). In Finland, SCS and production traits are currently evaluated using test-day data applying longitudinal models (Faba Breeding, 2007). Therefore, to

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include CM in selection schemes, knowledge of the genetic correlations during lactation between CM and test-day traits in the selection objective is beneficial.

Negussie et al. (2006c) considered SCS and milk production traits measured at different stages of lactation as separate but correlated traits in a multitrait analysis. They reported genetic correlations between these traits and CM. Treating test-day SCS or MY as separate traits by stage of lactation requires estimation of covariance components for all traits, which leads to a nonparimonious and computing-intensive model. However, covariance functions could offer alternatives to multivariate analyses. They provide a method of treating traits measured at different stages of lactation as separate traits with a correlation structure between them. This may be considered as infinite-dimensional equivalent to covariance matrices for a given number of traits across a continuous scale (e.g., lactation).

Random regression models (RRM) induce a covariance structure along a given trajectory (van der Werf et al., 1998). They can therefore be used for estimation of genetic associations between traits over time. In dairy cattle, RRM have been applied to describe the covariances of SCS (e.g., Ødegård et al., 2003; Koivula et al., 2004; Negussie et al., 2006b), MY (e.g., Jamrozik and Schaeffer 1997; Schaeffer et al., 2000; Kettunen et al., 2000; Lidauer et al., 2003), and CM (e.g., Heringstad et al., 2003; Chang et al., 2004a; Negussie et al., 2006d). Random regression models have also been used to describe genetic correlations between longitudinal traits such as BCS, milk yield, and BW with fertility traits measured once during lactation (Veerkamp et al., 2001; Berry et al., 2003). The objectives of the present study were 1) to estimate covariances and associated parameters of test-day SCS, MY, and CM traits, and 2) to estimate the genetic associations during lactation between CM and each of the test-day traits using RRM.

MATERIALS AND METHODS

Data and Trait Definition

Data were extracted from the Finnish health and production database of Faba Breeding (Helsinki, Finland). Data included information on CM, test-day SCS, and MY of first-lactation Finnish Ayrshire cows whose calving dates were from 1995 through 2000 and with age at first calving from 22 to 30 mo. Somatic cell score was the natural logarithm of SCC ($\log_e \text{SCC}$), which was recorded every 2 mo and measured in 1,000 cells/mL. A test-day record comprised observations on SCS and MY recorded from 8 to 365 DIM.

Since 1983, veterinarians in Finland have been recording the date, diagnosis, treatment, and the drug used in treating CM cases. This has been compiled into

Table 1. Description of data

Item	
Cows, n	27,557
Records, n	138,048
Sires, n	506
Daughters per sire, mean n	54
Year-month of calving classes, n	72
Age classes, n	9
Herd-test-days, n	40,428
Herd-year classes, n	4,773
Frequency of CM1, ¹ %	7.2
Frequency of CM2, ² %	7.8
Mean test-day SCS, $\log_e$ 1,000 cells/mL	4.0
Mean test-day milk yield, kg	24

¹CM1 = Percentage of cows with at least one record of veterinary treatment of clinical mastitis in the period from 7 d before to 30 d after calving.

²CM2 = Percentage of cows with at least one record of veterinary treatment of clinical mastitis in the period from 31 to 300 d after calving, culling, or second calving.

the Finnish dairy health-recording database. For this study, information on CM cases was extracted from the database and merged with test-day SCS and MY records. Two CM traits were defined. The first CM trait included cases of veterinary-treated clinical mastitis in first lactation from 7 d before to 30 d after calving (CM1). The second CM trait included cases of veterinary-treated clinical mastitis in first lactation from 31 to 300 d after calving, culling, or second calving, whichever occurred first (CM2). Within these 2 intervals, the absence or presence of mastitis was scored as "0" or "1", respectively. A cow was considered to have had mastitis, and scored 1, if a veterinary treatment was recorded at least once or a cow was culled for mastitis reasons within this interval. Otherwise, she was scored 0.

In this study the 2 CM traits (CM1 and CM2) were chosen because a previous analysis on part of the same data showed a genetic correlation much lower than 1 (0.51) between these traits (Negussie et al., 2006c). Furthermore, this definition of CM traits is consistent with the definition currently used in the national genetic evaluation of CM for Finnish Ayrshire (Faba Breeding, 2007). Preliminary bivariate longitudinal analysis of CM, where CM was classified into 11 intervals, resulted in very low frequencies within each interval and led to erratic estimates and computational difficulties.

The data included a total of 27,557 cows, of which 21,259 had records for all traits. Details of the data are given in Table 1. The sire pedigree file had 684 males of which 506 had daughters with data.

Data Analysis

Model. Initially, univariate analyses of test-day SCS and MY were performed to determine the appropriate

order of polynomials for random regressions. The univariate RRM was

$$y_{hiklmno} = f_i + hy_h + ym_k + \sum_{r=0}^t \phi_\pi(d)_r b_1 \\ + htd_o + \sum_{r=0}^t \phi_\alpha(d)_r p_m + \sum_{r=0}^t \phi_\alpha(d)_r a_n + e_{hiklmno},$$

where $y_{hiklmno}$ = observation of test-day SCS or MY for daughter m of sire n , recorded in herd test-day o , year $\times$ month of calving k , herd-year h on a cow m belonging to the calving age class i , calving-year $\times$ calving-season class l , and measured on DIM d ; f_i = fixed effect of age at first calving i ; hy_h = fixed effect of herd-year h ; ym_k = fixed effect of year $\times$ month of calving k ; htd_o = random effect of herd test-day o ; b_1 = fixed regressions to describe the shape of lactation curve within calving year $\times$ calving-season classes l ; p_m = vector of random regressions of permanent environmental effect within cow m ; a_n = vector of random regressions of additive genetic effect of sire n ; and $e_{hiklmno}$ = random residual.

The covariables for coefficients b_r ($r = 0, \dots, 4$) were

$$\phi_\pi(d) = [c_0 \ c_1 \ c_2 \ c_3 \ \exp(wd)]^T, \quad [1]$$

where $c_0 \ c_1 \ c_2 \ c_3$ represent coefficients of the third-order orthogonal Legendre polynomial at DIM d , and w is coefficient of the exponential term of the Wilink function (Wilink, 1987). The most appropriate coefficients of the exponential term (w) that fitted the current data for modeling the fixed lactation curves were estimated to be -0.09 (Negussie et al., 2006b) for SCS and -0.05 for MY (Lidauer et al., 2003). For both test-day SCS and MY, 4 different models with order of random regressions ranging from intercept to third-order were tested.

The covariance structure for models with random htd effect was defined as

$$\text{Var} \begin{bmatrix} htd \\ \mathbf{a} \\ \mathbf{p} \\ \mathbf{e} \end{bmatrix} = \begin{bmatrix} \mathbf{H} & 0 & 0 & 0 \\ 0 & \mathbf{G} \otimes \mathbf{A} & 0 & 0 \\ 0 & 0 & \mathbf{P} \otimes \mathbf{I} & 0 \\ 0 & 0 & 0 & \mathbf{R} \end{bmatrix}$$

where $\mathbf{H}$ is a diagonal matrix of the form $\mathbf{I}\sigma_h^2$, and σ_h^2 is the variance of the random htd effect, $\mathbf{A}$ is the matrix of additive genetic relationships among sires, $\otimes$ is the Kronecker product, $\mathbf{G}$ and $\mathbf{P}$ are covariance matrices of the random regression coefficients for sire additive genetic and cow permanent environmental effects, $\mathbf{R}$ is the diagonal matrix of the form $\mathbf{I}\sigma_e^2$, and σ_e^2 is residual variance. The fit of the different RRM with varying orders of Legendre polynomials was assessed using log-likelihoods, Akaike's information criteria (AIC; Burn-

ham and Anderson, 2002), and estimates of residual variances. In addition, eigenvalues of the additive genetic covariance matrices were analyzed to assess the importance of adding further parameters.

The log-likelihood of a model given the data reflects the overall fit of the model and smaller values indicate poor fit. Because the likelihood tends to favor complex models with many parameters, the more conservative AIC has been suggested (Burnham and Anderson, 2002). The AIC penalizes models for the addition of parameters, and thus selects a model that fits well but has a minimum number of parameters (i.e., simplicity and parsimony; Burnham and Anderson, 2002). All of the above criteria were considered and the model with the highest log-likelihood, lowest AIC, and residual variance was considered to be the most appropriate. As a result, in the multitrait RRM analyses of CM traits with test-day SCS or MY, the sire additive genetic and permanent environmental effects for test-day SCS and MY were modeled by second- and third-order orthogonal Legendre polynomials, respectively. Only the intercept term was fitted for the CM1 and CM2 traits. The modeling of fixed effects was the same for all traits with the exception of the lactation curve, which was modeled only for the test-day traits.

For instance, the description of the multitrait RRM for test-day SCS, CM1, and CM2 traits was

$$\begin{bmatrix} y_{SCS} \\ y_{CM1} \\ y_{CM2} \end{bmatrix} = \begin{bmatrix} f_{SCS_i} \\ f_{CM1_i} \\ f_{CM2_i} \end{bmatrix} + \begin{bmatrix} ym_{SCS_k} \\ ym_{CM1_k} \\ ym_{CM2_k} \end{bmatrix} + \begin{bmatrix} hy_{SCS_h} \\ hy_{CM1_h} \\ hy_{CM2_h} \end{bmatrix} \\ + \begin{bmatrix} \sum_{r=0}^4 \phi_\pi(d)_r b_{SCS_1} \\ 0 \\ 0 \end{bmatrix} + \begin{bmatrix} htd_{SCS_o} \\ 0 \\ 0 \end{bmatrix} \\ + \begin{bmatrix} \sum_{r=0}^2 \phi_\alpha(d)_r p_{SCS_m} \\ p_{CM1_m} \\ p_{CM2_m} \end{bmatrix} + \begin{bmatrix} \sum_{r=0}^2 \phi_\alpha(d)_r a_{SCS_n} \\ a_{CM1_n} \\ a_{CM2_n} \end{bmatrix} + \begin{bmatrix} e_{SCS} \\ e_{CM1} \\ e_{CM2} \end{bmatrix},$$

where y_{SCS} , y_{CM1} , and y_{CM2} are test-day SCS observations, and observations (0 or 1) on CM1 and CM2, respectively, recorded in herd test-day o , in year $\times$ month of calving k , herd-year h , on a cow m belonging to the calving age class i , calving-year $\times$ calving-season class l and measured on DIM d . Fixed effects were age at calving (f), year $\times$ month of calving (ym), herd $\times$ year (hy), and regression coefficients (b) describing the shape of the lactation curve within calving-year $\times$ calving-season classes. The age effect (in months) consisted of 9 classes, and there were 72 ym classes in first lacta-

tion, starting from January 1995. The calving seasons were October to February, March to June, and July to September. The herd sizes are small in Finland and therefore the herd effect was modeled by fixed herd-year and random herd-test-day (**htd**) components. For CM traits, because htd classes were too small for reliable estimation a random herd-year and no htd effect were fitted. The number of hy and htd classes are in Table 1.

Random genetic effects were a_{SCSn} , a_{CM1n} , and a_{CM2n} . The a_{SCSn} represented random genetic effects for test-day SCS with

$$\phi_\alpha(d) = [c_0 \ c_1 \ c_2]^T \quad [2]$$

representing a second-order orthogonal Legendre polynomial at DIM d . Random effects p_{SCSm} , p_{CM1m} , and p_{CM2m} were nongenetic animal effects for a cow m with $\phi_\alpha(d)$ as in [2] for test-day SCS, CM1, and CM2, respectively. Random e_{SCS} , e_{CM1} , and e_{CM2} were measurement errors.

To facilitate accurate variance component estimation for the CM traits, the multitrait RRM has to be defined as both the test-day and CM traits would be repeated observations but never recorded simultaneously. Thus, the variance between daily residuals was assumed uncorrelated, but the animalwise environmental covariance among traits was modeled by permanent environmental effect. The estimation of this component for CM traits was accomplished during REML analyses by restricting the residual variance of CM traits to a predetermined operationally small value. As a result, most of the residual variance was absorbed into the permanent environmental component facilitating the estimation of permanent environmental correlation between CM and test-day traits. The resulting covariance components of the random regression coefficients for sire additive genetic and cow permanent environmental effects were then used for estimation of the necessary parameters. All analyses were made using the DMU package (Madsen and Jensen, 2000).

Estimation of Genetic Parameters. Daily sire variance of SCS or MY at time d_i can be written as

$$\sigma_s^2(d_i) = \phi'_\alpha(d_i) \mathbf{G}_s \phi_\alpha(d_i),$$

where $\mathbf{G}_s$ is the covariance matrix of the random sire regression coefficients for SCS or MY. Heritability of a trait at any time d_i along the lactation trajectory was estimated as

$$\hat{h}_{d_i}^2 = 4\hat{\sigma}_s^2(d_i)/[\hat{\sigma}_e^2 + \hat{\sigma}_p^2(d_i) + \hat{\sigma}_s^2(d_i) + \hat{\sigma}_{htd}^2],$$

where $\hat{\sigma}_p^2$ is the variance of permanent environmental effects given as $(\phi'_\alpha(d_i) \mathbf{P} \phi_\alpha(d_i))$. Genetic correlation between a test-day and CM trait at times d_i was estimated as:

$$\hat{r}_g(d_i) = \frac{(\phi'_\alpha(d_i) \mathbf{G}_{scs,cm} \mathbf{1})}{[\hat{\sigma}_{scs}^2(d_i) \hat{\sigma}_{cm}^2]^{1/2}},$$

where $\mathbf{G}_{scs,cm}$ are random regression coefficients of the sire genetic covariance between the test-day trait and CM, and $\hat{\sigma}_{cm}^2$ is the sire genetic variance of the CM trait.

RESULTS AND DISCUSSION

Model Selection

Model comparisons of the RRM were based on estimates of log-likelihoods, AIC, residual variance and proportion of the total variance explained by each eigenvalue of the additive genetic covariance function for the different orders of fit (Table 2). Estimates of the log-likelihoods for both test-day SCS and MY showed that increased order gave better fit up to second order for SCS and up to third order for MY. The lowest AIC was for the quadratic model for SCS and the cubic model for MY. The analyses of eigenvalues of the genetic covariance function showed that the largest eigenvalue accounted for over 80% of the additive genetic variation for both SCS and MY. However, for SCS, the last eigenvalue of the cubic RRM accounted for much less than 1% of the variation suggesting that the quadratic RRM is the most appropriate.

With the increase in the order of the random regression, there was a decrease in the estimates of residual variance (Table 2). However, the decrease in the residual variance estimates tended to decline as the order of the random regression increased. For instance, in going from quadratic to cubic RRM the decline in the residual variance for test-day MY was about 13%, whereas for test-day SCS a slight increase in the residual variance between quadratic and cubic RRM was observed. The model comparison indicated that second- and third-order orthogonal Legendre polynomials were appropriate for modeling genetic variances for test-day SCS and MY, respectively, across DIM. This is consistent with models from previous studies for SCS (Ødegård et al., 2003; Koivula et al., 2004) and MY (Pool et al., 2000; Lidauer et al., 2003).

In this study, the permanent environmental effect for both test-day SCS and MY was modeled with the same function as the additive genetic effect. This is to ensure that both curves had equal flexibility (Olori et al., 1999; Pool and Meuwissen, 2000) and because such models are easier to implement.

Table 2. Log-likelihoods (log-L), Akaike information criteria (AIC), residual variance (σ_e^2), and proportion of the variation explained by each eigenvalue for the first 4 orders of test-day SCS and milk yield

Trait	Order ¹	Log-L	AIC	σ_e^2	Proportion of variation (%)			
SCS	LP ₀	-74,339	148,683	0.59	100.0			
	LP ₁	-73,293	146,595	0.50	93.0	7.0		
	LP ₂	-72,373	144,759	0.44	91.2	6.7	2.1	
	LP ₃	-72,453	144,923	0.45	84.9	9.0	5.9	0.1
Milk yield	LP ₀	-237,636	475,276	7.42	100.0			
	LP ₁	-226,722	453,451	5.90	90.2	9.8		
	LP ₂	-216,253	432,518	4.62	85.0	13.0	2.0	
	LP ₃	-212,286	424,587	4.00	84.1	11.7	2.4	1.6

¹LP = Legendre polynomials; LP₀ = intercept; LP₁ = linear; LP₂ = quadratic; and LP₃ = cubic random regression models.

Heritability

Heritabilities of CM1, CM2, test-day SCS, and MY for selected DIM of first lactation are in Tables 3 and 4. For CM1 and CM2, the heritabilities from the multitrait RRM with either test-day SCS or MY were 0.03 and 0.02, respectively. Using a linear sire model, Heringstad et al. (2001) and Svendsen and Heringstad (2006) reported heritability of 0.03 for the period of 15 d before to 30 d after calving for the Norwegian Red cattle. For Danish Red cattle, Lund et al. (1999) reported heritability of 0.05 and 0.02 for CM traits defined as 10 d before to 50 d after calving and 50 to 350 d after calving, respectively. The estimates from the present study fall within the range of most values (0.02 to 0.03) reported for heritability of CM from analyses with the linear models based on data from the Nordic health-recording systems (Heringstad et al., 2000).

Heritability of test-day SCS during first lactation ranged from 0.07 to 0.12 (Table 3). The estimates were

less than 0.09 in early lactation and increased gradually, reaching peak at 0.12 at 160 d after calving (Table 3). Possible explanations for the lower estimates of heritability at the beginning of lactation could be large environmental variation during the early stages of lactation, or low genetic variance. It is also important to note here that if a cow has udder infections at the test-day, the farmer does not take SCC measurements. This usually occurs in early lactation (when the incidence of mastitis is high) and might have caused a reduction in the amount of available information on SCS and consequently increased the environmental components leading to low heritability estimates. This is in line with earlier studies (Koivula et al., 2004; Silvestre et al., 2006) that have shown lower estimates of heritability for the early stages of first lactation. They are, however, in contrast to the results of Liu et al. (2001) and de Roos et al. (2003) who reported a fairly homogeneous daily heritability of about 0.09 for first-lactation Ger-

Table 3. Heritabilities (diagonal, in bold), genetic (below diagonal), and phenotypic (above diagonal) correlations between test-day SCS and clinical mastitis traits CM1 and CM2 for selected DIM estimated with the multitrait random regression model

		SCS							CM1 ¹	CM2 ²
	DIM	30	60	110	160	210	260	310		
SCS	30	0.07	0.62	0.58	0.53	0.48	0.43	0.38	0.02	0.19
	60	0.99	0.08	0.64	0.61	0.57	0.52	0.45	-0.01	0.20
	110	0.96	0.99	0.09	0.67	0.65	0.61	0.52	-0.03	0.20
	160	0.93	0.97	0.98	0.12	0.68	0.65	0.56	-0.05	0.19
	210	0.90	0.94	0.97	0.99	0.10	0.68	0.60	-0.05	0.17
	260	0.85	0.90	0.94	0.97	0.98	0.09	0.64	-0.04	0.15
	310	0.78	0.83	0.87	0.91	0.95	0.98	0.07	-0.02	0.11
CM1		0.77	0.66	0.60	0.55	0.50	0.45	0.41	0.03	0.09
CM2		0.34	0.49	0.54	0.58	0.63	0.68	0.71	0.46	0.02

¹CM1 = clinical mastitis trait scored as 0 or 1 depending on whether a cow had at least one veterinary treatment of clinical mastitis or was culled due to mastitis reasons in the period from 7 d before to 30 d after calving.

²CM2 = clinical mastitis trait scored as 0 or 1 depending on whether a cow had at least one veterinary treatment of clinical mastitis or was culled due to mastitis reasons in the period from 31 to 300 d after calving, culling, or second calving.

Table 4. Heritabilities (diagonal, in bold), genetic (below diagonal), and phenotypic (above diagonal) correlations between test-day milk yield and clinical mastitis traits CM1 and CM2 for selected DIM estimated with the multitrait random regression model

	DIM	Milk yield						CM1 ¹	CM2 ²
		30	60	110	160	210	260	310	
Milk yield	30	0.18	0.73	0.64	0.57	0.49	0.39	0.30	-0.03
	60	0.98	0.19	0.76	0.71	0.60	0.46	0.33	-0.01
	110	0.93	0.98	0.19	0.79	0.69	0.55	0.39	-0.03
	160	0.88	0.94	0.98	0.19	0.76	0.65	0.48	0.02
	210	0.79	0.84	0.91	0.97	0.20	0.76	0.61	-0.06
	260	0.67	0.71	0.79	0.88	0.97	0.20	0.73	-0.06
	310	0.52	0.57	0.66	0.77	0.89	0.97	0.17	-0.05
CM1		0.51	0.44	0.36	0.34	0.31	0.24	0.13	0.03
CM2		0.66	0.59	0.57	0.60	0.65	0.63	0.49	0.02

¹CM1 = clinical mastitis trait scored as 0 or 1 depending on whether a cow had at least one veterinary treatment of clinical mastitis or was culled due to mastitis reasons in the period from 7 d before to 30 d after calving.

²CM2 = clinical mastitis trait scored as 0 or 1 depending on whether a cow had at least one veterinary treatment of clinical mastitis or was culled due to mastitis reasons in the period from 31 to 300 d after calving, culling, or second calving.

man Holsteins and heritability ranging from 0.12 to 0.19 for early-lactation Dutch Holsteins, respectively.

The heritability of MY during lactation ranged from 0.17 to 0.20 (Table 4). The heritability was lowest from calving until peak of lactation, rose toward mid lactation, and then declined after 250 DIM. The highest heritability was around 200 DIM. This is because of the high genetic variance in the mid to late stages of lactation and decreasing residual and permanent environmental variances. On the other hand, the low heritability at the beginning and the decline observed at the end of the lactation could be explained by the strong influence of nongenetic effects accumulated before calving and associated with the farmer's decision regarding drying off at the end of the lactation. Similar estimates of heritability curves have been reported in studies using multitrait analysis (Liu et al., 2000; Druet et al., 2003) and RRM (Pool et al., 2000).

Genetic Correlations

Genetic Correlations Between Test-Day SCS and CM Traits. The genetic correlation between CM1 and test-day SCS during lactation ranged from 0.41 to 0.77 (Table 3). The highest correlation was found in the interval between calving and 30 d after calving where the correlations between the 2 traits ranged from 0.68 to 0.78. The genetic correlation between CM2 and test-day SCS during lactation ranged from 0.34 to 0.71 (Table 3). It was lowest in the early part of lactation (ranging from 0.34 to 0.50) and highest in the late stages of lactation (ranging from 0.60 to 0.72). The high genetic correlation between CM1 and SCS in the early part of lactation and between CM2 and SCS in the late stages of lactation was as expected and indicates that CM and

SCS from the same interval may have more similar genetic bases than those from different intervals. Generally, genetic associations between test-day SCS and CM traits varied during lactation. Across lactation, the genetic correlations between the traits were much lower than unity, suggesting that SCS seems to measure something other than CM. Thus, combining information from CM traits with the test-day SCS information should increase the accuracy of genetic evaluation for udder health (Pösö and Mäntysaari, 1996; Negussie et al., 2006a).

The variability of genetic correlations between SCS and CM traits during lactation is consistent with the reports from an earlier multitrait analysis by Negussie et al. (2006c). In addition, the high genetic correlation observed between CM1 and test-day SCS, particularly during the early part of the lactation, is in agreement with Heringstad et al. (2006). Using a bivariate threshold-linear sire model, they reported that with high probability the genetic correlation between CM and test-day SCS in the first 30-d interval of early lactation lies between 0.53 and 0.71.

In the literature, most estimates of genetic correlations between CM and SCS have been from lactation-average models (Pösö and Mäntysaari, 1996; Rupp and Boichard, 1999; Carlén et al., 2004; Negussie et al., 2006a) and are reported to average 0.6 (Heringstad et al., 2000). The estimates from this study were greater than 0.6 for some parts of lactation and varied across the lactation. One limitation of the lactation-average model is inefficient use of available information, because it does not account for stage of lactation effects (Ødegård et al., 2003; Negussie et al., 2006b). Moreover, with the lactation-average model, longitudinal description of the genetic association between these traits dur-

ing lactation is not possible. The practical utility of the results from this study would therefore be the ability to combine test-day SCS information with CM traits (defined over the different stages of lactation) in the genetic evaluation of animals for udder health. This will offer increased accuracy of evaluations for mastitis. In addition, with the multitrait RRM analyses, daily breeding values for all animals and different selection criteria can be calculated. This may enable testing of sires at a younger age offering an early prediction of animals' genetic merit.

Genetic Correlations Between Test-Day MY and CM Traits. Genetic correlations between test-day MY and CM traits from the multitrait RRM were all positive and moderate (Table 4). In early lactation (before 60 d), the genetic correlations between test-day MY and CM1 and between test-day MY and CM2 ranged from 0.44 to 0.51 and from 0.59 to 0.66, respectively. The positive and moderate genetic correlations between these traits confirm the antagonistic relationship between udder health and milk production. Furthermore, genetic correlations between both CM traits and test-day MY were greatest during early lactation (Table 4). The observed pattern of changing correlations during lactation between the traits may suggest that selection for high MY alone in early lactation will have a more deleterious effect on mastitis than selection on higher milk yield in late lactation.

There are no previous studies on genetic correlations during lactation between CM traits and test-day MY. All literature estimates of genetic correlations between CM and MY are from lactation-average models and ranged from 0.24 to 0.55 (Heringstad et al., 2000; Hansen et al., 2002). The estimates from the present study were slightly higher and varied during lactation.

Final Remarks

The most common approach when utilizing mastitis data in genetic evaluations is to consider mastitis as a binomial trait and to apply linear models, which assume a normal distribution of the data (Heringstad et al., 2000). Threshold models are an alternative to linear models and are theoretically more appropriate. Because of software problems, all estimates of parameters in the present study were obtained from the linear model analyses.

In the Nordic countries, selection for improved mastitis resistance is mainly based on CM records (Heringstad et al., 2000; Negussie et al., 2006a), whereas in other countries genetic improvement of udder health mainly relies on selection for reduced SCC and possibly other traits such as udder type traits. Thus, strategies for selection to improve mastitis resistance vary across

countries (Negussie et al., 2006a) as do the definitions of CM traits. Most commonly, CM traits have been defined on a lactation basis and analyzed fitting lactation-average models. Such definition of CM traits does not fully utilize the information available from dairy health recording schemes. Recently, there has been increased interest in the longitudinal modeling of CM (e.g., Heringstad et al., 2003; Chang et al., 2004a; Negussie et al., 2006d).

The advantages of a longitudinal model for CM include the ability of accounting for stage of lactation as well as for short-term environmental effects. However, earlier results have shown unrealistic estimates of parameters at the ends of the lactation period (Heringstad et al., 2003; Chang et al., 2004a; Negussie et al., 2006d). A longitudinal threshold model analysis of CM (Heringstad et al., 2003) has also concluded that a longitudinal model may be unable to capture differential gene expression in different parts of the lactation because the genetic variance-covariance structure is static. Furthermore, the dynamics of the model are induced by the Legendre function (which does not have a genetic component) and hence a multiple trait definition of CM has been suggested as the most logical specification (e.g., Heringstad et al., 2001; Chang et al., 2004b; Negussie et al., 2006c).

In this study, a 2-trait definition of CM was used for the analysis of CM with the test-day traits. One of the reasons for the definition of only 2 CM traits instead of several CM traits during lactation was to circumvent a high degree of parameterization. In addition, definition of several CM traits during lactation would lead to a complex model requiring more computational effort.

Information on the genetic associations between fertility and health traits with test-day traits such as SCS and MY is scant in the literature. There are few studies that have considered genetic associations between traits with different data structures (Pryce et al., 2001; Veerkamp et al., 2001; Banos et al., 2004) and this has been thoroughly discussed in Mäntysaari (2006). For instance, using RRM, Berry et al. (2003) reported genetic associations between fertility traits measured once during lactation and traits such as BCS, MY, and BW. Recently, Banos et al. (2006) used regressions of SCC, CM, and udder problems other than mastitis on genetic evaluations for BCS, energy content, and cumulative effective energy balance to calculate genetic correlations between these groups of traits. They pointed out that estimates of genetic correlations with this method could be a practical alternative to bivariate RRM models with the longitudinal modeling of CM, which is computationally difficult. They also indicated that the method could be sensitive to the genetic variance estimates assumed in genetic evaluations. Thus,

the approach used in this study could offer a means for estimation of genetic associations between traits with different data structures directly from performance data and without undue influence of assumed genetic variance estimates.

CONCLUSIONS

Genetic associations between the 2 CM traits and test-day SCS or test-day MY varied during lactation. In some parts of the lactation, estimates of the genetic correlations from the multitrait RRM were greater than most estimates in the literature, which are based on lactation-average models. The approach used in this study for estimation of genetic associations between CM and test-day traits offers a means to combine information from traits with different data structures. In particular, the ability to combine information from test-day SCS with CM traits in a multitrait RRM would increase the accuracy of genetic evaluation for mastitis resistance and address the diversity of udder health problems.

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