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Genetic Parameters for Clinical Mastitis, Somatic Cell Score, and Production in the First Three Lactations of Swedish Holstein Cows

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ABSTRACT

Using a mixed linear animal model, genetic parameters were estimated for clinical mastitis (MAST), lactation average somatic cell score (LSCS), and milk production traits in the first 3 lactations of more than 200,000 Swedish Holstein cows with first calving from 1995 to 2000. Heritability estimates for MAST (0.01 to 0.03) were distinctly lower than those for LSCS (0.10 to 0.14) and production traits (0.23 to 0.36). The genetic correlation between MAST and LSCS was high for all lactations (mean 0.70), implying that selection for low LSCS will reduce the incidence of mastitis. Undesirable genetic relationships with production were found for both MAST and LSCS with genetic correlations ranging from 0.01 to 0.45. This emphasizes the need for including udder health traits in the breeding goal. Genetic correlations across lactations for the same trait were positive and high for both MAST (>0.7), LSCS (>0.8), and production traits (>0.9), with the strongest correlations between second and third parity for all traits (>0.9 for udder health traits and close to unity for production traits).
(**Key words:** genetic correlation, heritability, health, dairy cattle)

Abbreviation key: LSCS = lactation average somatic cell score, **MAST** = clinical mastitis.

INTRODUCTION

Mastitis is one of the most common and costly diseases in dairy cattle. In Sweden, the number of veterinary-treated cases of mastitis per 100 lactations was 18.3 in year 2000–2001, and udder diseases, together with high SCC, were the second leading reason for culling in year 2001, accounting for nearly 24% of culled cows (Svensk Mjölk, 2002). Economic losses are considerable and associated with reduced milk yield, discarded milk, reduction in milk price due to high SCC, veterinary and treatment costs, increased labor, and increased culling rate. Animal welfare and ethical aspects, such as the use of antibiotics, are also strong arguments for reducing the frequency of mastitis.

Selection has traditionally focused on production traits. Today it is generally accepted that undesirable genetic relationships exist between production and health disorders, including mastitis (e.g., Rauw et al., 1998). According to several studies, milk production is unfavorably genetically correlated with both clinical mastitis and SCC (e.g., Emanuelson et al., 1988; Nielsen et al., 1997; Rupp and Boichard, 1999; Heringstad et al., 2000; Castillo-Juarez et al., 2002; Hansen et al., 2002), although some authors have reported favorable genetic associations between production and SCC in later parities (Pösö and Mäntysaari, 1996; Haile-Mariam et al., 2001a).

The heritability of clinical mastitis is low, especially when analyzed with linear models (Pösö and Mäntysaari, 1996; Rupp and Boichard, 1999; Lassen et al., 2003). Owing to the higher heritability of SCC and its high genetic correlation with clinical mastitis, it can be used for indirect selection to improve mastitis resistance (Mrode and Swanson, 1996; Heringstad et al., 2000). However, selection has been proven to be most efficient when information on clinical cases and SCC are combined (e.g., Philipsson et al., 1995). In the Swedish national genetic evaluation, bulls receive breeding values for clinical mastitis, based on information on clinical mastitis (veterinary treatments and culling due to mastitis) and SCC in first lactation daughters (Svensk Mjölk, 1999).

Mastitis is, however, not only a problem in first lactation. Actually, both mastitis frequency (Pösö and Mäntysaari, 1996; Nielsen et al., 1997) and level of SCC (Da et al., 1992; Reents et al., 1995; Nielsen et al., 1997)

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Table 1. Number of observations, means, and standard deviations for production and udder health traits in the first 3 lactations of Swedish Holstein cows.

	Lactation								
	1			2			3		
Trait[1]	n	$\bar{X}$	SD	n	$\bar{X}$	SD	n	$\bar{X}$	SD
MILK	192,652	7575	1552	81,715	8832	1713	36,273	9308	1736
FAT	192,652	303	61	81,715	354	68	36,273	373	71
PROT	192,652	249	48	81,715	291	53	36,273	303	53
LSCS	206,033	0.787	0.426	115,107	0.891	0.488	54,562	0.995	0.501
MAST	221,104	0.104	0.305	122,280	0.121	0.326	59,233	0.149	0.357

[1]MILK = Milk production (kg) in 305 d, FAT = fat production (kg) in 305 d, PROT = protein production (kg) in 305 d, LSCS = lactation average somatic cell score from 5 to 150 d after calving (expressed in 10,000 cells/mL) transformed to a logarithmic scale with base 10, MAST = veterinary treatments of, and culling due to, mastitis observed from 10 d before to 150 d after calving (0/1).

increase with increasing parity. Ideally, genetic evaluation for mastitis resistance also would include information from later lactations. Depending on the genetic parameters, multiple lactation records can be considered either as different traits in a multi-trait model or as repeated manifestations of the same trait in a single-trait repeatability model (Da et al., 1992; Reents et al., 1995).

The primary objectives were to estimate heritabilities of, and genetic correlations between, clinical mastitis and lactation average somatic cell score (**LSCS**) and to estimate genetic correlations between these udder health traits and production traits in the first 3 lactations of Swedish Holstein cows. A further aim was to estimate genetic correlations for udder health traits across lactations.

MATERIALS AND METHODS

Data

Data on clinical mastitis, SCC, and production were extracted from the Swedish milk recording scheme, and they were edited to include records from the first 3 lactations of Swedish Holstein cows having their first calving between 1995 and 2000. Although information on lactation number was given, a general restriction of age at calving was constructed to exclude cows with wrong lactation number. The defined minimum and maximum ages for first, second, and third calving were 20 to 38, 32 to 52, and 43 to 66 mo, respectively. If age at calving at a particular lactation was below or above the allowed period, records for that lactation were not used in analyses. The same was true if a cow belonged to a herd-year class with fewer than 2 observations. Cows from sires with fewer than 50 daughters in the data before editing were excluded. The number of sires and number of herd-year classes for lactation 1 to 3 were 838, 784, 673 and 31,511, 22,023, 13,570, respectively. The number of observations after editing for analyzed traits in the 3 lactations are given in Table 1. To make bivariate analysis computationally feasible, the total data set (1) was split into 2 smaller data sets of equal size (2 and 3) by assigning herds randomly to either data set. All known pedigree information of the cows was traced back as far as possible, resulting in relationship matrices of 539,919, 288,809, and 286,171 animals for data sets 1, 2, and 3, respectively.

The data sets contained information on udder health traits, 305-d production (milk, fat, and protein yield), days open, as well as proportion of North American Holstein and proportion of heterosis. Days open was calculated as the number of days from calving to last insemination. Cows not inseminated after calving and cows inseminated <30 d or >250 d after calving were assigned the average value for days open. The proportion of North American Holstein was calculated for each individual animal from proportion of North American Holstein of the sire and the dam, respectively, and originally derived from imported North American Holstein sires with proportion 1. The proportion of heterosis was estimated using the formula: s (1-d) + d (1-s), where s (d) is the proportion of North American Holstein of the sire (dam). The Swedish Holstein breed can currently be considered a synthetic population of the original Swedish Friesian and foreign Holstein, as extensive use of Holstein sires, mainly from the United States and Canada, has taken place during the last decades (Koenen et al., 1994). The proportion of North American Holstein genes for cows in this study, which were born from 1988 to 1999, increased from about 50 to 75%. The degree of heterosis showed an opposite trend as a consequence of the increased level of North American Holstein genes in both the female and male population during these years.

Definition of Traits

Mastitis, SCC, and production were defined in the same way as in the Swedish national genetic evalua-

tion. A case of mastitis (**MAST**) was defined as a veterinary-treated clinical mastitis (with or without teat injury) from 10 d before to 150 d after calving, or culling for mastitis within that period. The restricted time period was used to reduce bias due to culling. Mastitis was defined as a binary trait distinguishing between cows with at least one reported case during the defined period (1) and cows without cases (0). Lactation average somatic cell score (LSCS) was the arithmetic mean of monthly test day SCC from 5 to 150 d after calving, expressed in 10,000 cells/mL, and transformed to a logarithmic scale with base 10 before averaging. Production of milk, fat, and protein (kg) was based on completed 305-d lactations. For interrupted lactations of >45 d length, and ongoing lactations of >100 d length, production was extended to 305-d yield. Real 305-d yield was analyzed for lactations of >305 d length, and for completed lactations of <305 d length total production in that lactation was analyzed without extension (i.e., MILK, FAT, PROT).

Statistical Analysis

(Co)variance components were estimated by REML, and analyses were performed with the DMU package (version 6, release 4) developed by Madsen and Jensen (2000). Both convergence criteria were set to 10^{-6}. Estimates of heritabilities were derived from univariate analyses on data set 1 and estimates of correlations between traits and between lactations for the same trait were averages from 2 bivariate analyses on data sets 2 and 3. The following linear animal model was used for the production traits:

$$y_{ijkl} = hy_i + ym_j + age_k + a_l + b_1 Het_l + b_2 Hol_l + b_3 DOP_l + e_{ijkl}$$

where y_{ijkl} is the observation; hy_i is the fixed effect of ith herd by year of calving; ym_j is the fixed effect of jth year by month at calving; age_k is the fixed effect of kth age in months at calving (one month per class); a_l is the random effect of lth animal; b_1 is the fixed regression coefficient on the proportion of heterosis of animal l (Het_l); b_2 is the fixed regression coefficient on the proportion of North American Holstein of animal l (Hol_l); b_3 is the fixed regression coefficient on days open of animal l (DOP_l); and e_{ijkl} is the random residual effect. The same model, without the regression on days open, was used for the udder health traits. Random effects were assumed to have zero means and the covariance structure for bivariate analysis was:

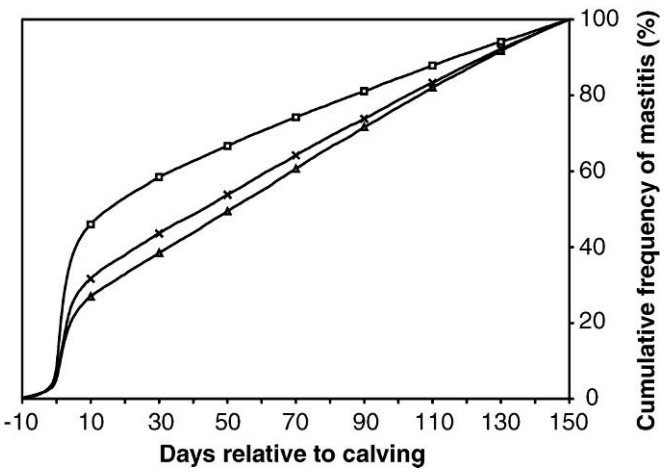

Figure 1. The cumulative relative frequency of the total number of mastitis cases within 150 d of lactation one (□), two (△), and three (×) of Swedish Holstein cows.

$$V\begin{bmatrix}\mathbf{a}_1\\ \mathbf{a}_2\\ \mathbf{e}_1\\ \mathbf{e}_2\end{bmatrix} = \begin{bmatrix}\mathbf{A}\sigma^2_{a_1} & \mathbf{A}\sigma_{a_{12}} & 0 & 0\\ & \mathbf{A}\sigma^2_{a_2} & 0 & 0\\ & symm. & \mathbf{I}\sigma^2_{e_1} & \mathbf{I}\sigma_{e_{12}}\\ & & & \mathbf{I}\sigma^2_{e_2}\end{bmatrix}$$

where **A** is the additive relationship matrix, **I** is the identity matrix, and the indices represent the 2 traits in the bivariate analysis.

RESULTS

Basic Statistics

The overall means for the traits can be seen in Table 1. Figure 1 shows the cumulative relative frequencies of mastitis from 10 d before to 150 d after calving in the first 3 lactations. Mastitis frequency was highest at calving and in the beginning of all lactations, and it was higher for first-lactation cows than for cows in later lactations. About 46% of all cases up to 150 d in first lactation occurred within 10 d after calving, whereas the same level was reached after 44 and 34 d in the second and third lactations, respectively. For all lactations, the total number of cases within 150 d of lactation constituted about 60 to 65% of all cases in completed lactations. Thus, the opportunity period (–10, 150) captures a large part of all cases. Even though only a few percent of all cases occurred before calving, it has been previously shown that heritability increases when these days are included in the registration period for mastitis (Heringstad et al., 1997).

Table 2. Estimated effects of increasing age at calving by 1 mo on production traits, lactation average somatic cell score (LSCS) and clinical mastitis in the first 3 lactations of Swedish Holstein cows.[1]

	Lactation		
Trait[2]	1	2	3
MILK	61	67	45
FAT	3.2	3.0	2.2
PROT	2.2	2.1	1.3
LSCS	0.0056	0.0038	0.0010
MAST	0.0035	0.0018	0.0015

[1]Effects shown are regressions of class estimates on age class in the intervals 23 to 35, 35 to 47, and 47 to 59 months of age at calving for lactation 1, 2 and 3, respectively.

[2]MILK = milk production (kg) in 305 d, FAT = fat production (kg) in 305 d, PROT = protein production (kg) in 305 d, LSCS = lactation average somatic cell score from 5 to 150 d after calving (expressed in 10,000 cells/mL) transformed to a logarithmic scale with base 10, MAST = veterinary treatments of, and culling due to, mastitis observed from 10 d before to 150 d after calving (0/1).

Effects of Systematic Environmental Effects

Presented estimates of fixed effects are from univariate analyses on the total data set. Increased age at calving was associated with an increase in both mastitis frequency, level of LSCS, and production for all 3 lactations (Table 2). No clear pattern could be seen for the effect of year and month at calving (January 1995 to December 2000) on mastitis frequency, whereas LSCS tended to be lower and production traits higher for cows calving during the last 6 mo of a year (results not shown).

Estimated effects of heterosis (100 vs. 0%) and proportion of North American Holstein (100% North American Holstein vs. 100% original Swedish Friesian) on mastitis, LSCS, and production from univariate analyses are shown in Table 3. The effect of heterosis was less MAST, lower LSCS, and higher production. The effect of North American Holstein was more cases of MAST, higher LSCS, and higher production.

For production traits a third covariate was included in the model, namely regression on number of days open. When days open increased with 1 d, production for first-parity cows increased with 6.2, 0.19, and 0.22 kg of milk, protein, and fat, respectively. The corresponding figures for second- and third-parity cows were 7.9, 0.24, and 0.29 kg and 7.6, 0.23, and 0.30 kg, respectively.

Heritabilities and Correlations

Heritabilities and correlations between traits within parities are provided in Table 4. Heritabilities of MAST were low: 0.03 for first lactation and 0.01 for later lactations. For LSCS, heritabilities were considerably higher than those of MAST, but here also estimates decreased slightly with increasing parity, from 0.14 to 0.10. Heritabilities of milk, fat, and protein production were of moderate size, ranging from 0.23 to 0.36.

Table 3. Estimated effects of heterosis and proportion North American Holstein on production traits, LSCS, and clinical mastitis in the first 3 lactations of Swedish Holstein cows.

		100% heterosis		100% N. Am Holstein	
Lactation	Trait[1]	Effect	SE	Effect	SE
1	MILK	157.1	23.7	342.7	71.5
	FAT	8.38	0.95	7.14	2.86
	PROT	5.25	0.71	10.12	2.13
	LSCS[2]	–0.034	0.007	0.048	0.020
	MAST	–0.014	0.005	0.040	0.012
2	MILK	35.4	38.0	669.7	102.7
	FAT	5.71	1.62	13.14	4.51
	PROT	2.43	1.15	17.29	3.12
	LSCS[2]	–0.041	0.011	0.033	0.029
	MAST	–0.024	0.007	0.016	0.014
3	MILK	111.8	60.0	802.0	145.6
	FAT	5.21	2.60	16.47	6.39
	PROT	3.06	1.82	19.68	4.40
	LSCS[2]	–0.057	0.017	0.017	0.038
	MAST	–0.028	36.7	0.028	38.8

[1]MILK = Milk production (kg) in 305 d, FAT = fat production (kg) in 305 d, PROT = protein production (kg) in 305 d, LSCS = lactation average somatic cell score from 5 to 150 d after calving (expressed in 10,000 cells/mL) transformed to a logarithmic scale with base 10, MAST = veterinary treatments of, and culling due to, mastitis observed from 10 d before to 150 d after calving (0/1).

[2]The estimates for lactation 1 to 3 correspond to about 4600, 7000, and 12,200 cells less and 7100, 6100, and 4000 cells more for heterosis and North American Holstein, respectively. The differences in number of cells were calculated by adding the effect to the mean value for somatic cell score and compare the difference in number of cells with and without the effect.

Table 4. Estimated parameters[1] for production traits, LSCS, and clinical mastitis in the first, second, and third lactations of Swedish Holstein cows. Heritabilities in bold on diagonal, genetic correlations above diagonal and environmental correlations below diagonal. Genetic standard deviations (σ_a) on last line for each lactation. The subscripts are the approximated standard errors for the estimates.[2]

Lactation trait	Trait[3]				
	MILK	FAT	PROT	LSCS	MAST
First lactation					
MILK	**0.34**$_{0.01}$	0.53$_{0.02}$	0.87$_{0.01}$	0.22$_{0.04}$	0.32$_{0.06}$
FAT	0.88	**0.36**$_{0.01}$	0.67$_{0.01}$	0.17$_{0.04}$	0.22$_{0.06}$
PROT	0.97	0.89	**0.31**$_{0.01}$	0.23$_{0.04}$	0.29$_{0.06}$
LSCS	–0.22	–0.17	–0.19	**0.14**$_{0.01}$	0.68$_{0.05}$
MAST	–0.13	–0.11	–0.11	0.14	**0.030**$_{0.003}$
σ_a	712.2	28.98	20.13	152.4	0.0511
Second lactation					
MILK	**0.25**$_{0.01}$	0.21$_{0.05}$	0.76$_{0.02}$	0.13$_{0.06}$	0.45$_{0.11}$
FAT	0.88	**0.32**$_{0.02}$	0.52$_{0.03}$	0.03$_{0.06}$	0.12$_{0.12}$
PROT	0.96	0.89	**0.25**$_{0.01}$	0.18$_{0.06}$	0.37$_{0.11}$
LSCS	–0.15	–0.13	–0.14	**0.13**$_{0.01}$	0.66$_{0.09}$
MAST	–0.07	–0.07	–0.06	0.17	**0.012**$_{0.003}$
σ_a	612.2	29.65	18.67	163.1	0.0348
Third lactation					
MILK	**0.23**$_{0.02}$	0.11$_{0.09}$	0.74$_{0.04}$	0.13$_{0.11}$	0.26$_{0.20}$
FAT	0.88	**0.25**$_{0.02}$	0.47$_{0.07}$	0.02$_{0.11}$	0.01$_{0.20}$
PROT	0.96	0.88	**0.23**$_{0.02}$	0.13$_{0.11}$	0.19$_{0.20}$
LSCS	–0.13	–0.11	–0.11	**0.10**$_{0.01}$	0.77$_{0.15}$
MAST	–0.06	–0.07	–0.06	0.15	**0.012**$_{0.004}$
σ_a	608.3	27.45	18.19	146.5	0.0374

[1]Estimates of heritabilities and genetic standard deviations (σ_a) are from single-trait analyses on full dataset using full pedigree, correlations are averages from 2 bivariate analysis on split data, using full pedigree of respective dataset.

[2]Standard errors are average estimates where applicable. Standard errors for environmental correlations ranged from 0.001 to 0.008, 0.002 to 0.013, and 0.004 to 0.018, for first, second, and third lactations, respectively.

[3]MILK = milk production (kg) in 305 d, FAT = fat production (kg) in 305 d, PROT = protein production (kg) in 305 d, LSCS = lactation average somatic cell score from 5 to 150 d after calving (expressed in 10,000 cells/mL) transformed to a logarithmic scale with base 10, MAST = veterinary treatments of, and culling due to, mastitis observed from 10 d before to 150 d after calving (0/1).

Estimated genetic correlations between MAST and LSCS were about 0.7 to 0.8, with the highest estimate found for the third lactation. Environmental correlations between MAST and LSCS for the 3 first lactations were low with a mean of 0.15.

Genetic correlations between milk production traits in the first 3 parities varied between 0.11 and 0.87, although most estimates were moderate to high. The highest correlation for all lactations was between milk and protein, whereas the correlation between milk and fat was lowest. The strength of the correlations declined with increasing parity, especially between milk and fat. Estimated environmental correlations were all high, about 0.9 to 1.0, and again the highest estimates were between milk and protein.

Estimated genetic correlations between milk production traits and udder health traits were all positive, which indicates an undesirable relationship. The magnitude of the correlations, however, varied considerably between traits and lactations. For instance, MAST was most strongly correlated to milk (0.26 to 0.45) and least to fat. The correlation between MAST on one hand and milk or protein on the other was highest in the second lactation, whereas the correlation between MAST and fat decreased with increasing parity down to near zero in the third lactation. However, the estimates of correlations are averages of 2 bivariate analyses, and for the third lactation, the genetic correlations between MAST and all 3 production traits for the 2 subsets (2 and 3) differed considerably, with estimates for data set 2 being close to zero or slightly negative. The same was true for the correlation between MAST and fat in the second lactation. The corresponding standard errors for these genetic correlations were high. Environmental correlations between MAST and production traits were low and negative for all traits and lactations, ranging between –0.06 and –0.13.

Estimated genetic correlations between LSCS and production traits were lower than the corresponding correlations between MAST and production traits. The highest estimates of LSCS were found for the first lactation (0.17 to 0.23). In later lactations the correlations in our study decreased to about 0.1 and 0.2 for LSCS and milk or protein, respectively, and they were close to

Table 5. Estimated genetic (r_g) and environmental (r_e) correlations of production and udder health traits across the first three lactations in Swedish Holstein cows. The subscripts are the approximated standard errors for the estimates.[1]

	r_g			r_e		
Trait[2]	1–2	1–3	2–3	1–2	1–3	2–3
MILK	$0.90_{0.01}$	$0.90_{0.02}$	$0.99_{0.01}$	0.40	0.34	0.34
FAT	$0.91_{0.01}$	$0.92_{0.02}$	$0.99_{0.01}$	0.38	0.32	0.31
PROT	$0.91_{0.01}$	$0.88_{0.03}$	$0.99_{0.01}$	0.42	0.33	0.36
LSCS	$0.88_{0.02}$	$0.81_{0.04}$	$0.98_{0.02}$	0.22	0.13	0.26
MAST	$0.76_{0.10}$	$0.70_{0.15}$	$0.92_{0.14}$	0.04	0.03	0.05

[1]Standard errors are average estimates. Standard errors for environmental correlations ranged from 0.005 to 0.018.

[2]MILK = milk production (kg) in 305 d, FAT = fat production (kg) in 305 d, PROT = protein production (kg) in 305 d, LSCS = lactation average somatic cell score from 5 to 150 d after calving (expressed in 10,000 cells/mL) transformed to a logarithmic scale with base 10, MAST = veterinary treatments of, and culling due to, mastitis observed from 10 d before to 150 d after calving (0/1).

zero for LSCS and fat. In similarity with the estimates between MAST and production in the third lactation, the correlations between LSCS and production in the third lactation are less precise and are averages based on 2 estimates that differed markedly. Environmental correlations between LSCS and production traits were estimated at about –0.2 in first lactation and somewhat weaker in later lactations.

The correlations across lactations for the same trait are given in Table 5. Estimated genetic correlations of MAST across the first 3 lactations were all above 0.7. The highest estimate was between second and third lactation (>0.9), whereas the lowest was between first and third lactation. However, the average value between first and third parity is calculated from 2 estimated genetic correlations (0.46 and 0.98) that differed considerably. One of the 2 bivariate analyses for the genetic correlation between parities 2 and 3 had convergence problems, even though parameter estimates changed very slowly, and it was therefore interrupted after 50 iterations. Environmental correlations between MAST across lactations were positive but close to zero.

Estimated genetic correlations of LSCS across lactations, ranging from 0.8 to 1.0, were higher and associated with lower standard errors than the corresponding estimates of MAST. In similarity with MAST, the highest estimate was between second and third lactation and the lowest between first and third. Environmental correlations were about 0.1 to 0.3. Also here, the lowest estimate was found between parities with the longest time interval between them.

For production traits, estimated genetic correlations across lactations were high, around 0.9 between first and later lactations, and near unity between the second and third lactations. Environmental correlations across lactations for all production traits were about 0.3 to 0.4.

DISCUSSION

Heritabilities

Heritability estimates of MAST (0.03 and 0.01 for first and later lactations, respectively) are in the range of reported estimates from other studies using linear models. In a review by Heringstad et al. (2000) estimates of heritabilities of clinical mastitis from 13 studies based on Nordic data were between 0.001 and 0.06, with most values in the interval 0.02 to 0.03. Other estimates reported for first lactation range from 0.02 to 0.06 (Rupp and Boichard, 1999; Sørensen et al., 2000; Hansen et al., 2002; Lassen et al., 2003). Few studies have taken later parities into account and results are inconsistent. Pösö and Mäntysaari (1996) found higher heritabilities for lactation 2 and 3 in comparison with lactation one, whereas Nielsen et al. (1997) did not find any differences in estimates between lactations. Heritability estimates on the linear scale are, however, influenced by frequency level, and estimates from different studies are, therefore, not easily comparable (Emanuelson, 1988; Heringstad et al., 2000).

Estimated heritabilities of LSCS (0.10 to 0.14) are in agreement with previously reported estimates. In a review, Mrode and Swanson (1996) reported estimates between 0.05 and 0.47, with weighted average heritabilities of SCC of 0.11 (SD 0.04) and 0.11 (SD 0.07) for first and later lactations, respectively. A later review reports estimates ranging from 0.08 to 0.19 (Heringstad et al., 2000), and more recent estimates are of similar size varying between 0.09 and 0.18 (Haile-Mariam et al., 2001b; Castillo-Juarez et al., 2002; Søndergaard et al., 2002; Mrode and Swanson, 2003; Ødegård et al., 2003). In most studies including later lactations, only a slight variation of heritability of SCC in various lactations was found. However, Da et al. (1992), for example, observed increases in heritability with increasing par-

ity for the first 3 lactations (0.05 to 0.11), whereas Banos and Shook (1990) reported that heritability decreased with increasing parity for the first 3 lactations (0.14 to 0.11).

In our study, the heritability of all traits decreased with increasing lactation number. This was mainly an effect of increasing residual variances but also, in some cases, due to decreasing genetic variances. The lower heritability in later lactations could also partly be explained by selection in first parity. This was confirmed in the analyses with 2 lactations for the same trait, where estimated heritabilities were more similar for different lactations than from the univariate analysis, especially for production traits (for example, for protein in lactation 1 to 3, average heritabilities estimated from bivariate analysis were 0.29, 0.27, and 0.27, whereas heritabilities from univariate analysis were 0.31, 0.25, and 0.23).

Correlations Between Udder Health Traits

The high estimates of genetic correlations between MAST and LSCS (around 0.7 to 0.8) found in this study are in the upper range of reported estimates cited in the literature (Mrode and Swanson, 1996; Rupp and Boichard, 1999; Heringstad et al., 2000), although estimates close to unity have been found (Lund et al., 1994). Heringstad et al. (2000) reviewed 7 studies based on Nordic field data, where estimates of genetic correlations between clinical mastitis and SCC ranged from 0.3 to 0.8, with an average of 0.6. Not many studies have taken later lactations into account. In our study, genetic correlation was higher in third lactation. This is in agreement with the results from Pösö and Mäntysaari (1996), where the largest increase was between first (0.4) and later parities (0.6 and 0.7 in the second and third lactations, respectively) and Nielsen et al. (1997), who found the highest correlation for the third lactation for one of their data sets and no clear difference in the other data set.

Correlations Between Udder Health Traits and Production Traits

In the literature, the genetic correlations between clinical mastitis and production traits have generally been unfavorable. This corresponds to results in our study, where estimates ranged from 0.01 to 0.45, with higher estimates found for first and second parities. Estimates of genetic correlation between mastitis susceptibility and milk yield based on Nordic data ranged from 0.24 to 0.55 (Heringstad et al., 2000). Other reported estimates for first-lactation cows, between clinical mastitis on one hand and milk, protein, or fat yield on the other, also ranged from 0.2 to 0.5 (Emanuelson et al., 1988; Uribe et al., 1995; Nielsen et al., 1997; Van Dorp et al., 1998; Heringstad et al., 1999; Rupp and Boichard, 1999; Hansen et al., 2002). In agreement with our results, Rupp and Boichard (1999) found the lowest correlation between clinical mastitis and fat (0.15) and the highest between clinical mastitis and milk (0.45) in first-lactation Holstein cows.

Estimated genetic correlations between LSCS and production traits for the first 3 lactations ranged from near zero to 0.2, with the strongest correlations for first lactation. These are in the upper range of previously reported estimates. Mrode and Swanson (1996) found, for first lactation, a weighted average genetic correlation between SCC and milk, fat, and protein yields of 0.14 (SD 0.04 to 0.05). More recent estimates for first lactation were between close to zero and 0.3 (Pösö and Mäntysaari, 1996; Charfeddine et al., 1997; Luttinen and Juga, 1997; Nielsen et al., 1997; Pösö et al., 1997; Rupp and Boichard, 1999; Castillo-Juarez et al., 2002). In similarity with previous studies, we found a lower genetic correlation between LSCS and fat yield than between LSCS and milk or protein yield (Charfeddine et al., 1997; Rupp and Boichard, 1999; Castillo-Juarez et al., 2002).

The strength of genetic correlations between LSCS and production traits in our study decreased with increasing parity, although estimates remained positive. Other authors reported that the genetic correlation between SCC and milk production, changed from positive, thus unfavorable, in the first lactation, to negative in later lactations (Banos and Shook, 1990; Pösö and Mäntysaari, 1996; Haile-Mariam et al., 2001a). Two possible explanations for the changes in genetic correlation between parities have been given (Banos and Shook, 1990). First, partly different genes may affect SCC in first vs. later lactations because different pathogens may be mainly responsible for the mastitis cases. Second, it has been argued that culling practices, especially during first lactation, that remove low-producing cows with high occurrence of mastitis and high levels of SCC may have an influence on genetic correlations. However, we would not expect that culling practice to give the observed change in genetic correlation, rather the opposite.

Correlations Across Lactations for Udder Health Traits

For both MAST and LSCS the highest genetic correlations across lactations were between second and third parities and the lowest between first and third parities, probably due to the longer time interval between them. For MAST (0.7 to 0.9) this was in agreement with re-

Table 6. The accuracy (r_{TI}) in selection for mastitis resistance based on different index traits (somatic cell score (LSCS), clinical mastitis (MAST) or both LSCS and MAST) and on different daughter group sizes (50, 100 and 150).

Daughter group size	Traits[1] in index		
	LSCS	MAST	LSCS + MAST
50	0.54	0.53	0.64
100	0.60	0.66	0.74
150	0.62	0.73	0.79

[1]LSCS = lactation average somatic cell score from 5 to 150 d after calving (expressed in 10,000 cells/mL) transformed to a logarithmic scale with base 10, MAST = veterinary treatments of, and culling due to, mastitis observed from 10 d before to 150 d after calving (0/1).

sults from Pösö and Mäntysaari (1996) and Nielsen et al. (1997). Although the size of our estimates were very similar to those estimated by Pösö and Mäntysaari (1996), they were lower overall compared with the estimates by Nielsen et al. (1997) (0.9 to 1.0). The high genetic correlation between parities 2 and 3 could be used as an argument for a multi-trait model with first and later lactations as separate traits.

Estimates of LSCS across lactations (0.8 to near unity) were similar to previously reported estimates. Mrode and Swanson (1996) summarized genetic correlations across lactations for SCC and found simple averages of 0.77, 0.76, and 0.87 between lactations 1 and 2, lactations 1 and 3, and lactations 2 and 3, respectively. More recent studies report genetic correlations around 0.7 to 0.9 between first and second parities, 0.7 to 0.8 between first and third parities, and 0.9 to near unity between second and third parities (Pösö and Mäntysaari, 1996; Boichard and Rupp, 1997; Nielsen et al., 1997; Boettcher et al., 1998; Mrode and Swanson, 2003). Our results suggest that LSCS should be considered as the same trait genetically for lactations 2 and 3, and as a separate but highly correlated trait for lactation one. Thus, based on genetic correlations only, a multi-trait model with first and later lactations as separate traits can be proposed.

Accuracy in Selection for Mastitis Resistance

To compare the accuracy in selection for mastitis resistance when selection is based on MAST, LSCS, or a combination of both measures, selection index theory was used. Accuracy is defined as the correlation between the true breeding goal, which in this case is freedom from clinical cases of mastitis, and the indices, being composed of LSCS, MAST, or LSCS + MAST. Parameters assumed were those estimated in first lactation. The progeny group sizes used were 50, 100, and 150 daughters. The resulting accuracies are shown in Table 6.

For all progeny group sizes, the accuracy was naturally highest when both measures, LSCS and MAST, were combined. If only one trait was considered and the daughter group size was small (up to about 50 daughters), selection based on LSCS was more efficient than selection based on MAST. However, for larger daughter groups, selection based on MAST was more efficient. That selection based on both traits was most efficient in reducing mastitis was expected and confirmed the results by Philipsson et al. (1995).

CONCLUSIONS

The unfavorable genetic correlation between udder health and production emphasizes the need to select for improved mastitis resistance, to prevent an increase in mastitis frequency as a consequence of selection for yield only. Heritability estimates of MAST were low (0.01 to 0.03). The higher heritability of LSCS (0.10 to 0.14), and its high genetic correlation with MAST (0.66 to 0.77), makes it a suitable indirect trait when selecting against mastitis, in this population. When only one trait was considered and the daughter group size was small (<50), LSCS was more efficient in improving mastitis resistance than selection directly on MAST, but for larger daughter groups, direct selection was more efficient. However, irrespective of daughter group size, accuracy was highest when both traits were combined in an index.

Mastitis frequency and level of LSCS increased with increasing parity. Therefore it is important that selection programs seek to improve mastitis resistance in all parities. Waiting for information from later lactations before selecting young bulls would create a prolonged generation interval, which is not desirable, and because genetic correlations between parities were relatively high (>0.7) for both MAST and LSCS, resistance in later lactations will be improved even if only first-lactation records are used. However, even with these rather high correlations, inclusion of later-parity information in the genetic evaluation would be expected to enhance accuracy somewhat, through inclusion in pedigree information. Given the estimated correlations, a multi-trait model with first and later lactations as separate traits can be suggested for both MAST and LSCS.

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