GENETICS AND BREEDING

Genetic and Environmental Relationships Among Somatic Cell Count, Bacterial Infection, and Clinical Mastitis

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ABSTRACT

Incidence of bacterial infection in 9784 lactations of 7763 cows in 31 herds, SCC in 32,448 lactations of 19,764 cows from 54 herds, and incidence of first parity mastitis recorded in the first lactations of 148,143 cows in 828 herds were analyzed. Bacterial infection was analyzed dichotomously by both threshold and linear models. The effects of parity, season, stage of lactation, and parity by stage of lactation interaction on SCC were estimated. Heritability of mean lactation log SCC—corrected for the effects of parity, season, and stage of lactation—varied from .13 to .27 for all parities in different data sets. Heritability of bacterial infection was .04 for the threshold model and .02 for the linear models. Heritability of field-recorded mastitis was .01. The genetic correlation between bacterial infection and SCC was near unity, but the genetic correlation between SCC and mastitis was .3. Selection for lowered SCC should reduce incidence of bacterial infection by 2% per unit of selection intensity.

(**Key words**: somatic cell count, mastitis, genetic correlations, threshold model)

Abbreviation key: **BAC** = incidence of bacterial infection, **HYS** = herd-year-season, **LSCC** = log of SCC, **MSCC** = mean corrected log SCC per lactation.

INTRODUCTION

Mastitis has a major economic effect on commercial milk production, and reduction of mastitis incidence could have a major effect on herd profitability (4, 10, 23). The additive genetic standard deviations for the cost of clinical mastitis in first and later parities were estimated as $13.49 and $56.20 compared with $82 for mature equivalent milk production (10). Although major efforts are directed to controlling udder infection by improved herd management techniques, only minimal effort has been directed toward breeding for reduced susceptibility. The main reason for the lack of emphasis on udder health traits in selection is that these traits in general display very low heritability (4, 6, 7, 22, 23). However, whether the low heritability estimates obtained are due to a lack of genetic variance or due to inaccurate recording of field data for this trait is not clear. Furthermore, mastitis is recorded categorically, and heritability estimates of categorical traits on a continuous scale are generally low (11, 17, 25, 26).

Numerous studies have indicated a positive relationship between milk SCC and udder disease (4, 5, 6, 7, 15, 23). Heritability for log-transformed SCC (**LSCC**) is generally higher than direct measures of udder infection (1, 7, 13, 14, 16, 19, 20, 21, 22). Estimates range

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from 0 to .38; the mean is close to .1. In addition, the same equipment used to assay milk components can also be used to estimate SCC. Thus, a number of studies have suggested that it may be possible to select indirectly for reduced incidence of mastitis by selection for lowered SCC (4, 6, 7, 10, 14, 22, 23). Because SCC is affected by many factors other than bacterial udder infection, elevated SCC alone is not a conclusive indication of udder infection (15). Genetic correlations between SCC and clinical mastitis are higher than the phenotypic correlations but do not approach unity (4, 7, 10, 23, 28). Whether these moderately high correlations accurately reflect the true genetic relationships or are due to inaccurate recording or inappropriate analysis procedures is not clear.

Incidence of bacterial subclinical infection (**BAC**) may be a more accurate measure of udder health than clinical mastitis for several reasons. In addition to incidences of clinical mastitis that are not detected by the herd manager, there may be additional incidences of subclinical infection, which may also result in elevated SCC. Only a few studies (4, 28) attempted to estimate genetic parameters among SCC, clinical mastitis, and udder infection, and those studies were based on small samples and suboptimal statistical procedures.

Most previous studies did not correct SCC scores for systematic environmental effects in analysis, even though some factors have major effects (27). For continuous traits, multitrait REML is the method of choice to estimate genetic and environmental parameters. Most previous studies have used other methods. An exception is Schutz et al. (19), who corrected for age and month of calving and estimated variance components using both Henderson's method 3 and REML. Previous studies that analyzed BAC used methods inappropriate for discrete variables (4, 28). Recently, methods based on the threshold model were developed that are more appropriate for analysis of discrete variables (9, 12). Those methods have already been applied to analyses of several discrete traits of interest, including dystocia, calf mortality, and twinning rate (11, 17, 25, 26). The goals of the present study were to develop lactation measures of SCC for Israeli Holsteins; to determine the genetic and environmental relationships among SCC, clinical mastitis, and udder infection, as recorded by veterinary laboratories using multitrait REML; and to estimate genetic and environmental parameters for BAC using both linear and threshold models.

MATERIALS AND METHODS

Three data sets were analyzed. Set 1 consisted of 9784 lactations of 7763 cows in 31 herds that were assayed for udder bacterial infection between January 1988 and June 1990. Four regional laboratories participated in the trial under the supervision of the Israel Mastitis Reference Center. Bacterial assays were performed on a composite milk sample from all four quarters. Sampling and storing of composite samples and the biological procedures for diagnosis of udder pathogens conformed to the recommendations of the Research Committee of the US National Mastitis Council (3). The major bacterial species recorded were *Staphylococcus aureus,* coagulase-negative staphylococci (micrococci), *Streptococcus* sp. other than *Streptococcus agalactiae (Streptococcus uberis* and *Streptococcus dysgalactiae*), and coliform organisms (*Escherichia coli, Aerobacter aerogenes, Klebsiella* sp., and *Serratia* sp). Of the 9784 lactations that were assayed, 4355 (45%) were assayed at least twice, and 1677 (17%) were assayed at least three times. There were 19,125 bacterial assays taken in 9784 lactations. Fifty-three lactations without SCC records were deleted, leaving 9731 valid lactation records. The SCC was recorded on 57,351 monthly tests of these lactations.

Data set 2 consisted of 32,448 lactations of 19,764 cows in 54 herds with at least one valid monthly SCC record, recorded between January 1988 and June 1990. There were 133,313 valid SCC records in this data set. This data set included the SCC data from data set 1. The additional herds included in this data set were under lesser scrutiny than those in data set 1. Thus, the mean number of SCC records per lactation was lower. Annualized milk and fat production, adjusted for parity, calving age, month of calving, and days open were computed as described by Ezra et al. (8). Incomplete lactations were extended for production traits as described by Bar-Anan et al. (2). Means for production traits were 9465 kg of milk, 299 kg of fat, and 3.18% fat.

TABLE 1. Basic description of the data sets.

Data set	Mastitis variable	Records	Lactations	Cows	Herds	Sires
		(no.)				
1	Bacterial assays	19,125	9731	7763	31	312
	SCC	57,351	9731	7763	31	312
2	SCC	133,313	32,448	19,764	54	215[1]
3	Clinical mastitis	148,143	148,143	148,143	828	292

[1]Includes only sires of daughters with valid SCC lactation records.

Data set 3 consisted of 148,143 first lactation records of cows that freshened between January 1985 and June 1990 from 828 herds that were field-recorded for clinical mastitis until May 1991. Of these, 5502 had valid first parity records for the lactation measure of SCC computed from data set 2. Four categories were determined by the herd manager: 0, no mastitis; 10, one incidence of mastitis; 15, two incidences of mastitis; and 20, more than two incidences of mastitis. Basic description of the data sets is in Table 1.

As assayed in data set 1, BAC was analyzed by the following model:

$$BAC_{ijklmn} = G_i + S_{ij} + P_k + MIM_l + HYS_m + e_{ijklmn}, \quad [1]$$

where BAC_{ijklmn} is the result of the first bacterial assay of parity k of a cow in herd-year-season (**HYS**) m, taken at month in milk l, daughter of sire j from group i of sires; G_i is the effect of group i of sires; S_{ij} is the effect of sire j of group i; P_k is the effect of parity k; MIM_l is the effect of month in milk l, HYS_m is the effect of HYS m, and e_{ijklmn} is the random residual associated with each record. Sires were grouped biennially by year of birth in this analysis and in the model of Equation [4]. In this model, sire, HYS, and residual effects were considered to be random, and all other effects were assumed to be fixed. Because of software limitations, sires were considered to be unrelated in this analysis. The BAC was scored dichotomously either as 0, when no pathogen was detected, or as 1, when a pathogen was detected. Only daughters of sires with at least 10 valid daughter records were included in the analysis. Two seasons, beginning in April and October, were defined by freshening date for each herd-year. The HYS effect was considered to be random for this analysis because of the dichotomous nature of the variable (25, 26). This model was analyzed by both linear and threshold models by the methods of Misztal et al. (12). Variance components were estimated by REML for the linear model and the counterpart of REML for the threshold model analysis (12). Because the scale of the effects is arbitrary for the threshold model, solutions and variance components were scaled so that its residual variance component was equal to the residual variance component of the linear model analysis, following the procedure of Weller and Gianola (25) and Weller et al. (26).

The SCC lactation curves were computed from data set 2 as the mean $\log_{10}$ SCC cells per microliter (LSCC) for all cows within a parity-stage of lactation class. Stage of lactation classes were defined as 15-d intervals starting with parturition. This allowed for sufficient data in each class to obtain relatively smooth curves. A lactation measure of SCC was computed as the lactation mean of LSCC (**MSCC**) and corrected for the effects of parity, calendar month of test, and DIM at test. Various models with different combinations of interactions were tested. After elimination of nonsignificant effects, the following model was most appropriate to compute the effects of these factors on LSCC:

$$LSCC_{ijkl} = P_i + H_j + M_k + b_1 SDIM + b_2 DIM + b_3(DIM)^2 + b_{4i}P_i \times SDIM + b_{5i}P_i \times DIM + e_{ijkl}, \quad [2]$$

where $LSCC_{ijkl}$ is the LSCC for test l, recorded in calendar month k, of a cow from

herd j in parity i; P_i is the effect of parity i; H_j is the effect of herd j; M_k is the effect of calendar month k of test; SDIM is the square root of DIM at test; DIM is DIM at test; $P_i \times$ SDIM and $P_i \times$ DIM are the interactions of parity with SDIM and DIM; b_1, b_2, b_3, b_{4i}, and b_{5i} are regression coefficients; and e_{ijkl} is the random residual associated with each record. Parities greater than 3 were combined in the analysis. This analysis model differs from the model of Wiggans and Shook (27) chiefly by the inclusion of a parity effect and parity by DIM interactions instead of separate analysis by parity. Corrected LSCC (CLSCC) was then computed as follows:

$$\begin{aligned} CLSCC_{ijkl} = {} & LSCC_{ijkl} - P_i - M_k \\ & - b_1SDIM - b_2DIM \\ & - b_3(DIM)^2 - b_{4i}P_i \times SDIM \\ & - b_{5i}P_i \times DIM. \end{aligned} \quad [3]$$

Then, for all lactations with more than 3 valid SCC records MSCC was computed as the mean of corrected LSCC.

Variance and covariance components for MSCC, milk, fat, and fat percentage were computed from data set 2 by multitrait REML using the method of VanRaden and Jung (24) and the following linear model:

$$T_{ijklm} = G_{ij} + S_{ijk} + HYS_{il} + e_{ijklm} \quad [4]$$

where T_{ijklm} is the record for trait i, recorded for lactation m of a cow in HYS l, daughter of sire k, from group j of sires; G_{ij} is the effect of group j of sires for trait i; S_{ijk} is the effect of sire k of group j on trait i; HYS_{il} is the effect of HYS l on trait i; and e_{ijklm} is the random residual for trait i associated with record m. Group and HYS effects were fixed, whereas sire and residuals were random. Although HYS effects were considered to be random in the model of Equation [1], the multitrait analysis required that all traits be analyzed by the same model. Preliminary results from the analysis based on Equation [1] indicated that the HYS effect was >.2 of the residual variance. Because the number of records per HYS was relatively large, nearly identical results should be obtained whether HYS is assumed to be random or fixed. Variance components were estimated using records from all parities and using only first parity records.

Equation [4] was also used in data set 1 to estimate variance and covariance components of the four traits just considered and for BAC corrected for parity and month in milk using the effects derived from the linear model analysis of Equation [1]. In addition, the model of Equation [4] was used to compute variance components for first parity lactations with valid records for MSCC and clinical mastitis and variance components for all valid first parity records for clinical mastitis in data set 3. Thus, in addition to the single-trait analyses of BAC by Equation [1], five different variance component analyses were computed using Equation [4].

Records of daughters of sires with less than 10 valid records per sire were deleted in the

TABLE 2. Number of records, herd-year-seasons (HYS), sires, and sire relatives in the variance component analyses.

Model	Traits	Parities	Records	HYS	Sires[1]	Relatives[2]
				(no.)		
1	BAC[3]	All	7610	43	90	. . .
4	MSCC,[4] milk, fat, fat %	All	16,221	191	313	121
		1	5268	187	122	91
	MSCC, BAC, milk fat, fat %	All	4998	111	120	82
	MSCC, mastitis	1	5502	72	123	80
	Mastitis	1	148,143	2841	292	116

[1]Only sires with at least 10 daughters were included in the single-trait analyses for BAC and mastitis. Sires with at least 5 daughters were included in the multitrait analyses.

[2]Includes dams of sires and sires of sires. In the single-trait analysis of BAC, sires were assumed to be unrelated, so no additional relatives were included.

[3]Incidence of bacterial infection.

[4]Mean corrected log SCC per lactation.

TABLE 3. Incidence of the major pathogens in the first assay of each lactation in data set 1.

Pathogen	Infected samples	
	Frequency	Percentage
Staphylococcus aureus	1369	14.1
Coagulase-negative staphylococci	349	3.6
Coliforms	165	1.7
Streptococcus sp. other than *Streptococcus agalactiae*	145	1.5
Other pathogens	187	1.9
Uninfected	7516	77.2
Total	9731	100.0

TABLE 4. Mean frequency of incidence of bacterial infection (BAC) and solutions for fixed effects by threshold (TM) and linear models (LM) with Equation [1].

Effect	Level	Mean BAC	Solution	
			TM	LM
Parity	1	.166	0	0
	2	.216	.100	.066
	3	.266	.186	.125
	≥4	.315	.234	.164
Months in milk	1	.161	0	0
	2	.214	.061	.039
	3	.226	.069	.045
	4	.218	.076	.049
	5	.218	.060	.038
	6	.254	.108	.074
	7	.320	.173	.120
	8	.283	.128	.082
	9	.284	.110	.073
	10	.249	.047	.027
	11	.240	.059	.034
	12	.249	.111	.074
Group[1]	1	.326	0	0
	2	.237	−.015	−.014
	3	.160	−.017	−.012
	4	.164	−.040	−.032

[1]Sires were grouped biennially by year of birth.

analysis of mastitis from data set 3. In the other analyses, which included less data, records of daughters of sires with fewer than 5 valid records per sire were deleted. Relationships between sires and between sires and dams of sires were included in the model. Only lactations with valid records for all traits were included in the analysis. The number of records, HYS, sires, and relatives of sires for the six variance component analyses are in Table 2.

Heritabilities were estimated from the models of Equations [1] and [4] as four times the sire component of variance divided by the sum of the sire and residual variance components. Standard errors of the heritability estimates were computed by the method of VanRaden and Jung (24). The HYS component of variance was not included in order to facilitate comparison of the three heritability estimates for BAC: linear and threshold model analyses by Equation [1] and the linear multitrait analysis by Equation [4]. Genetic correlations were computed from the variance and covariance component estimates as follows:

$$r_{gxy} = \frac{\sigma_{sxy}}{(\sigma^2_{sx})(\sigma^2_{sy})}, \quad [5]$$

where r_{gxy} is the genetic correlation between traits x and y, σ_{sxy} is the sire component of covariance between these traits, and σ^2_{sx} and σ^2_{sy} are the sire variance components for traits x and y. Environmental correlations were computed similarly with the residual variance and covariance components instead of the sire variance components.

RESULTS AND DISCUSSION

Incidence of the major pathogens in the first assay of each lactation in data set 1 is given in Table 3. Of 9731 lactation records, 22.8% were infected in the first assay. The most common pathogen was *Staph. aureus,* followed by coagulase-negative staphylococci, coliforms, and *Streptococcus* sp. other than *Strep. agalactiae.* Infection rates for any other pathogens were less than 1%. Relative frequencies of pathogens differed from the results of Coffey et al. (4), who found *Staphylococcus epidermidis* and *Corynebacterium bovis* to be the most prevalent pathogens. *Staphylococcus aureus* was the main pathogen in 62% of the infected samples. In the following analyses, only two infection states were considered: infected with pathogen (given a value of 1) or uninfected (given a value of 0).

Means of BAC by fixed effect levels and solutions for fixed effects for the analysis of BAC by the threshold and linear models using Equation [1] are in Table 4. The first level of

TABLE 5. Variance components and heritability estimates of incidence of bacterial infection (BAC) by threshold and linear models.

Model	Variance component			h^2
	HYS[1]	Sire	Residual[2]	
Threshold	.0493	.00167	.1479	.045
Linear	.0316	.00089	.1479	.024

[1]Herd-year-season.

[2]The residual variance component of the threshold was arbitrarily set equal to the corresponding linear model variance component.

each fixed effect was set to 0 in all analyses. The BAC increased with parity and decreased by group number, but these two effects were confounded. Sires were grouped by birth year, and the earlier groups had more daughters in later parities. The BAC increased until 7 mo in milk and then decreased. Variance components and heritability estimates by both models are in Table 5. There were only 2801 first parity records. This sample was considered to be too small for a separate first parity analysis. As in previous studies (17. 25, 26), variance components and heritability were nearly doubled by the threshold model, but heritability was .045. The correlation between sire solutions derived by the linear and threshold models was .99, which was similar to results for other traits (17, 25, 26). Variance components for HYS

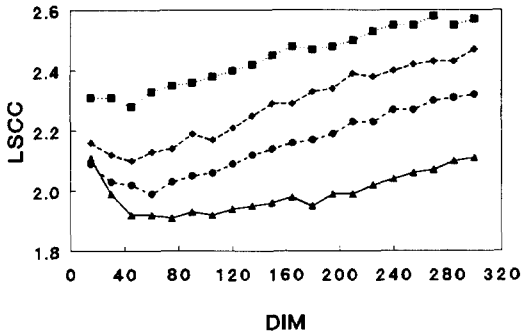

Figure 1. Lactation curves for the $\log_{10}$ of SCC (LSCC = log cells per microliter). Each point represents the mean LSCC for all cows within a parity-stage of lactation class. Stage of lactation classes were defined as 15-d intervals starting with parturition. ▲ = Parity 1; ● = parity 2; ♦ = parity 3; and ■ = parity ≥4.

TABLE 6. Mean log SCC (LSCC) and solutions for the discrete effects in the fixed model analysis of LSCC with Equation [2].

Effect	Level	Mean LSCC	Solution
Parity	1	2.00	0
	2	2.15	−.213
	3	2.28	−.204
	≥4	2.43	−.036
Month	Jan	2.18	0
	Feb	2.20	.015
	Mar	2.21	.034
	Apr	2.16	−.030
	May	2.18	−.008
	Jun	2.21	−.026
	Jul	2.20	−.022
	Aug	2.21	−.001
	Sep	2.20	−.019
	Oct	2.15	−.043
	Nov	2.15	−.050
	Dec	2.16	−.002

effects were quite large in both analyses, .2 to .3 of the residual. Thus, treating this effect as fixed would have very little effect on solutions.

Lactation curves for LSCC are in Figure 1. The LSCC lactation curves are "inverted" relative to production lactation curves. The first parity lactation curve was markedly different from the later parity curves. These results are similar to the LSCC lactation curves of Schutz et al. (18), except that those authors found high LSCC values at the beginning of the lactation for all parities. Solutions for the discrete effects included in the analysis of LSCC by the model of Equation [2] are in Table 6, and the regression coefficients for the continuous effects are in Table 7. All effects included in the model were significant at $P < .001$, but the coefficient of determination was only .19. The parity effects were computed relative to 0 DIM. Thus, the first parity effect was highest, even though mean LSCC is lowest in first parity. Corresponding to the lactation curves in Figure 1, the effect of the square root of DIM was negative for all parities and of greatest absolute value for first parity; LSCC was highest in February and March and lowest in October and November.

Genetic and environmental correlations and heritabilities for MSCC, adjusted milk and fat production, and fat percentage are in Table 8 for the analyses of all parity records and of

TABLE 7. Partial regressions of log SCC on functions of DIM in the fixed model analysis of Equation [2].

Effect	Parity	Regression
$DIM^{.5}$	1	−.154
	2	−.103
	3	−.084
	≥4	−.074
DIM	1	.00937
	2	.00784
	3	.00718
	≥4	.00650
DIM^2	. . .	.00000667

first parity records from data set 2. Genetic and environmental correlations and heritability estimates for these four traits and for BAC over all parities from data set 1 are in Table 9. Standard errors of the heritability estimates are also given. Because only lactations with valid records for all traits were included in each analysis, the number of records for the five-trait analysis was only 4998, whereas the four-trait all parity analysis included 16,221 records (Table 2). These differences may explain to some degree the differences among the three heritability estimates derived for MSCC and production traits. Heritability was highest for MSCC in the four-trait all parity analysis (.27) and lowest in the five-trait analysis (.13). The former value is higher than most estimates in the literature (1, 7, 13, 14, 16, 19, 20, 21, 22); however, most previous estimates were computed on unadjusted SCC or LSCC scores. Monardes and Hayes (14) found lowest heritability for second parity. The heritability estimate for BAC from the multitrait analysis was slightly lower than the linear model single-trait analysis, but the difference was not significant. The genetic correlation between MSCC and BAC was nearly equal to unity, but the environmental correlation was only .3. The correlation between sire evaluations for MSCC and BAC was .87. Coffey et al. (4) found correlations of about .6 between sire evaluation for SCC and incidence of BAC but did not directly estimate the genetic correlation. The genetic correlation between MSCC and production traits was lowest in the four-trait all parity analysis and highest in the five-trait analysis. Similar to previous results, the genetic correlation between production and SCC was positive, i.e., economically unfavorable (7, 19, 20, 21).

These results indicate that, under Israeli field conditions, selection on MSCC will be more efficient in reduction of BAC than direct selection on BAC. The gain in selection by direct selection on BAC will be $ih^2\sigma_p$, where i is the selection intensity, h^2 is the heritability of BAC, and σ_p is the phenotypic standard

TABLE 8. Estimates of genetic correlations (above the diagonal), environmental correlations (below the diagonal), and heritabilities (on the diagonal) for mean corrected log SCC per lactation (MSCC), adjusted milk and fat production, and fat percentage from the all parity and first parity analyses.[1]

Parity[2]	Trait	MSCC	Milk	Fat	Fat %
All	MSCC	.273 (.059)	.069	.057	−.008
	Milk	−.153	.291 (.061)	.450	−.458
	Fat	−.139	.733	.353 (.066)	.587
	Fat %	.017	−.333	.384	.702 (.090)
1	MSCC	.194 (.097)	.387	.295	−.116
	Milk	−.061	.336 (.123)	.484	−.532
	Fat	−.034	.734	.338 (.123)	.483
	Fat %	.039	−.404	.312	.701 (.168)

[1]Standard errors are given in parentheses under the heritability estimates.

[2]There were 16,221 records in the all parities analysis and 5286 records in the first parity analysis.

TABLE 9. Estimates of genetic correlations (above the diagonal), environmental correlations (below the diagonal), and heritabilities (on the diagonal) for mean corrected log SCC per lactation (MSCC), incidence of bacterial infection (BAC), adjusted milk and fat production, and fat percentage from the all parity analyses.[1]

Trait	MSCC	BAC	Milk	Fat	Fat %
MSCC	.134 (.077)	.994	.266	.606	.283
BAC	.304	.016 (.035)	.220	.656	.359
Milk	−.117	−.032	.132 (.076)	.201	−.599
Fat	−.094	−.025	.733	.151 (.080)	.663
Fat %	.031	.008	−.333	.384	.440 (.128)

[1]4998 records were analyzed. Standard errors are given in parentheses under the heritability estimates.

deviation. (Although three values are presented for the heritabilities of BAC and MSCC, we will use the lowest values. Thus, these estimates of the expected responses to selection can be considered to be conservative.) From Table 9, h^2 = .016, and σ_p can be computed as .367. Thus, the response to direct selection on BAC will be i(.016)(.367) = .006i or .6% per unit of selection intensity. The correlated response (CR) of BAC to selection on MSCC can be computed as follows:

$$CR = ih_s r_g \sigma_b, \quad [6]$$

where h_s is the square root of the heritability of MSCC, r_g is the genetic correlation, and σ_b is the additive genetic standard deviation for BAC. The heritability of MSCC from the five-trait all parity analysis is .134. Thus, h_s = .366. The sire component of variance for BAC as in Table 9 for the linear model is .000536; thus, the additive genetic variance is .00214, and σ_b = .046. Thus, per unit of selection intensity, CR = (.366)(.994)(.046) = .017 or a decrease of 1.7% in the incidence of BAC. In addition to yielding a great in response to selection, it is easier and less expensive to score SCC than BAC.

Variance and covariance components were computed for 5502 first parity lactations with valid MSCC and clinical mastitis records from data set 2. In addition, variance components were estimated for clinical mastitis from the complete file for data set 3. Frequencies of the different mastitis scores in each data set are in Table 10 and were generally similar. Heritabilities were .213 and .009 for MSCC and clinical mastitis. The genetic correlation of MSCC and clinical mastitis was .299, and the environmental correlation was .110. Heritability of clinical mastitis from the complete data set 3 analysis was .010. Both the heritability estimates for mastitis and genetic correlation between mastitis and SCC were lower than most literature estimates (6, 7, 22, 23). The low genetic correlation and heritability for clinical mastitis may be due to inaccurate recording of field data. Because of the low heritability, direct selection on clinical mastitis will not be an efficient method to increase udder health.

TABLE 10. Frequency of mastitis scores in first parities from data sets 2 and 3.

Mastitis score[1]	Analysis 1[2] Frequency	%	Analysis 2[3] Frequency	%
0	5085	92.4	140,525	94.9
10	318	5.8	6046	4.1
15	68	1.2	987	.7
20	31	.6	585	.4
Total	5502	100.0	148,143	100.0

[1]0 = No incidence of mastitis, 10 = 1 incidence of mastitis, 15 = 2 incidences of mastitis, and 20 = more than 2 incidences of mastitis during the lactation.

[2]Included only lactations with valid records for both mastitis and mean corrected log SCC (data set 2).

[3]Included all valid first parity mastitis records (data set 3).

CONCLUSIONS

Heritability for MSCC—a lactation measure of LSCC, corrected for environmental factors—was similar to the heritability for production traits. Heritability of BAC was .02 by the linear model and .04 by the threshold model. The genetic correlation between MSCC and BAC was near unity, whereas the genetic correlation between MSCC and clinical mastitis as recorded in field data was only .3. Selection for lowered MSCC should reduce BAC by 2% per unit of selection intensity.

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