Phenotypic and genetic statistics of components of milk and two measures of somatic cell concentration

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Phenotypic and Genetic Statistics of Components of Milk and Two Measures of Somatic Cell Concentration

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ABSTRACT

Heritabilities, genotypic and phenotypic correlations for milk, fat, and protein yields, and two traits related to somatic cell concentration (cumulative lactation score and lactational somatic cell concentration) were estimated. A total of 18,416 first lactations of Holstein cows were analyzed by a new procedure for estimating variance components. Heritabilities were .21, .23, .19, .17, and .61 for milk, fat, and protein yields, cumulative lactation score, and lactational somatic cell concentration. Addition of protein yields to the current selection for two traits with nil economic value for protein would improve genetic gains for fat and milk yields in the northeastern United States. If cumulative lactation score and lactational somatic cell concentration were incorporated in current selection for two traits, restricted selection indexes should be used to avoid reduction in genetic gains for milk and fat yields.

INTRODUCTION

Increased milk and fat yields are the main goals of dairy cattle selection in the northeastern US, because current pricing makes both traits the most profitable objective of selection. However, the dairy industry has become interested in other traits such as protein production or mastitis resistance, which have potential economic value.

Milk, fat, and protein yields have moderate heritabilities. Butcher et al. (4) reported .28, .17, and .21 for milk, fat, and protein yields,

which are similar to those by Wilcox et al. (12) .23, .25, and .17. Phenotypic and genotypic associations among yields have been high. Butcher et al. (4) found phenotypic correlations of .87, .93, and .89 between milk and fat, milk and protein, and fat and protein and corresponding genotypic correlations of .66, .82, and .77. Similar results by Wilcox et al. (12) verify a general pattern where phenotypic associations are closer than genotypic associations, and correlations of milk with protein are higher than correlations of fat with protein and milk with fat (10).

Results related to mastitis resistance are inconsistent because of unclear definition of the disease. As a result, different kinds of measurements of mastitis such as clinical infections, bacterial cultures, or screening tests are used, and sampling strategy varies from taking a single observation per cow per lactation to combining several monthly tests into a score. Lush (8) classified mastitis resistance as binomial, infected, or noninfected; a cow was uninfected if she reached 9 yr of age without an infection. Legates and Grinnells (7) also used a binomial classification where a cow was considered susceptible if she had a somatic cell concentration (SCC) greater than 500,000 cells per milliliter in any of her quarters during the sampling period. Heritability of susceptibility to mastitis was .27 (7). Both reports stated that the binomial classification could lower heritabilities compared to a multicategorical classification. Alrawi et al. (1) estimated heritabilities .48±.07, .36±.08, .46±.15, and .23±.12 for first, second, third, and fourth lactations for a cumulative lactation score based on monthly California mastitis tests (CMT). These large heritabilities disagree with (13) with heritabilities from −.11 to .11, −.24 to .11, and −.01 to .24 for first, second, and later lactations of cows that had been tested once during lactation for clinical mastitis and presence of specific mastitis pathogens. Phenotypic as-

sociations among yields and traits related to mastitis resistance have been low. Legates and Grinnells (7) found correlations of .02 and .01 between milk and mastitis and fat and mastitis. Wilton et al. (13) estimated phenotypic correlations between milk yield and several kinds of udder infection were between .0 and −.10 but pointed out that these estimates could be affected in opposite ways by more infections reducing yield and higher infection rate in high-producing cows. They also reported the genetic correlation between milk yield and infections of first lactation cows was .30. Some reports (5, 11) suggested protein yield and SCC are related not only through milk yield but also by a specific relationship between both traits. Weaver and Kroger (11) found an increase of total protein and whey protein in samples of milk with high SCC and attributed this to blood proteins in the milk of infected udders. Haenlein et al. (5) reported total protein content was constant when SCC changed because reduction of certain proteins was compensated for by increase in other proteins.

The purpose of this research was to study genotypic and phenotypic associations between milk, fat, and protein yields and traits related to mastitis resistance.

MATERIALS AND METHODS

Monthly information for milk yield, fat, and protein percentages, and SCC corresponding to first lactations of 2-yr-old Holstein cows initiated between July, 1977, and June, 1980, were obtained from the Dairy Records Processing Laboratory (DRPL) at Cornell University. Data were limited to first lactation daughters of artificial insemination (AI) sires. All lactations had milk yield and fat percent measured, but many records for protein and

SCC information were not available. As a consequence, data were divided into four subsets (Table 1). Data sets in Table 1 were used selectively where they would provide the most information for a parameter.

Milk, fat, and protein 305-day lactation yields were estimated from monthly information by Dairy Herd Improvement (DHI) factors that estimate lactation yields from monthly data. Two traits based on monthly SCC were used — cumulative lactation score (CLS) and lactational somatic cell concentration (LSCC). Alrawi et al. (1) defined CLS by number of monthly CMT's showing infection and their position along the lactation curve. In our study, CLS was defined as in (1) except monthly SCC was used instead of CMT and a cow was considered infected when the SCC was over 400,000 cells/ml (R. P. Natzke, personal communication, 1980). Table 2 shows the method of assignment of CLS scores with the condition that a monthly test with an SCC greater than 400,000 cells/ml was an indicator of infection. High scores in early lactation were considered more detrimental than similar scores in later lactations. The highest assigned score of 21 was an indicator of resistance to infection, whereas low scores near zero indicated consistent SCC measurements over 400,000/ml per sample and susceptibility to infection. To compute LSCC, a total count of somatic cells per milliliter per lactation was obtained by combining the product of SCC monthly tests and milk weight by DHI factors that estimate lactation cell yields from monthly data and dividing by the corresponding estimate of milk yield to obtain an estimate of LSCC somatic cells per milliliter for the lactation.

The object of the statistical analysis to obtain genotypic and phenotypic correlations and heritabilities was achieved by paternal half-sister analysis such that:

TABLE 1. Summary of data.

Available records	Number of		
	Records	Sires	Herds
Milk and fat	18,416	872	535
Milk, fat, and protein	8,747	661	341
Milk, fat, and somatic cell concentration	7,226	561	348
Milk, fat, somatic cell concentration,			
and protein	1,562	271	120

$$\hat{h}^2 = 4\hat{v}_s/(\hat{v}_s + \hat{v}_e)$$

where

$\hat{h}^2$ = heritability in the narrow sense,
$\hat{v}_s$ = sire variance,
$\hat{v}_e$ = residual variance, and

the genetic or phenotypic correlation between traits i and j is

$$\hat{r}_{ij} = \hat{Cov}_{ij}/(\hat{v}_i \cdot \hat{v}_j)^{.5}$$

where

$\hat{Cov}_{ij}$ = genetic or phenotypic covariance between traits i and j, and

$$\hat{Cov}_{ij} = (\hat{v}_{i+j} - \hat{v}_i - \hat{v}_j)/2$$

and $\hat{v}_i$, $\hat{v}_j$, and $\hat{v}_{i+j}$ = genotypic (or phenotypic) variances of traits i and j and the sum of trait i and j.

The model to estimate v_e and v_s was:

$$y = Hh + Aa + Ss + e$$

TABLE 2. Coding for cumulative lactation score.[1]

Early lactation Tests 1, 2, 3	Mid-lactation Tests 4, 5, 6	Early lactation Tests 7, 8, 9	Assigned score	Early lactation Tests 1, 2, 3	Mid-lactation Tests 4, 5, 6	Early lactation Tests 7, 8, 9	Assigned score
			21	2		3	8
		1	20		3	2	8
	1		19	1	2	2	7
1			18	2	1	2	7
		2	17	1	3	1	7
	1	1	17	3		2	7
1		1	16	2	2	1	7
1	1		16	3	1	1	6
2			15	2	3		6
		3	15	3	2		6
	1	2	14	1	2	3	6
1		2	14		3	3	5
1	1	1	14	2	1	3	5
	2	1	13	3		3	4
1	2		13	1	3	2	4
	3		13	2	2	2	4
2		1	13	3	1	2	4
2	1		12	2	3	1	3
3			12	3	2	1	3
	1	3	12	3	3		3
1		3	11	1	3	3	3
	2	2	11	2	2	3	3
1	1	2	11	3	1	3	2
2		2	10	2	3	2	2
	3	1	10	3	2	2	2
1	2	1	10	3	3	1	2
1	3		10	2	3	3	1
2	1	1	10	3	2	3	1
3		1	9	3	3	3	0
2	2		9				
3	1		9				
	2	3	9				
1	1	3	8				

[1] Cumulative lactation score is assigned according to the number of somatic cell concentration tests in each one-third of the lactation that exceeds 400,000 cells. (Developed from Aĺrawi et al. (1).)

where:

> **y** = the vector of observed random variables,
>
> **H**, **A**, and **S** = known design matrices corresponding to herd-year-season, age at calving, and sire,
>
> **h** and **a** = vectors of unknown fixed effects for herd-year-season and age at calving,
>
> **s** = a vector of unknown random sire effects, and
>
> **e** = a vector of random residuals.

It was assumed that

$$E \begin{bmatrix} \mathbf{y} \\ \mathbf{s} \\ \mathbf{e} \end{bmatrix} = \begin{bmatrix} \mathbf{Hh} + \mathbf{Aa} \\ 0 \\ 0 \end{bmatrix}$$

and

$$V \begin{bmatrix} \mathbf{y} \\ \mathbf{s} \\ \mathbf{e} \end{bmatrix} = \begin{bmatrix} \mathbf{I}_N \sigma_e^2 + \mathbf{SS}'\sigma_s^2 & \mathbf{S}\sigma_s^2 & \mathbf{I}_N \sigma_e^2 \\ & \mathbf{I}_t \sigma_s^2 & 0 \\ \text{Symmetric} & & \mathbf{I}_N \sigma_e^2 \end{bmatrix},$$

where

> E(**y**) = the expected value of **y**,
>
> V(**y**) = the variance-covariance matrix of **y**,
>
> N = total number of observations, and
>
> t = number of sires.

The $\hat{v}_e$ was estimated as the pooled mean square within herd-year-season-age-sire subclasses, whereas $\hat{v}_s$ was estimated by a new method for estimation of variance components by Henderson (6). The method involves equating sums of squares of approximate best linear unbiased prediction (BLUP) solutions to their expectations, such that

$$\hat{v}_s = [\text{tr}(\mathbf{r}'\mathbf{D}^{-2}\mathbf{r}) - \hat{v}_e \, \text{tr}(\mathbf{CD}^{-2})] \, / \text{tr}(\mathbf{CD}^{-2}\mathbf{C})$$

where

> **r** = the vector of right-hand-sides corresponding to sires after absorption of all fixed effects,
>
> **C** = the coefficient matrix after absorption of all fixed effects, and
>
> **D** = a diagonal matrix with diagonal elements the sum of the diagonal elements of **C** plus the ratio $\hat{v}_e/\hat{v}_s$, estimated from reports.

RESULTS AND DISCUSSION

Means, standard deviations, heritabilities, genotypic correlations, and within herd-year-season-age phenotypic correlations are in Table 3. Heritability of .17 for CLS of first lactations is lower than the .48 obtained by Alrawi et al. (1), although they used CMT in computing the score whereas SCC from a Fossomatic unit was used in our study. The genotypic correlation of −.11 between CLS and milk yield suggests a moderate negative association between milk production and CLS score. This compares with the estimate in Alrawi et al. (1) of −.31 and agrees with the estimate of Wilton et al. (13). It could be concluded that genes for higher milk production are associated with genes for susceptibility to infection. However, phenotypic correlation between milk yield and CLS in Table 3 is close to zero, possibly because there are more infections in high producers and less production in infected cows, thus affecting the phenotypic correlation between milk yield and CLS in opposite ways.

Phenotypic and genotypic correlations for protein and fat yields with CLS are close to zero (Table 3). Low phenotypic and genotypic correlations between fat yield and resistance to infection were reported (7, 13). It is difficult to speculate on causes of opposite signs of correlations between fat and CLS and protein and CLS in Table 3.

The CLS and LSCC have correlations close to zero with milk and fat yields in Table 3. However, the genotypic association between LSCC and protein yield was unrealistic (−1.40) and can be attributed to sampling error. The phenotypic correlation of −.22 between protein yield and LSCC is also surprising because of reported positive and null associations between the traits (5, 11). Heritability of .61 for LSCC could indicate that selection for LSCC would be effective. However, high heritabilities in traits related to mastitis susceptibility are difficult to explain. Lush (8) reasoned that if mastitis has been highly heritable, mass selection would have reduced its incidence in dairy herds. This was not the case, although a positive association between mastitis susceptibility and milk production would explain prevalence of the disease. A response to the argument of Lush (8) by Legates and Grinnells (7) pointed out that mass selection

TABLE 3. Heritabilities (on the diagonal), phenotypic correlations (above the diagonal), genotypic correlations (below the diagonal), means, and standard deviations.

	Yields (kg)			Lactational somatic cell concentration ($\times 10^3$/ml)	Cumulative lactation score
	Milk	Fat	Protein		
Yields (kg)					
Milk	.21	.73	.90	−.02	.02
Fat	.43	.24	.80	−.06	.07
Protein	.79	.80	.19	−.22	−.05
Lactational somatic cell concentration	−.01	.08	−1.40	.61	−.76
Cumulative lactation score	−.11	−.02	.08	−.28	.17
Mean	6129	223	205	208	18.9
Standard deviation[1]	917	34	29	227	3.5

[1] Within herd-year-season-age standard deviations.

would have been ineffective because many susceptible cows were not detected until their third lactation, thus leaving susceptible progeny in the herd. Additionally, environmental effects increasing the incidence of inflammations, especially the widespread use of milking machines, could have obscured progress by mass selection.

The phenotypic association between CLS and LSCC of −.76 is not surprising as infected cows have high LSCC but low CLS, but the moderately negative genotypic correlation of −.28 between LSCC and CLS suggests the traits respond to different genetic mechanisms.

When selection indexes including milk, fat, and LSCC or CLS were built with nil economic values for LSCC and CLS, genetic gains for milk were almost equal to gains obtained through milk and fat selection. However, more infections would be expected with the index, suggesting the use of negative economic values for LSCC and positive economic values for CLS. This approach would lower genetic gains for milk and fat yields. A better alternative would be to use restricted selection indexes for including LSCC or CLS in an index (1) to limit the LSCC or CLS to current counts in selecting for production.

Heritabilities for yields in Table 3 are within the range typically reported in analyses of several lactations (4, 12) but appear low compared to estimates for first lactation (3, 9). One possible explanation is heritability tends to

decrease as the number of records per filled subclass approaches one (9). The average number of records per filled subclass was near one in all the data sets used in this study, although the exact distribution of the number of records was not investigated.

Selection indexes for milk, fat, and protein yields were built with phenotypic and genotypic variances in this study. Under the pricing scheme in the northeastern US, an index based on milk, fat, and protein yield with nil economic value for protein produced higher gross return per lactation (GRL) than the index based on milk and fat yields alone. However, when a pricing scheme included a differential of ±$.11 per protein point with a base protein percent of 3.2, the GRL was lower than that obtained by selection for milk and fat despite the higher gains in fat and protein yields for the index with protein priced. Results were similar for Anderson et al. (2) and are caused by a reduction in potential gains and price of the carrier, water. When an index including milk, fat, and protein yields with nil economic value for milk yield was computed, gains for fat and protein yields and GRL were highest and milk yield decreased.

CONCLUSIONS

High positive correlations between milk, fat, and protein yields were confirmed. Including protein yields with zero economic value in an

index improved genetic gains for milk and fat yields but not for protein yield. In future considerations of milk pricing, care must be taken in selecting a fair economic value for protein to avoid reductions of gross returns per lactation. Alternatives to the system of price differentials for protein penalize or ignore the carrier, water, in the milk price. These alternatives would generate resistance by dairymen.

The question of what trait could be used to select for mastitis resistance is unresolved. The LSCC and CLS showed a high negative phenotypic correlation but only a moderately negative genotypic correlation. Global measurements such as LSCC or CLS offer a compromise between accuracy and feasibility today, but with the expectation of cheaper testing techniques in the future, selection for resistance to specific bacteria insensitive to environmental control will be possible.

If selection for low LSCC or high CLS is wanted, restricted selection indexes should be used to avoid reductions in milk and fat yields.

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