GENETIC EVALUATION FOR SOMATIC CELL COUNT AND RELATIONSHIP WITH INBREEDING IN CANADIAN HOLSTEINS

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SUMMARY

An animal model evaluation of Canadian Holsteins for a lactation measurement $\log_2$ transformed somatic cell count (SCC) was completed based on data from Ontario and Québec. About 360,000 multiple lactation LSCS of over 200,000 cows with first lactation information in the data, based on two or more test-day records were processed. Weighting factors were computed from single test-day records and assigned to each observation according to the number of test-day records in LSCS.

Herd-year-season and age by parity fixed effects were included in a repeatability animal model for genetic evaluation. The additive relationship matrix included two generations of ancestors. Unknown animals were assigned to 23 phantom groups according to sex and year of birth. Solutions for age by parity showed a highly significant increasing trend, which was stepping up in subsequent parities. No genetic trend was detected for cows, while sire EBV showed a favorable but small decreasing trend from the mid eighties. Average inbreeding coefficients of daughters were plotted against their sire EBV for LSCS. The linear regression coefficient indicated a small but significant increase of SCC (about 1,000) per 1% of increase in inbreeding.

INTRODUCTION

SCC is accepted to be a useful indicator for mastitis, the highest ranking pathology affecting dairy production (Boettcher et al. 1992), and it is widely used as a valuable tool for management practices. Selection for milk yield has been proven to increase health problems and mastitis susceptibility, as supported by the positive (.2) genetic correlation between production and mastitis (Shook and Schutz, 1994). Coffey et al. (1986) reported a positive genetic relationship between measures of SCC in milk and rates of udder infection and suggested that selection for decreased SCC is a sensible step for reduction of mastitis incidence. In addition, heritability of SCC has been shown to be nearly double that of direct measures of mastitis (Schutz, 1994).

However, several questions still need to be answered on mastitis and SCC, as pointed out by Dekkers et al. (1993). High SCC is indeed the consequence and not the cause of inflammation of the mammary gland and the ability of the SCC to predict future mastitis incidence of a cow is reported to be very low (McDermott et al. 1982). When selecting for lower SCC, we assume that cows will be more resistant to mastitis, but more experimental evidence is needed to determine whether this relationship holds for the entire range of SCC. As an extreme condition, a genetically determined complete absence of neutrophilis (the major component of SCC) in extra vascular tissues can lead to high health risks (Kehrli and Shuster 1993).

Nevertheless, Shook and Schutz (1994) found that a selection index that includes SCS was nearly as effective in decreasing mastitis as an index that included clinical mastitis, along with milk production. They concluded that taking SCS into account, with the appropriate emphasis, in a selection index would not improve mastitis resistance but would slow down the current rate of increase in mastitis susceptibility, which is associated with selection for production.

Based on the above reasoning, national sire genetic evaluations by animal model of Canadian Holsteins has been planned. The purpose of this paper is establish procedures for genetic

evaluation for SCS, to present results of preliminary genetic evaluations for SCS based on Ontario and Québec data, and to explore the relationship between SCS and inbreeding.

MATERIALS AND METHODS

A widely accepted lactation measurement of SCC is the Lactation Somatic Cell Score (LSCS), a mean of $\log_2$ transformed test-days SCC, preadjusted for stage of lactation (Wiggans and Shook 1987).

Sets of additive factors to adjust test-day SCS for stage of lactation were first computed by lactation number from a fixed model that included cow stage of lactation, and calendar month. Based on similarities between lactations two to five, joint factors were subsequently computed for these lactations. Preadjustment factors were deviated from an unweighted average over classes, such that the adjustment would have a minor effect on the average SCS. Lactation measures for SCS (LSCS) were obtained as the average of preadjusted test-day SCS.

It has been suggested (Zhang et al., 1994) that in a routine genetic evaluation model, LSCS based on fewer test-days should receive less emphasis than LSCS based on a complete recorded lactation. In order to run a complete animal model procedure for routine genetic evaluation in Canada, weighting factors needed to be computed. This was accomplished using test-day SCS from 137,765 lactations on official recording, with 8 or more test-day records, preadjusted for stage of lactation. Two sets of squared correlations, were selected for first and later lactations and used as weighting factors in the animal model evaluation.

The weighted animal model, including 43,447 herd-year-season fixed effects and 27 age by parity fixed classes, was set up to evaluate the random additive genetic effect of 5677 sires of 205,236 recorded cows with 360,343 LSCS (37% from Ontario and 63% from Québec). Birth year of most cows was between 85 and 90 for Québec and between 83 and 89 for Ontario. Only cows with a first lactation record in the data were included in order to exclude selection bias. For the same reason LSCS based on as few as 2 test-days were included. The model also included a random permanent environmental effect. The additive relationship matrix included animals two generations back, and unknown ancestors were assigned to 23 phantom groups defined by sex and year of birth.Heritability and repeatability were assumed .11 and .27, as proposed by Zhang et al. (1994). The (co)variance matrix of error terms was diagonal with elements equal (weight)$^{-1}\sigma^2_e$. Equations were solved by Jacobi iteration.

Previously computed inbreeding coefficients (Miglior and Burnside 1994) for each cow in the data set were collected. Sire EBV for LSCS were then linearly regressed on the average inbreeding coefficient of their daughters.

RESULTS

Sample means and SD of LSCS by parity are reported in table 1. Weighting factors for LSCS based on number of test-days records are reported in table 2 and additive preadjustment factors for stage of lactation are in table 3.

Table 1. Sample statistics for LSCS.

Parity	N	Mean	SD
1	205,236	2.327	1.226
2	98,877	2.713	1.343
3	41,235	3.092	1.431
4	12,840	3.378	1.503
5	2,155	3.594	1.609
all	360,343	2.565	1.335

Table 2. Weighting factors for first and later lactations by number of test-days included in LSCS.

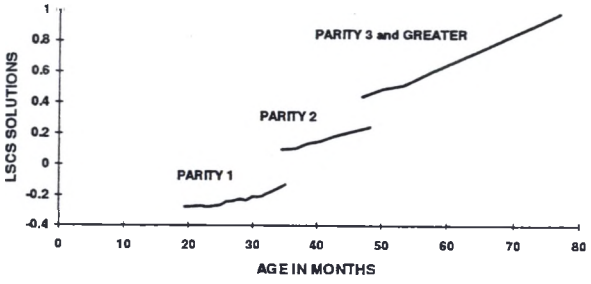
No. of test-days	Lactation 1	Lactation ≥ 2
2	0.59	0.66
3	0.74	0.78
4	0.84	0.87
5	0.91	0.93
6	0.96	0.97
7	0.99	0.99

Table 3. Additive adjustment factors of test-day SCS for stage of lactation.

Stage of lactation	Lactation 1	Lactation ≥ 2	Stage of lactation	Lactation 1	Lactation ≥ 2
1 - 10	-0.867	-0.333	121 - 150	+0.167	+0.114
11 - 20	-0.078	+0.441	151 - 180	+0.077	-0.099
21 - 30	+0.244	+0.706	181 - 210	+0.000	-0.302
31 - 60	+0.431	+0.762	211 - 240	-0.080	-0.494
61 - 90	+0.401	+0.586	241 - 270	-0.180	-0.720
91 -120	+0.278	+0.340	271 - 305	-0.384	-1.003

The weighting factor for LSCS based on 8 or more test-days was 1.0. The overall estimated mean LSCS was 2.659 corresponding to a mean SCC of 78,960. Age by parity solutions from the animal model showed the increasing trend reported in figure 1. As an example, estimated averages of SCC for a 2yr old calving heifer and a 5yr old cow were 65,300 and 120,080 SCC. The two fixed factors in the animal model accounted for 32% of the variation in LSCS.

Figure 1. Age and parity effect on LSCS.



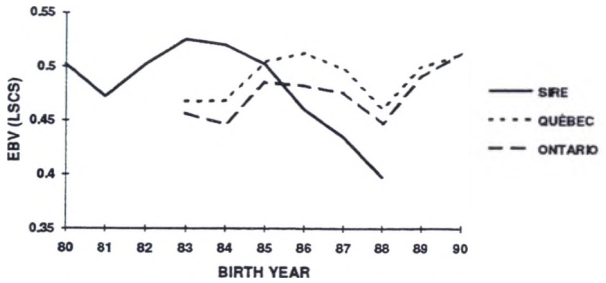
Sire EBV were approximately normally distributed. Their distribution by number of daughters is shown in table 4.

Table 4. Distribution of sire EBV and SD by number of daughters.

No. of daughters	No. of sires	Average EBV	σ_{EBV}
1	1,801	.497	.123
2	649	.496	.137
3 - 5	876	.503	.158
6 - 10	584	.497	.191
11 - 20	479	.494	.228
21 - 30	371	.511	.281
31 - 50	534	.469	.298
50 - 100	187	.479	.301
101 - 500	132	.483	.361
501 & >	64	.501	.397
36.5	*5,677*	*.495*	*.201*

As the number of progeny increased, the SD of EBV increases, as expected because of the higher accuracy of prediction. Genetic trend computed from sire EBV by birth year is shown in figure 2.

Figure 2. Genetic trend of LSCS.



Starting in 1983 a slow but favorable genetic trend can be detected with an estimated yearly decrease of 0.024 LSCS. Over the same period, cow EBV did not decrease, although patterns were similarin Quèbec and Ontario.

The weighted linear regression coefficient of EBV for LSCS of 4873 sires on the average inbreeding of their daughters was 0.019 ± 0.007 (P = .008). An increase of 1% in inbreeding is therefore responsible for an extra 1,000 SCC for SCC around the mean.

DISCUSSION

Figure 1, reporting age by parity effects on LSCS, shows a clearly increasing pattern associated with age of cows. First parity spans over the first 14 classes while second parity covers classes 15 to 21. A larger variation between vs. within parities is evident.

Zhang et al. (1994) found that no genetic trend was detectable on Ontario data looking at cow EBV for first lactation. In that study, only LSCS of lactations with more then 5 test-day records were processed. As a result, short records from cows culled for mastitis were excluded. Although this potential source of selection bias was removed here, no genetic trend was found in the present study in cows. A surprising favorable although small genetic trend for sires was detected. This is possibly due to a correlated response to selection for udder conformation.

The result reported for the effect of inbreeding on LSCS indicates a small but significant contribution to SCC variation. The regression coefficient was consistent with the value of 0.012 found by Miglior et al. (1994) for first lactations only and indicates that higher inbreeding is associated with higher SCC and can be related to higher susceptibility to mastitis and to other diseases.

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