Phenotypic and Genetic Relationships Between Somatic Cell Counts and Clinical Mastitis in French Dairy Holstein Cows

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Abstract

This paper summarizes three already published studies about the relationships between somatic cell counts (SCC) and clinical mastitis (CM) in French Holstein. These studies were based on CM data collected by the milk recording technicians at each test-day since September 1995 in Western France. The first two studies focused on the putative risk of CM associated with very low SCC. The first one analyzed the risk of the first CM occurrence according to the SCC level at first test-day by survival analysis. The second one analyzed the risk of CM in second lactation by logistic regression according to SCC criteria measured in first lactation. Results were consistent and showed that lower the SCC level, lower the risk of CM. Decreasing SCC level in the population should not increase CM risk and there is no intermediate optimum. In the third analysis, genetic parameters were estimated. The genetic correlation between SCC and CM was high (0.72) and selection for lower SCC level should increase resistance to both clinical and subclinical mastitis.

Keywords: clinical mastitis, dairy cow, somatic cell count, genetic parameters, survival analysis, logistic regression

1. Introduction

Direct selection against clinical mastitis (CM) is difficult, because in most countries CM events are not recorded, and because the corresponding heritability is very low, close to 0.02 (Emanuelson et al., 1988 ; Weller et al., 1992 ; Lund et al., 1994 ; Pöso and Mäntysaari, 1996). Conversely, somatic cell counts (SCC) are routinely recorded in most milk recording systems and are available on a large scale at a moderate cost. The heritability of SCC, close to 0.15 (Mrode and Swanson,1996), is much greater than for CM. Consequently, it is believed that selection for decreased SCC would reduce susceptibility to both clinical and subclinical mastitis (Colleau and Le Bihan-Duval, 1995). The efficiency of such an indirect selection against CM based on SCC is dependent, however, on the genetic correlation between both traits. Most estimates are positive but range from 0.3 to 0.7. More accurate estimates are needed to narrow this wide interval and predict the efficiency of this approach.

The linearity of the relationship between SCC and CM is also questioned. Some authors (Kehrli and Schuster, 1994 ; Coffey et al., 1986; Schukken et al., 1994) were concerned by the recommendation of continuously decreasing SCC by selection, and argued that such a trend could impair the cow's capacity for leukocyte recruitment and, therefore, her ability to respond to intra-mammary infection. The question is raised whether SCC should be decreased to the lowest possible value or should not be lower than a critical threshold. This paper summarizes two contributions to answer that question and a third study about estimates of genetic parameters.

2. Material

The data consisted of Holstein cows from Morbihan and Finistère regions in Western France, with first calving between September 1, 1995 and August 31, 1996. CM events, which are not routinely reported in the national data base, were declared by farmers and collected every month by Milk Recording technicians. Reliability and completeness of recording was assessed by a survey. Herds (36%) for which recording was considered to be incomplete were excluded from the study. The final data set included 25,833 cows, out of them 5,156 (20%) had at least one CM in first lactation.

3. Relationship between initial SCC and risk of CM (Rupp and Boichard, 2000)

The effect of SCC level at first test-day on the risk of subsequent CM was analyzed by survival analysis. To avoid the effect of a previous CM event on the first SCC, 1,940 cows with a CM recorded before 35 days after first calving were discarded. The relationship between early SCC and clinical mastitis was studied in a range of low to moderate cell counts and only cows with a first SCC lower than 400,000 cells/ml were considered. For these discarded cows (4,719), CM frequency was high (51.2 %) and 90% of these clinical events occurred before 100 days after first calving. The final data set consisted of 20,422 cows in 2,611 herds, with 13% of these cows having at least one CM.

The variable analyzed was the time interval to the first CM event occurring in first or, if necessary, in second lactation. If no CM occurred during the first lactation and if the cow did not calve a second time, her record was censored the day of her last test day. If a cow was still present in the herd and still unaffected at the end of the period under study (March 1st 1997), her record was censored at the end of the period.

In addition to a Weibull baseline hazard function, the proportional hazard model included a random time-dependent herd-year effect and the fixed effects of lactation stage (time-dependent), month of first calving, initial SCC (ISCC), and initial milk yield. ISCC and milk yield at first test day were pre-adjusted for days in milk. Six levels : (1) < 35,000 cells/ml, (2) 35,000-50,000, (3) 50,000-75,000, (4) 75,000-150,000, (5) 150,000-215,000, and (6) 215,000-400,000 cells/ml.

Estimates were obtained with the 'Survival Kit' (Ducrocq and Sölkner, 1994). The analysis was carried out on the complete data set and on different herd subgroups. Two subgroups were defined according to herd CM frequency (less or more than 20% affected lactations), which was calculated from data of all parities and all cows in each herd. Two other subgroups were also defined according to herd somatic cell score (SCS) mean (below or above 3.4), where $SCS=\log_2(SCC/100000)+3.$).

Table 1 and Figure 1 present the relationship between ISCC and risk of first CM. In the overall analysis, the relative hazard gradually increased with ISCC. Cows with the lowest ISCC, *i.e.*, less than 35,000 cells/ml, were at lowest risk. When compared with cows with the lowest ISCC, the relative hazard ratio reached 1.75 for cows with the highest ISCC, *i.e.*, between 215,000 and 400,000 c/ml. Estimates of the ISCC effect were similar for groups of herds with low or high SCS level. In contrast, estimates of ISCC effect revealed somewhat different patterns for herds with high CM frequency when compared with herds with fewer clinical cases. In the latter herds, no significant increase in risk was observed until up to 75,000 cells/ml. Relative hazard was significantly higher for ISCC from 75,000 to 215,000 cells/ml, and seemed to decrease for the highest class. In all cases, however, the lowest SCC level was never associated to an increase in risk of CM.

Table 1. Relative hazard ratio for Initial SCC

Initial SCC (1000 cells/ml)	All herds	subgroups of herds			
		CM-	CM+	SCS-	SCS+
< 35	1.00	1.00	1.00	1.00	1.00
35 to 50	1.17*	1.03	1.27*	1.13	1.14
50 to 75	1.28*	1.06	1.38*	1.26*	1.21
75 to 150	1.39*	1.31*	1.41*	1.48*	1.24*
150 to 215	1.86*	2.00*	1.73*	1.79*	1.75*
215 to 400	1.88*	1.51*	2.06*	1.80*	1.76*

- Risk significantly ($p<0.05$) different from 1.00.

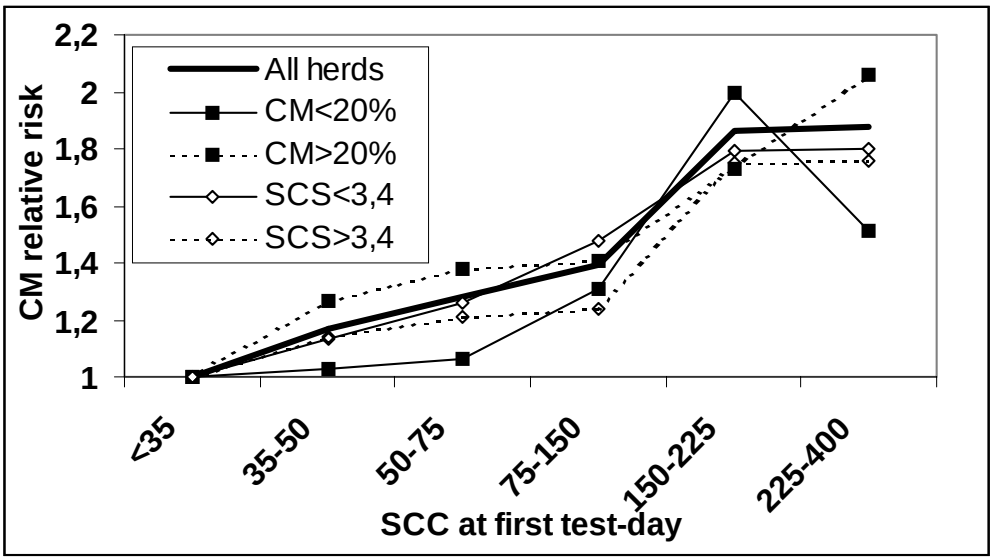

Figure 1. Evolution of the relative CM hazard ratio with Initial SCC.

4. Relationship between SCC in first lactation and risk of CM in second lactation (Rupp et al, 2000)

The goal of this study was to analyze the association between the SCC level in first lactation and the probability of CM in second lactation. Only data from the Finistère region, with a longer recording period, were used. The analyzed data set included 10,205 cows with two consecutive lactations. The outcome variable was defined by the presence (=1) or absence (=0) of at least one case of CM in second lactation and was analyzed by logistic regression, following Foulley and Gianola (1996). The model included the random effect of herd and the fixed effects of milk yield in first lactation, age at first calving, month of second calving, presence/absence of CM in first lactation, and one out of the 10 following SCC criteria defined from first lactation data : arithmetic mean of monthly test-day SCC or SCS; mean of the last two SCC or SCS; SCS at the first test-day adjusted for days in milk; proportion of monthly SCCs above 300 000 or 800 000 cells/ml, or below 20 000, 35 000, or 50 000 cells/ml.

The analyses were performed on the complete data set and on 4 different subgroups of herds, defined by combining herd lactation incidence risk of CM (< or ≥20% of lactations with at least one CM) with herd SCS mean level (< or ≥3).

The 10 criteria provided consistent results (Table 2). Lower (higher) mean cell level, higher (lower) proportion of low SCC values, lower (higher) proportion of high SCC values were all associated with a lower (higher) risk of CM in the second lactation. Odds ratios for high (versus low) cell level during the whole or at the end of the first lactation were around 2.0. Corresponding odds ratio for proportion of SCC lower than 20 000 cells/ml or higher than 800 000 cells/ml were smaller (around 1.5). This result shows that the latter criteria are less relevant for analyzing CM occurrence, probably because they are more sensitive to random variation and also because cow distribution across levels is strongly unbalanced. Odds associated with high SCC at the first test-day were lower (1.4) but still significant, although this measure is highly variable and quite distant from the second lactation.

Figure 2 show the results obtained in the four subgroups of herds for lactation mean SCS. Results were consistent between subgroups of herds and, regardless of the epidemiological status of the herd towards udder health, cows with the lowest cell level in the first lactation were at the lowest risk of CM in the second lactation.

Table 2. Odds ratios of CM in the 2nd lactation for different cell-level descriptors in the 1st lactation (* Odds ratio significantly different from 1 with a probability $p<0.05$)

Variable	Odds ratio	Variable	Odds ratio
		Percentage of SCC <20,000 cells/ml	
Lactation mean SCS			
≤ 1.4	1.00	>40	1.00
]1.4-2.1]	1.17	]20-40]	1.12
]2.1-2.8]	1.49*	]12-20]	1.28*
]2.8-3.5]	1.63*	]0-12]	1.23*
>3.5	2.05*	0	1.42*
		Percentage of SCC <35,000 cells/ml	
Lactation mean SCC (*1000 cells/ml)			
≤ 40	1.00	>75	1.00
]40-60]	1.23	]50-75]	1.10
]60-100]	1.52*	]25-50]	1.40*
]100-200]	1.82*	]0-25]	1.53*
>200	2.10*	0	1.74*
		Percentage of SCC <50,000 cells/ml	
Mean of the last 2 SCS			
≤ 1.4	1.00	>75	1.00
]1.4 - 2.1]	1.36*	]50-75]	1.27*
]2.1 - 2.8]	1.41*	]25-50]	1.46*
]2.8 - 3.5]	1.54*	]0-25]	1.55*
> 3.5	2.02*	0	1.81*
		Percentage SCC >300,000 cells/ml	
Mean of the last 2 SCC			
≤ 40	1.00	0	1.00
]40-60]	1.21*	]0-12]	1.28*
]60-100]	1.29*	]12-20]	1.37*
]100-200]	1.47*	]20-40]	1.46*
>200	1.96*	>40	1.82*
		Percentage SCC >800,000 cells/ml	
First SCS			
≤1.4	1	0	1.00
]1.4-2.1]	1.01	]0-12]	1.19
]2.1-2.8]	1.39*	]12-18]	1.15
]2.8-3.5]	1.25*	]18-30]	1.70*
>3.5	1.38*	>30	1.56*

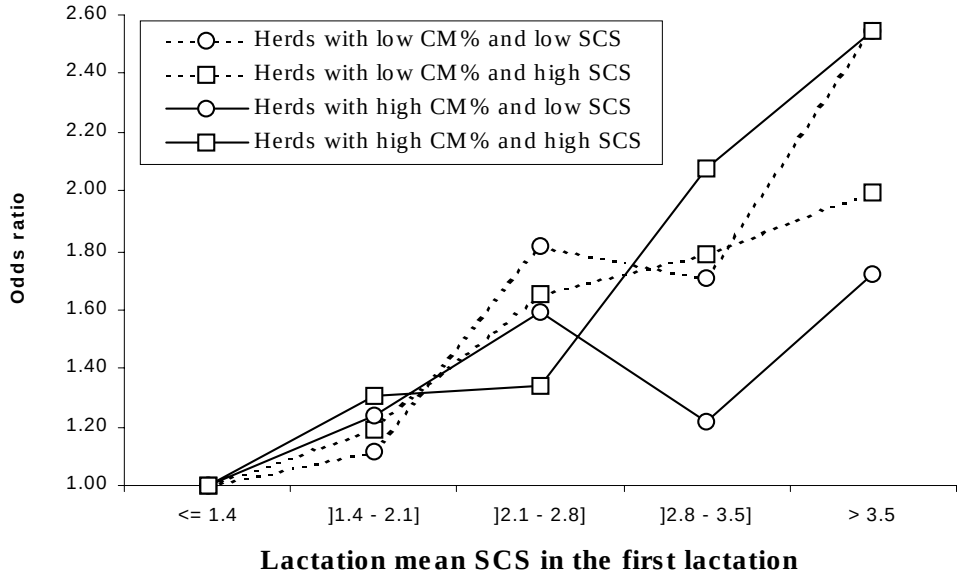

Figure 2. CM odds ratio in the 2nd lactation associated with lactation mean SCS in the 1st lactation, for subgroups of herds defined according to CM risk and lactation mean SCS.

These two studies showed no increase in susceptibility to CM for cows with the lowest SCC level. This result was observed in contrasted epidemiological situations regarding CM frequency and herd cell level. Actually, these results suggest that selection for decreased SCC may effectively reduce CM incidence and that the breeding goal should favor cows with the lowest observed SCC. This result is valid under the current situation and will need periodic confirmation on the long term, if the average SCC level substantially decreases in the population.

5. Estimation of genetic parameters (Rupp and Boichard, 1999)

Finally, the data set was used to estimate genetic parameters between CM (1=presence or 0=absence of at least one clinical case), mean lactation SCS, production, udder conformation (UC), and milking speed in first lactation by REML. The analysis was performed with the VCE package (Neumaier and Groeneveld, 1998). Models included fixed effects of herd, month of calving, age at calving and the random animal effect. For UC and milking ease, the model also included the fixed effects of the classifier and days in milk at scoring.

The heritability estimate for CM was 2,4% and was much lower than for SCS (0.17). The genetic correlation estimates, (Table 3) showed : 1) a strong association between SCC and CM (0.72), although this correlation was lower than 1 ; 2) a stronger opposition of production with CM (~0.4) than with SCS (<0.2); 3) similar associations between UC traits and LSCS or CM, with significant correlations with udder depth, udder balance and fore udder attachment, but not with teat length ; 4) no association between milking speed and CM, in spite of a strong and unfavorable association between SCS and milking speed.

The genetic correlation between CM and SCS is illustrated in Figure 3. This plot presents the raw CM frequency in first lactation of 50 progeny groups in the two regions where CM information was available, as a function of the official SCS evaluation of these sires (estimated on a national basis with a large number of daughters with up to 3 lactations analyzed, Boichard and Rupp, 1997).

Note that the sign of the EBVs is changed, i.e. positive values mean less SCC, which explains that the observed correlation is negative. CM frequency ranged from 7 to 32 % between progeny groups and showed that genetic variability for CM was large, in spite of a low heritability.

Table 3. Estimates of genetic correlations with LSCS and CM

Trait	Genetic correlation with LSCS(1)	Genetic correlation with CM(2)
Lactation SCS	-	0.72[1]
CM	-	-
Milk yield	0.15	0.45
Protein content	0.20	-0.24
Fat content	-0.02	-0.26
Protein yield	0.27	0.41
Fat yield	0.11	0.15
Udder depth	- 0.40	- 0.46
Udder cleft	- 0.10	- 0.03
Udder balance	- 0.29	- 0.32
Fore udder attachment	- 0.32	- 0.36
Rear udder height	0.13	- 0.03
Teat length	0.08	0.12
Teat placement	0.14	0.06
Front teat distance	0.16	0.20
Side teat distance	0.26	0.20
Milking ease	0.44	0.06

(1) SE between 0.030 and 0.070
(2) SE between 0.080 and 0.120

Conclusion

From these series of study, it appears that 1) no increase in CM susceptibility is observed for cows with very low SCC and, therefore, selection could be applied to decrease SCC as much as possible, without any intermediate optimum ; 2) although the environmental correlation between SCC and CM is rather low (0.21), the corresponding genetic correlation is high (0.7) and makes it possible to use SCC to select for both clinical and subclinical mastitis resistance ; 3) including production and SCC in the breeding objective leads usually to favorable responses on SCC level but to unfavorable responses on CM, because of the stronger opposition of production with CM (Colleau and Le Bihan, 1995) ; to obtain favorable responses on CM, SCC are not sufficient and CM data appear to be absolutely necessary.

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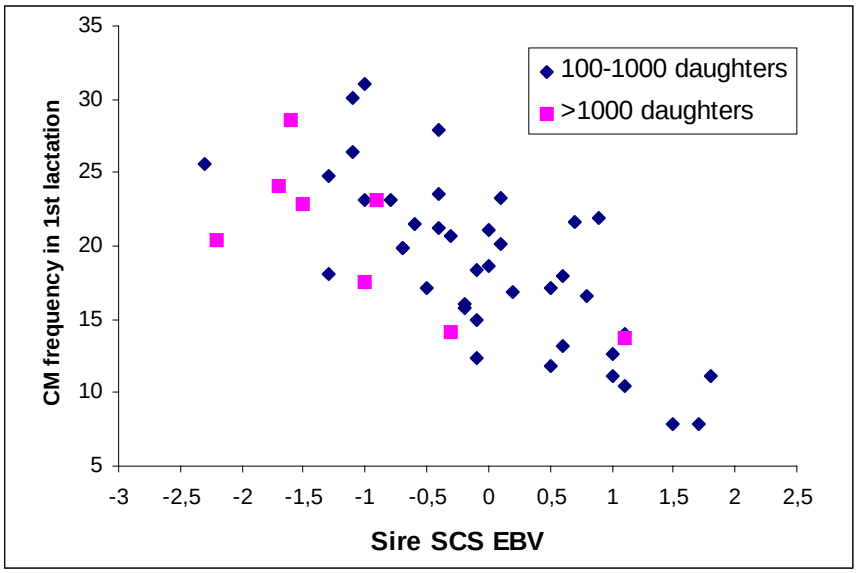

Figure 3. Relationship between SCS EBV of 50 service sires and the average CM frequency in first lactation of their progeny.