Genetic Correlations between Somatic Cell Count and Milkability in the First Three Lactations in Fleckvieh

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Abstract

Test-day records for log-transformed somatic cell count (SCS) and average flow rate (AFR) from the routine dairy recording from Bavarian Fleckvieh cows were analysed. Three data sets with observations of approximately 20,000 cows each were sampled from the total data set. For each of the first three lactations six time periods with up to 33 days were defined. Multiple-trait REML analyses were carried out to estimate variance components for these time periods. Estimates of heritability for SCS ranged from 0.05 to 0.13. Estimates of heritability for AFR ranged from 0.21 to 0.41. Phenotypic correlations obtained from a large data set indicated, that the relationship between SCS and AFR varied across lactations as well as throughout lactation. Estimation of genetic correlations between SCS and AFR for time periods confirmed this. Throughout lactation 1 as well as early in later lactations, SCS and AFR were positively correlated. However, estimates of genetic correlation changed as lactation progressed. Late in lactations 2 and 3, a clearly favourable relationship between SCS. Since results from the literature indicated a similar relationship between SCS and milk yield, further research will include milk yield as well as other milkability traits in order to clarify the causal relationship among these traits.

1. Introduction

In the joint genetic evaluation in Austria and Germany (Baden-Wuerttemberg, Bavaria) for somatic cell count (SCC) and milking speed a multiple trait model with five traits is applied: somatic cell count from lactations 1 to 3, average flow rate (AFR) from Austria and Baden-Wuerttemberg, and AFR from Bavaria. These milkability traits are from different recording systems and, therefore, are considered to be different traits (Sprengel et al., 2001). Only AFR from lactation 1 is included, even though in Bavaria AFR from all lactations is available for approximately 75% of the cows. Genetic correlations between SCC and AFR are assumed to be positive, based on results by Sprengel et al. (2000). The unfavourable genetic correlation between SCC and milking speed is well documented (see, e.g., Boettcher et al., 1998; Rupp and Boichard, 1999).

Dairy farmers place considerable emphasis on milkability, because slow milking cows are hindering the milking process of the herd, especially in milking parlours. At auctions, where daily milk yield as well as milkability of freshly calved heifers are announced, milkability had a significant impact on the price (Krogmeier *et al.*, 2006). Faster milking cows raised better prices. However, the relationship was not linear since cows with very high milkability seemed to get penalized. This indicated that farmers consider very fast milking cows to be more susceptible for mastitis.

A previous study by Dodenhoff *et al.* (1999) indicated that AFR in later lactations is larger than in first lactation and that the shape of lactation curve is different in later lactations as compared to first lactation. Therefore, AFR from later lactations might give additional information about milkability of cows and increase accuracy of genetic evaluation. However, very little is known about the relationship between SCC and milkability in later lactations. Therefore, objective of this study was to estimate genetic correlations between SCC and AFR in the first three lactations.

2. Materials and Methods

Data for this study included test-day records of Fleckvieh cows from Northern Bavaria, recorded from 1999 to 2007. The data were from the routine dairy recording, where AFR was derived based on threshold flow rates (Worstorff *et al.*, 1992). Depending on the recording method (two milkings per day or alternative milking) one or two observations per test-day were available. A large data set was extracted to analyze phenotypic variability of SCC and AFR during lactations and to calculate phenotypic correlations between these traits. This data set comprised test-day records from all cows born after 2000. There were 328,422 cows in lactation 1 which had on average 6.5 SCC observations and 11.1 AFR observations. Naturally, there were fewer cows in lactation 2 (185,311 cows, on average 6.3 SCC observations and 10.8 AFR observations, respectively), and in lactation 3 (89,107 cows, on average 5.9 SCC observations and 10.0 AFR observations, respectively) because of selection or because they had not yet entered later lactations.

For the estimation of variance components three smaller data sets with records from herds larger than average were sampled. Data were edited with respect to age at calving, days in milk (DIM, 8 to 305), and number of observations per herd test-day. Cows were required to have at least one observation in the first lactation. These edits left 18,719, 16,937, and 19,041 cows in data sets 1, 2, and 3, respectively. For each cow, seven generations of paternal pedigree information and two generations of maternal pedigree information were added. Only informative ancestors were kept so that the total number of animals in the relationship matrix was 46,216, 40,725, and 46,695, respectively.

Lactations were divided into periods which were considered to be different traits. 18 time periods (DIM 8-20, 31-63, 64-96, 130-162, 196-228, 275-305 in lactations 1, 2, and 3, respectively) were defined. The first time period in each lactation included only 13 days in order to obtain precise information about the beginning of lactation. If two milkings per cow and test-day were available, the one where the component sample was taken remained in the data. Preliminary studies revealed that AFR from morning milkings and AFR from evening milkings can be considered to be the same trait, even though AFR from morning milkings is slightly larger (uneven milking intervals). To improve normality, a square root transformation was applied to AFR, and SCC was logtransformed to somatic cell score (SCS).

Due to the definition of time periods, in lactation 1 fewest observations were in the first time period (DIM 8-20), ranging from 5,023 to 5,484 observations. The second time period had the most observations (ranging from 13,995 to 15,325 observations). As could be expected, the number of observations decreased towards the end of lactations (cows drying off) as well as across lactations (cows being culled). The last time period in lactation 3 included 2,779 to 4,820 observations. Means across data sets were fairly similar for SCS and AFR, respectively.

For each data set multiple trait analyses with four traits were carried out for various combinations of the time periods for both traits in order to fill the diagonal as well as the offdiagonals of the 36 x 36 matrix of parameter estimates (18 SCS traits, 18 AFR traits). To account for selection, each analysis included time period 2 from lactation 1 for both SCS and AFR. In the models, fixed effects of herd x year and calving year x calving month were included. Days in milk and age at first calving were linear covariates. Components of variances were estimated by REML using an average information algorithm implemented in the DMU package (Madsen and Jensen, 2000).

3. Results and discussion

3.1 Phenotypic variability

AFR curves in later lactations were different from the curve in lactation 1 (Figure 1). In the first part of the lactation, AFR in lactations 2 and 3 was considerably larger than in lactation 1. However, throughout the lactation AFR in the second and third lactation decreased stronger as compared to lactation 1. Therefore, from day 200 onwards AFR in lactation 1 was actually larger than in lactations 2 and 3. The reason for this somewhat surprising fact may be that the degree of udder filling plays an important role in milk ejection (Bruckmaier, 2001). Figure 2 shows that the corresponding pattern of lactation curves for milk yield per milking was very similar to the pattern for AFR. Towards the end of lactation cows in the first lactation had milk yields comparable to cows in later lactations. But since first lactation cows will in general have smaller udders than older cows, their udders most likely had a larger degree of filling, thus leading to a larger AFR.



Figure 1. Lactation curves for average flow rate (running averages, 5 days) of Fleckvieh cows.



Figure 2. Lactation curves for milk yield per milking (running averages, 5 days) of Fleck-vieh cows.

Lactation curves for log-transformed SCC (SCS) are presented in Figure 3. Early in lactation, SCS was fairly high in all lactations, and it decreased until around day 35. From this point on, SCS increased during the remainder of lactation. However, the increase was stronger in later lactations as compared to lactation 1. An effect of parity and stage of lactation on somatic cell count was also found in previous studies (e.g., Schepers *et al.*, 1997). For SCS, there seemed to be a larger difference between lactations 2 and 3 than for AFR and milk yield, respectively.



Figure 3. Lactation curves for somatic cell score (running averages, 5 days) of Fleckvieh cows.

Figure 4 shows that correlations between SCS and AFR on each day of lactation were generally small. Early in lactation 1 the correlation was close to zero. After a short increase to approximately 0.10 at day 45 it decreased as lactation progressed, and even turned negative in the last third of the lactation. The shape of the curves was similar in later lactations but the correlations were negative from the beginning of lactation. These findings were unexpected. The well known unfavourable relationship between SCS and AFR was confirmed only for the first half of lactation 1, albeit at a lower level than what might have been expected. Later in lactation 1 as well as in lactations 2 and 3, respectively, SCS and AFR were actually favourably correlated, i.e., cows with larger AFR tended to have smaller SCS. This relationship became more favourable as the lactation progressed.



Figure 4. Lactation curves for phenotypic correlations (running averages, 5 days) between somatic cell score and average flow rate of Fleckvieh cows.

3.1 Variance components

Patterns of variance components across data sets were fairly consistent as were the patterns of estimates of heritabilities and correlations (not presented). Since the data sets were sampled from the same population, the estimates from the three data sets were combined.

The combined estimates of heritability (h^2) for SCS and AFR are presented in Table 1. For SCS, they ranged from 0.05 to 0.13, slightly increasing during lactation which was also observed by Druet *et al.* (2005). As in Liu *et al.* (2000), h^2 in the first part of the lactation was smaller in lactation 2 and 3, respectively, as compared to lactation 1.

For AFR, h^2 in lactation 1 ranged from 0.29 to 0.41, agreeing well with results from other studies (Sprengel *et al.*, 2000; Ilahi and Kadarmideen, 2004; Norberg and Rasmussen, 2007). Slightly lower h^2 were found in lactations 2 and 3. In all lactations, h^2 was largest for DIM 31-63 and for DIM 64-96, and smallest for DIM 275-305.

Estimates of the phenotypic correlation between corresponding time periods of SCS and AFR (Table 1) were in line with the correlations from the large data set (Figure 4). They were close to zero early in lactation and then became slightly negative (lactation 1) or clearly negative (lactations 2, 3) in later time periods.

In lactation 1, estimates of the genetic correlation (r_a) between SCS and AFR ranged from 0.21 to 0.47 (Table 1). Such unfavourable genetic correlations were also found by Boettcher *et al.* (1998), Rupp and Boichard (1999), and Sprengel *et al.* (2000). Time periods later in lactation had slightly smaller r_a as compared to time periods earlier in lactation. Positive r_a of the same magnitude were found for the first three time periods in lactation 2 and lactation 3, respectively, but r_a decreased sharply towards the end of lactation, resulting in r_a for DIM 275-305 as negative as -0.34 (lactation 2) and -0.21 (lactation 3).

Similar results have been reported for the genetic correlation between milk yield and log SCC as summarized by (Mrode and Swanson, 1996). Genetic correlations between test-day SCC and milk yield changed from low positive to negative during the first lactation and from near zero to negative in later lactations (Haile-Mariam *et al.*, 2001). Both Schutz *et al.* (1990) and Haile-Mariam *et al.* (2001) suggested that

early in lactation high yielding cows were more susceptible to mastitis, resulting in a positive genetic correlation, while later in lactation and in subsequent lactations, respectively, mastitis caused lower milk yields, resulting in a negative genetic correlation.

Because of the close relationship of milkability with milk yield, the changing genetic correlation between SCS and AFR possibly has a similar background. Early in lactation, the correlation is positive because the defences of the teat duct appear to be limited by the rate of milk flow (Hillerton, 1986) and, therefore, cows with high flow rates are easier to infect (Boettcher et al., 1998). This correlation is certainly enhanced by the fact that high vielding cows have larger AFR and are more susceptible to mastitis. As lactations progress cases of clinical mastitis or subclinical mastitis, indicated by increased SCS, lead to reduced milk yield, a lower degree of filling of the udder and thus to a lower AFR. Even lower AFR are possible if mastitis causes damage to the udder tissue which could lead to a prolonged milking time.

One could also think of an opposite causal relationship between SCS and AFR if evenness of milk removal from single quarters is considered. AFR can decrease if milk is removed unevenly. Early in lactation, milk will most likely be removed completely from all quarters at the same time. As lactation progresses, udder morphology may change so that milk from fore quarters is removed earlier than from hind quarters. If milk is removed unevenly, some quarters may be more drained than others, resulting in larger SCS since stripping milk has considerably more cells than foremilk (Miller *et al.*, 1986). Also, overmilking of single quarters may be disadvantageous for udder health.

	1 0	Heritability		Correlation	
Lactation	DIM	SCS	AFR	phenotypic	genetic
1	8 - 20	0.09	0.32	0.02	0.26
	31 - 63	0.10	0.40	0.07	0.47
	64 - 96	0.10	0.41	0.06	0.45
	130 - 162	0.10	0.39	0.02	0.32
	196 - 228	0.11	0.37	0.01	0.33
	275 - 305	0.10	0.29	-0.04	0.21
2	8 - 20	0.05	0.34	0.03	0.40
	31 - 63	0.06	0.37	0.02	0.40
	64 - 96	0.10	0.37	-0.01	0.28
	130 - 162	0.11	0.33	-0.06	0.07
	196 - 228	0.13	0.30	-0.16	-0.19
	275 - 305	0.13	0.28	-0.20	-0.34
3	8 - 20	0.06	0.34	0.01	0.38
	31 - 63	0.07	0.34	0.00	0.40
	64 - 96	0.08	0.36	-0.02	0.26
	130 - 162	0.10	0.32	-0.09	-0.03
	196 - 228	0.12	0.28	-0.17	-0.14
	275 - 305	0.09	0.21	-0.21	-0.21

Table 1. Estimates of heritability for SCS and AFR and estimates of phenotypic and genetic correlations between corresponding time periods for SCS and AFR.

4. Conclusions

Phenotypic correlations between SCS and AFR, obtained from a large data set, were close to zero and became slightly more negative throughout lactation. Heritability estimates for SCS in Fleckvieh, obtained from three smaller data sets, were in good agreement with results from other studies. Heritability estimates for AFR in the first three lactations were of the same magnitude (ranging from 0.21 to 0.41) and decreased toward the end of lactation. An unfavourable genetic relationship between SCS and AFR, as known from the literature, was confirmed for lactation 1 and for time periods early in lactations 2 and 3. Results indicated the relationship became considerably more negative over the course of the lactation, .i.e., at the end of lactations 2 and 3 SCS and AFR were favourably correlated. Obviously, the relationship of SCS with AFR is very similar to its relationship with milk yield with the same mechanisms being responsible. Additionally, evenness, or rather unevenness, of milk removal from single quarters may play a role.

Further research on the correlation between SCS and AFR will include milk yield and possibly other milkability traits, e.g., maximum flow rate, in order to help clarify the causal relationship between these traits.

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