Alternative use of somatic cell counts in genetic selection for clinical mastitis

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Abstract
Three sets of somatic cell count (SCC) traits were defined in this study: (1) lactation-averages of SCC, (2) traits derived from the proportion of test-day SCC above 150,000 cells/ml, (3) patterns of peaks in SCC. Genetic parameters were estimated for these SCC-traits, as well as their genetic correlation with mastitis-traits. Mastitis-traits were clinical and subclinical mastitis (CM and SCM, respectively) and were both scored as binary traits. Data was available from farms with a PC Management Information System, and the dataset contained 56,726 lactations of 30,145 cows on 272 herds. Variance components for sire and permanent animal effects were estimated using ASREML. The estimated heritabilities for CM and SCM were both low at 3%. Heritabilities for SCC-traits ranged from 1 to 13%; lowest for the patterns of peaks in SCC, and highest for lactation-average SCC. A range of genetic correlations was estimated between SCC-traits and CM or SCM, varying from 0.55 to 0.93 for CM, and from 0.68 to 0.98 for SCM. Strongest genetic correlation was estimated between CM and the pattern of peak that describes a quick recovery in SCC (0.93), and between SCM and SCC averaged between 151 and 400 days (0.95). Therefore, to improve the overall udder health, information from a combination of SCC-traits is expected to be most successful.

Introduction
Current selection indices realize an increase in milk yield and simultaneously monitor udder health by selecting for lower lactation-average somatic cell count (**SCC**). However, the estimated moderate genetic correlation of ≈ 0.7 between lactation-average SCC and udder health indicates that, genetically, they are not identical traits (Mrode and Swanson, 1996). Therefore, it could be worthwhile to establish if other SCC-traits provide additional information for selection that aims to decrease genetic susceptibility to clinical and subclinical mastitis (**CM** and **SCM**, respectively), in comparison to the information provided by one lactation-average SCC.

The objective of this study was, therefore, to develop alternative measures from SCC, and to estimate their genetic relationship with CM and SCM. These alternative measures take into account the length of the observed lactation-period, patterns of peaks in SCC, and indicators of excessive SCC.

Materials and Methods
To analyze alternative SCC-traits as mastitis-indicators for genetic selection, three sets of SCC-traits were defined. The first set of SCC-traits consisted of SCC averaged over different lengths of observation. The second set of SCC-traits was derived from the proportion of excessive test-day SCC-recordings. The third set of SCC-traits corresponded to patterns of peaks in SCC (De Haas et al., 2004).

Trait definitions
Somatic cell counts were averaged over shorter lactation-lengths, because it is known that most first cases of CM occur in the first weeks of lactation. To be able to find an optimum, SCC were averaged per lactation over the test-day records up to 400, 300 and 150 days in milk (**DIM**). This average SCC was transformed to SCS=1000+100*(2log(SCC/1,000,000) resulting in SCS400, SCS300 and SCS150, respectively. To be able to compare the first *versus* second part of the lactation, SCC was averaged over day 151 to 400 and this average was log-transformed (SCS151-400).

Excessive test-day SCC indicated the suspicion of a SCC to originate from an intramammary infection (**IMI**). Each test-day SCC was classed individually as a binary trait; when the test-day SCC was above 150,000 cells/ml it was registered as 1, otherwise it was scored as 0. Based on these classifications, two SCC-traits were defined to describe the dynamics of SCC during lactation. The hypothesis is that these

dynamics differ between healthy and mastitic cows. The first SCC-trait will be referred to as 'suspected', and distinguishes only between presence (1) or absence (0) of test-day SCC above 150,000 cells/ml, independent of how many high SCC-recordings were registered in the lactation. The second SCC-trait will be referred to as 'seriousness', and is calculated as the mean of the classified individual test-days within one lactation.

Patterns of peaks in SCC distinguish between lactations with short or longer periods of increased SCC, and also between lactations with and without recovery within three test-day records. Two patterns of peaks in SCC are genetically analyzed. The first pattern describes a quick rise in SCC followed by an immediate decrease in SCC; i.e. consecutive test-day recordings of SCC had to be low-high-low (P1). The second pattern captures a longer increased SCC; i.e. one test-day with a low SCC recorded followed by two test-days with high SCC, and no recovery took place within two test-day recordings (P2). The sum of P1 and P2 (i.e. all patterns of peaks (= P_ALL), without specifying what kind of peak exactly) was also analyzed.

Analyzed udder health traits were CM and SCM, scored as present (1) or absent (0) when in a lactation at least one case of CM, respectively SCM, was recorded. Cases of CM were registered by farmers in their PC Management Information System, and they allowed NRS to upload and use these data. Cases of SCM were derived from test-day SCC, and defined as a conversion from two consecutive SCC test-days below a certain cut-off value to one above this value (Van den Borne et al., 2006). The cut-off values in the current study were the same as those used in the national milk recording; i.e. 150,000 cells/ml for heifers, and 250,000 cells/ml for multiparous cows.

Data editing

Data editing was done by excluding lactations (1) of fourth parity and higher, (2) with ages at first calving less than 640 days, and (3) of cows with less than 75% Holstein-Friesian genes. Only test-day SCC-recordings between 5 and 400 DIM were included, and only observations on CM between -15 and 400 DIM. For the analyses, a pedigree file was constructed based on sires and maternal grandsires (MGS) of cows in the data. This file contained 3,436 AI bulls with 2,446 sires plus 2,471 MGS (of which 1,791 were sire as well), and 310 unique identities of fathers of the sires or MGS. Cows with unknown pedigree, and cows with sires with less than five offspring (as sire or MGS) in the dataset, were deleted. The final dataset consisted of 56,726 lactations of 30,145 cows on 272 herds.

Statistical analyses

ASREML (Gilmour et al., 2006) was used to estimate variance components. Heritabilities were estimated with univariate analyses, using a linear model. The model included random effects for sire and MGS and for cow, to account for the permanent animal effects across repeated lactations. The model used was:

$$Y = \mu + \text{fixed effects} + S_{sire} + \tfrac{1}{2} S_{mgs} + PERM_{animal} + e$$

Fixed effects included were an interaction between herd and year of calving (with 1,647 classes), parity (with three classes), and month of calving (with twelve classes). A linear polynomial was included for age at calving.

Bivariate analyses were carried out to estimate correlations between CM, SCM and SCC-traits, using linear models. Fixed effects were the same as mentioned for the univariate analyses. Genetic parameters were calculated from the estimated variance components.

Results

The proportion of lactations with at least one case of CM was calculated to be approximately 11%. The proportion of lactations with at least one case of SCM was much higher; 33%. The mean SCS increased with increasing length of observation from 601 to 647, but was highest for SCS151-400 (659.5). The proportion of lactations with presence of a SCC above 150,000 cells/ml was roughly 60%. The proportion of lactations with presence of any kind of peak was 22%, equally divided among P1 and P2.

Heritabilities

The heritability of CM and SCM was 2 and 3% (Table 1). Higher heritabilities were estimated for SCS of lactation-average SCC, varying from 8 to 13%, increasing with longer lengths of lactation. Heritabilities of SCC-traits describing the dynamics of SCC were low at 6 and 11%, but higher than the heritabilities for CM and SCM. Patterns of peaks in SCC showed low heritabilities (1 to 5%).

Table 1. *Heritabilities (h^2) and genetic variances (σ^2_a) of udder health traits (clinical and subclinical mastitis) and several alternative somatic cell count traits, with their respective standard errors as subscripts*

	h^2	σ^2_a
Clinical mastitis	$0.02_{.01}$	$0.002_{.001}$
Subclinical mastitis	$0.03_{.01}$	$0.007_{.001}$
SCS150	$0.08_{.01}$	$1{,}830_{252.1}$
SCS300	$0.12_{.02}$	$2{,}044_{255.2}$
SCS400	$0.12_{.02}$	$1{,}986_{245.6}$
SCS151-400	$0.13_{.02}$	$2{,}047_{254.8}$
Suspected	$0.06_{.01}$	$0.014_{.002}$
Seriousness	$0.11_{.01}$	$0.008_{.001}$
Any pattern in SCC	$0.05_{.01}$	$0.009_{.002}$
Quick recovery pattern	$0.01_{.00}$	$0.001_{.000}$
No recovery pattern	$0.04_{.01}$	$0.004_{.001}$

Genetic correlations

The strongest genetic correlations between CM and the newly defined SCC-traits were estimated with the patterns of peaks in SCC (Table 2). Between SCM and the newly defined SCC-traits, the strongest genetic correlations were estimated with the lactation-averages, especially those SCS of SCC averaged over longer periods.

When comparing the estimated genetic correlations of SCC-traits with either CM or SCM, stronger genetic correlations were estimated for SCM than for CM with SCS300, SCS400, SCS151-400 and the traits describing the dynamics of SCC. Weaker genetic correlations were estimated with the patterns of peaks in SCC (Table 2).

The estimated genetic correlation between CM and SCM was 0.50 (s.e. 0.12).

Table 2. *Genetic correlations between udder health traits (clinical mastitis (CM) and subclinical mastitis (SCM)) and several somatic cell count traits, with their respective standard errors as subscripts*

	CM	SCM
SCS150	$0.73_{.07}$	$0.75_{.06}$
SCS300	$0.67_{.08}$	$0.89_{.04}$
SCS400	$0.66_{.08}$	$0.91_{.03}$
SCS151-400	$0.55_{.09}$	$0.95_{.02}$
Suspected	$0.60_{.09}$	$0.94_{.02}$
Seriousness	$0.64_{.09}$	$0.85_{.05}$
Any pattern in SCC	$0.89_{.06}$	$0.73_{.07}$
Quick recovery pattern	$0.93_{.06}$	$0.68_{.10}$
No recovery pattern	$0.88_{.06}$	$0.76_{.07}$

The genetic correlation between CM and SCS150 was stronger than the genetic correlation between CM and SCS151-400 (Table 2). This seems to suggest that genetic selection on lower SCC during early lactation also decreases the occurrence of CM. For SCM stronger genetic correlations were estimated with SCC averaged over long (≥ 300 DIM) and late lactations (151-400 DIM) than over short lactations (150 DIM). This seems to suggest that selection for lower SCC, especially in late lactation, will decrease the frequency of SCM.

Discussion

Several studies have shown that the frequency of cases of CM is much higher in the first half of the lactation than in the second half (Emanuelson et al., 1988; Barkema et al., 1998). Therefore, the hypothesis was that SCC in the first half of the lactation is more informative as a (clinical) mastitis-indicator, than lactation-average SCC including test-day recordings in the second half of the lactation. We have indeed estimated a stronger or equally strong genetic correlation between CM and SCC in the first half of the lactation (SCS150) than between CM and SCC in the second half of the lactation (SCS151-400) or between CM and SCC through the whole lactation (i.e. SCS300 or SCS400). Emanuelson et al. (1988) reported similar results as well in Swedish data, and Barkema et al. (1998) have reported this in other Dutch data. Also for genetic selection it is interesting to keep the period of data collection short, so that information becomes sooner available and genetic selection can occur faster.

However, an average does still not give full justice to the dynamic fluctuations in SCC. The most advantageous cow would respond very quickly to an infection and then return to normal levels. Such a picture is not necessarily reflected in an average. Therefore, we aimed to summarize SCC in more biologically informative ways, by analyzing (a) patterns of peaks in SCC and interpreting (b) excessive cell counts.

Genetically, reducing pathogen-specific cases of CM is not of big interest, because the Dutch breeding objective is not to distinguish between contagious and environmental mastitis, but to decrease genetic susceptibility to overall CM (and SCM). The estimated heritabilities of the patterns of peaks in SCC were similar to the estimated heritabilities for CM and SCM, and a strong genetic correlation was estimated between

P_ALL and CM. This suggests that the proportion of cows with at least one case of CM can be effectively reduced by genetic selection on diminishing presence of peaks in SCC.

The idea behind emphasizing excessive test-day SCC was to attribute risks of IMI to cows. It relied on the hypothesis that higher SCC is linked to an IMI. This is confirmed by the strong genetic correlation between SCM and the trait 'suspected', that identified lactations with at least one cell count above 150,000 cells/ml. Apparently, cases of SCM can be better captured by identifying excessive cell count than by presence of patterns of peaks in SCC or by lactation-average SCC. Only SCC averaged over long lasting lactations (400 DIM) showed a similar strong genetic correlation with SCM. This can be confirmed by the current knowledge about SCM, that SCM can be identified by increased SCC and usually even repeated increased SCC-recordings, with no clinical signs visible (Zadoks, 2002).

Further research is needed to determine which traits should be included in a (final) selection index to improve the overall udder health by genetics. As stated before, a combination of traits will most likely be needed, because of the wide range of genetic correlations that was estimated between SCC-traits and CM or SCM, varying from 0.55 to 0.93 for CM, and from 0.68 to 0.98 for SCM. The current study has already shown some very promising results for some of the alternatively defined SCC-traits, but further refinement of the SCC-, CM- or SCM-traits might even improve the results. Selection indices can be calculated with different combinations of the SCC-traits to test what combination of traits will result in the highest accuracy and most genetic progress.

Conclusions

A wide range of genetic correlations between udder health traits and several SCC-traits is estimated in this study, and very promising results are shown for some of the alternatively defined SCC-traits. Further work to refine the definitions of the SCC-, CM- and/or SCM-traits might even improve the results. Selection index calculations will have to be carried out eventually, to come up with a selection of the most informative SCC-traits to be included in an index to improve overall udder health (i.e. both CM and SCM).

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