

Improving Selection on Udder Health by using Different Trait Definitions of Somatic Cell Count

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Abstract

Two sets of somatic cell count (SCC) traits were defined in this study: (1) traits derived from the proportion of test-day SCC above 150,000 cells/ml, (2) 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 11%; lowest for the patterns of peaks in SCC, and highest for SCC-traits describing the dynamics in SCC. A range of genetic correlations was estimated between SCC-traits and CM or SCM, varying from 0.60 to 0.93 for CM, and from 0.68 to 0.94 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 the presence of at least one test-day SCC above 150,000 cells/ml (0.94). Therefore, to improve the overall udder health, information from a combination of SCC-traits is expected to be most successful.

1. 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 approximately 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.

2. Materials and Methods

To analyze alternative SCC-traits as mastitis-indicators for genetic selection, two sets of SCC-traits were defined. The first set of SCC-traits corresponded to patterns of peaks in SCC (De Haas *et al.*, 2004). The second set was derived from the proportion of excessive test-day SCC-recordings.

2.1 Trait definitions

Patterns of peaks in SCC distinguished 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 were analyzed. The first pattern described a quick rise in SCC followed by an immediate decrease in SCC; i.e. consecutive test-day SCC-records had to be low-high-low (P1). The second pattern captured a longer increased

SCC; i.e. one test-day with a low SCC recorded followed by two test-days with high SCC, so no recovery took place within two test-day recordings (P2). The sum of P1 and P2 (= P_ALL) was also analyzed.

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, three 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 distinguished 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 was calculated as the mean of the classified individual test-days within one lactation. The last SCC-trait is 'duration' and can only be created within the 'suspected' group. 'Duration' was defined as the number of test-day recordings during the longest episode of consecutive cell counts above 150,000 cells/ml.

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. 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.

2.2 Data editing

Data editing was done by excluding lactations (1) of fourth parity and higher, (2) with ages at 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.

2.3 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:

$$Y = \mu + \text{fixed effects} + S_{\text{sire}} + \frac{1}{2} S_{\text{mgs}} + \text{PERM}_{\text{animal}} + e$$

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.

3. 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 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.

3.1 Heritabilities

The heritability of CM and SCM was 2 and 3% (Table 1). Heritabilities of SCC-traits describing the dynamics of SCC were low between 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_a^2) 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_a^2 |
|----------------------|---------------------|-----------------------|
| Clinical mastitis | 0.02 _{.01} | 0.002 _{.001} |
| Subclinical mastitis | 0.03 _{.01} | 0.007 _{.001} |
| Suspected | 0.06 _{.01} | 0.014 _{.002} |
| Seriousness | 0.11 _{.01} | 0.008 _{.001} |
| Duration | 0.08 _{.01} | 0.406 _{.057} |
| Any peak | 0.05 _{.01} | 0.009 _{.002} |
| Short peak | 0.01 _{.00} | 0.001 _{.000} |
| Long peak | 0.04 _{.01} | 0.004 _{.001} |

3.2 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). For SCM, the strongest genetic correlations were estimated with the newly defined SCC-traits describing the dynamics in SCC.

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 |
|-------------|---------------------|---------------------|
| Suspected | 0.60 _{.09} | 0.94 _{.02} |
| Seriousness | 0.64 _{.09} | 0.85 _{.05} |
| Duration | 0.62 _{.09} | 0.88 _{.04} |
| Any peak | 0.89 _{.06} | 0.73 _{.07} |
| Short peak | 0.93 _{.06} | 0.68 _{.10} |
| Long peak | 0.88 _{.06} | 0.76 _{.07} |

4. Discussion

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. 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).

4.1 Selection indices

To test which SCC-traits are genetically most informative as mastitis-indicators, we have calculated accuracies of several selection indices (Hazel, 1943). These selection indices consisted of 1, 2 or 3 newly defined SCC-trait.

Comparisons were made between sires with 100 or 10,000 daughters; i.e. young *versus* proven bulls.

When only the currently used lactation-average SCC, with a genetic correlation of 0.65 was included, the young sires with 100 daughters accomplished an accuracy of 57% (Table 3a). Lower accuracies were accomplished for the newly defined SCC-traits that describe the dynamics in SCC. The highest accuracy was accomplished for P_ALL (67%).

Table 3a. Accuracies of udder health indices including 1, 2 or 3 somatic cell count traits, based on sires with information from 100 daughters

| | 1 trait | SCC + 1 trait | SCC + any peak + 1 traits |
|-------------|---------|------------------|---------------------------------|
| SCC | 0.565 | | |
| Suspected | 0.466 | 0.587 | 0.699 |
| Seriousness | 0.550 | 0.611 | 0.706 |
| Duration | 0.518 | 0.599 | 0.702 |
| Any peak | 0.665 | 0.701 | |
| Short peak | 0.416 | 0.611 | |
| Long peak | 0.624 | 0.678 | |

Including one newly defined trait in the index with a genetic correlation of 0.9 with the lactation-average SCC, resulted in higher accuracies. An index with lactation-average SCC and ‘any peak’ (i.e. P_ALL) showed the highest accuracy of 70% (Table 3a). Indices with three traits; i.e. lactation-average SCC, plus P_ALL and a newly defined trait describing the dynamics in SCC did not prove to be better for young bulls than indices with two traits.

Table 3b. Accuracies of udder health indices including 1, 2 or 3 somatic cell count traits, based on sires with information from 10,000 daughters

| | 1 trait | SCC + 1 trait | SCC + any peak + 1 traits |
|-------------|---------|------------------|---------------------------------|
| SCC | 0.649 | | |
| Suspected | 0.598 | 0.650 | 0.984 |
| Seriousness | 0.639 | 0.662 | 0.974 |
| Duration | 0.619 | 0.654 | 0.979 |
| Any peak | 0.887 | 0.944 | |
| Short peak | 0.912 | 0.951 | |
| Long peak | 0.876 | 0.925 | |

When selecting well-proven sires with 10,000 daughters, selection against presence of short peaks (i.e. P1) in SCC was most effective to decrease the frequency of CM (Table 3b). Also for proven bulls, indices with three traits; i.e. lactation-average SCC, plus P_ALL and a newly defined trait describing the dynamics in SCC did not appear to be better than indices with two traits (Table 3b).

5. Conclusions

A wide range of genetic correlations between udder health traits and alternatively defined 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-traits might even improve the results. More selection index will be calculated 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).

Acknowledgements

The farmers are greatly acknowledged for providing information on occurrence of cases of clinical mastitis. This study is part of the five-year mastitis program of the Dutch Udder Health Center and was financially supported by the Dutch Dairy Board.

References

- De Haas, Y., Veerkamp, R.F., Barkema, H.W., Gröhn, Y.T. & Schukken, Y.H. 2004. Associations between pathogen-specific clinical mastitis and somatic cell count patterns. *J. Dairy Sci.* 87, 195-105.
- Gilmour, A.R., Gogel, B.J., Cullis, B.R. & Thompson, R. 2006. *ASREML User Guide Release 2.0* VSN International Ltd., Hemel Hempstead, HP1 1ES, United Kingdom.
- Hazel, L.N. 1943. The genetic basis for constructing selection indexes. *Genetics* 28, 479.
- Mrode, R.A. & Swanson, G.J.T. 1996, Genetic and statistical properties of somatic cell count and its sustainability as an indirect means of reducing the incidence of mastitis in dairy cattle. *Anim. Breed. Abstr.* 64, 847-857.
- Zadoks, R.N. 2002. Molecular and mathematical epidemiology of *Staphylococcus aureus* and *Streptococcus uberis* mastitis in dairy herds. *Ph.D. Thesis*, Utrecht University, Utrecht, The Netherlands.