

Genetic Associations between Somatic Cell Score and Pathogen Specific Subclinical Mastitis in Norwegian Red Cows

K. Haugaard, B. Heringstad*† and A. C. Whist‡*

**Department of Animal and Aquacultural Sciences, Norwegian University of Life Sciences, PO Box 5003, N-1432 Ås, Norway. †GENO Breeding and A. I. Association, PO Box 5003, N-1432 Ås, Norway. ‡ Norwegian School of Veterinary Science, PO Box 8146 Dep, N-0033 Oslo, Norway*

Abstract

The aim of this study was to estimate heritabilities of and genetic correlations among pathogen specific subclinical mastitis (SCM) and lactation mean somatic cell score (LSCS) in Norwegian Red. Four binary pathogen specific SCM traits, *Staphylococcus aureus*, *Streptococcus dysgalactiae*, *Streptococcus uberis* and coagulase negative staphylococci (CNS), were analyzed together with unspecific SCM and LSCS using a multitrait linear sire model. Estimated heritability was 0.139 for LSCS, 0.075 for unspecific SCM, and ranged from 0.002 (*Streptococcus uberis*) to 0.013 (CNS) for pathogen specific SCM. Genetic correlations were positive and high, ranging from 0.68 to 0.99. Most of the genetic correlations were significantly lower than 1, indicating that subclinical mastitis caused by different pathogens can be considered as different traits.

Keywords: pathogen specific mastitis, subclinical mastitis, linear model

Introduction

Mastitis is the most frequent disease in dairy cattle worldwide. The pattern of disease varies from mild subclinical to severe clinical cases and mastitis can be caused by several different pathogens. Somatic cell count (SCC) is associated to mastitis, and often used in genetic evaluation as an indirect measure of disease. Since 2000, bacteriological milk sample results from the mastitis laboratories have been recorded routinely in the Norwegian Dairy Herd Recording System. This additional source of data may provide valuable information on pathogen specific mastitis. Haugaard *et al.* (2011) used this data for genetic analyses of pathogen specific clinical mastitis, in this study the aim was to analyse pathogen specific subclinical mastitis. Our objective was to examine whether data from the mastitis laboratories could be used to identify pathogen specific subclinical mastitis, and to do a first genetic analysis of these traits for Norwegian Red.

Material and Methods

Information on calving, testday SCC, clinical mastitis (CM) and pathogens from milk samples on first lactation Norwegian Red cows, calving from January 2001 through December 2009, was extracted from the Norwegian Dairy Herd Recording System. The cows were sired by an AI-bull, and were 20-36 months of age at calving. First lactation was defined from day of calving to 400 days after calving, culling or date of next calving. Unspecific- and pathogen specific subclinical mastitis were defined based on the cows testday SCC records and bacteriological milk sample results (pathogen data) as illustrated in Figure 1. Subclinical mastitis (SCM) was score as 1 if two consecutive test day records had SCC > 100,000 cells/ml, where neither was associated with a case of CM, and 0 otherwise. SCC was assumed elevated due to CM if a CM record occurred within 27 days before or 1 day after the SCC-record. SCC records from the first 14 days after calving was

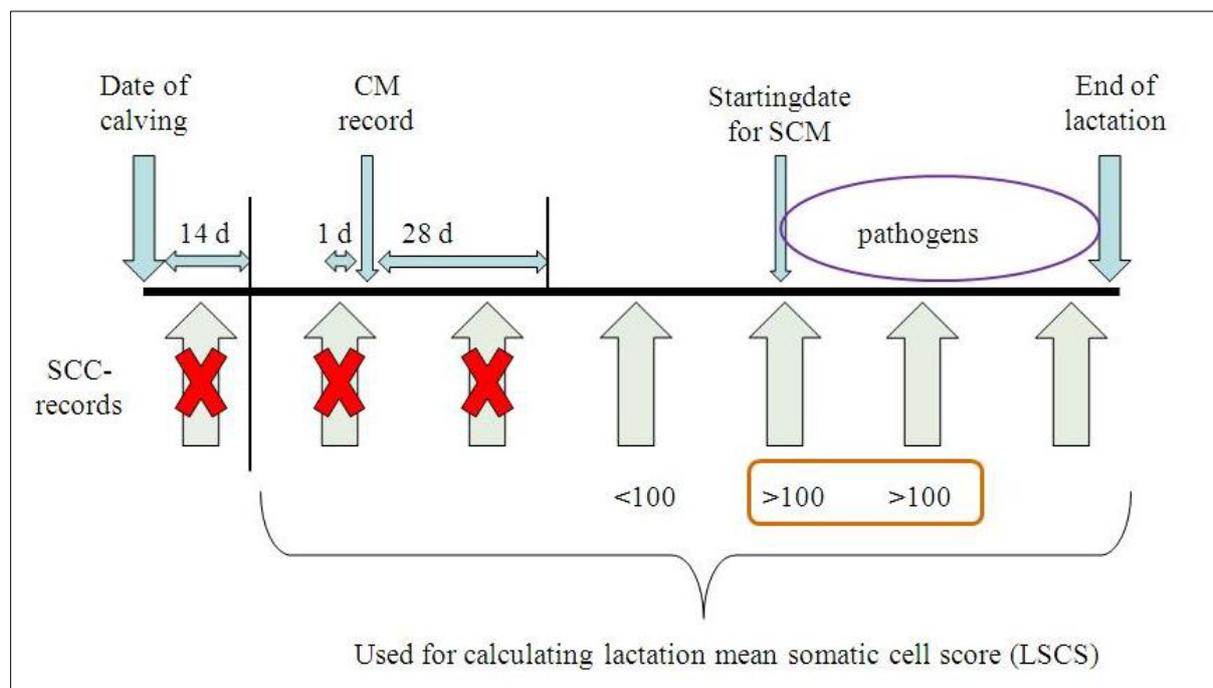


Figure 1. Definition of unspecific- and pathogen specific subclinical mastitis based on testday SCC records and bacteriological milk sample results (pathogen data).

not considered, as these are naturally elevated and gives little information about udder health. Cows with only one SCC record after the first 14 days of lactation were discarded, leading to lactation lengths no shorter than 40 days. The starting date for defining pathogen specific SCM was set to the date of the first of the elevated SCC records. Pathogen information from all milk samples collected after the starting date, excluding those that could be connected to a CM-record (+/- 7 days), was used in the analyses. Several different pathogens were classified from the milk samples, of which many had too low frequency to be used for genetic analyses. *Staphylococcus aureus*, *Streptococcus dysgalactiae*, *Streptococcus uberis* and coagulase negative staphylococci (CNS) were the most frequent pathogens that could be associated with SCM. Five binary (0/1) SCM-traits were defined, one for each of the four pathogens and one for unspecific SCM (USCM). USCM was defined as a case of SCM with either 1) no milk samples analyzed, 2) milk samples analyzed but no pathogens was found or 3) milk samples analyzed and pathogens other than the four previously mentioned was found. It is important to note that sending milk samples to the mastitis laboratory is not mandatory, only some of the cows have this information. The

frequencies of pathogen specific SCM were therefore quite low; *Staph.aureus* 2.44 %, *Strep.dysgalactiae* 0.45 %, *Strep.uberis* 0.21 %, and CNS 1.98 %, whilst for USCM the frequency was 23.2 %. The overall frequency of SCM was 27.4 %.

In addition to the 5 SCM-traits, lactation mean somatic cell score (LSCS) was included. For each cow LSCS was calculated based on all SCC-records from 14 days after calving to end of lactation, using the following formula:

$$LSCS = \frac{1}{n} \sum_{i=1}^n \left(\log_e \left(\frac{SCC}{1000} \text{ cells/ml} \right) \right)$$

where n = number of records pr cow.

Only herds with at least one milk sample that could be connected to a case of SCM were included. From the original 757,621 first lactation cows, 285,755 were included in the analyses. Herd-5-year classes were defined by using two time periods of approximately five years (2001-2004 and 2005-2009). A sire pedigree-file with 3,304 bulls was conducted by tracing the sires and maternal grandsires of the 1,549 bulls with daughters in the dataset back as far as possible.

A multivariate linear sire-model was used for analyses of LSCS, USCM, and the 4 pathogen specific SCM traits. In matrix notation:

$$y = X\beta + Z_h h + Z_s s + e$$

where y is a vector of observations, β is a vector of systematic effects, including age at first calving (17 single month classes) and month-year of calving (108 classes), h is a vector of random herd-5-year effects (10,482 levels), s is the random effect of sire (3304 bulls), e is the vector of residual effects, and X , Z_h and Z_s are the corresponding incidence matrices. As no cows could be positive for both USCM and any of the pathogen specific traits, the residual co-variances involving USCM were set to 0.

The (co)variance components were estimated using the dmuai procedure in DMU (Madsen and Jensen, 2007).

Results and Discussion

Estimated heritabilities, genetic- and residual correlations are shown in Table 1. Heritability was 0.139 LSCS, 0.075 for USCM, and ranged from 0.013 to 0.002 for the pathogen specific SCM. These heritabilities were lower than those found by Sørensen *et al.* (2009), who reported heritabilities of 0.14 for unspecific mastitis (CM and SCM), 0.045 for *Strep. dysgalactiae*, 0.057 for CNS, 0.039 for *Staph.aureus* and 0.079 for *Strep.uberis*. However, heritabilities of binary traits estimated with linear models are frequency dependent and low frequencies give low heritabilities. Sørensen *et al.* (2009) used a threshold model, which is not affected by frequency. Our heritability estimates are therefore not directly comparable.

The genetic correlations (SE) were positive and high, varying from 0.68 (0.07) between *Staph.aureus* and CNS to 0.99 (0.10) between

Table 1. Estimated heritabilities¹ (diagonal), and genetic (below diagonal, SE in parantheses) and residual² (all SE≤0.002) (above diagonal) correlations between somatic cell score (LSCS), unspecific-, *Staphylococcus aureus*-, *Streptococcus dysgalactiae*-, *Streptococcus uberis*- and coagulase negative staphylococci (CNS) subclinical mastitis (SCM).

	LSCS	Unspecific SCM	<i>Staphylococcus aureus</i> SCM	<i>Streptococcus dysgalactiae</i> SCM	<i>Streptococcus uberis</i> SCM	CNS SCM
LSCS	0.139	0.66	0.30	0.13	0.08	0.24
Unspecific SCM	0.97 (0.01)	0.075	0	0	0	0
<i>Staphylococcus aureus</i>	0.87 (0.04)	0.82 (0.05)	0.007	0.11	0.06	0.22
<i>Streptococcus dysgalactiae</i>	0.76 (0.06)	0.79 (0.06)	0.76 (0.08)	0.004	0.06	0.13
<i>Streptococcus uberis</i>	0.80 (0.08)	0.81 (0.08)	0.78 (0.10)	0.99 (0.10)	0.002	0.07
CNS	0.74 (0.04)	0.77 (0.04)	0.68 (0.07)	0.75 (0.07)	0.75 (0.10)	0.013

¹ Heritabilities was computed as: $h^2 = 4\sigma_s^2 / (\sigma_s^2 + \sigma_h^2 + \sigma_e^2)$

² The residual correlations between unspecific SCM and the pathogen specific SCM traits were set to 0.

Strep.dysgalactie and *Strep.uberis*. Except for the correlation of LSCS/USCM (0.97) and *Strep.dysgalactiae/Strep.uberis* (0.99), the genetic correlations were significantly lower than 1, indicating that SCM caused by different pathogens can be considered as different traits. As both USCM and LSCS are based on test

day SCC, their high genetic correlation was expected.

The genetic correlations between LSCS and the SCM traits varied from 0.74 (CNS) to 0.97 (USCM). These correlations were higher than those found by Sørensen *et al.* (2009), who

reported genetic correlations between lactation mean somatic cell score (5-170 days) and the mastitis traits, ranging from 0.44 (*Staph.aureus*) to 0.71 (unspecific mastitis) using bivariate threshold models. Our higher correlations may be explained by different trait definitions. Here we included only SCM, and high testday SCC is more often related to subclinical or chronic cases of mastitis than to CM.

The residual correlations were positive and low (0.03-0.3), with the exception of the correlation between LSCS and USCM (0.66).

Conclusions

This was a first genetic analysis of pathogens specific SCM in Norwegian Red. Heritabilities for all the pathogens specific mastitis traits were low. However, this was a linear model analysis, which not take into account the binary nature of the traits. Further analyses will

be done using threshold models. The genetic correlations between traits were positive and high, but most of them were significantly lower than 1, indicating that SCM caused by different pathogens can be considered to be different traits.

References

- Haugaard, K., Heringstad, B. & Whist, A.C. 2011. Genetic analyses of pathogen specific clinical mastitis in Norwegian Red cows. *J. Dairy Sci.* (Submitted).
- Madsen, P. & Jensen, J. 2007. *An user's guide to DMU. A package for analyzing multivariate mixed models.* University of Aarhus, Research Center, Foulum, Tjele, Denmark.
- Sørensen, L.P., Mark, T., Madsen, P. & Lund, M.S. 2009. Genetic correlations between pathogen-specific mastitis and somatic cell count in Danish Holsteins. *J. Dairy Sci.* 92, 3457-3471.