Genetic Parameters for Somatic Cell Score According to Udder Infection Status in Valle del Belice Dairy Sheep

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Introduction

Somatic cell count (SCC), and therefore somatic cell score (SCS), has been widely promoted as an indirect method of predicting mammary infections and as a selection criterion to improve mastitis resistance (e.g., Heringstad, B., Klemetsdal, G., and Ruane, J. (2000); Barillet (2007)). An elevated SCC in milk is an indication of the occurrence of infection in the udder. However, a possible complicating factor is that SCC in healthy and infected animals may be considered as different traits (Detilleux and Leroy (2000)). In principle, therefore, SCC from healthy and infected animals should be analyzed separately. But, because intramammary infection status is generally unknown, statistical models are usually applied indifferently to SCC obtained from infected or non infected animals. Test-day SCC may, therefore, be regarded as a mixture of observations from animals with unknown health status (Ødegård, J., Jensen, J., Madsen, P. et al. (2003)). Ødegård, J., Jensen, J., Madsen, P. et al. (2003) and Gianola, D., Ødegård, J., Heringstad, B. et al. (2004) described a mixture model for SCS containing fixed and random effects affecting baseline SCS, with mastitis producing a change in the mean and variance of the distribution.

In the present study we used a data set for which SCC as well as bacteriological data are available, indicating whether an animal was infected at the time of sampling. Therefore, instead of using mixture models to determine the infection status, we were able to analyze SCS, separately in healthy and infected animals. The aims of this study, therefore, were: i) to estimate the heritability of SCS, according to whether the animals were healthy or infected; and ii) to estimate the phenotypic and genetic correlation between the bacteria negative SCS (i.e., healthy animals) and the bacteria positive SCS (i.e., infected animals).

Material and methods

Dataset. Data consisted of 8,843 test-day records from 2,047 lactations of 1,120 ewes. Data were collected at approximately 1-mo intervals in 4 Valle del Belice flocks between 2004

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and 2007. At the same time, milk samples were collected aseptically from each animal for bacteriological analyses. Ewes were considered infected if > 5 colony forming units (CFU) per 10µl of milk of one species of bacteria were isolated, and the data used in this study were the apparent presence or absence of infection for each milk sample. The pedigree file included 1,603 animals. The data was also divided in two sub-datasets: one sub-dataset including records with SCC information and bacteria positive response (2,866 test-day records from 1,263 lactations of 805 ewes), and the other one including records with SCC information and bacteria negative response (5,977 test-day records from 1,805 lactations of 1,062 ewes). Of the 1,120 ewes of the original data, 744 were included in both sub-datasets.

Statistical analyses. The test-day traits analyzed as response variables were SCS, obtained after log-transformation of test-day SCC, using a base 2 logarithmic function: SCS = $\log_2$ (SCC/100) + 3 (Ali and Shook (1980)). Variance components and genetic parameters for SCS (whole dataset as well as bacteria negative and positive subsets) were estimated using ASReml (Gilmour, A.R., Gogel, B.J., Cullis, B.R. et al. (2002)), with the following repeatability test-day animal model:

$$y_{ijklmn} = \mu + FTD_i + YPS_j + P_k + LS_l + \beta_1 DIM_{ijklmn} + \beta_2 \exp\left(-0.05 * DIM_{ijklmn}\right) + A_m + PE_m + PE_{km} + e_{ijklmn}$$

where y_{ijklmn} was the SCS test-day measurement; μ was the population mean; FTD_i was the random effect of flock by test-day interaction *i* (91 levels); YPS_j was the fixed effect of year by season of lambing interaction *j* (6 levels); P_k was the fixed effect of the parity (3 levels, 1, 2, and ≥3); LS_l was the fixed effect of litter size class *l* (2 levels, single or multiple lambs); DIM_{ijklmn} and $\exp\left(-0.05 * DIM_{ijklmn}\right)$ were two covariates used to model the shape of lactation curves (Wilmink (1987)); A_m was the random additive genetic effect of the individual *m* (1,603 levels); PE_m was the general random permanent environmental effect of ewe *m* across lactations (1,120 levels); PE_{km} was the random permanent environmental effect on the individual *m* within parity class *k* (1,662 levels); e_{ijklmn} was the random residual effect. The same model was used for the analysis of the two sub-datasets.

Phenotypic and genetic correlations between SCS in the bacteria negative and positive subsets were estimated using bivariate analyses. Given the data structure, i.e. non-contemporaneous bacteria negative and positive SCS observations for any individual, the environmental covariance between the two traits was assumed to be zero and not estimated when the genetic correlation was estimated. However, to estimate an approximate phenotypic correlation, the dataset was restructured and reduced to adjacent pairs of bacteria negative and positive SCS data, i.e. the bacteria negative and positive SCS observations closest together within a lactation were used. The same fixed and random effects, as previously described, were fitted in the model.

Results and discussion

Arithmetic means, standard deviations and range of SCC and SCS test-day traits are given in Table 1. Although the arithmetic mean SCC for infected animals was approximately 3-fold higher than that for uninfected animals, the ranges of SCC for uninfected and infected animals were similar. The difference between bacteria positive and bacteria negative SCC may have been higher if SCC and infection status were considered per udder half. However,

we only had information at the animal level (summarizing the whole udder); therefore a dilution effect due to mixing of milk having high SCC coming from infected glands with low SCC from a healthy gland has to be considered.

Table 1 - Descriptive statistics of SCC and SCS traits

	N. of records	Mean	SD	Range
Whole data SCC ($\times 10^3$ cells/ml)	8,843	1,812	4,150	13 - 31,268
Whole data SCS		5.01	2.37	0.06 - 11.29
Bacteria negative SCC ($\times 10^3$ cells/ml)	5,977	1,077	3,084	13 - 29,368
Bacteria negative SCS		4.34	2.06	0.06 - 11.20
Bacteria positive SCC ($\times 10^3$ cells/ml)	2,866	3,346	5,462	16 - 31,268
Bacteria positive SCS		6.42	2.36	0.36 - 11.29

Phenotypic variances after adjustment for systematic environmental effects, the heritabilities, and the repeatabilities within and across lactations for SCS traits are presented in Table 2. The heritability estimate for SCS was 0.09, which is generally in the range reported in literature for repeatability test-day models (e.g., Baro, J.A., Carriedo, J.A., and San Primitivo F. (1994); Hamann, H., Horstick, A., Wessels, A.et al. (2004)).

Table 2 - Phenotypic variance (σ^2_p), heritability (h^2) and repeatability within (r_w) and across (r_a) lactations (± SE) for SCS traits

	$\sigma^2 p$	$h^2 \pm$ SE	$r_w \pm$ SE	$r_a \pm$ SE
Whole data SCS	5.467	0.09 ± 0.04	0.29 ± 0.04	0.33 ± 0.02
Bacteria negative SCS	2.225	0.10 ± 0.06	0.21 ± 0.04	0.30 ± 0.03
Bacteria positive SCS	5.573	0.03 ± 0.03	0.20 ± 0.05	0.31 ± 0.04

Heritability estimates for bacteria negative and bacteria positive SCS were respectively 0.10 and 0.03. This genetic difference could be due to the different sub-datasets (i.e., different animals and different number of records) used for the analysis. Therefore, an analysis was carried out in which only the animals present in both sub-datasets were considered. However, this analysis had little effect on the estimated heritabilities. Repeatability estimates within lactation ranged between 0.20 and 0.29, and were in the range (0.22 to 0.38) generally reported in literature for sheep (e.g., El-Saied, U.M., Carriedo, J.A., and San Primitivo, F. (1998); Hamann, H., Horstick, A., Wessels, A. et al. (2004)). Repeatability estimates across lactations ranged between 0.30 and 0.33, and were higher than the within-lactation values. Repeatabilities were similar for bacteria negative and bacteria positive SCS.

Table 3 - Genetic and phenotypic correlations (± SE) between baseline SCS and SCS response

		Bacteria positive SCS
Bacteria negative SCS	Genetic correlation:	0.62 ± 0.06
	Approximated phenotypic correlation:	0.19 ± 0.02

The phenotypic and genetic correlations between bacteria negative and bacteria positive SCS

are presented in Table 3. The approximated phenotypic correlation was 0.19 (s.e. 0.02), whereas the genetic correlation was 0.62 (s.e. 0.06). Whilst this genetic correlation is moderate and positive, it indicates that ewes having a high SCS when healthy are also more likely to have a greater SCS when infected. Moreover, it suggests it may be more appropriate to consider bacteria negative and bacteria positive SCS as different traits. Our result was similar to the one reported by Madsen, P., Shariati, M.M., and Ødegård, J. (2008) in Danish Holsteins, but higher than the 0.13 reported by Boettcher, P.J., Caraviello, D., and Gianola D. (2007) in US Holsteins. It might be hypothesized that ewes with high bacteria negative SCS have a higher capacity to react, in terms of increase in SCS, in response to an infection. However, a somewhat different interpretation is also possible. The bacteria positive SCS actually consists of the baseline SCS (i.e. the SCS ewes would have had in the absence of infection) along with the true response to infection. Therefore, it is likely that the positive genetic correlation is picking up the baseline that is contributing to both measures, with the true response (i.e. the extra) SCS possibly being only weakly correlated. The sum of the two results in a trait that is genetically correlated with bacteria negative SCS, but has a low phenotypic correlation (0.19).

Conclusion

Our results suggested that bacteria negative and bacteria positive SCS may be partially independent traits, confirming that SCC from healthy and infected animals should be analyzed separately, wherever possible. The genetic correlation of liability to infection with either bacteria negative SCS or bacteria positive SCS will be estimated as our next step, to investigate whether the approach of selecting animals for decreased SCS, particularly bacteria negative SCS, will help to reduce the prevalence of mastitis.

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