Livestock Science 151 (2013) 134–139

Contents lists available at SciVerse ScienceDirect

Livestock Science

journal homepage: www.elsevier.com/locate/livsci

Estimation of genetic and phenotypic parameters for bacteriological status of the udder, somatic cell score, and milk yield in dairy sheep using a threshold animal model

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ARTICLE INFO

Article history:
Received 29 May 2012
Received in revised form
14 November 2012
Accepted 20 November 2012

Keywords:
Infection status
Somatic cell score
Milk yield
Sheep
Threshold model

ABSTRACT

The objective of this study was to estimate the genetic parameters for infection status (INF), as indicator of mastitis, SCS (i.e., log-transformed SCC), and milk yield (MY), by using a Gibbs sampling algorithm. The data comprised 17,843 test-day records of 2040 ewes. The pedigree file included 2948 animals. A bivariate variance component analysis was performed using the TM software. Fixed effects considered in the analysis were litter size, parity, flock by test-day interaction, year by season of lambing interaction, and stage of lactation; whereas the animal, and the permanent environmental effect within and across lactations were considered as random as well as the error. Flat priors were used for both fixed effects and variance components. Parameters were drawn from the posterior conditional distributions. The posterior means of heritability for MY, SCS and INF were equal to 0.14, 0.09, and 0.09, respectively; whereas the repeatability within lactation was around 0.30 for the three traits, and ranged between 0.29 and 0.41 across lactations. The genetic correlation between INF and SCS was equal to 0.93, suggesting that selection for low SCS would also lead to a reduced incidence of mastitis. On the other hand, the positive and moderate genetic correlation between mastitis and milk yield (0.59) confirms the antagonistic association between udder health and milk yield. Therefore, in breeding programs that emphasize milk yield, the unfavorable genetic correlation between milk yield and mastitis, may result in an increased incidence of the latter.

1. Introduction

Mastitis is one of the major diseases in dairy ewes and cows, which leads to economic losses, mainly due to discarded milk, reduced milk production and quality, early culling, and increased health care costs in both dairy ewes (Leitner et al., 2003, 2004) and cows (i.e., Bennett et al., 1999; Wellenberg et al., 2002). Mastitis has therefore motivated extensive research towards improved udder sanitation and mastitis control (El-Saied et al., 1998). However, genetic evaluation of mastitis is particularly difficult because of the low heritability and the categorical nature of the trait. As a consequence, correlated traits have been suggested to increase the efficiency of selection for mastitis resistance. In particular, SCC has been promoted as an indirect method of predicting mammary infections (Boettcher, 2005) and as a selection criterion to improve mastitis resistance (Gonzalo et al., 2003). It has been indeed demonstrated that mastitis causes an increase in SCC in small ruminants (Leitner et al., 2004; Sanchez et al., 1999; Zeng et al., 1997) and cattle

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http://dx.doi.org/10.1016/j.livsci.2012.11.014

(e.g., Heringstad et al., 2006; Olde Riekerink et al., 2007). Moreover, mastitis data are difficult and expensive to collect, whereas SCC is currently recorded in several milk recording schemes in both dairy sheep (Astruc et al., 2004) and cattle (Boettcher, 2005).

Estimates of genetic correlations between infection status (i.e., mastitis), SCC, and production traits are essential for the calculation of optimal selection indices. However, one problem in this sense is that the categorical nature of mastitis is usually ignored and genetic parameters have been often estimated using methodologies developed to analyze normally distributed traits, which is considered to be not optimal for categorical traits (e.g., Gianola and Foulley, 1983). The definition of mastitis as a binary trait, however, does not fully use all information provided by the data, because some animals can have more than one case of mastitis (Hinrichs et al., 2005). Rekaya et al. (1998) suggested the development of test-day models for longitudinal binary response for the analysis of mastitis field data in cattle. Test-day models should in fact allow considering the dynamic nature of mastitis, which is usually ignored by lactation models.

Whereas several estimates of genetic correlation between mastitis and SCC and production traits are available in cattle (Carlen et al., 2004; Koivula et al., 2005; Rupp and Boichard, 2003); such an information is lacking in sheep. Therefore, the objective of this study was to estimate the genetic parameters for infection status (INF), as indicator of mastitis, SCS (i.e., log-transformed SCC), and milk yield (MY), by using a test-day model implemented with a Gibbs sampling algorithm.

2. Materials and methods

All procedures involving animals were performed according to the principles and specific guidelines on animal care and welfare as required by Italian law.

2.1. Data and trait definitions

The original data consisted of 17,923 test-day records from 3406 lactations of 2046 ewes. Test-day records for MY and SCC were collected at approximately 1-month intervals, following an A4 recording scheme (ICAR, 2003), by the University of Palermo in four Valle del Belice flocks between 2004 and 2011. At milking time, cases of clinical mastitis were identified by the technicians and test-day weights and milk samples of those ewes were not considered. Clinical mastitis was reported for the evident signs of udder inflammation, or abnormal milk, or both.

All ewes were milked twice daily, and the milk of both daily milkings was analyzed; SCC were calculated as the weighted average of the morning and evening milking, where weighting is according to the corresponding milk yield. SCC was log-transformed to SCS, using Ali and Shook (1980) formula.

At the same time, milk samples were collected aseptically from each animal for bacteriological analyses, which were performed by conventional techniques, on 5% sheep blood agar plates, incubated at 37 °C, and examined after 10–24 h and 36–48 h incubation. Several bacteriological colonies were considered, mainly of genera *Staphylococcus*, *Streptococcus*, *Pasteurella*, *Escherichia*, and *Pseudomonas*. The information on the presence/absence of mastitis-causing pathogens was used to create an infection status variable, i.e. 0 if no pathogens were isolated, 1 otherwise, without considering the different pathogenicity of these bacteria. Ewes were considered infected if >5 colony forming units (CFU) per 10 μl of milk of one species of bacteria were isolated.

All test-day records used in the analysis were required to have information regarding MY, SCS, and INF. After editing, the data comprised 17,843 test-day records from 3000 lactations of 2040 ewes. The average number of test-day records per ewe per lactation was 4.84 ± 3.36. The pedigree file included 2948 animals. In addition to the 2040 animals with records, 158 sires and 750 dams were included.

2.2. Model

Bivariate variance component analyses were performed, in which the binary variable (i.e., INF) was analyzed with each of the two continuous traits (i.e., SCS and MY). For the infection status, the threshold concept was applied. The threshold model postulates an underlying continuous random variable, liability (λ), such that an observed binary response takes the value of 1 if λ is larger than a fixed threshold (τ), and 0 otherwise. Given the mean and the variance, liability was assumed to be normally distributed. Since with binary data the threshold (τ) and the residual variance (σ^2_e) are not identifiable, these parameters are usually set to arbitrary values: $\tau=0$ such that INF=1 if $\tau>0$ and 0 otherwise and $\sigma^2_e=1$. The model was formulated in a Bayesian context, in which the data vector was augmented with the unobservable liabilities. Liabilities were later integrated out of the joint posterior distribution, using Gibbs sampling.

The model for the observable continuous traits (either MY or SCS), denoted as y_1 and the augmented underlying liability for the INF, denoted as y_2, was as follows:

$$\begin{bmatrix} y_1 \\ y_2 \end{bmatrix} = \begin{bmatrix} X_1 & 0 \\ 0 & X_2 \end{bmatrix} \begin{bmatrix} \beta_1 \\ \beta_2 \end{bmatrix} + \begin{bmatrix} Z_{\text{pew1}} & 0 \\ 0 & Z_{\text{pew2}} \end{bmatrix} \begin{bmatrix} \text{pe}_{\text{w1}} \\ \text{pe}_{\text{w2}} \end{bmatrix} + + \begin{bmatrix} Z_{\text{pea1}} & 0 \\ 0 & Z_{\text{pea2}} \end{bmatrix} \begin{bmatrix} \text{pe}_{\text{a1}} \\ \text{pe}_{\text{a2}} \end{bmatrix} + \begin{bmatrix} Z_1 & 0 \\ 0 & Z_2 \end{bmatrix} \begin{bmatrix} a_1 \\ a_2 \end{bmatrix} + \begin{bmatrix} e_1 \\ e_2 \end{bmatrix}$$

where X_1 and X_2 are the design matrices relating fixed effects in β_1 and β_2 to y_1 and y_2, respectively. The β's included effects of litter size (2 levels: single or multiple born lambs), parity (three levels: first, second, and third or higher parity), flock by test-day interaction (187 levels), year by season of lambing interaction (14 levels), where the season of lambing was coded as 1 if a ewe gave birth in the period January through June, otherwise it was coded as 2 (according to Riggio et al., 2007), and stage of lactation (9 levels, each of thirty days in milk, from weaning – i.e.,~30 days after lambing – to the end of lactation). The design matrices Z_{pew1} and Z_{pew2} are related to the random permanent environmental effect within lactation (indicated as pe_{w1} and pe_{w2}, respectively),

whereas the Z_{pea1} and Z_{pea2}, to the random permanent environmental effect across lactations (indicated as pe_{a1} and pe_{a2}, respectively). The random animal effects are included in a_1 and a_2, and linked to the appropriate records via the design matrices Z_1 and Z_2, whereas the residual effects are included in e_1 and e_2.

To complete the Bayesian specification of the model, priors distribution for all the parameters model have to be declared. For random effects (both permanent environmental and animal effects), a multivariate normal prior distribution with zero mean and appropriate variance–covariance matrices was used. Flat priors were used for both fixed effects and variance components. Parameters were drawn from the posterior conditional distributions using Gibbs sampling, as implemented in the TM software (Legarra et al., 2008, http://snp.toulouse.inra.fr/~alegarra/). CODA package of the R language/environment (Plummer et al., 2006) was used to assess the convergence by visual inspection of the trace plots (a plot of the iteration number against the value of the draw of the parameter at each iteration), and based on that a chain of 700,000 iterations was run for each trait, with a burn-in of 100,000 rounds, keeping every 100th sample for inference of posterior features (6000 samples). Heritabilities were computed in the usual way, as the ratio between the additive genetic and the phenotypic variances; whereas, repeatabilities within and across lactations were computed as the ratio between the additive genetic and the permanent environment (within and across lactations, respectively) variance components and the phenotypic variance.

3. Results and discussion

This paper has estimated the genetic relationship between the infection status (i.e., mastitis) and both milk yield and SCS in Valle del Belice dairy sheep. Given that in case of clinical mastitis data were not collected, our infection status trait is most likely an indicator of subclinical mastitis. On the other hand, it is also possible that the bacteriological analysis reveals the presence of a clinical mastitis causing pathogen, even if the clinical symptoms are not evident yet.

To our knowledge, information regarding the genetic relationship of mastitis with both SCC and milk production traits is lacking in sheep. Therefore, results were mostly compared with those reported for dairy cattle. Moreover, estimates available for milk production traits in sheep literature are mostly obtained with frequentist approaches.

Mastitis is one of the greatest problems affecting commercial milk production. In the present study, the frequency of infection (i.e., bacteriological status) was 42%, i.e. the percentage of test-days coded as mastitis days. The infection frequency in this study was in agreement with those reported by Las Heras et al. (1999) and Bergonier et al. (2003) for dairy ewes and with those reported by Fourichon et al. (2001) for dairy cattle, but higher than those reported by Heringstad et al. (1999, 2001, 2003) (from 17% to 30%) in cattle. However, it should be noted that the results from Heringstad et al. were only based on first lactations data.

Descriptive statistics for the continuous traits considered (i.e., MY and SCS) both considering the whole sample and according to the health status (i.e. infected or not infected) are reported in Table 1. The daily average MY was 1289 g and 1367 g for infected and not infected animals, respectively, whereas the mean SCS was 6.04 and 4.27, respectively. The mean SCS for both groups (i.e., infected and not infected) were similar to those reported by Riggio et al. (2010) in the same breed and by Ariznabarreta et al. (2002) in Churra sheep and Leitner et al. (2003) in Israeli-Assaf and Awassi sheep. Considering the whole data, the mean SCS was higher than the value of 3.34 reported by Barillet et al. (2001) in the Lacaune breed and the 3.80 reported by Serrano et al. (2003) in the Manchega breed, using a lactation mean. However, this value is lower than those reported in literature for test-day models, ranging from 5.26 to 12.1 (i.e., El-Saied et al., 1998; Othmane et al., 2002).

In order to illustrate the behavior of the Gibbs sampler, Fig. 1 shows traces of the sample sequences obtained for heritability of MY, SCS, and INF. The Gibbs sampler seemed to visit the effective parameter space at random, even before the burn-in period.

Posterior means and the standard deviations of the variance components, heritabilities, and repeatabilities are given in Table 2. The posterior means of heritability for MY, SCS and INF were equal to 0.14, 0.09, and 0.09 respectively; whereas repeatability within lactation was around to 0.30 for the three traits, and ranged between 0.29 and 0.41 across lactations. The posterior mean estimate of heritability for MY estimated in this study was lower than the value of 0.20 reported by Ugarte et al. (1996) in blond-faced Laxta sheep, using Bayesian approach, and lower than those obtained for other sheep breeds with a frequentist approach, which are between 0.15 and 0.24 (Barillet et al., 2001; El-Saied et al., 1998; Othmane et al., 2002). No heritabilities estimated with Bayesian approach were found in literature for SCS for sheep. However, the heritability estimate for SCS in this study falls within the range (0.04–0.16) reported in the literature for sheep (e.g., Barillet et al., 2001; Baro et al., 1994; Hamann et al., 2004). Using the same Bayesian approach, Penasa et al. (2010) obtained a value of 0.05 for

Table 1
Descriptive statistics for milk yield and somatic cell score both considering the whole sample and according to the health status (infected or not infected).

	N	Milk yield		Somatic cell score	
		Mean ± SD	Min–max	Mean ± SD	Min–max
Infected	7452	1289 ± 545	90–3943	6.04 ± 2.47	0.39–11.29
Not infected	10,391	1367 ± 564	112–4140	4.27 ± 2.13	0.08–11.26
Total	17,843	1224 ± 574	90–4140	5.0 ± 2.40	0.08–11.29

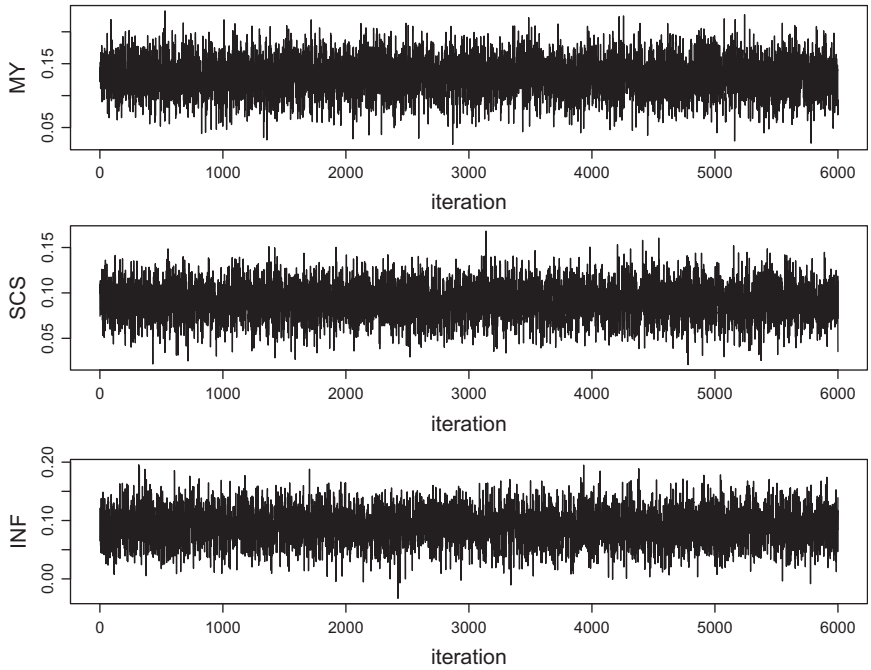

Fig. 1. Traceplots of the iteration numbers against the value of the draw of the heritability for MY, SCS and INF at each iteration.

Table 2

Posterior means and standard deviation of additive genetic (σ^2_a), within $\left(\sigma^2_{pew}\right)$ and across $\left(\sigma^2_{pea}\right)$ lactation permanent environmental variances, heritability $\left(h^2\right)$ and repeatability within (r_w) and across lactation (r_a) for the traits considered.

	σ^2_a	σ^2_{pew}	σ^2_{pea}	h^2	r_w	r_a
MY	$24{,}309 \pm 6068$	$32{,}294 \pm 4939$	$49{,}049 \pm 2712$	0.14 ± 0.03	0.32 ± 0.02	0.41 ± 0.04
SCS	0.48 ± 0.13	1.14 ± 0.12	1.16 ± 0.07	0.09 ± 0.02	0.30 ± 0.02	0.30 ± 0.02
INF	0.18 ± 0.05	0.38 ± 0.05	0.39 ± 0.04	0.09 ± 0.03	0.29 ± 0.02	0.29 ± 0.03

MY: milk yield; SCS: somatic cell score; INF: infection status.

the SCS heritability in a small autochthonous dairy cattle breed; whereas, other authors found higher values in German Holstein (Hinrichs et al., 2005) and Danish Holstein breed (Madsen and Odegard, 2006).

The posterior mean estimate of heritability for liability to mastitis (i.e., infection status) was equal to 0.09, which is the same as the estimate reported by Riggio et al. (2010) in a previous study on the same breed, using a threshold animal model, assuming a probit link function. To our knowledge, no other estimates for either subclinical or clinical mastitis are reported in literature for sheep. In cattle, heritability estimates of clinical mastitis tend to vary based on the type of data and method of analysis. In a review, Heringstad et al. (2000) concluded that most estimates of heritability for this trait from traditional linear methods on the observable scale range from 0.001 to 0.06, with most values in the interval 0.02–0.03, whereas heritability of liability to clinical mastitis from threshold models range from 0.06 to 0.12. The authors also reported that designed field studies (e.g., Lyons et al., 1991; Uribe et al., 1995) have given somewhat higher estimates of heritability, due most likely to a more accurate data recording. Moreover, it should be noted that heritability estimates of all-or-none traits are functions of incidence, and differences in estimates between different studies may be caused by real differences between populations and countries, but also be due to somewhat different definitions of mastitis traits (Heringstad et al., 2000).

One of the reasons why SCS is usually preferred to the direct trait in selection programs for mastitis resistance is that the former has usually higher heritability. However, in our study the heritability estimate for the infection status was the same as the one for SCS (0.09). Nevertheless, it is worth to mention that compared to collecting information on infection status, it is easier, cheaper, and less time-demanding for farmers to collect information on SCC/SCS, as this can be regularly recorded during milk recording at low cost (Riggio, 2012).

Despite that the literature in dairy sheep is limited compared with dairy cattle, some studies showed that the selection for mastitis resistance based on SCS is feasible (e.g., Barillet et al., 2001). When including mastitis in the breeding goal, it is useful to know what measure of the trait is most appropriate and its relationship to the primary production traits and indicator traits in the population.

Table 3
Posterior genetic and phenotypic correlations between infection status (INF) and milk production traits (MY and SCS).

	Genetic correlation	Phenotypic correlation
INF–MY	0.59 ± 0.31	0.14 ± 0.02
INF–SCS	0.93 ± 0.06	0.46 ± 0.01

Table 3 shows the posterior means and standard deviations for phenotypic and genetic correlations between INF and milk production traits (i.e., MY and SCS). The genetic correlation between INF and SCS was equal to 0.93, suggesting that selection for decreased SCS would lead to a reduced incidence of mastitis as well. This result is higher than the value of 0.51 reported by Riggio et al. (2010) in a previous study. However, these authors were investigating the genetic correlation between bacteria negative SCS and the infection status, and they suggested that animals with lower SCS, assessed when apparently not infected, are genetically less likely to be infected (across all time points). In cattle, most estimates of genetic correlation between SCS and mastitis range from 0.50 to 0.85 (Carlen et al., 2004; Hinrichs et al., 2005; Koivula et al., 2005; Rupp and Boichard, 2003); whereas, the genetic correlation between SCS and bacteriological infection was estimated to be near unity (Weller et al., 1992) in cattle, being this very similar to our result. These results, therefore, seem to indicate that SCS and subclinical infections are essentially the same trait in both cattle and sheep.

Heringstad et al. (2003) estimated different genetic correlations between different stages of lactation (varying between -0.19 and 0.98) in cattle. However, in other studies (e.g., Hinrichs et al., 2005; Kadarmideen et al., 2000; Rekaya et al., 1998) mastitis was treated as the same trait at each day of lactation in all lactations, and this approach was also considered in the current study.

The positive and moderate genetic correlation between mastitis and milk yield (0.59), confirms the antagonistic association between udder health and production traits (e.g., Carlen et al., 2004; Heringstad et al., 2005; Negussie et al., 2008). Therefore, in breeding programs that emphasize milk yield, which is still the most important selection criterion in most dairy sheep breeds, the unfavorable genetic correlation between milk yield and mastitis, may result in an increased incidence of the latter.

4. Conclusions

Mastitis is still one of the major problems and causes of considerable economic losses in the dairy sector both in cattle and sheep. The genetic correlation estimated in this study between infection status of the udder (i.e., mastitis) and SCS was high, suggesting that selection for mastitis resistance through selection for SCS can be feasible. Moreover, the unfavorable genetic correlation between mastitis and milk yield would lead to a further decrease in udder health if mastitis is ignored in breeding programs.

Conflict of interest statement

The authors declare no conflict of interest.

References

Ali, A.K.A., Shook, G.E., 1980. An optimum transformation for somatic cell concentration in milk. J. Dairy Sci. 63, 487–490.

Ariznabarreta, A., Gonzalo, C., San Primitivo, F., 2002. Microbiological quality and somatic cell count of ewe milk with special reference to staphylococci. J. Dairy Sci. 85, 1370–1375.

Astruc, J.M., Barillet, F., Fioretti, M., Gabina, D., Gootwine, E., Mavrogenis, A.P., Romberg, F.J., Sanna, S.R., Stefanake, E., 2004. Report of the working group on milk recording of sheep. In: Proceedings of the 34th Biennial Session of the International Committee for Animal Recording (ICAR), vol. 113. EAAP Publication, Sousse, Tunisia, pp. 315–322 .

Barillet, F., Rupp, R., Mignon-Grasteau, S., Astruc, J.M., Jacquin, M., 2001. Genetic analysis of mastitis resistance and somatic cell score in French Lacaune dairy sheep. Genet. Sel. Evol. 33, 397–415.

Baro, J.A., Carriedo, J.A., San Primitivo, F., 1994. Genetic parameters of test day measures for somatic cell count, milk yield and protein percentage of milking ewes. J. Dairy Sci. 77, 2658–2662.

Bennett, R.M., Christansen, K., Clifton-Hadlay, R., 1999. Preliminary estimates of the direct costs associated with endemic diseases of livestock in Great Britain. Prev. Vet. Med. 39, 155–171.

Bergonier, D., De Crémoux, R., Rupp, R., Lagriffoul, G., Berthelot, X., 2003. Mastitis of dairy small ruminants. Vet. Res. 34, 689–716.

Boettcher, P., 2005. Breeding for improvement of functional traits in dairy cattle. Ital. J. Anim. Sci. 4, 7–16.

Carlen, E., Strandberg, E., Roth, A., 2004. Genetic parameters for clinical mastitis, somatic cell score, and production in the first three lactations of Swedish Holstein cows. J. Dairy Sci. 87, 3062–3070.

El-Saied, U.M., Carriedo, J.A., San Primitivo, F., 1998. Heritability of test day somatic cell counts and its relationship with milk yield and protein percentage in dairy ewes. J. Dairy Sci. 81, 2956–2961.

Fourichon, C., Seegers, H., Beaudeau, F., Verfaille, L., Bareille, N., 2001. Health-control costs in dairy farming systems in western France. Livest. Prod. Sci. 68, 141–156.

Gianola, D., Foulley, J.L., 1983. Sire evaluation for ordered categorical data with a threshold model. Genet. Sel. Evol. 15, 201–224.

Gonzalo, C., Ariznabarreta, A., Othmane, M.H., Carriedo, J.A., De La Fuente, L.F., San Primitivo, F., 2003. Genetic parameters of somatic cell count in dairy sheep considering the type of mammary pathogen effect. J. Anim. Breed. Genet. 120, 282–287.

Hamann, H., Horstick, A., Wessels, A., Distl, O., 2004. Estimation of genetic parameters for test day milk production, somatic cell score and litter size at birth in East Friesian ewes. Livest. Prod. Sci. 87, 153–160.

Heringstad, B., Klemetsdal, G., Ruane, J., 1999. Clinical mastitis in Norwegian cattle: frequency, variance components, and genetic correlation with protein yield. J. Dairy Sci. 82, 1325–1330.

Heringstad, B., Klemetsdal, G., Ruane, J., 2000. Selection for mastitis resistance in dairy cattle—a review with focus on the situation in the Nordic countries. Livest. Prod. Sci. 64, 95–106.

Heringstad, B., Rekaya, R., Gianola, D., Klemetsdal, G., Weigel, K.A., 2001. Bayesian analysis of liability of clinical mastitis in Norwegian cattle with a threshold model: effects of data sampling method and model specification. J. Dairy Sci. 84, 2337–2346.

Heringstad, B., Rekaya, R., Gianola, D., Klemetsdal, G., Weigel, K.A., 2003. Bivariate analysis of liability to clinical mastitis and to culling in first lactation cows. J. Dairy Sci. 86, 653–660.

Heringstad, B., Chang, Y.M., Gianola, D., Klemetsdal, G., 2005. Genetic associations between susceptibility to clinical mastitis and protein yield in Norwegian dairy cattle. J. Dairy Sci. 88, 1509–1514.

Heringstad, B., Gianola, D., Chang, Y.M., Ødegård, J., Klemetsdal, G., 2006. Genetic associations between clinical mastitis and somatic cell score in early first-lactation cows. J. Dairy Sci. 89, 2236–2244.

Hinrichs, D., Stamer, E., Junge, W., Kalm, E., 2005. Genetic analyses of mastitis data using animal threshold models and genetic correlation with production traits. J. Dairy Sci. 88, 2260–2268.

ICAR, International Committee for Animal Recording, 2003. International Regulations for Milk Recording in Sheep. Department Génétique ei Contrôle des Performances, Institut de l'élevage, Paris.

Kadarmideen, H.N., Thompson, R., Simm, G., 2000. Linear and threshold model genetic parameters for disease, fertility and milk production in dairy cattle. Anim. Sci. 71, 411–419.

Koivula, M., Mäntysaari, E.A., Negussie, E., Serenius, T., 2005. Genetic and phenotypic relationships among milk yield and somatic cell count before and after clinical mastitis. J. Dairy Sci. 88, 827–833.

Las Heras, A., Domínguez, L., López, I., Fernández-Garayzábal, J.F., 1999. Outbreak of acute ovine mastitis associated with *Pseudomonas aeruginosa* infection. Vet. Rec. 145, 111–112.

Legarra, A., Varona, L., López de Maturana, E., 2008. Manual TM. 〈http://snp.toulouse.inra.fr/~alegarra/〉.

Leitner, G., Chaffer, M., Caraso, Y., Ezra, E., Kababea, D., Winkler, M., Saran, A., 2003. Udder infection and milk somatic cell count, NAGase activity and milk composition—fat , protein and lactose—in Israeli Assaf and Awassi sheep. Small Rumin. Res. 49, 157–164.

Leitner, G., Chaffer, M., Shamay, A., Shapiro, F., Merin, U., Ezra, E., Saran, A., Silanikove, N., 2004. Changes in milk composition as affected by subclinical mastitis in sheep. J. Dairy Sci. 87, 46–52.

Lyons, D.T., Freeman, A.E., Kuck, A.L., 1991. Genetics of health traits in Holstein cattle. J. Dairy Sci. 74, 1092–1100.

Madsen, P., Odegard, J., 2006. Analysis of test day somatic cell score using a liability-normal mixture model. In: Proceedings of the 2006 Interbull Meeting Kuopio. June 4–6 , 2006, Finland.

Negussie, E., Strandén, I., Mäntysaari, E.A., 2008. Genetic association of clinical mastitis with test-day somatic cell score and milk yield during first lactation of Finnish Ayrshire cows. J. Dairy Sci. 91, 1189–1197.

Olde Riekerink, R.G.M., Barkema, H.W., Veenstra, W., Berg, F.E., Stryhn, H., Zadoks, R.N., 2007. Somatic cell count during and between milkings. J. Dairy Sci. 90, 3733–3741.

Othmane, M.H., De La Fuente, L.F., Carriedo, J.A., San Primitivo, F., 2002. Heritability and genetic correlations of test day milk yield and composition, individual laboratory cheese yield, and somatic cell count for dairy ewes. J. Dairy Sci. 85, 2692–2698.

Penasa, M., Cecchinato, A., Battagin, M., De Marchi, M., Pretto, D., Cassandro, M., 2010. Bayesian inference of genetic parameters for test-day milk yield, milk quality traits, and somatic cell score in Burlina cows. J. Appl. Genet. 51, 489–495.

Plummer, M., Best, N., Cowles, K., Vines, K., 2006. CODA: Convergence Diagnosis and Output Analysis for MCMC. R News 6, 7–11.

Rekaya, R., Rodriguez-Zas, S.L., Gianola, D., Schook, G.E., 1998. Test day models for longitudinal binary responses: an application to mastitis in Holsteins. In: Proceedings of the 49th Meeting of the European Association for Animal Production. Warsaw, Poland.

Riggio, V., Finocchiaro, R., van Kaam, J.B.C.H.M., Portolano, B., Bovenhuis, H., 2007. Genetic parameters for milk somatic cell score and relationships with production traits in primiparous dairy sheep. J. Dairy Sci. 90, 1998–2003.

Riggio, V., Portolano, B., Bovenhuis, H., Bishop, S.C., 2010. Genetic parameters for somatic cell score according to udder infection status in Valle del Belice dairy sheep and impact of imperfect diagnosis of infection. Gen. Sel. Evol. 42, 30.

Riggio, V., 2012. Genetic Aspects of Somatic Cell Count and Udder Health in the Italian Valle del Belice Dairy Sheep. Ph.D. Thesis. Wageningen University.

Rupp, R., Boichard, D., 2003. Genetics of resistance to mastitis in dairy cattle. Vet. Res. 34, 671–688.

Sanchez, A., Contreras, A., Corrales, J.C., 1999. Parity as a risk factor for caprine subclinical intramammary infections. Small Rumin. Res. 31, 197–201.

Serrano, M., Pérez-Guzmán, M.D., Montoro, V., Jurado, J.J., 2003. Genetic analysis of somatic cell count and milk traits in Manchega ewes. Mean lactation and test-day approaches. Livest. Prod. Sci. 84, 1–10.

Ugarte, E., Urarte, E., Arrese, F., Arranz, J., Silio, L., Rodriguez, C., 1996. Genetic parameters and trends for milk production of Blond-faced Latxa sheep using Bayesian analysis. J. Dairy Sci. 79, 2268–2277.

Uribe, H.A., Kennedy, B.W., Martin, S.W., Kelton, D.F., 1995. Genetic parameters for common health disorders of Holstein cows. J. Dairy Sci. 78, 421–430.

Wellenberg, G.J., van der Poel, W.H., van Oirschot, J.T., 2002. Viral infections and bovine mastitis: a review. Vet. Microbiol. 88, 27–45.

Weller, J.I., Saran, A., Zeliger, Y., 1992. Genetic and environmental relationships among somatic cell count, bacterial infection, and clinical mastitis. J. Dairy Sci. 75, 2532–2540.

Zeng, S.S., Escobar, E.N., Popham, T., 1997. Daily variation in somatic cell count, composition and production of Alpine dairy goats. Small Rumin. Res. 26, 253–260.