Combination of Test Day Somatic Cell Count and Incidence of Mastitis for the Genetic Evaluation of Udder Health

T.C. Pritchard, R. Mrode, M.P. Coffey and E. Wall
SAC, Roslin Institute Building, Easter Bush, Midlothian, EH25 9RG U.K.
E-mail: tracey.pritchard@sac.ac.uk

Abstract

Genetic parameters were estimated in a joint analysis of somatic cell count (SCC) and mastitis (MAS) in Holstein/Friesian cows for the first three parities using a random regression model (RRM). There were 67,175, 30,617, and 16,366 cows with records for SCC in parities 1, 2 and 3, respectively. The incidence of MAS was 14, 20, and 25% in parities 1, 2, and 3, respectively. Average daily heritability estimates (h^2) were 0.11, 0.14, and 0.15 for SCC in parities 1, 2, and 3, respectively. Corresponding estimates were 0.05, 0.07, and 0.09 for mastitis as a binary trait (MAS) and 0.06, 0.07, and 0.12 as a count trait (NMAS). Genetic correlations (r_g) between parities were different to one; thus indicating that separate parities should be treated as separate traits. Genetic correlations between SCC at days in milk (DIM) and MAS within parities were generally medium in value, varying from 0.41 to 0.74. It is intended that the new parameters will be used in setting up a national evaluation system for the joint analysis of SCC and MAS.

1. Introduction

Mastitis is a recurrent and costly disease, affecting dairy farms worldwide, impacting on production, welfare, and the quality of milk produced. The udder health sub-index in the UK national Profitable Lifetime Index (£PLI) includes lactational average somatic cell count (LSCC) and the udder composite type trait as indirect predictors of udder health. Due to a deficiency in mastitis recording, SCC is used widely internationally as an indicator trait, for both clinical and subclinical mastitis, and was introduced into genetic evaluations in the UK in 1998. Strong genetic correlations (but less than unity) between mastitis and SCC (Heringstad *et al.*, 2000) imply that additional genetic gain could be achieved if mastitis were routinely recorded and evaluated, and selected for directly, in addition to SCC.

Some UK herds voluntarily record mastitis as part of herd management and levels of recording in more recent years have increased. A test-day model has become the method of choice for the analysis of production traits in the UK but a repeatability model is currently used for SCC. Mrode and Swanson (2003) estimated genetic parameters for UK SCC using a test-day RRM. However, there has not been any study examining the relationship between test-day SCC and mastitis in the UK. Thus, the availability of data on mastitis in recent years allows the opportunity for such joint analysis of both traits. This study undertakes the joint analysis of test-day SCC and mastitis in the first three lactations fitting a RRM.

2. Materials and Methods

2.1 Data description

Recording of mastitis events was performed voluntarily and the data was made available by two Milk Recording Organisations in the UK. Mastitis events recorded from 0 to 305 days after calving were considered and were analysed as either a binary trait (MAS) or as a count trait (NMAS). For MAS, affected animals were classed as 1 irrespective of the number of mastitis episodes recorded within that lactation and non-affected animals were classed as 0. For NMAS, the number of unique episodes of mastitis during each lactation period was considered.

The dataset consisted of the first three lactations of Holstein Friesian cows, calving from 1996 to 2009. The dataset was built up by identifying animals affected by mastitis and their contemporaries in the same herd-year-season. Edits were: 1) calving ages for 1st, 2nd

and 3rd parity were within the ranges of 18-42 months, 30-62 months, and 42 to 70 months, respectively, 2) sires were born from 1992 onwards, 3) sires had at least 20 eligible daughters, and of these up to the first 250 daughters born were selected, 4) test-day records from 4 to 305 days of calving, 5) at least 6 tests/animal/parity and only the first 12 tests were included for SCC, 6) at least 5 animals per herd-year-season, 7) at least 1 animal with mastitis per herd-year-season 8) at least 1% of animals with mastitis per herd-year-season, and 9) every animal required data in lactation one. After editing, the dataset consisted of 1,045,283 test-day somatic cell counts (SCC) from 67,175 animals, sired by 4,785 bulls (Table 1). The animal pedigree consisted of 165,142 animals, including three generations.

Table 1. Summary of data in terms of observations in different parities.

Parity	SCC		Mastitis
	No. Cows	No. tests	No. (%) cows affected
1	67,175	615,844	9,070 (13.5)
2	30,617	279,971	6,009 (20.0)
3	16,366	149,468	4,012 (24.5)

2.2 Model

The model for SCC included herd-test-day, fixed lactation curves nested with calving year-groups (1996-1999, 2000-2003, 2004-2006, and 2007-2009), age in months and month at calving, random regressions with Legendre-polynomials of order 2 for animal and permanent environmental (PE) effects. The model for both mastitis traits included fixed herd-year-season, age in months and month of calving, random animal and PE effects.

All analyses were implemented using Gibbs sampling algorithm based on Gauss-Seidel iterative best linear unbiased prediction scheme for solving the mixed model equations. A flat prior was assumed for the fixed effects and sampling was block-wise. A uniform prior distribution was assumed for animal and PE effects, and sampling was block-wise per animal from the full conditional distribution, which is proportional to a scaled inverted chi-squared distribution. The chain length of 120,000 was generated, of which the first 40,000 were discarded as burn-in period. One out of the five iterations was then saved and the marginal posterior means obtained were regarded as estimates of variance and covariance components. Genetic variances and h^2 for SCC, covariances between SCC and MAS or NMAS at different DIM were calculated from the covariance matrix generated from the random regressions for animal effects. Posterior standard deviations were computed from the variation among genetic parameters estimated from each sample saved from the Gibbs sampling.

3. Results and Discussion

3.1 Incidence of mastitis

There were 15,902 cows (23.7%) which were affected by mastitis at least once during the first three parities. As a binary trait, mastitis incidence increased with parity from 13.5 to 24.5%. The observed trend of increased mastitis incidence with increasing parity and the proportions of animals affected in the first three parities were similar to other studies although incidence level was at the higher end of the range (Pösö and Mäntysaari, 1996). Mastitis incidence was 16.7% across all lactations, which reflects the greater effort in farmer recording since the study of Kadarmideen *et al.* (2000), also based on farmer recorded data, who reported an incidence of 6%.

3.2 Heritability estimates of SCC and mastitis

In general, genetic variances of SCC increased with parity and were high at the beginning of the lactation and decreased with DIM until about 90 days and increased subsequently for the remaining part of the lactation. The trend in PE variances was similarly high at the beginning of the lactation but declined until about 50-70 DIM and thereafter increased, peaking at about 130-150 DIM, followed by another decline with values increasing towards the end of lactation. The residual variances generally decreased with DIM. Generally, daily SCC h^2 increased with DIM and was higher in later parities compared to the first

parity (Figure 1). Similar trends and estimates were reported on an earlier study using UK data (Mrode and Swanson, 2003). The increase in daily h^2 could be attributed to declining estimates of residual variance with DIM, and the increasing trend in genetic variances with days, especially after about 70 days. Average daily h^2 were 0.11, 0.14, and 0.15 respectively for parities 1, 2 and 3.

The h^2 estimates of mastitis were low and increased with parity, ranging from 0.05 to 0.12, yet were at the higher end of estimates generally obtained from a linear model (Heringstad *et al.*, 2000). Low heritability is common for functional traits, such as health and fertility. The low heritability estimate for mastitis can be explained by large environmental influences, as well as the classification of mastitis as a binary/count trait, which results in a loss of information and a low observed variation among cows. The large environmental influence suggests that some improvements could be made by changes in herd management or improved recording practices. Increasing h^2 estimates with parity could be explained by the higher proportion of animals treated, as h^2 estimates from linear models fitted to binary traits depend upon frequency. Mastitis defined as a binary trait implies that animals with one mastitis event are equally at risk to mastitis as those animals with multiple cases, thus it is perceived that there is a loss of information when the trait is treated as binary. Estimates were generally higher when repeated cases of mastitis within parities were accounted for, as more variability is observed among cows.

3.3 Genetic correlations

As expected, r_g estimates For SCC, within each parity were highest between adjacent DIM, usually about 0.97 to 0.98 and declined as DIM got further apart. In general, the within parity r_g were particularly higher within the first parity relative to the other parities, even when DIM were farther apart.

The r_g among SCC across lactations were highest at the same DIM and declined gradually as DIM got further apart. For instance, the r_g for SCC for the same DIM in parities 1 and 2 (r_{g12}) were medium value ranging from 0.48 to 0.72, peaking at about 150-180 DIM. While corresponding r_g between parities 1 and 3 (r_{g13}) followed the same pattern, values were much lower (0.35) at the beginning and end of lactation. However, r_g between parities 2 and 3 were high, ranging from 0.88 to 0.94, and showed little variation at different stages of lactation.

For both SCC and mastitis r_g between parities showed that adjacent parities (i.e. 1 and 2, 2 and 3) were more strongly correlated than non-adjacent parities (i.e. 1 and 3), as would be expected. The r_{g12}, r_{g13} and r_{g23} computed for 305-day LSCC were 0.75 (0.03) 0.64 (0.05) and 0.92 (0.02) respectively, further confirming the higher r_g between parities 2 and 3. The r_{g12}, r_{g13} and r_{g23} for MAS were 0.56 (0.06), 0.48 (0.10), and 0.89 (0.03) and for NMAS were 0.62 (0.06), 0.42 (0.09) and 0.85 (0.03), respectively. The r_g between parities were different to one; therefore indicating that separate parities should be treated as separate traits. All animals in the dataset had data from their first parity, but some cows may not have fulfilled all three lactations. Thus, the lower r_g between parities 1 and 3 could be due to some animals affected by mastitis in their first parity leading to removal from the herd and consequently with no observations in later parities.

The r_g between SCC at DIM and MAS within parities were generally of medium value, varying from 0.41 to 0.74, but were highest in parity 3. Similarly, Negussie *et al.* (2010) obtained genetic correlations between test-day SCC and mastitis that ranged from 0.47 to 0.70.

The r_g within parities were positive between MAS and LSCC and ranged from (0.53 to 0.68); indicating that reducing LSCC also reduces the incidence of mastitis. However, a r_g of less than one indicates that genetically they are not the same trait. Despite this, the moderate to high values supports the use of SCC as a good predictor of udder health in current evaluations, and the addition of mastitis as a direct trait could add some useful information. For instance, cows might not be milk sampled during a mastitis infection and treatment, therefore higher SCC measures at

these times associated with mastitis would not be recorded. Average r_g between mastitis and LSCC from literature were 0.60 from Nordic field data (Heringstad *et al.*, 2000). Across parity genetic correlations between both mastitis traits and LSCC ranged from 0.34 to 0.75 and were lowest between parities 1 and 3.

Conclusion

The genetic parameters and trends for SCC and genetic correlations with mastitis obtained in this study are in line with other studies. A test-day model that accounts for the variations in heritabilities and genetic correlations throughout lactation in combination with mastitis as a direct trait should result in more accurate evaluations compared with the current lactation average repeatability model.

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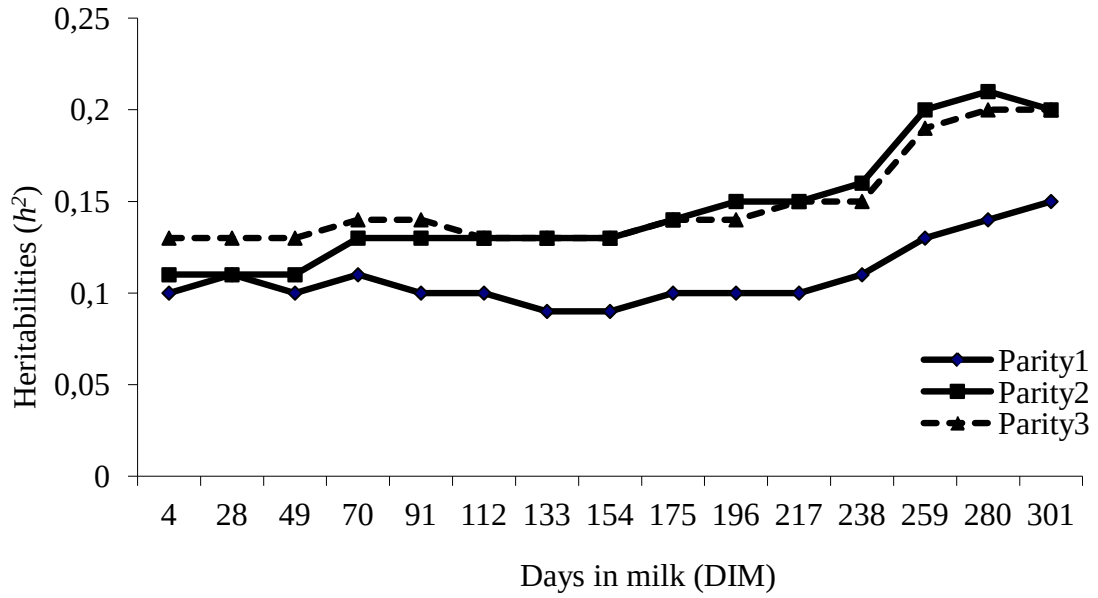

Figure 1. Daily heritabilities for SCC in the first three parities.