Estimation of variance components for clinical mastitis and somatic cell scores for the Nordic dairy cattle populations

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Abstract

Clinical mastitis (CM) is a disease which causes great losses to the dairy industry. Due to the low incidence of CM and its discrete nature, somatic cell scores (SCS), which are measured on a regular basis, are often included in genetic evaluation. As such, determining the genetic architecture of udder health traits at different risk stages is important. Thus, the objectives of this study were to estimate variance components for (i) CM events at two risk stages (early and late lactation) and at three lactations, and for (ii) SCS at three lactations. Data consisted of CM and SCS records for Holstein (HOL), Jersey (JER), and Red dairy cattle (RDC) cows. For CM, each risk period of each lactation was considered as a correlated trait, and for SCS each lactation was considered as a separate trait modelled using random regression. The genetic component was modelled using sire information (sire model). Variance components were estimated using Monte Carlo expectation-maximization residual maximum likelihood. Mean CM incidence ranged from 3.5% to 13.2% for HOL, from 5.6% to 13.6% for JER, and from 3.10% to 10.9% for the RDC breed. Combined heritability of CM was 5.4%, 6.2%, and 6.3% for HOL, JER, and the RDC breed, respectively. Heritability estimates for individual lactations and periods ranged from 0.73% to 3.48% for HOL, 1.11% to 2.82% for JER, and 1.62% to 3.39% for RDC. In addition, genetic correlations among CM traits ranged from 0.28 to 0.95, from 0.32 to 0.98, and from 0.44 to 0.93 for the HOL, JER, and RDC breeds, respectively. On the other hand, the combined heritability of SCS of 305 days in milk and for 1st, 2nd, and 3rd lactations were 0.14, 0.17, and 0.19 for the HOL breed, 0.17, 0.18, and 0.16 for the JER breed, and 0.18, 0.19, and 0.20 for the RDC breed. Genetic correlations for SCS among lactations were high (> 0.80) for all breeds. Furthermore, genetic correlations between CM and SCS traits ranged from 0.41 to 0.78 for HOL, from 0.29 to 0.69 for JER, and from 0.27 to 0.66 for RDC. Overall, the heritability estimates for traits related to udder health, including CM and SCS was low or moderate for all the breeds considered. On the other hand, genetic correlations among CM traits, and among SCS traits were moderately high to high.

Key words: Udder health, heritability, genetic correlations

Introduction

Clinical mastitis (**CM**) is a costly disease affecting dairy cattle, causing not only direct losses due to a reduced production and early culling of affected cows, but also due to changes in management necessary for the treatment of affected cows (Rollin et al., 2005). The risk of clinical mastitis is not constant throughout the life of an individual, but instead is greater at the beginning of the lactation and increases for latter lactations, as compared to the first lactation (Valde et al., 2004). As such, trait definition should properly reflect these risks.

Due to the binary nature of CM, variance component estimation may be challenging. In

addition, depending on management and environmental conditions, the incidence of clinical mastitis may be low such that many records may be necessary for estimation of variance components. On the other hand, somatic cell scores (**SCS**) are regularly recorded and may provide additional information for estimation of variance components related to udder health (Nash et al., 2000).

Due to the interest in efficiently using the joint reference population, EuroGenomic member countries have agreed to harmonize traits and adopt the "gold standard" definition of CM traits which identifies early (up to 60d) and late (> 60d) risk periods for each lactation. Thus, the objective of the current work was to estimate variance components, including heritability and genetic correlations for (i) early and late risk periods for CM at three lactations, and for (ii) SCS at three lactations for the Holstein (**HOL**), Jersey (**JER**), and Red dairy cattle (**RDC**) breeds.

Materials and Methods

Data

Data for the HOL and RDC breeds were sampled from herds in Sweden, while for the JER breed were sampled from herds in Denmark. For all breeds phenotypic records were considered starting in 2010, and herds with a minimum of 20 and a maximum of 100 first year calves were included in the analyses.

Records corresponding to 68,422, 64,194, and 71596 HOL, JER, and RDC cows, respectively, were included. These cows were sired by 2159, 986, and 1258 bulls for the HOL, JER, and RDC, respectively. Mean CM incidence ranged from 3.50% to 13.20% in HOL, from 5.65% to 13.58% in JER, and from 3.11% to 10.86% in RDC. The largest incidence of CM was observed at the late period of the third lactation. As part of the EuroGenomic trait harmonization strategy, CM records were transformed into Snell scores (Snell, 1964). Mean SCS (in logarithmic scale) was 4.04, 4.40, and 4.70 for HOL, 4.19, 4.32 and 4.49 for JER, and 4.07, 4.41 and 4.71 for RDC.

Model

For CM, six traits were included consisting of the two risk periods defined earlier for each of three lactations. In addition, one SCS trait was defined for each lactation and analyzed with a multi-trait random regression model. All nine traits were analyzed using a multi-trait linear mixed model. For CM, fixed effects included herd-year and age while for SCS a fixed lactation curve was also included. Regression effects for both CM and SCS traits included heterosis, recombination loss, and inbreeding.

Random effects for both CM and SCS traits included a sire and a permanent environmental effect, both of which were modeled using random regression. For CM, only an intercept was fitted, while SCS was modeled using a quadratic Legendre polynomial (intercept, linear, and quadratic) plus an exponential (Wilmink) term. For the sire effect, pedigree information was pruned to four generations.

Variance components corresponding to the nine traits defined earlier were estimated using the Monte Carlo Expectation Maximization REML algorithm in MiX99 (Vuori et al., 2006).

Results & Discussion

Overall, estimates of heritability were low (Table 1), ranging from 0.74% to 3.48% for the early and late period of the first and third lactation, respectively, in the HOL breed. Heritability in the JER breed was slightly lower overall and remained consistent across periods and lactations ranging from 1.11% to 2.82%, while the RDC was similar to the HOL ranging from 1.62% to 3.39%. These estimates resemble those reported by Negussie et al. (2011) for first lactation in Finnish Ayrshire.

Table 1. Heritability (%) for CM traits at three lactations and two risk periods (early, late) in the HOL, JER, and RDC breeds.

Breed	Lactation	Early	Late
HOL	1	0.74	1.24
	2	1.38	2.08
	3	1.43	3.48
JER	1	1.11	1.22
	2	1.38	2.32
	3	1.51	2.82
RDC	1	1.62	1.13
	2	1.29	2.61
	3	1.61	3.39

Genetic correlations among CM traits ranged from 0.35 to 0.95 for the HOL, 0.33 to 0.98 for the JER, and from 0.47 to 0.91 for the RDC. Genetic correlations were, in general, larger for subsequent risk periods. On the other hand, phenotypic correlations were low for all breeds.

Heritability for SCS traits, as a function of days in milk, ranged from 0.05 to 0.12 for the HOL, from 0.04 to 0.13 for the JER, and from 0.07 to 0.14 for the RDC (Figure 1). Overall, the largest estimates of heritability were found in the third lactation. Genetic correlations among

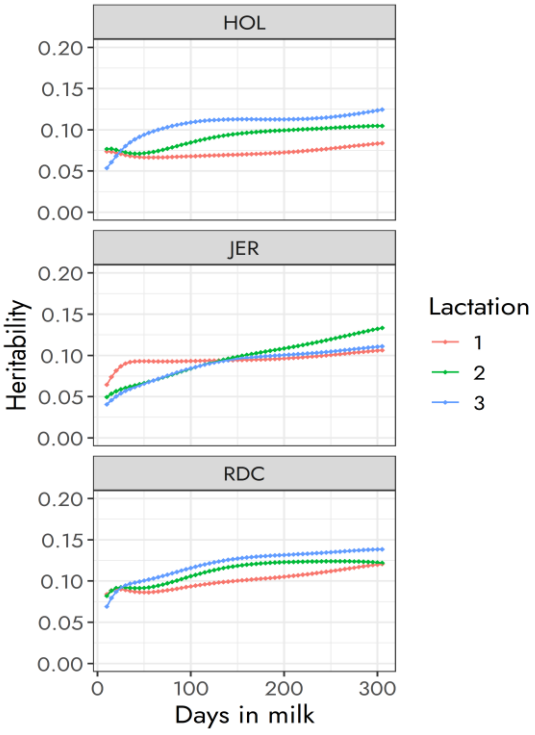

Figure 1. Heritability of SCS as a function of days in milk for HOL, JER, and RDC at three lactations.

daily SCS were high within a lactation, ranging from 0.79 to 0.99, and decreased slightly across lactations ranging from 0.59 to 0.84.

In general, combined CM heritability was about 6% for all three breeds, while the 305-d SCS heritability ranged from 14% to 21% for all breeds (Table 2). Genetic correlations among 305-d SCS traits were high and ranged from 0.84 to 0.98. On the other hand, phenotypic correlations for those traits were smaller, ranging from 0.18 to 0.51.

Genetic correlations between combined CM and 305-d SCS were moderate to large and increased with each lactation (Table 2). For the HOL these ranged from 0.61 to 0.74, for the JER they ranged from 0.43 to 0.63, and for the RDC they ranged from 0.47 to 0.63. Due to the magnitude of these genetic correlations, information from SCS can be useful for the estimation of CM traits.

Table 2. Heritability (diagonal), genetic correlations (upper triangle), and phenotypic correlations (upper triangle) for combined CM and 305-d SCS for HOL, JER and RDC breeds.

Breed	Trait[1]	CM	SCS1	SCS2	SCS3
HOL	CM	0.058	0.147	0.191	0.208
	SCS1	0.614	0.146	0.404	0.321
	SCS2	0.733	0.861	0.172	0.478
	SCS3	0.736	0.836	0.982	0.198
JER	CM	0.061	0.108	0.182	0.227
	SCS1	0.433	0.179	0.408	0.328
	SCS2	0.486	0.911	0.188	0.488
	SCS3	0.627	0.851	0.952	0.169
RDC	CM	0.064	0.128	0.175	0.191
	SCS1	0.474	0.182	0.443	0.336
	SCS2	0.564	0.913	0.201	0.506
	SCS3	0.632	0.845	0.960	0.215

1. Combined heritability for CM, and 305-d heritability for SCS at each lactation.

Conclusions

Overall, heritability for CM traits was low for all breeds but increased for the late period of each lactation and was larger for the third lactation. On the other hand, heritability for SCS traits was larger. Because of this, and due to the moderate to large genetic correlations

between SCS and CM traits, it would be beneficial to include both sets of traits to aid in the genetic evaluation of udder health traits in the Nordic evaluation.

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