

028 Genetic parameters for clinical mastitis in primi- versus multiparous cows

Saskia Bloemhof, Gerben de Jong, Yvette de Haas^{*}
 NRS, Arnhem, The Netherlands
^{*} Haas.Y@nrs.nl

Introduction: Clinical mastitis (CM) is one of the major diseases in dairy cattle. Mastitis control programs are often designed to provide guidelines to reduce CM on farms. Genetic selection is another strategy to combat CM. Although genetic selection is a slow process, it results in a permanent change in the genetic composition of the dairy herd⁵. The (first) breeding value for udder health of a bull is based on the performance of his daughters in their first lactation. However, CM is not a problem in first lactation only¹. Actually, both frequency of CM³, and level of somatic cell count (SCC)⁴ increase with increasing parity. Therefore, the aim of this study was to estimate genetic parameters for CM and lactation-average SCC for the first 3 lactations of Dutch Holstein cows.

Materials and Methods: Records on CM treatments were available from Management Information Systems on Dutch dairy farms. Data recording was done on a voluntary basis and was performed by the farmers themselves. Data on CM were recorded from June 1998 until May 2006, and all herds participated in the national milk recording system. The data was edited with criteria on age at calving, parity, lactation length, Holstein-Friesian% and availability of pedigree information. After editing the data, the final dataset consisted of 35,379 lactations from 21,064 cows on 250 farms. A pedigree file was constructed based on sires and maternal grandsires (MGS) of the cows in the final dataset, and contained 3,855 bulls with their parents.

Clinical mastitis was defined on a lactation basis as an all-or-none trait; either a cow had CM in a certain lactation (1) or not (0). Through this, only first cases of CM per lactation were taken into account. The cell counts were transformed to somatic cell score ($SCS = 1000 + 100 * (2 \log(SCC/1000))$), and was averaged per lactation over test-day records between 5 to 335 days in milk (DIM). Also, SCS in the first half of the lactation (5-150) was compared with SCS in the second half (151-335).

ASREML² was used to estimate variance components for CM and SCS using a linear sire/MGS-model. Univariate analyses were carried out for CM and SCS, with the following model: $Y = \mu + \text{fixed effects} + S_{\text{sire}} + \frac{1}{2} S_{\text{mgs}} + e$

The random sire effect was identified by sire and MGS. The sire effects were linked using the relationship matrix, and were assumed to be normally distributed (σ^2_s). Fixed effects included were parity (3 classes), age at calving, an interaction between herd and year of calving (with 948 classes), and month of calving (12 classes).

Bivariate analyses were carried out to estimate genetic correlations between CM and SCS. The applied model for the bivariate analyses was the same as the one applied for the univariate analyses.

Results: The mean proportion of cows that had CM at least once during lactation was 15.8%, and was lowest for heifers (13.4%) and highest for 3rd parity cows (19.6%). Of all 2nd parity cows 16.1% got CM at least once during lactation. The proportion of heifers with CM increased rapidly up to 50 DIM (Figure 1). Actually, half of all first cases of CM in heifers occurred before 20 DIM, whereas, half of the total proportion of 2nd and 3rd parity cows with CM was approached around 70 DIM. Before 150 DIM 75% of all first cases of CM had occurred. This shows that cows are more susceptible for cases of CM during the first half of lactation, than during the latter half.

The heritabilities for CM in parity 1, 2 and 3 were all around 3% (Table 1). The genetic correlations between CM in consecutive parities were high (~0.9), but low between parity 1 and 3 (0.6). This implies that CM has not the same genetic background in these parities.

The genetic correlations between CM and lactation-average SCS were 0.8 in parity 2 and 3 (Table 2), but somewhat lower in parity 1 (0.6). Even stronger genetic correlations were estimated for SCS in the first half of lactation, but SCS in the latter half of lactation showed weak correlations with CM. From a biological point of view, it is logical that SCS in the first half of lactation is strongest correlated to CM, because 75% of the first cases of CM occur before 150 DIM.

Conclusions: Genetic correlations between CM in consecutive parities are high, but somewhat lower between further aparted parities. Somatic cell score averaged over first half of lactation was found to be strongest genetically correlated with CM.

Acknowledgements: This study is part of the five-year mastitis programme of the Dutch Udder Health Centre and was financially supported by the Dutch Dairy Board.

References:

1. Carlén et al. (2004). Genetic parameters for clinical mastitis, somatic cell score, and production traits in the first three lactations of Swedish Holstein cows. *J. Dairy Sci.* 87: 3062-3070.
2. Gilmour et al. (2002). ASREML reference manual 2nd edition, Release 1.0 NSW Agriculture Biometrical Bulletin 3. NSW Agriculture, Orange, NSW 2800, Australia.
3. Pösö and Mäntysaari (1996). Relationships between clinical mastitis, somatic cell score, and production for the first three lactations of Finnish Ayrshire. *J. Dairy Sci.* 79: 1284-1291.
4. Reents et al. (1995). Estimation of genetic parameters for test day records of somatic cell score. *J. Dairy Sci.* 78: 2847-2857.
5. Shook (1989). Selection for disease resistance. *J. Dairy Sci.* 72: 1349-1362.

Table 1: Estimated genetic parameters for clinical mastitis in parity 1, 2 and 3 (CM1, CM2 and CM3, respectively). Heritabilities on diagonal, phenotypic correlations below diagonal and genetic correlations above diagonal, with standard errors as subscripts

	CM1	CM2	CM3
CM1	0.03 _{0.01}	0.88 _{0.13}	0.63 _{0.22}
CM2	0.06 _{0.01}	0.03 _{0.01}	0.91 _{0.12}
CM3	0.05 _{0.02}	0.12 _{0.01}	0.04 _{0.01}

Table 2: Genetic correlations between clinical mastitis in parity 1, 2 and 3 (CM1, CM2 and CM3, respectively), and somatic cell scores averaged from 5 to 335 days, from 5 to 150 days and from 151 to 335 days (SCS5-335, SCS5-150 and SCS151-335, respectively), with standard errors as subscripts

	CM1	CM2	CM3
SCS5-335	0.64 _{0.12}	0.79 _{0.10}	0.79 _{0.10}
SCS5-150	0.65 _{0.11}	0.86 _{0.08}	0.88 _{0.09}
SCS151-335	0.50 _{0.15}	0.59 _{0.14}	0.61 _{0.14}

Figure 1: The proportion of cows with at least one case of clinical mastitis during parity 1, 2 or 3 (CM1, CM2 and CM3, respectively) per day in milk.

