Somatic Cell Count as an Indicator of Subclinical Mastitis. Genetic Parameters and Correlations with Clinical Mastitis

Morten Svendsen[1] and Bjørg Heringstad[1,2]
[1]GENO Breeding and A.I. Association, P.O Box 5003, N-1432 Ås, Norway.
[2]Dep. of Animal and Aquacultural Sciences, Norwegian University of Life Sciences. P.O Box 5003, N-1432 Ås, Norway.
Phone: +47 64965227 Fax: +47 64965101 E-mail: morten.svendsen at umb.no
Phone: +47 64965117 Fax: +47 64965101 E-mail: bjorg.heringstad at umb.no

Key words: Dairy cattle; somatic cell count; mastitis; binary trait; multivariate; sire model

1. Introduction

Both clinical mastitis (CM) and soma-tic cell count (SCC) have complex biological background. Schukken *et al.* (1997) reviewed defence mechanisms against mastitis, such as mechanical prevention of microorganisms entering the udder; cellular innate immunity for immediate response to infection; and adaptive immunity involving antibody producing B-lymfocytes and T-lymfocytes. Bacteria causing intramammarian infection can be grouped in three major classes. Gram-negative bacteria like *E.coli*; *S.aureus*; and non-agalactiae streptococci (*S.uberis* and *S.dysgalactiae*), the last two groups being gram-positive. These bacteriaclasses differ with regard to prevalence through lactation, ability to cause clinical symptoms, defense mechanisms activated in the udder, and pattern in SCC response (de Haas *et al.,* 2002; Schukken *et al.,* 1997).

Heringstad *et al.* (2004) analyzed CM by dividing each of the first 3 lactations into 4 periods, treating each period as a binary CM trait. Genetic correlations varying from .24 to .73 indicate that CM should not be regarded as the same trait in different parts of lactation or in different lactations. The Norwegian mastitis index has now been expanded to include both early and late lactation traits in 3 lactations (Svendsen and Heringstad, 2006).

SCC has so far not been used in genetic evaluation of mastitis in Norway. In the Norwegian Dairy Herd Recording System milk yield is recorded every month, but samples for analysis of milk constituents and SCC are only mandatory every second month. With such long intervals between testdays, SCC can't be expected to reflect well the frequency of clinical mastitis. Especially not short term infections. Subclinical and chronic infections however, could cause longterm elevated SCC (de Haas *et al.,* 2002) covering several testdays.

Our objective was to express sub-clinical and chronic mastitis as a binary trait based on prolonged elevated SCC and estimate genetic parameters and correlation to clinical mastitis for such trait.

2. Material and Methods

Data were from the Norwegian Dairy Herd Recording System and included mastitis information and testday SCC for the first 3 lactations of cows with first calving from 1988 to 1997 in herd by year classes of at least five.

Clinical mastitis (CMi) was defined as a binary trait within each of seven intervals (i=1,..,7) such that first lactation was divided in 3 intervals: (-15 to 30), (31 to 120), and (121 to 305), whereas second and third lactations were divided in 2 intervals: (-15 to 30) and (31 to 305) days postpartum. Number of observations and proportion of treated cows for each trait can be found in Svendsen and Heringstad (2006).

Subclinical or chronical mastitis (SM) was defined as a binary trait within each lactation.

If SCC on 2 testdays 2 mo apart both were above a fixed threshold the binary variable was defined as 1, 0 otherwise. In herds with monthly milk samples, three consecutive testday SCC had to be above the threshold. Four thresholds were investigated, 50,000, 100,000, 150,000, and 200,000 cells/ml. Only testdays between 30 and 270 days in milk were considered. Numbers of observations were 566,000, 392,000 and 241,000 in first, second and third lactation, respectively. The proportions of cows with SM defined by the 4 SCC thresholds are given in Table 1.

The gaussian linear sire model used for analysis had fixed effects of age at calving in months and year by month of calving, and random effects of herd by year, sire, and residual. Acceptable age at calving was 21 to 31 mo at first calving; 32 to 47 mo at second calving; and 43 to 61 mo at third calving. Pedigrees of sires were traced at least 3 generations. Variance components were estimated from bivariate models using the 'dmuai' program (Madsen and Jensen, 2005).

Expected genetic progress in each binary trait from progeny testing of bulls was calculated using selection index theory (Lin, 1978). Assumed information sources and datastructure were as in Svendsen and Heringstad (2006). Bivariate estimates from SCC-threshold of 100,000 cells/ml were combined with CM estimates into 10-variate positive semidefinite covariance matrices by using the 'itsumcov' program (Henshall and Meyer, 2002).

3. Results and Discussion

The distribution of testday SCC across first lactation is depicted in Figure 1. During the first month there were many high SCC records, but the situation improved after peak lactation before getting worse in the last trimester. Fifteen to twenty percent of the samples had SCC below 20,000 cells/ml and about half of the samples were below 50,000 cells/ml.

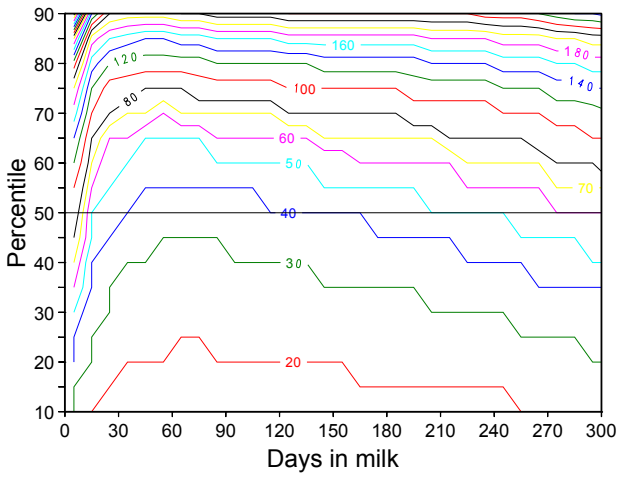

Figure 1. Testday SCC (10^3 cells/ml) in first lactations since 2001. Percentiles calculated for 10 days periods throughout the lactation.

The proportions of affected cows were higher for SM (Table 1) than for CM (Svendsen and Heringstad, 2006) which is advantageous when analyzing a binary trait with a Gaussian linear model. For thresholds of 50,000 and 100,000 cells/ml the heritability estimates were between .076 and .086 (Table 1) which were close to those found for liability of CM in a Bayesian threshold model (Heringstad *et al.,* 2004). SM defined by 200,000 cells/ml showed higher heritabilities than CM traits (Svendsen and Heringstad, 2006) with similar proportions affected cows.

Within lactations, SM traits were automatically correlated as the lower SCC threshold included all affected cows of the higher SCC threshold. The genetic correlations between the most different traits defined by 50,000 and 200,000 cells/ml were between .89 and .92. Bull ranking will there-fore not be severely affected by choice of SCC threshold. Herd by year correlations were between .74 and .79, and the residual correlations ranged from .31 to .37.

Table 1. Subclinical mastitis defined by SCC threshold in first, second, and third lactation. Proportion of affected cows (p), variance ratios of herd by year (c^2) and sire times four (h^2) in %.

SCC threshold	p			c^2			h^2		
10^3 cells/ml	1st	2nd	3rd	1st	2nd	3rd	1st	2nd	3rd
50	45.5	62.5	69.8	5.9	7.0	7.3	8.6	8.2	7.6
100	25.2	37.1	43.4	4.2	5.6	6.3	7.8	7.7	7.7
150	16.5	25.1	29.6	3.4	4.9	5.6	5.9	5.9	6.1
200	11.6	18.1	21.7	2.9	4.2	4.8	4.5	4.9	5.1

Table 2. Residual correlation (r_e), herd by year correlation (r_c) and genetic correlation (r_g) between subclinical mastitis, defined by the same SCC threshold across lactations.

SCC threshold	r_e			r_c			r_g		
10^3 cells/ml	1-2	2-3	1-3	1-2	2-3	1-3	1-2	2-3	1-3
50	.28	.32	.18	.80	.95	.77	.90	.99	.83
100	.28	.33	.19	.75	.91	.73	.94	.98	.87
150	.25	.31	.17	.71	.92	.69	.96	.98	.90
200	.23	.29	.15	.71	.92	.70	.96	.98	.91

For SM defined by the same SCC threshold across lactations (Table 2), second and third lactation were slightly higher correlated than first to second and first to third lactation. The genetic correlations between SM traits varied between .83 and .99 and were slightly higher than what was found between CM traits by Svendsen and Heringstad (2006).

Correlations between SM and CM in the same lactation are given in Table 3. Resid-ual correlations were all close to zero. Herd by year correlations were slightly negative with CM in early lactation. In herds with high frequency of early CM, carry over effects of treatments, dried up quarters and early culling may reduce high SCC later in lactation.

Genetic correlations tended to be higher when the SCC threshold defining SM was higher (Table 3). The average result for 200,000 cells/ml was similar to the estimated genetic correlation between lactation SCC and CM of 0.53 in a recent Norwegian study (Ødegård *et al.,* 2004). The correlations were stronger to CM in late lactation (CM2, CM3, CM5 and CM7) than to CM in early lactation. This was consistent over all SCC thresholds and lactations. This may be because mastitis caused by gram-positive bacteria are more frequent in late lactation and these bacteria often cause chronic infections with high SCC. Adaptive immunity plays a major role to fight these infections, whereas innate immunity and mechanical defense are most important factors to prevent *E.coli* infections from reaching a clinical stage in early lactation (Schukken *et al.,* 1997). If these mechanisms show indepen-dent inheritance, low genetic correlation is to be expected.

The new Norwegian mastitis index (Svendsen and Heringstad, 2006) is expected to give genetic progress for SM (Table 4) almost at the same rate as for CM in late lactation. However, including the SM traits (New+SM in Table 4) does not improve the CM traits and does only marginaly improve the SM traits themselves.

Table 3. Residual correlations (r_e), herd by year correlations (r_c), and genetic correlations (r_g) between subclinical mastitis defined by SCC thresholds (SM) and clinical mastitis traits (CMi) in the same lactation.

	Threshold 10^3 cells/ml	SM1 CM1	SM1 CM2	SM1 CM3	SM2 CM4	SM2 CM5	SM3 CM6	SM3 CM7
r_g	**50**	.26	.49	.40	.31	.41	.32	.38
	100	.29	.52	.45	.42	.50	.39	.47
	150	.33	.58	.51	.46	.57	.38	.57
	200	.35	.61	.56	.47	.62	.42	.61
r_c	**50**	-.21	.02	-.02	-.09	.00	-.10	.04
	100	-.19	.03	.01	-.13	.02	-.07	.02
	150	-.16	.01	.03	-.14	.01	-.08	.01
	200	-.14	.02	.04	-.15	-.01	-.12	.00
r_e	**50**	-.04	.03	.08	-.02	.06	-.02	.05
	100	-.03	.04	.09	-.01	.09	-.01	.08
	150	-.02	.05	.10	.00	.10	.01	.09
	200	-.01	.05	.11	.01	.10	.02	.10

Table 4. Expected genetic gain in CM and SM traits from selection based on the new mastitis index (1/3 CM1 + 1/3 CM2 + 1/3 CM3) with different sources of information, relative to expected gain of 100 in the old mastitis index (0.7 CM1 + 0.3 CM2). Traits providing information are underlined.

Mastitis index	CM1	CM2	CM3	CM4	CM5	CM6	CM7	SM1	SM2	SM3
Old	100	100	100	100	100	100	100	100	100	100
New	93.9	120.4	129.9	100.0	128.6	100.0	122.3	119.7	121.3	123.4
New +SM	93.9	120.4	129.9	100.0	128.6	100.0	122.3	121.9	123.1	126.0
New -CM	20.4	56.3	57.4	34.4	65.7	38.0	51.4	202.1	202.8	221.4

Because of few SCC testdays, the SM traits were constructed at the end of lactation. Therefore no records from own daughters would be available at the time of first evaluation. In a situation where only SM is available (New –CM in Table 4), the rate of improvement would double for SM, but only half the progress in late lactation CM and a third in early lactation CM would be achieved.

4. Conclusions

Choice of SCC-threshold defining SM will not severly affect bull ranking.

Genetic correlations between SM in different lactations were high.

Genetic correlations between SM and CM were stronger with higher SCC thresholds defining SM and with CM in late lactation.

Including information from SM in the mastitis index will only yield a marginal improvement.

5. Acknowledgement

The Norwegian Dairy Herd Recording System and the Norwegian Cattle Health Ser-vice are acknowledged for providing access to data. Dr. Erling Sehested and Dr. Astrid Karlsen are acknowledged for preparing the testday SCC data.

6. References

de Haas, Y., Barkema, H.W. & Veerkamp, R.F. 2002. The effect of pathogen-specific clinical mastitis on the lactation curve for somatic cell count. *J. Dairy Sci. 85,* 1314-1323.

Henshall, J.M. & Meyer, K. 2002. 'PDMATRIX' – Programs to make matrices positive definite. In *'Proceedings of the 7th World Congress on Genetics Applied to Livestock Production'*. Communication No. 28-12.

Heringstad, B., Chang, Y.M., Gianola, D. & Klemetsdal, G. 2004. Multivariate threshold model analysis of clinical mastitis in multiparous Norwegian Dairy cattle. *J. Dairy Sci. 87,* 3038-3046.

Lin, C.Y. 1978. Index selection for genetic improvement of quantitative characters. *Theor. Appl. Genet. 52,* 49-56.

Madsen, P. & Jensen, J. 2005. *A user's guide to DMU - A package for analyzing multivariate mixed models.* Version 6, release 4.5. Danish Institute of Agricultural Sciences, Research Centre Foulum, Denmark, 25 pp.

Ødegård, J., Heringstad, B. & Klemetsdal, G. 2004. Short communication: Bivariate genetic analysis of clinical mastitis and somatic cell count in Norwegian Dairy Cattle. *J. Dairy Sci. 87,* 3515-3517.

Schukken, Y.H., Lam, T.J.G.M. & Barkema, H.W. 1997. Biological basis for selection on udder health traits. *Interbull Bulletin 15,* 27-33.

Svendsen, M. & Heringstad, B. 2006. New genetic evaluation for clinical mastitis in multiparous Norwegian Red cows. *Proceedings Interbull Open Meeting,* June 4-6, 2006, Kuopio, Finland. *Interbull Bulletin 35,* 8-11.