New Genetic Evaluation for Clinical Mastitis in Multiparous Norwegian Red Cows

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1. Introduction

The breeding plan for the Norwegian Red (NRF) population was designed for a broad breeding goal including traits with low heritability. Each year since 1972, about 125 test bulls has sired 40% of the cows, enabling progeny testing based on 250 to 300 daughters per sire. The remaining 60% of the cows has been sired by elite bulls selected based on a total merit index. The use of any particular elite sire in a herd has been limited to at most every fifth cow. This practice has helped preserve a broad genetic basis of the population and keep up the effective daughter contribution from small herds.

Clinical mastitis has been included in the total merit index since 1978. The weight the first years was large enough to counteract unfavourable correlated effects from selection for increased milk yield, but was increased in 1990 to achieve genetic progress for mastitis resistance. In the genetic evaluation, clinical mastitis has been defined as a binary trait, where one or more treatments during the interval from -15 to 120 d postpartum was scored as 1, and 0 otherwise.

Until 1999 the yearly cohort of test bulls were evaluated once by a linear sire model based solely on own daughters and relationships among the bulls (Fimland, 1984). Since then, data from 1978 onwards on daughters of both test and elite bulls has been analysed simoultanously in a linear sire model accounting for all relationships among bulls (Svendsen, 1999). This far, first and second lactation have been analysed separately with selection of elite bulls based on first lactation results only. Older bulls had first and second lactation results combined in an index.

Heringstad *et al.* (2004) investigated further utilization of the Norwegian health recordings by dividing each of the first 3 lactations into 4 periods, treating each period as a binary trait. This way clinical mastitis in second and third lactation could be utilized, and multiple treatments during lactation, culling of cows and records in progress could be taken into account. Genetic correlations varied from .24 to .73 and were found too low to consider a repeatability model. Furtermore, periods in early lactation were more strongly correlated to other early periods than to any late period. Similarly, late periods showed stronger correlation to other late periods than to any of the early periods. This may be caused by multiple mechanisms of mastitis resistance, with different impact in early and late lactation. Schukken *et al.* (1997) reviewed the biological basis for such mechanisms of mastitis resistance. The estimates of genetic change by Heringstad *et al.* (2004) showed that there was a strong response to the selection carried out in early first lactation, and also similar indirect responses in early second and third lactations. The correlated responses for periods in late lactations were only half in size.

Heringstad *et al.* (2004) applied a Bayesian multivariate threshold model. The computational burden is prohibitive for a routine genetic evaluation at present time. However, to rank sires a gaussian linear sire model produce similar results as a threshold model (Heringstad, 2003) at a fraction of the computational effort.

Our objectives were to simplify the model of Heringstad *et al.* (2004) and implement it as a multivariate gaussian linear sire model for routine genetic evaluation. Further, to estimate appropriate genetic parameters for this implementation, and to device a suitable selection criterion for clinical mastitis to be included in the total merit index.

2. Material and Methods

Data were from the Norwegian Dairy Herd Recording System and included mastitis information for the first 3 lactations of 2.3 million cows with calving from September 1978 onwards. First lactation was divided in 3 intervals: (-15 to 30), (31 to 120), and (121 to 305), while second and third lactations were divided in 2 intervals: (-15 to 30) and (31 to 305) days postpartum. Clinical mastitis (CMi) was defined as a binary trait within each of these seven intervals (i=1,..,7). Number of observations and the proportion of treated cows for each trait are given in Table 1.

A subset of data, including cows with first calving from 1988 to 1997 from herd by year classes with at least five cows, was extracted for estimation of variance components (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. Before 2003 data from herd by year classes with no reported treatments of any disease were discarded. Such herds were assumed not participating in the health recording system, but are now handeled as not having disease treatments to report. Pedigrees of sires were traced at least 3 generations.

The genetic evaluation was imple-mented as a 7-variate gaussian linear sire model using the 'dmu5s' program (Madsen and Jensen, 2005). To estimate variance components for this implementation, bivariate models were analysed by use of the 'dmuai' program of Madsen and Jensen (2005). The resulting bivariate estimates were combined into 7-variate positive semidefinite covariance matrices by using the 'itsumcov' program (Henshall and Meyer, 2002). The full seven-variate genetic evaluation contained 4.1 million equations in 2,500 fixed effects, 3.98 million herd by year equations and 132,000 equations in sire pedigree. The system was solved by 900 iterations in 4 hours on a PC with 3GHz Intel P4 processor and 2 Gb RAM in dual channel configuration.

Each sire gets 7 breeding values for mastitis resistance that have to be combined. Because only CM1, CM2 and CM3 will con-tribute daughter information at the time when elite sires are selected, the mastitis index was defined as: CMI_{new} = 1/3 EBV_{CM1} + 1/3 EBV_{CM2} + 1/3 EBV_{CM3}. The other traits only contribute information through correlations in the 7-variate equation system. The old mastitis index corresponds in terms of the new binary traits to: CMI_{old} = 0.7 EBV_{CM1} + 0.3 EBV_{CM2}.

Table 1. Binary clinical mastitis traits (CMi) in the first 3 lactations. Number of cows (x1000) and proportion of treated cows (%) in total data and in a subset used for variance component estimation.

Trait	Lact-ation	Days in milk	Total data n	Total data p	Subset n	Subset P
CM1	1	-15, 30	2,336	10.0	544	11.9
CM2	1	31, 120	2,334	4.4	560	5.0
CM3	1	121, 305	2,180	6.3	558	7.8
CM4	2	-15, 30	1,759	10.5	384	12.1
CM5	2	31, 305	1,603	15.3	381	18.2
CM6	3	-15, 30	1,198	13.2	234	14.8
CM7	3	31, 305	1,063	17.9	231	20.7

Table 2. Information sources and datastructure in the selection index of a test bull at first genetic evaluation. Assumed effective number of daughters distributed by culling pattern.

Relative	rel. coeff.	CM-trait	Eff. dg.	CM-trait	Eff. dg.	CM-Trait	Eff. dg.
Own dg.	.5	1 and 2	31	1	140		
Sire 1st batch dg.	.25	1 to 7	88	1 to 5	56	1 to 3	39
Sire 2nd batch dg.	.25	1 to 7	791	1 to 5	503	1 to 3	351
Mgs 1st batch dg.	.125	1 to 7	88	1 to 5	56	1 to 3	39

Expected genetic response in each binary trait to progeny testing of bulls was calculated using selection index theory (Lin, 1978). Table 2 shows the information sources and datastructure assumed in the selection index of a test bull at first genetic evaluation.

3. Results and Discussion

The binary traits in this new implementation were constructed by use of original data from the Norwegian Dairy Herd Recording System. This improved some issues on missing data in the old imple-mentation (Svendsen, 1999).

When test bulls receive their first genetic evaluation, about half their daughter-group has reached 150 days in milk. In the new implementation almost the whole daugh-tergroup will provide information on CM1, whereas earlier a daughter would be missing unless both CM1 and CM2 were observed. First lactation was divided into 3 periods. This facilitates early use of information on resist-ance mechanisms manifesting late in lact-ation. However, it also cause lower proportions of treated cows in each of CM2 and CM3 (Table 1), which is disadvantageous when fitting binary traits with gaussian linear models.

Variance ratios and correlations resulting from the bent 7-trait (co)variance matrices of herd by year, sire and residual are given in Table 3. Compared to the multi-variate threshold model results of Heringstad *et al.* (2004), heritabilities were much lower (.008 to .03) and correlations were higher (.43 to .90). However, the pattern of strong correlations among early lactation traits as well as strong correlations among late lactation traits was also found here.

The new mastitis index will receive the same weight in the total merit index as before. However, internally there is increased weight on late lactation mastitis resistance. The correlation with the old mastitis index was between .85 and .90. As seen from Table 4 the new mastitis index is expected to result in equal or faster genetic improvement for all traits except CM1. The major improvements are expected in late lactation, where Heringstad *et al.* (2004) found less genetic improvement than in early lactation.

Table 3. Variance ratios (x100) of sire times four (h^2) and herd by year (c^2). Herd by year correlations above diagonal and genetic correlations below diagonal.

	h^2	c^2	CM1	CM2	CM3	CM4	CM5	CM6	CM7
CM1	3.00	6.62		.49	.43	.74	.43	.68	.43
CM2	0.83	2.34	.56		.65	.47	.71	.46	.71
CM3	1.48	3.60	.44	.90[b]		.45	.80	.47	.81
CM4	2.03	5.39	.81[a]	.60	.56		.59	.85	.55
CM5	2.58	5.93	.44	.87[b]	.91[b]	.58		.53	.90
CM6	2.15	5.98	.73[a]	.54	.46	.92[a]	.54		.59
CM7	2.26	6.09	.48	.78[b]	.82[b]	.53	.94[b]	.58	

[a)] Correlations among early lactation traits. [b)] Correlations among late lactation traits.

Table 4. Expected genetic gain in CM traits from selection based on the new mastitis index, relative to the expected gain using the old mastitis index.

Mastitis index	CM1	CM2	CM3	CM4	CM5	CM6	CM7	Average
Old	100	100	100	100	100	100	100	100
New	93.9	120.4	129.9	100.0	128.6	100.0	122.3	108.9

4. Conclusions

Data on all cows in the Norwegian Dairy Herd Recording System is now utilized.

Information on multiple treatments and treatments in second and third lactation is now used.

Records in progress at time of first genetic evaluation are handled more effec-tively.

Resistance to mastitis in early and late lactation is treated as separate traits.

Faster genetic progress is expected overall, and especially in late lactation.

5. Acknowledgement

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