

Preliminary Results from a Genetic Analysis of Clinical Mastitis Data

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Abstract

First lactation clinical mastitis records from Norwegian health recording were analysed. These preliminary analyses utilised data on 70,861 daughters from 2 annual batches of a total of 257 young bulls. Genetic parameters of mastitis resistance were estimated with both a threshold and a traditional linear model analysis, resulting in heritability estimates of 0.09 and 0.05 respectively. Mastitis information from the period 10 days prior to 90 days after calving resulted in the highest heritability estimate. Estimated breeding values and ranking of bulls were compared for the threshold and linear models. Correlations using either criterion were both greater than 0.99.

1. Introduction

Mastitis is the most frequent and costly disease affecting dairy cattle. In recent years genetic evaluation for mastitis resistance has received more attention in dairy cattle breeding. Denmark, Finland, Norway and Sweden are the only countries which includes mastitis resistance directly in their dairy cattle breeding programs (Heringstad et al., 1996). All the four Nordic countries have well-established, national recording systems for health data and use field records of veterinary treated cases of clinical mastitis in breeding evaluation.

However, there are some differences between the Nordic countries in the way mastitis resistance is defined (Ruane and Klemetsdal, 1996). Mastitis is generally considered as an all-or-non trait, with cows reported with mastitis in a defined period of the lactation as diseased and cows with no report in this period as healthy. The considered period varies between countries, covering the period 10 days before to 180 days after calving in Denmark; 7 days before to 150 days after calving in Finland; 15 days before to 120 days after calving in Norway and 10 days before to 150 days after calving in Sweden. The main reason for using information only from a short period of the

lactation is to avoid bias due to culling of cows. In the first part of lactation culling rate is low and, according to Syväjärvi et al. (1986), about 2/3 of all mastitis treatments occur within 2 months after calving.

The most common approach when utilising mastitis data in genetic evaluation, has so far been to apply linear models. Linear models assume normal distribution of the data, while mastitis data is non-normal. The threshold model (e.g. Falconer, 1989) takes into account the binary nature of mastitis data and can therefore be advantageous for variance component and breeding value estimation (Gianola and Foulley, 1983). In a simulation study, Meijering and Gianola (1985) compared linear and non-linear methods of sire evaluation for categorical data, and found significantly larger selection response with non-linear methods for binary traits, with moderate to high heritability on the underlying scale, and incidence in the population below 25 %.

The aims of this preliminary analysis were to analyse the effect of the period of the lactation on the heritability of mastitis resistance, to estimate heritability of mastitis resistance both with linear and threshold models and to compare ranking of bulls based on results from the two models. Further details can be found in Karlsen (1997).

2. Material and methods

2.1. Data

Data was extracted from the research data base generated at the Department of Animal Science, Ås (Ruane et al., 1997), from first lactation daughters of 2 annual batches of young bulls progeny tested in 1994 and 1995. A record was included if: i) the sire-ID number was valid, ii) age at first calving was between 450 and 1200 days, and iii) the lactation was carried out in only one herd and from a normal calving. Clinical mastitis data from 30 days before calving to the end of first lactation was sampled. If the cow had more than one observation of clinical mastitis in this period, the last observation before calving and the first observation after calving were kept. The dataset included a total of 70,861 first lactation daughters bred by 257 sires, from 34,620 different herd by year subclasses. The average number of daughters per sire was 276, and the overall frequency of clinical mastitis was 26.3 %.

2.2. Definition of the trait

Mastitis resistance was defined as a binary trait, on the basis of whether or not the cow had mastitis in the following periods;

- C from 30 days before to 30, 60, 90, 120, 150, 180 and 240 days after calving.
- C from 20 days before to 30, 60, 90, 120, 150, 180 and 240 days after calving.
- C from 10 days before to 30, 60, 90, 120, 150, 180 and 240 days after calving.
- C from calving to 30, 60, 90, 120, 150, 180 and 240 days after calving.
- C from 15 days before to 120 days after calving.
- C from 7 days before to 150 days after calving

In all analyses cows culled before the end of the relevant period were included if they had mastitis and excluded if they did not. This is because cows culled before the end of the period were not considered to have had the same chance to express this trait.

2.3. Model

The model was:

$$Y_{ijklm} = A_i + M_j + HY_k + S_l + E_{ijklm}$$

where

Y_{ijklm} is an observation of mastitis,

A_i is the fixed effect of i 'th age at calving in 15 classes, where < 20 months is the first class and > 32 months the last class and the other classes are in single months,

M_j is the fixed effect of j 'th month of calving in 12 classes,

HY_k is the random effect of k 'th herd by year subclass,

S_l is the random effect of l 'th sire, and

E_{ijklm} is a random error term.

The data was analysed by both linear and threshold sire models by use of CMMAT 2 (Miztal, 1989, 1991 and Miztal et al. 1989), developed from the non-linear evaluation procedure of Gianola and Foulley (1983). This program estimates sire variance components by a REML-type algorithm. Sires were assumed unrelated in the study.

The herd by year effects were considered as random. Initial variance ratios ($\sigma^2_{\text{error}} / \sigma^2_{\text{herd*year}}$) for herd by year were estimated on 6 subsets of the data, on randomly drawn herds and periods. The average estimate was 9.79 for the threshold model and 9.42 for the linear model, and these variance ratios were used in the main analyses which absorbed the effect of herd by year subclasses.

3. Results and discussion

3.1. Heritability estimates for different periods of first lactation

A sire threshold model was run to analyse what effect the period of the lactation had on the heritability of mastitis resistance. Figure 1 shows the heritability of different information periods.

Including information on mastitis before calving resulted in higher heritabilities than if the period started at calving. There were no major differences in the size of the estimates whether the information period started 10, 20 or 30 days before calving. The heritability estimates of mastitis ranged 0.084 to 0.088 if mastitis information before calving were included, and 0.075 to 0.079 if the information period started at calving.

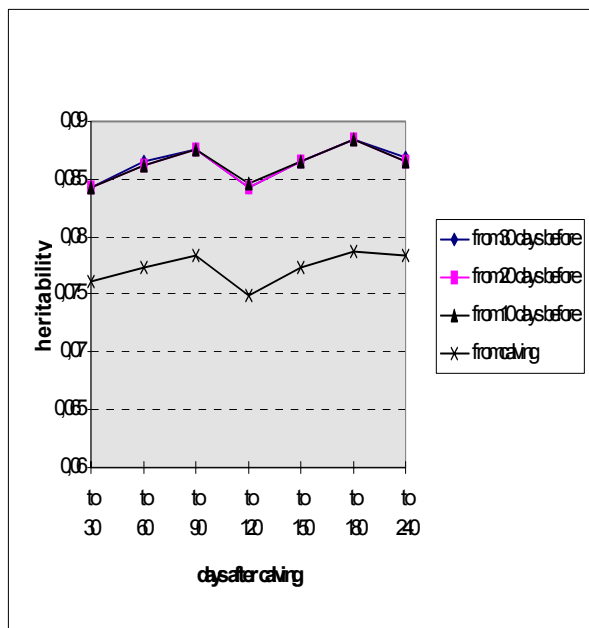


Figure 1. Heritability estimates of mastitis resistance by use of different information periods.

Waage et al. (1990) showed that the relative proportion of different bacteria causing mastitis varied with stage of lactation, especially before and after calving. Before calving, *A. Pyogenes* showed a higher and *E. Coli* a lower relative frequency than after calving. This indicates that mastitis resistance may, to a certain degree, be considered as different traits in different stages of lactation, and this may explain the different heritability from including mastitis information before calving.

The curve of heritability estimates however is flat, but tends to have a maximum at 90 and 180 days after calving, and a corresponding minimum when the period of information ends at 120 days. It may therefore be concluded that information on mastitis from at least 10 days before calving to about 90 days after calving results in the highest heritability estimate of mastitis resistance. In an analysis of Danish clinical mastitis records, the

highest genetic variance for mastitis was found when using data from the period -10 to 50 days after calving (Lund and Jensen, 1996).

3.2. Threshold model vs. linear model.

Heritabilities of mastitis resistance estimated with linear and threshold model, using the current mastitis-information periods in the four Nordic countries, are presented in Figure 2. Linear models gave heritability estimates in the range of 0.049 to 0.051, while estimates from the threshold models ranged 0.085 to 0.088.

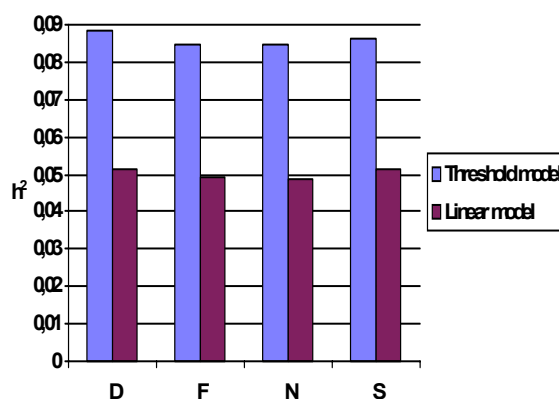


Figure 2. Heritability estimates of mastitis resistance by use of the threshold and linear models, and the current information periods in the Nordic countries; D=Denmark (-10 to 180 days), F=Finland (-7 to 150 days) N=Norway (-15 to 120 days) and S=Sweden (-10 to 150 days).

The figure shows that a threshold model resulted in a heritability that was roughly twice that estimated with a linear model. There are no basic differences in the heritability estimates from the four time periods, for either model.

Weller et al. (1988) found 2 to 5 times higher heritability estimates for binary scored traits with a threshold model than those from a linear model. Matos et al. (1997) estimated heritabilities of a binary fertility variable that were up to 19 times greater for a threshold model than for a linear model. More moderate increases in heritability of fertility traits with more than 2 categories were also calculated by Matos et al. (1997), 1.4 to 2.8 times, with a threshold sire model.

3.3. Ranking of bulls

Sire evaluation from the threshold and linear model were compared, by use of mastitis data from the period 10 days prior to 90 days after calving. Correlation between estimated breeding values with the two models were 0.998. Rank correlation (using Spearman's correlation test) was roughly the same, 0.999.

This is not unexpected as Meijering (1985) showed that differences between threshold and linear models may only be minor with large number of progeny per sire. Similar results were found by Weller et al. (1988), estimating the correlation between linear and threshold model evaluation greater than 0.9.

From the threshold analysis, the probabilities of mastitis among daughters, across fixed effects, were computed for each sire. This is a way to present results from the threshold analysis on the visible scale. Figure 3 shows that the daughters of the 3 sires with the least favourable estimated breeding value of mastitis resistance are expected to have twice the mastitis frequency (32%) of daughters from the 3 best bulls (17%). The figure also demonstrates that the genetic variance of mastitis resistance is substantial.

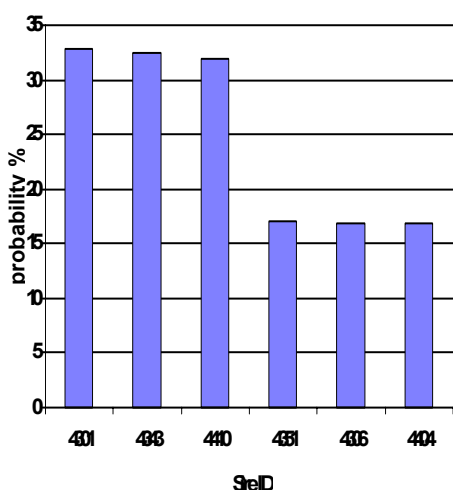


Figure 3. Probability (%) of mastitis among daughters, from the highest and lowest ranking bulls, across fixed effects.

3.4. Future research plans

The presented results is from a preliminary study. The next step is to analyse the whole dataset, including clinical mastitis records from 1978 onwards, and to take account of relationships between sires. Initial plans is to analyse the genetic parameters of mastitis resistance with different models and different definitions of the trait. On this basis, the genetic trend for mastitis resistance will be estimated.

4. Conclusions

The period before calving gives important information about mastitis resistance. Information on clinical mastitis in the period from at least 10 days before calving to 90 days after calving results in the highest heritability estimates of mastitis resistance in this study. Heritability estimates with a threshold model were roughly twice the linear model estimates. Correlation of almost one were found between estimated breeding values and between sire ranking using either models.

5. Acknowledgement

I. Miztal is acknowledged for the use of his CMMAT 2-program

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