GENETICS AND BREEDING

Clinical Mastitis in Norwegian Cattle: Frequency, Variance Components, and Genetic Correlation with Protein Yield

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ABSTRACT

Records of clinical mastitis in first lactation Norwegian Cattle from 1978 onward were analyzed. Variance components for clinical mastitis were estimated with a linear sire model using records of more than 1.2 million cows from 2043 sires, resulting in heritability estimates of 0.035. Different strategies for extracting data gave very similar results, and estimated heritability for mastitis was the same with univariate and bivariate (with protein yield) analyses, which indicates that selection bias caused by correlated responses from other traits in the breeding goal is not a problem with this data set. The estimated genetic correlation between clinical mastitis and protein yield is 0.25.

(**Key words**: clinical mastitis, variance components)

Abbreviation key: **FCD** = first-crop daughters, **FSCD** = first- and second-crop daughters.

INTRODUCTION

Mastitis is the most frequent and costly disease affecting dairy cattle. Strategies to reduce mastitis frequency are important to reduce costs of production and use of antibiotics as well as for ethical and animal welfare reasons. In recent years, genetic evaluation for mastitis resistance has received increasing attention in dairy cattle breeding. Denmark, Finland, Norway, and Sweden, however, are the only countries with national recording systems for health data in dairy cattle, and Norway was the first country to introduce a nationwide recording system (5).

The Norwegian health recording system, which involves recording of all veterinary treatments on an individual animal basis, was introduced nationwide in 1975 (11). Since then, disease traits have been recorded in most milk-recorded herds. Figures for 1996 show that 90% of all cows were included in the dairy cattle recording service, and 98% of these participated in the health card system (9). Each cow has an individual health card that is updated each time the veterinarian treats her. Because antibiotics can only be prescribed by veterinarians in Norway, health recording is very reliable. Field records of veterinarian-treated cases of clinical mastitis have also been used in the breeding evaluation of bulls since 1978. The current health card system includes 63 different traits, and mastitis is observed as acute clinical mastitis, chronic clinical mastitis, or subclinical mastitis (9). In this study, records on clinical mastitis were used with no distinction between acute and chronic, which is in agreement with current practice for breeding evaluation in Norway.

The main aim of this study was to describe Norwegian data and to estimate variance components and heritability of clinical mastitis in first lactation Norwegian Cattle using data from 1978 onward. This data set is the largest yet analyzed to investigate the genetics of clinical mastitis. Previous estimates in the Norwegian population have been based on data from much shorter periods (4, 10, 12).

To obtain variance components that are not affected by selection, all data upon which selection was based and complete pedigree information should be included in a mixed model analysis (3). To fulfill this condition, a multiple-trait analysis is required because selection in Norwegian Cattle is based on several traits (13), and mastitis is correlated with other traits in the breeding goal. This type of analysis would be very computationally demanding, but alternative strategies exist to take account of selection. A commonly used alternative is to use first-crop daughters (**FCD**) only [e.g., (8)]. Another strategy would be to account for selection in a bivariate analysis of mastitis and protein yield, as the latter trait has been heavily weighted in the breeding goal. A by-product of the bivariate analysis is estimation of the genetic correlation between mastitis and protein yield. In this paper, the two strategies to avoid selection bias were compared with results from a univariate analysis with use of first- and second-crop daughters (**FSCD**).

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MATERIALS AND METHODS

Data

A research database containing all phenotypic information from the Norwegian Dairy Recording Service (Ås, Norway), from 1978 onward, on an individual cow basis, has been constructed [described by Ruane et al. (9)]. Data are included from different sources: health cards, slaughterhouses, insemination reports, laboratory milk analyses, and farmers. The research database was built up using annual copies of tapes from the Norwegian Dairy Recording Service containing all data on cows culled each year in addition to a copy of all data for animals still alive. Thus, data for all animals recorded since September 1, 1978 are available in the research database at the Department of Animal Science (Agricultural University of Norway, Ås, Norway). The database includes, for example, more than 6 million calving records and more than 6 million health card records, including 2 million of clinical mastitis.

From this data, a pedigree file on an individual basis was constructed for the Norwegian Cattle population. The file contains records on more than 3 million females; the first cow was born in 1958, and, from 1978 onward, an average of 152,000 cows were born each year. Only cows included in the pedigree file were used in the genetic analyses.

From the pedigree file and health card data, a data file was constructed containing information on clinical mastitis during the first lactation for cows calving between September 1, 1978 and December 31, 1995. A record was accepted if age at first calving was between 450 and 1200 d and the lactation started with a normal calving (lactations starting with an abortion or calving in another herd were omitted). To ensure participation in the health recording system, only data from herds with at least one health card recording during the year the cow calved were accepted. The data set contained a total of 1,745,155 first lactation cows. Altogether, 89% of the cows were daughters of 3083 AI sires. A total of 28,672 herds and 358,826 herd by year subclasses were present in the data. The overall frequency of clinical mastitis was 21%, defined as a binary trait based on whether or not the cow had mastitis in the period from 30 d before first calving to 305 d after calving (or to the date of 2nd calving or to the date of culling if either of these events occurred before d 305). These data were used for the calculation of mastitis frequency.

Univariate analysis. For estimating variance components, only daughters of AI bulls that were progeny tested in the years 1978 to 1995 were used. Records of AI sires with fewer than 20 daughters were deleted. Daughters of young bulls only (i.e., FCD) were kept in one data set as one strategy to avoid selection bias. Alternatively, the FSCD of AI bulls were kept in another data set. Records from AI bulls with second-crop daughters only were excluded. The structure of the two data sets is given in Table 1.

TABLE 1. Summary statistics of the data used for univariate variance component analysis. One data set was with first-crop daughters (FCD) only, and one data set was with both first- and second-crop daughters (FSCD).

	FCD	FSCD
Records, no.	514,028	1,229,196
Sires, no.	2043	2043
Herd × year classes, no.	246,960	324,025
Daughters per sire, mean no.	252	602
Records per herd × year class, mean no.	2.1	3.8
Mastitis frequency, %	16	17
Calving, yr	1978–1995	1978–1995

Mastitis resistance was defined as a binary trait on the basis of whether or not the cow had clinical mastitis in the period from 15 d before calving to 120 d after calving, which is the current information period used for calculation of breeding values in Norway. Cows culled before 120 d after calving were included if they had had mastitis. Otherwise, they were excluded because cows culled before the end of the period were not considered to have had a sufficient chance to express the trait.

Bivariate analysis. Selection bias may also be avoided by using a bivariate analysis of mastitis and protein yield. Hence, a data file with first lactation protein yield was constructed using the pedigree file and the same criteria for calving period, age at calving, start of lactation, and minimum number of offspring per sire as for mastitis. Only cows with complete 305-d lactation records were considered. The 305-d protein yield data were merged with the FSCD mastitis data set and were restricted to cows with first calving between September 1, 1978 and December 31, 1989 because of computing limitations. The merged data set included a total of 750,013 cow records of which 728,269 had mastitis data, 617,132 had protein yield data, and 595,388 (79%) had information on both traits. The structure of the data set is given in Table 2.

Sire pedigree file. The pedigree file was built up using sires of cows in the data set as the starting point, and their pedigrees were traced back as far as possible. The pedigree file used in the univariate analyses contained 2159 bulls, the first was born in 1940. In total, 50 bulls had unknown sires, which

TABLE 2. Summary statistics of the data used in the bivariate analysis of clinical mastitis and protein yield.

	Mastitis	Protein yield	Mastitis and protein yield
Records, no.	728,269	617,132	595,388
Sires, no.	1415	1415	1415
Herd × year classes, no.	204,199	205,133	194,534
Daughters per sire, mean no.	515	436	420
Records per herd × year class, mean no.	3.6	3.0	3.1
Mastitis frequency, %	14	...	11
Mean 305-d protein yield, kg	...	166.6	167.1
Calving, yr.	1978–1989	1978–1989	1978–1989

represented the base population. The pedigree file for the bivariate analysis contained 1522 bulls of which 45 had unknown sires.

Model

Univariate analysis. Variance components for mastitis were estimated using the following linear sire model:

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

where

Y_{ijklm} = observation of mastitis (0 = healthy, 1 = diseased);
A_i = fixed effect of age i at calving in 15 classes, where <20 mo is the first class, >32 mo is the last class, and the other classes are in single months;
M_j = fixed effect of month j of calving in 12 classes;
HY_k = fixed effect of herd × year class k;
S_l = random effect of sire l; and
E_{ijklm} = random error term.

Bivariate analysis. In the bivariate analysis, the same model was used for mastitis as in the univariate analysis, and protein yield was analyzed using the following model:

$$Y_{ijklmn} = A_i + M_j + D_k + HY_l + S_m + E_{ijklmn}$$

where

Y_{ijklmn} = observation of 305-d lactation protein yield (in kilograms); and
D_k = fixed effect of class k of days open in 14 classes, where <30 d is the first class, >149 d is the last class, and the other classes are in 10-d intervals.

Other effects are as defined previously for mastitis.

An additive relationship matrix containing the relationship between sires was included in the analyses. (Co)variance components for random effects were estimated with REML using the program VCE4 (6).

RESULTS

Figure 1 shows mastitis frequency per year in the total data set (1,745,155 first lactation cows) in which mastitis is a binomial trait depending on whether the cow had at least one record of clinical mastitis during first lactation. First lactation was defined as the period from 30 d before calving to 305 d after calving or to second calving or culling if they occured before d 305. Mastitis frequency increased from 1978 onward.

Figure 2 shows cumulative mastitis frequency for the same cows and illustrates at which stage of lactation the first case of clinical mastitis occurred. The days around calving were important, as the period from 1 d before calving to 5 d after calving covered more than 35% of all mastitis cases.

Variance components were estimated using either FCD only or all daughters of a sire given that his FCD were present in the data (FSCD). Table 3 shows that the two strategies gave very similar estimates of variance components, although heritability estimates were slightly higher (0.037 vs. 0.035) when based on FCD than on FSCD. The difference was not statistically significant.

Table 4 shows estimated (co)variance components from the bivariate analysis of mastitis and protein

TABLE 3. Variance components and heritability for clinical mastitis estimated with a univariate analyses of data sets with first-crop daughters (FCD) only or with first- and second-crop daughters (FSCD).

	FCD	FSCD
Variance component sire	0.00115[a]	0.00114[b]
Variance component residual	0.12314[a]	0.12810[b]
Total	0.12429	0.12924
Heritability	0.037	0.035

[a]Standard error of the ratios (variance component/total) = 0.0004.

[b]Standard error of the ratios (variance component/total) = 0.0003.

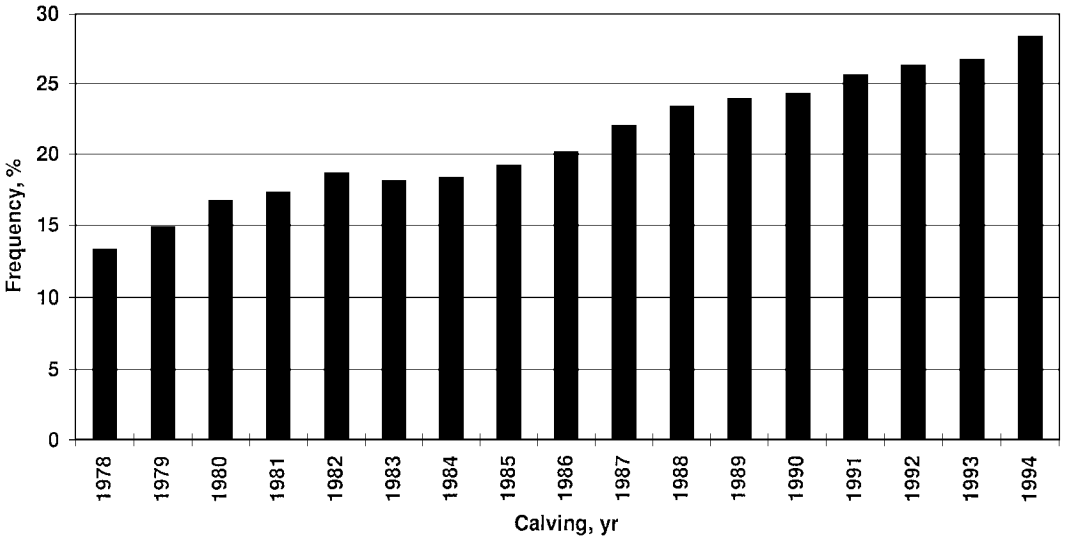

Figure 1. Mastitis frequency per year expressed as the percentage of cows with clinical mastitis during first lactation.

yield in addition to variance components from a univariate analysis of mastitis using the same data. The heritability of clinical mastitis was 0.029 in both the bivariate and univariate analyses. The estimated genetic correlation between mastitis resistance and protein yield was 0.25.

DISCUSSION

This data set is by far the largest yet used to investigate the genetic inheritance of clinical mastitis. The heritability values estimated were in agreement with results previously published. Heringstad et al. (5) summarized the heritability estimates of clinical mastitis based on Nordic field data analyzed with traditional linear models, and they found that estimates of heritability ranged from 0.001 to 0.06; most values were in the interval from 0.02 to 0.03.

Mastitis information for the period from 15 d before calving to 120 d after calving was used for estimation of variance components. The main reason for using

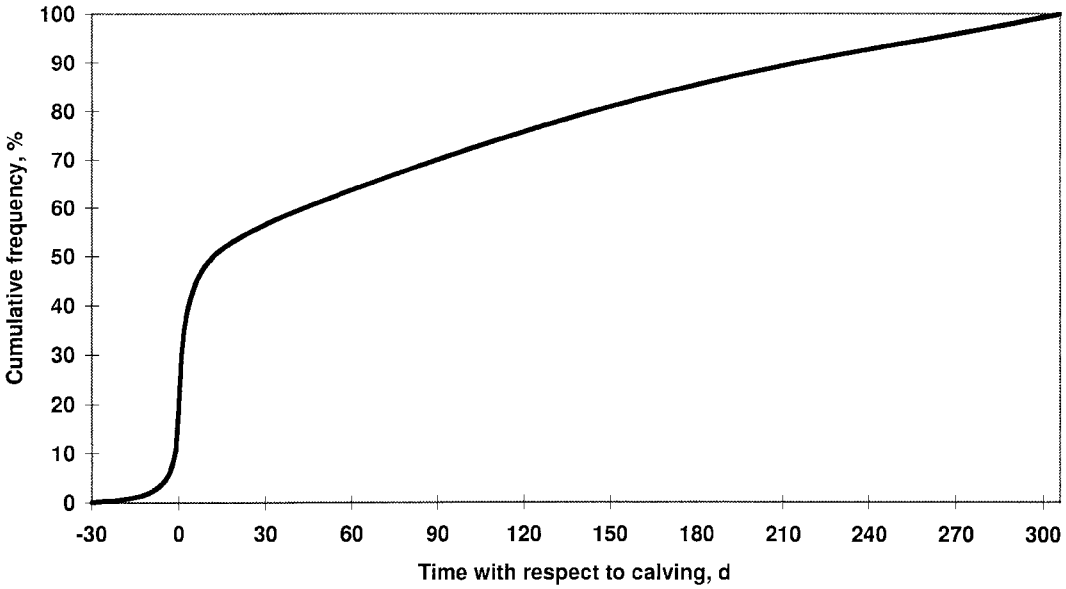

Figure 2. Cumulative mastitis frequency for cows with clinical mastitis during first lactation.

TABLE 4. (Co)variance components for mastitis and protein yield estimated in a bivariate analysis together with a univariate analysis of mastitis on the same data.

	Univariate	Bivariate		
	Mastitis	Mastitis	Protein yield	Mastitis and protein yield
(Co)variance component sire	0.00084[a]	0.00085[b]	22.98296[c]	0.03439[d]
(Co)variance component residual	0.11436[a]	0.11436[b]	477.52070[c]	–0.17783[c]
Total	0.11520	0.11521	500.50366	–0.14344
Heritability	0.029	0.029	0.184	. . .
Genetic correlation	. . .	. . .	. . .	0.25

[a]Standard error of the ratios (variance component/total): 0.0003.

[b]Standard error of the ratios [(co)variance component/total]: 0.0004.

[c]Standard error of the ratios [(co)variance component/total]: 0.001.

[d]Standard error of the ratios [(co)variance component/total]: 0.025.

mastitis information from only a short period of lactation is to avoid bias from culling of cows. In the first part of lactation, the culling rate is low, and, by using the period of 15 d before to 120 d after calving, roughly 76% of all first cases of clinical mastitis should be included in the data (Figure 2). A preliminary study on the effect of sampling period, using a threshold model on a subset of the data, showed that inclusion of information on mastitis before calving resulted in higher heritability estimates than if the period started at calving (4). However, no major differences were found between starting 10, 20, or 30 d prior to calving, and increasing the period after calving to more than 90 d did not influence the heritability estimate. Hence, the chosen sampling period was not thought to have had a significant effect on the size of the heritability estimate.

Although the heritability estimate (Table 3) based on FCD only was higher than the estimate based on FSCD, the difference was small. By including or excluding second-crop daughters, very similar estimates of variance components were found, which indicates that selection bias caused by correlated responses from other traits in the breeding goal is not a major problem in the univariate analysis of mastitis with the amount and structure of data used here. However, using all daughters of a sire, given that FCD were present in the data, improved connectedness of data over years. The effect on heritability estimates was small but using all daughters may be more important in an analysis of genetic trend.

The estimated heritability of mastitis from the bivariate analysis of mastitis and protein yield and the univariate analysis of mastitis on the same data (Table 4) was 0.029, indicating again that with this data, correlated responses caused by selection on protein yield did not bias the estimates. The variance components can therefore be estimated with a univariate analysis as multivariate analyses are much more demanding computationally.

Compared with data set FSCD covering the entire time period (Table 3), the estimated heritability of mastitis was lower (0.029 vs. 0.035) when data from 1990 onward were omitted (Table 4). In addition to the fact that the data set was larger when covering 1978 to 1995 (1,229,196 vs. 728,269 records), the difference between the estimates could also have been due to differences in mastitis frequency for the two data sets (14% vs. 17%). As frequencies increase from 0 to 50% heritability, estimates from linear models when applied to categorical data are expected to increase (2). Mastitis frequency increased from 1978 onward (Figure 1). In the period from 1978, somatic cell counts were introduced as a quality criteria for milk price, and the quality limit has gradually been strengthened over time. This change might have influenced the attention paid by the farmers to mastitis and thus increased the frequency with which the farmer would call the veterinarian to treat the cow. Recording may thus have been improved over time.

The genetic correlation between the continuous trait, protein yield, and the binary trait, mastitis, was estimated with a linear model, and, in theory, this is equal to the genetic correlation between the continuous trait and the underlying liability of the binary trait (2, 14). The estimate of 0.25 found here was in the lower range of estimates of genetic correlation between mastitis and milk yield based on Nordic data (5), ranging from 0.24 to 0.55 with a mean of 0.43. However, simulation studies have demonstrated that linear models under certain conditions, such as low

heritability and low incidence rate, underestimate the genetic correlation (1, 7). In addition, Gates et al. (1) found a greater downward bias for the genetic correlation from a sire model than from an animal model. Because heritability for mastitis is low and mastitis frequency in the data set was 11% (Table 2), the estimated genetic correlation between clinical mastitis and protein yield may have been underestimated with the linear sire model.

CONCLUSIONS

Using the largest data set yet studied for this purpose, clinical mastitis in first lactation Norwegian Cattle was estimated to have a heritability of 0.035, and did not seem to be affected by selection for other traits in the breeding goal. From a bivariate analysis, the genetic correlation between clinical mastitis and protein yield was estimated to be 0.25 and highlighted again the consequences of selecting mainly for milk yield and ignoring mastitis in dairy cattle breeding programs.

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