Bivariate Analysis of Liability to Clinical Mastitis and to Culling in First-Lactation Cows

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ABSTRACT

Records taken on 13,070 first-lactation daughters of 250 Norwegian Cattle sires were used to examine associations between susceptibility to clinical mastitis and to culling. Clinical mastitis was defined as a binary trait, whereas culling was treated as either binary (culled or not culled) or continuous (length of opportunity period) for two sampling periods (120 or 300 d of lactation). Two Bayesian models were employed; 1) a bivariate threshold model with both mastitis and culling as binary traits, and 2) a bivariate model with mastitis as a threshold binary variable and time to culling as Gaussian. The heritability of liability to clinical mastitis was not affected by either the length of sampling period (120 vs. 300 d) or by whether culling (binary) or length of opportunity period was the second trait in the bivariate analysis. The posterior mean (standard deviation) of heritability of liability to clinical mastitis was 0.06 to 0.07 (0.02) in all analyses. The heritability estimate of length of opportunity period was less than 0.001. Culling (threshold trait) in first lactation had a low heritability, but a high genetic correlation with clinical mastitis. The posterior means (standard deviation) for heritability of liability to culling were 0.01 (0.006) for 120 d and 0.02 (0.009) for 300 d, and the posterior means (standard deviation) of the genetic correlation between liability to clinical mastitis and to culling were 0.48 (0.24) and 0.53 (0.21) for 120 and 300 d, respectively.

(Key words: Bayesian methods, clinical mastitis, (co)-variance components, threshold model)

Abbreviation key: CM = clinical mastitis, CU = culling defined as a binary trait, LOP = length of opportunity period, NRF = Norwegian cattle.

INTRODUCTION

Culling of cows results in incomplete lactation records or, statistically speaking, in missing data (Little and Rubin, 1987; Im et al., 1989). In analysis of dairy cattle field records, either including or excluding records of culled cows in the analysis may cause data sampling biases, since culled cows are seldom a random sample. In genetic evaluation of clinical mastitis (CM) in Norway, CM is treated as a binary trait based on whether or not a cow has had at least one case of veterinary treated CM during the period from 15 d before calving to 120 d after first calving (Heringstad et al., 2000). In this setting, a valid question is whether or not records on cows culled before the end of this data sampling period should be included in the analysis? Heringstad et al. (2001a) showed that the data sampling strategy (include or exclude records of culled cows from the analyses) affected heritability estimates of liability to CM. Including all cows, whether culled or not, resulted in a sharper and more symmetric posterior distribution of the heritability. However, culled cows do not have an equal risk of contracting mastitis, relative to cows with complete records, since they have a shorter period of exposure to the disease. To account for this, Heringstad et al. (2001a) included a regression of liability to CM on the “length of opportunity period” in the model. For each cow, this latter variable was the number of days from the start of the sampling period for CM to date of culling or end of sampling period, whichever was first. Another possible method of addressing this problem is to carry out a bivariate analysis of CM and culling. Here, culling can be treated either as a categorical (“culled” or “not culled” during the sampling period) or as a continuous (length of opportunity period) trait.

The objective of this study was to examine associations between liability to CM and liability to culling (binary) or length of opportunity period, respectively, in first-lactation Norwegian cattle (NRF) cows. The focus

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Table 1. Descriptive statistics of the two datasets, 120 d and 300 d.

	120 d	300 d
Number of records	13,070	13,070
Number of sires	250	250
Number of herds	1868	1868
Length of opportunity period, mean, d	139	278
Length of opportunity period, SD, d	29	93
Length of opportunity period, range, d	31–150	31–330
Mastitis frequency, % cows with CM ¹ = 1	18.1	23.3
Culling rate, % cows with CU ² = 1	14.8	30.3
Culling rate for cows with CM ¹ = 1, %	19.9	33.8
Culling rate for cows with CM ¹ = 0, %	13.6	29.2
Mastitis frequency for cows with CU ² = 1, %	24.4	26.0
Mastitis frequency for cows with CU ² = 0, %	17.0	22.0

¹CM = Binary trait based on whether the cow had clinical mastitis (CM = 1) or not (CM = 0) before the end of the sampling period (120 d or 300 d).

²CU = Binary trait based on whether the cow was culled (CU = 1) or not (CU = 0) before the end of the sampling period (120 d or 300 d).

was on inferring genetic parameters, especially the genetic correlation between the two traits. Bivariate analyses suitable for clinical mastitis data were used for this purpose.

MATERIALS AND METHODS

Data and Trait Definitions

The data consisted of CM and culling information for the 13,070 first-lactation cows from the NRF population analyzed by Heringstad et al. (2001a). Data on first-lactation daughters of all young sires having their first progeny test in 1993 or 1994 were used, and only records from herds with at least five first-lactation daughters of these test bulls were kept. A total of 250 sires and 1868 herds were represented in the data, and the pedigree file included 482 sires. Two sampling periods were considered, going from 30 d before calving to either 120 or 300 d after calving (datasets 120 and 300 d, respectively). These sampling periods were chosen because 120 d is the period currently used in genetic evaluation of CM in Norway, whereas 300 d represents information from a full first lactation.

Mastitis was defined as a binary trait, based on whether or not the cow had at least one veterinary treatment of CM during a sampling period. Culling (CU) was a binary trait, based on whether or not the cow had been culled before the end of the sampling period. Length of opportunity period (LOP) for each cow was defined as the number of days from 30 d before calving to date of culling or to the end of sampling period, whichever occurred first. Descriptive statistics for the two datasets (120 d and 300 d) are given in Table 1. The frequency of CM was 18.1% in dataset 120 d, and 23.3% in dataset 300 d. About 15% of the cows were culled before 120 d of lactation, and 30% were

culled before 300 d. The mean of LOP was 139 d in dataset 120 d, and 278 d in dataset 300 d. This indicates roughly that cows stayed in the herd, on average, for about 93 and 84% of the opportunity periods considered in datasets 120 d and 300 d, respectively. Culling rate for cows with CM were higher than for those that did not contract the disease, as expected, and the mastitis frequency was higher in culled cows.

Model

Two statistical models for the analysis of joint binary and continuous responses were used. Model 1 was a bivariate threshold model with both CM and CU as binary traits possessing an underlying bivariate normal distribution. The application of such models in the animal breeding field goes back to Hoeschele et al. (1986) and Foulley et al. (1987), using marginal maximum likelihood or empirical Bayes methods. These are an extension of the univariate threshold model of Gianola and Foulley (1983). However, recent advances in Markov chain Monte Carlo (MCMC) methods facilitate the implementation of multivariate binary threshold models and make their use possible, even with large datasets. Model 2 was a bivariate model, with CM as binary (threshold) and log(LOP) as Gaussian. Here, it was postulated that liability to CM and log(LOP) were jointly Gaussian. This model was developed by Foulley et al. (1983).

Models 1 and 2 were fitted to data for each of the sampling periods (120 and 300 d), and the following linear sire model was used for all three traits:

$$y_{ijkl} = AS_i + h_j + s_k + e_{ijkl}$$

where y_{ijkl} is the liability to CM or to CU, or log(LOP), of daughter l of sire k calving in herd j and age $\times$ season

class i ; AS_i is the effect of age $\times$ season class i , in 20 classes from 5 age (<21, 21 to 23, 24 to 26, 27 to 29, and >29 mo) and 4 season (March–May, June–August, September–November, December–February) levels; h_j is the effect of herd j ; s_k is the transmitting ability of sire k , and e_{ijkl} is a residual.

In matrix notation, each of the two models can be represented as:

$$\mathbf{y} = \mathbf{X}\beta + \mathbf{Zs} + \mathbf{e}$$

where, $\mathbf{y}$ is a vector of liabilities to CM and liabilities to CU or log(LOP), β is a vector of systematic effects (age $\times$ season and herd effects) of order p , $\mathbf{s}$ is a vector of sire effects of order r , and $\mathbf{e}$ is a vector of residual effects. $\mathbf{X}$ and $\mathbf{Z}$ are the corresponding incidence matrices with the appropriate dimensions.

Bayesian Analysis

A Bayesian approach was adopted for inferences, with MCMC sampling employed in the implementation (e.g., Sorensen et al., 1995). Conditionally on the model parameters, it was assumed that the sampling distribution of the observations was:

$$p(\mathbf{y} | \beta, \mathbf{s}) \sim N(\mathbf{X}\beta + \mathbf{Zs}, \mathbf{I} \otimes \mathbf{R}_0) \quad [1]$$

where $\mathbf{R}_0$ is a 2×2 variance-covariance matrix with the following structure:

$$\mathbf{R}_0 = \begin{bmatrix} \sigma_{e1}^2 & \sigma_{e12} \\ \sigma_{e21} & \sigma_{e2}^2 \end{bmatrix} \text{ with } \begin{cases} \sigma_{e1}^2 = \sigma_{e2}^2 = 1 \text{ for Model 1 and} \\ \sigma_{e1}^2 = 1 \text{ for Model 2} \end{cases}$$

In Model 1, the two residual variances were set equal to 1, since the parameters are not identifiable.

Prior distributions. The vector of systematic effects was partitioned into two sub-vectors $\beta = (\beta'_R, \beta'_H)'$, where β_R and β_H are vectors of age $\times$ season effects and of herd effects, respectively. The prior distribution for the vector β_R was:

$$p(\beta_R | \sigma_R^2) \sim N(\mathbf{0}, \mathbf{I}\sigma_R^2) \quad [2]$$

with $\sigma_R^2 = 10^6$. The implication is that the systematic effects had independent, vague, prior distributions. The average number of records per herd was small, so that many herds were either all 0s or all 1s for any of the two binary traits. This causes well-known problems in analysis of threshold models. Hence, herd effects were assigned a normal prior with unknown mean and variance. This is based on results of Rekaya et al. (2000), who found that this prior distribution for herd effects

alleviated the “extreme category” problems in such data. The prior distribution for β_h was:

$$p(\beta_H | \eta, \mathbf{H}) \sim N(\eta, \mathbf{I} \otimes \mathbf{H}) \quad [3]$$

where $\eta = (\mathbf{1}'\eta_1, \mathbf{1}'\eta_2)'$ is a vector of mean herd effects, with η_1 = mean herd effect for CM and η_2 = mean herd effect for CU or log(LOP). Further,

$$\mathbf{H} = \begin{bmatrix} \sigma_h^2 & \sigma_{h12} \\ \sigma_{h12} & \sigma_{h2}^2 \end{bmatrix}$$

is a 2×2 (co)variance matrix and $\mathbf{I}$ is an identity matrix of order 1868×1868 . Both η_1 and η_2 and the elements of $\mathbf{H}$ were assumed unknown and, hence, priors were specified as follows:

$$p(\eta_i) \sim U[-10, 10], i = 1, 2. \quad [4]$$

The diagonal elements of $\mathbf{H}$ were assigned the following uniform prior distributions:

$$\begin{aligned} p(\sigma_{hi}^2) &\sim U[0, 1] \text{ for } i = 1, 2 \text{ in model 1 and} \\ &\text{for } i = 1 \text{ in model 2, and} \\ p(\sigma_{h2}^2) &\sim U[0, 100] \text{ in model 2.} \end{aligned} \quad [5]$$

For the unknown off-diagonal element of $\mathbf{H}$, the following uniform prior was used to ensure that correlations were between -1 and 1 :

$$p(\sigma_{h12}) \sim U\left[-\sqrt{\sigma_{h1}^2 \sigma_{h2}^2}, \sqrt{\sigma_{h1}^2 \sigma_{h2}^2}\right] \quad [6]$$

Note that the parameter space includes other parameters. This implies that a maximum likelihood analysis could not be undertaken because of lack of regularity conditions. This does not pose any difficulties in our Bayesian analysis. A multivariate normal distribution was adopted as prior for the sire effects:

$$p(\mathbf{s} | \mathbf{A}, \mathbf{G}_0) \sim N(\mathbf{0}, \mathbf{A} \otimes \mathbf{G}_0) \quad [7]$$

where $\mathbf{G}_0 = \begin{bmatrix} \sigma_{s1}^2 & \sigma_{s12} \\ \sigma_{s12} & \sigma_{s2}^2 \end{bmatrix}$ is the between-sire (co)variance matrix, and $\mathbf{A}$ is the additive relationship matrix between sires, of order 482×482 . The following uniform bounded priors were assigned to the elements of matrix $\mathbf{G}_0$:

$$\begin{aligned} p(\sigma_{si}^2) &\sim U[0, 1/3] \text{ for } i = 1, 2 \text{ in model 1 and} \\ &\text{for } i = 1 \text{ in model 2,} \end{aligned}$$

$p(\sigma_{s2}^2) \sim U[0, 200/3]$ in model 2,

and

$$p(\sigma_{s12}) \sim U\left[-\sqrt{\sigma_{s1}^2 \sigma_{s2}^2}, \sqrt{\sigma_{s1}^2 \sigma_{s2}^2}\right] \quad [8]$$

For the residual (co)variance matrix $\mathbf{R}_0$, the diagonal elements were set to 1 in model 1. Hence, the only unknown in the matrix is the covariance, for which the following bounded prior was specified:

$$p(\sigma_{e12}) \sim U[-1, 1].$$

For model 2, the following priors were assigned to the residual variance of log(LOP) and to the residual covariance between CM and log(LOP):

$$\begin{aligned} p(\sigma_{e2}^2) &\sim U[0, 200] \quad \text{and} \\ p(\sigma_{e12}) &\sim U\left[-\sqrt{\sigma_{e2}^2}, \sqrt{\sigma_{e2}^2}\right]. \end{aligned} \quad [9]$$

For model 2, for example, the joint prior density had the form:

$$\begin{aligned} p(\beta_R, \eta_1, \eta_2, \mathbf{H}, \mathbf{G}_0, \sigma_{e12}, \sigma_{e2}^2) = \\ p(\beta_R) p(\beta_H|\eta_1, \eta_2, \mathbf{H}) p(\eta_1) p(\eta_2) p(\mathbf{H}) p(\mathbf{s}|\mathbf{G}_0) p(\mathbf{G}_0) p(\sigma_{e12}) p(\sigma_{e2}^2) \end{aligned}$$

Posterior density. The joint posterior density is proportional to the product of the density of the conditional distribution of the observations times the joint prior density. In model 1, the sampling models were of a multinomial type where, for each cow, there are four possibilities: (CM⁺, CU⁺), (CM⁺, CU⁻), (CM⁻, CU⁺), (CM⁻, CU⁻). For example (CM⁺, CU⁺) denotes the event that a cow had mastitis and that she was culled before the end of the current opportunity period. Over all cows (dataset 300 d) the raw frequencies of cows in the 4 classes was: 0.54 (CM⁻, CU⁻), 0.23 (CM⁻, CU⁺), 0.15 (CM⁺, CU⁻), and 0.08 (CM⁺, CU⁺). The probability of observing an elementary event was modeled as a bivariate standard normal integral with correlation σ_{e12} (here, the covariance is equal to the correlation). Since a sire model was used then, given β_R , β_H , and $\mathbf{s}$, the observations for a given cow are conditionally independent of those from any other cow. In model 2, the sampling model is one where the observations were Gaussian for log(LOP). Again, given the effects, LOP observations are independent between cows. Further, given LOP and all effects, the records for CM are also independent between cows, with a probability of CM that is equal to an univariate normal integral where the mean of the

underlying distribution depends on log(LOP), and the variance is equal to $1 - \frac{\sigma_{e12}^2}{\sigma_{e2}^2}$.

Draws from the conditional posterior distribution of all the parameters were obtained using a Gibbs sampler with data augmentation (Sorensen et al., 1995). For model 1, the joint posterior was augmented with the bivariate normal liabilities for each cow. In model 2, the joint posterior was augmented with the univariate normal liabilities for CM. After augmentation, all the fully conditional posterior distributions of all model parameters can be derived as described by Albert and Chib (1993) and Sorensen et al. (1995). These are as follows: 1) normal for the location parameters and sire effects, 2) truncated normal for the liabilities, and 3) scaled-inverted Wishart distributions for the sire and herd variances. Liabilities were sampled from inverse cumulative normal distributions (Devroye, 1986). The conditional distributions of the residual covariance σ_{e12} (for models 1 and 2) and of the residual variance of log(LOP), σ_{e2}^2 (model 2), can be derived as follows. First note that σ_{e12} and σ_{e2}^2 are parameters of the residual structure so, given the data and all other location parameters, one can act as if the true residuals were known.

For [1], we have:

$$p(\mathbf{e}_1, \mathbf{e}_2|\mathbf{R}_0) \sim N(\mathbf{0}, \mathbf{I} \otimes \mathbf{R}_0).$$

The joint density of $\mathbf{e}_1$ and $\mathbf{e}_2$ can be written as the product of the marginal and conditional densities:

$$p(\mathbf{e}_1, \mathbf{e}_2|\mathbf{R}_0) = p(\mathbf{e}_1|\mathbf{R}_0) p(\mathbf{e}_2|\mathbf{e}_1, \mathbf{R}_0),$$

where

$$p(\mathbf{e}_1|\mathbf{R}_0) \sim N(\mathbf{0}, \mathbf{I}\sigma_{e1}^2), \quad [10]$$

$$\text{and, } p(\mathbf{e}_2|\mathbf{e}_1, \mathbf{R}_0) \sim N\left(\frac{\sigma_{e12}}{\sigma_{e1}^2}\mathbf{e}_1, \mathbf{I}\sigma_{e2}^{2*}\right)$$

where

$$\sigma_{e2}^{2*} = \sigma_{e2}^2 \left(1 - \frac{\sigma_{e12}^2}{\sigma_{e1}^2}\right). \quad [11]$$

From [10] and [11], we can write,

$$\mathbf{e}_2 = \sigma_{e12} \left(\frac{\mathbf{e}_1}{\sigma_{e1}^2}\right) + \mathbf{e}_2^* = \sigma_{e12}\boldsymbol{\varsigma} + \mathbf{e}_2^* \quad [12]$$

$$\text{where } \boldsymbol{\varsigma} = \frac{\mathbf{e}_1}{\sigma_{e1}^2}$$

and

$$\mathbf{e}_2^* \sim N(\mathbf{0}, \mathbf{I}\sigma_{e2}^{2*}).$$

For model 1, the diagonal elements of $\mathbf{R}_0$ are known, and the only element that needs to be sampled from the residual (co)variance matrix is σ_{e12} . Using [12], the fully conditional posterior distribution is:

$$p(\sigma_{e12}|\mathbf{e}_1, \mathbf{e}_2, \sigma_{e2}^{2*}) \sim N[(\zeta'\zeta)^{-1}\zeta'\mathbf{e}_2, (\zeta'\zeta)^{-1}\sigma_{e2}^{2*}]. \quad [13]$$

For model 2, the second diagonal element of $\mathbf{R}_0$, σ_{e2}^{2*} , and σ_{e12} are unknown. The conditional distribution of the latter is as in [13]. For σ_{e2}^{2*} , using [12], it can be shown that its conditional distribution is the scaled-inverted chi square process:

$$p(\sigma_{e2}^{2*}|\mathbf{e}_1, \mathbf{e}_2, \sigma_{e12}) \sim (N-2) \mathbf{e}_2^* \mathbf{e}_2^* \chi_{N-2}^{-2} \quad [14]$$

where $\mathbf{e}_2^* = \mathbf{e}_2 - \sigma_{e12}\zeta$.

Convergence diagnostics. Length of burn-in and of sampling period, and a measure of mixing rate were obtained by the method of Raftery and Lewis (1992), as implemented in the CODA software (Best et al., 1995), using the first 20,000 iterations of the Gibbs chain. Based on the diagnostics and visual inspection of trace plots, total chain lengths of 290,000 and 390,000 iterations were run, after a "burn in" of 10,000 rounds, for models 1 and 2, respectively. Monte Carlo standard errors of estimates of the posterior mean, calculated from between-batch variation, were all < 0.0005.

RESULTS AND DISCUSSION

Mean and standard deviations of the posterior distributions of the (co)variance components and of the corresponding heritabilities and correlations from the bivariate analyses are given in Table 2. The posterior means (SD) of heritability of liability to CM were between 0.06 and 0.07 (0.02) in all analyses (Table 2). These point estimates are in agreement with published results from analyses of Nordic mastitis data using threshold models, with estimates ranging from 0.06 to 0.12 (Simianer et al., 1991; Lund et al., 1999; Heringstad et al., 2001a). As shown in Figure 1, the posterior distribution of heritability of liability to CM was nearly invariant with respect to whether CU or log(LOP) was the second trait in the bivariate analysis, or with respect to the length of the sampling period (120 vs. 300 d). The latter is in agreement with Heringstad et al. (2001b), who found that heritability of CM estimated with linear models on the 0–1 scale and transformed to the liability scale were fairly constant with increasing length of the sampling period. Lund et al. (1999), using

linear models, found heritability of liability to clinical mastitis to be higher early in lactation. It is known that heritability estimates for "all or none" data tend to be frequency dependent and, therefore, are difficult to interpret (Dempster and Lerner, 1950; Gianola, 1982).

Figure 2 compares posterior distributions of heritability of liability to CM from our bivariate analyses with results from univariate analyses of the same data as published by Heringstad et al. (2001a). The posterior distributions in the bivariate analyses were more peaked and had a slightly higher mean. This illustrates that somewhat sharper inferences about CM can be obtained in the bivariate analysis. In the univariate case, a lower posterior mean was estimated in dataset 300 d than in 120 d, whereas in the bivariate analyses the posterior distributions for the two datasets were nearly identical. This suggests that the bivariate analyses may remove any effects of data sampling bias, caused by culling of cows, in analyses of CM data. The main reason for using CM information only from the first part of lactation in genetic evaluation (e.g., 120 d) is to avoid possible biases due to culling of cows (Heringstad et al., 2001a). Models and methods that can use information from a longer period of lactation and that eliminate sampling biases may be advantageous for genetic evaluation of CM.

Heritability of liability to CU was low, with posterior means below 0.02 (Table 2), and it had a skewed distribution (Figure 3), because of the proximity to the edge of the parameter space. The posterior distribution for 300 d had a slightly higher mean than for 120 d and it had a thicker right tail (Figure 3). This higher variation probably reflects that the culling rate was 30% for 300 d versus 15% for 120 d.

The posterior distribution of the genetic correlation between liability to CM and liability to CU was skewed and spanned essentially the positive part of the parameter space (Figure 4). The genetic correlation could not be estimated very precisely in any of the two datasets, but the results suggest a positive and probably high genetic correlation between CM and CU. The posterior means (SD) of the genetic correlation were 0.48 (0.24) and 0.53 (0.21) for datasets 120 d and 300 d, respectively (Table 2). A positive genetic correlation means that, genetically, cows with a higher liability to mastitis have a higher risk of being culled. Other studies have also reported a high genetic correlation between mastitis and culling. For example, in a bivariate analysis of CM and culling using linear models, Heringstad et al. (2001b) found a genetic correlation of 0.33 between the two traits. In analysis of mastitis and functional longevity with Danish Black and White dairy cattle, Neerhof et al. (2000) found that the correlation between predicted breeding values for mastitis resistance and risk

Table 2. Means and standard deviations (SD) of the posterior distribution of (co)variance components, heritabilities and correlations, for two bivariate models: 1) liability to clinical mastitis (CM) and liability to culling (CU), and 2) liability to clinical mastitis (CM) and log-length of opportunity period (log(LOP)), for datasets 120 d and 300 d.

	1) Bivariate CM and CU (trait 2 = CU)				2) Bivariate CM and log (LOP) (trait 2 = log (LOP))			
	120 d		300 d		120 d		300 d	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
σ_s^2 CM ¹	0.020	0.006	0.019	0.005	0.020	0.006	0.019	0.005
h^2 CM ²	0.067	0.019	0.065	0.018	0.067	0.018	0.064	0.018
σ_s^2 trait 2 ¹	0.003	0.002	0.005	0.003	1×10^{-5}	8×10^{-6}	1×10^{-5}	8×10^{-6}
h^2 trait 2 ²	0.011	0.006	0.017	0.009	0.0005	0.0003	0.0002	0.0001
σ_{s12} ³	0.004	0.003	0.005	0.003	-0.0004	0.0002	-0.0004	0.0002
r_s ³	0.48	0.24	0.53	0.21	-0.75	0.18	-0.75	0.17
σ_h^2 CM ¹	0.175	0.020	0.160	0.018	0.174	0.020	0.161	0.018
σ_h^2 trait 2 ¹	0.216	0.022	0.128	0.014	0.011	0.001	0.034	0.002
σ_{h12} ³	-0.020	0.013	-0.031	0.010	0.003	0.003	0.014	0.004
r_h ³	-0.11	0.07	-0.22	0.07	0.06	0.06	0.19	0.06
σ_e^2 CM ¹	1	0	1	0	1	0	1	0
σ_e^2 trait 2 ¹	1	0	1	0	0.091	0.001	0.291	0.004
σ_{e12} ³	0.192	0.023	0.114	0.018	-0.011	0.004	-0.015	0.007
r_e ³	0.19	0.02	0.11	0.02	-0.04	0.01	-0.03	0.01

¹ σ_s^2 = sire variance, σ_h^2 = herd variance, and σ_e^2 = residual variance.

² $h^2 = 4\sigma_s^2/(\sigma_s^2 + \sigma_h^2 + \sigma_e^2)$.

³ σ_{s12} , σ_{h12} , and σ_{e12} are the sire, herd, and residual covariances between the two traits, respectively, and r_s , r_h and r_e are the corresponding correlations.

of culling was -0.4, i.e., resistance against mastitis was genetically correlated with a lower risk of being culled.

The heritability of log(LOP) was nil. A strong genetic correlation, -0.75, was estimated between liability to CM and log (LOP) (Table 2). This indicates, essentially, that cows that are genetically susceptible to CM tend to be culled earlier. This estimate is not very reliable because the sire variance for log(LOP) was 1×10^{-5} and the posterior SD was 8×10^{-6} for both periods (Table 2). It should be noted that CM and LOP act recursively, in some sense. Cows may have a shorter LOP because

they have had mastitis and, conversely, they may have mastitis because they had a longer LOP. At least in context of maximum likelihood estimation, ignoring such recursiveness between dependent variables leads to inconsistent estimates (Johnston, 1972). Recursive estimation techniques are probably needed to account for this in a statistically optimal manner.

The correlation between herd effects was negative for CM and CU (-0.11 and -0.22 in datasets 120 d and 300 d, respectively), and positive for CM and log(LOP) (0.06 and 0.19 in datasets 120 d and 300 d, respectively) (Table 2). This suggests that herds in which CU rate

Figure 1. Posterior distribution of heritability ($h^2 = 4\sigma_s^2/(\sigma_s^2 + \sigma_h^2 + \sigma_e^2)$) of liability to clinical mastitis (CM) from bivariate analyses of liability to CM and liability to culling (CMCU) and from bivariate analyses of liability to CM and log length of opportunity period (CMLOP), for the two sampling periods (120 and 300 d).

Figure 2. Posterior distributions of heritability ($h^2 = 4\sigma_s^2/(\sigma_s^2 + \sigma_h^2 + \sigma_e^2)$) of liability to clinical mastitis (CM) from bivariate analyses of liability to CM and liability to culling (CMCU) and from univariate analyses of the same datasets (uni CM) for the two sampling periods (120 and 300 d).

Figure 3. Posterior distribution of heritability ($h^2 = 4\sigma_s^2/(\sigma_s^2 + \sigma_e^2)$) of liability to culling (CU) from bivariate analyses of liability to CM and liability to CU for the two sampling periods (120 and 300 d).

Figure 4. Posterior distribution of the genetic correlation between liability to clinical mastitis (CM) and liability to culling (CU) for the two sampling periods (120 and 300 d).

is higher tend to have lower liability to mastitis, as expected. Again, the estimates between CM and log(LOP) should be interpreted cautiously because of the recursive relationship between the traits.

The residual correlation between liability to CM and liability to CU was 0.19 (120 d) and 0.11 (300 d), whereas that between CM and log(LOP) was slightly negative (Table 2). This suggests that cow-specific environmental factors that make a cow more liable to contracting CM may make her more liable to culling and to having a shorter LOP as well.

It may be more efficient to reduce culling rate in first lactation by selecting against mastitis than by direct selection for decreased culling rate (or for a longer survival in first lactation). Assuming equal selection intensity, and linearity, the efficiency of indirect selection

relative to direct selection is given by $\alpha = \frac{r_g \sqrt{h_{CM}^2}}{\sqrt{h_{CU}^2}}$, where

h_{CM}^2 is the heritability of CM, h_{CU}^2 is the heritability of CU, and r_g is the genetic correlation between CM and CU, with all parameters in the probit scale. Using draws from the Gibbs sampler, the posterior mean (SD) of α was 1.27 (0.7) and 1.13 (0.54) in datasets 120 d and 300 d, respectively. Although the efficiency parameter cannot be estimated very precisely, the Bayesian analysis suggests that selection for increased resistance to mastitis may produce a larger response to selection for first-lactation survival than direct selection for increased survival.

CONCLUSION

The heritability of liability to CM was not affected much by either the length of the sampling period (120

d vs. 300 d) or by whether CU or log(LOP) was the second trait in the bivariate analysis. Culling, defined as a binary trait in first lactation, had a low heritability and a high genetic correlation with CM. The sire variance of log(LOP) was close to 0 for both sampling periods. Therefore, CU is probably a better trait in bivariate analyses, while LOP could be included as a regressor in a univariate analysis of CM. The results indicate that a bivariate analysis may be useful to remove effects of data sampling bias, caused by culling of cows.

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REFERENCES

- Albert, J., and S. Chib. 1993. Bayesian analysis of binary and polychotomous response data. *J. Am. Statist. Assoc.* 88:669–679.
- Best, N. G., M. K. Cowles, and S. K. Vines. 1995. CODA Manual version 0.30. MRC Biostatistics Unit, Cambridge, UK. 41 pp.
- Dempster, E. R., and I. M. Lerner. 1950. Heritability of threshold characters. *Genetics* 35:212–235.
- Devroye, L. 1986. *Non-Uniform Random Variate Generation*. Springer Verlag.
- Foulley, J. L., D. Gianola, and R. Thompson. 1983. Prediction of genetic merit from data on binary and quantitative variates with an application to calving difficulty, birth weight, and pelvic opening. *Genet. Sel. Evol.* 15:401–424.
- Foulley, J. L., S. Im, D. Gianola, and I. Hoeschele. 1987. Empirical Bayes estimation of genetic value for n binary traits. *Genet. Sel. Evol.* 19:197–224.

- Gianola, D. 1982. Theory and analysis of threshold characters. *J. Anim. Sci.* 54:1079–1096.
- Gianola, D., and J. L. Foulley. 1983. Sire evaluation for ordered categorical data with a threshold model. *Genet. Sel. Evol.* 15:201–223.
- Heringstad, B., G. Klemetsdal, and J. Ruane. 2000. Selection for mastitis resistance in dairy cattle—A review with focus on the situation in the Nordic countries. *Livest. Prod. Sci.* 64:95–106.
- Heringstad, B., R. Rekaya, D. Gianola, G. Klemetsdal, and K. A. Weigel. 2001a. Bayesian analysis of liability to clinical mastitis in Norwegian Cattle with a threshold model; Effects of data sampling method and model specification. *J. Dairy Sci.* 84:2337–2346.
- Heringstad, B., G. Klemetsdal, and J. Ruane. 2001b. Variance components of clinical mastitis in dairy cattle—effects of trait definition and culling. *Livest. Prod. Sci.* 67:265–272.
- Hoeschele, I., J. L. Foulley, J. J. Colleau, and D. Gianola. 1986. Genetic evaluation for multiple binary responses. *Genet. Sel. Evol.* 18:299–320.
- Im, S., R. L. Fernando, and D. Gianola. 1989. Likelihood inferences in animal breeding under selection—a missing-data theory view point. *Genet. Sel. Evol.* 21:399–414.
- Johnston, J. 1972. *Econometric Methods*. 2nd ed. McGraw-Hill, New York.
- Little, R. J. A., and D. B. Rubin. 1987. *Statistical Analysis with Missing Data*. Wiley, New York.
- Lund, M. S., J. Jensen, and P. H. Petersen. 1999. Estimation of genetic and phenotypic parameters for clinical mastitis, somatic cell deviance, and protein yield in dairy cattle using Gibbs sampling. *J. Dairy Sci.* 82:1045–1051.
- Neerhof, H. J., P. Madsen, V. P. Ducrocq, A. R. Vollema, J. Jensen, and I. R. Korsgaard. 2000. Relationships between mastitis and functional longevity in Danish Black and white dairy cattle estimated using survival analysis. *J. Dairy Sci.* 83:1064–1071.
- Raftery, A. L., and S. Lewis. 1992. How many iterations in the Gibbs sampler? Pages 763–774 in *Bayesian Statistics 4*. J. M. Bernardo, J. O. Berger, A. P. Dawid, and A. F. M. Smith, eds. Oxford University Press, Oxford, UK.
- Rekaya, R., K. A. Weigel, D. Gianola, B. Heringstad, and G. Klemetsdal. 2000. Methods for attenuating bias of variance component estimates in threshold models when herds are small. *J. Dairy Sci.* 83(Suppl. 1):56–57.
- Simianer, H., H. Solbu, and L. R. Schaeffer. 1991. Estimated genetic correlations between disease and yield traits in dairy cattle. *J. Dairy Sci.* 74:4359–4365.
- Sorensen, D. A., S. Andersen, D. Gianola, and I. Korsgaard. 1995. Bayesian inference in threshold models using Gibbs sampling. *Genet. Sel. Evol.* 27:229–249.