Short Communication: Genetic Analysis of Nonreturn Rate and Mastitis in First-Lactation Norwegian Red Cows

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ABSTRACT

Associations between clinical mastitis (CM) and nonreturn rate within 56 d after first insemination (NR56) were examined in Norwegian Red (NRF) cows. Records on absence or presence of CM within each of the intervals, –30 to 30, 31 to 150, and 151 to 300 d after first calving, and records on NR56 for 620,492 first-lactation daughters of 3,064 NRF sires were analyzed with a Bayesian multivariate threshold liability model. Point estimates of genetic correlations between NR56 and the 3 CM traits were between –0.05 and –0.02. Residual correlations were close to zero, and correlations between herd-5-yr effects on NR56 and CM in the 3 lactation intervals ranged from –0.15 to –0.17. It appears that CM and NR56 in first lactation are independent traits.

Key words: female fertility, genetic correlation, mastitis, multivariate threshold model

Mastitis and female fertility have been included in the total merit index used for selection of Norwegian Red (NRF) sires since the 1970s. The traits used in genetic evaluation have been absence or presence of clinical mastitis (CM) in the interval from 15 d before to 120 d after first calving (CM120), and nonreturn rate within 56 d after first insemination (NR56).

Andersen-Ranberg and Heringstad (2006) estimated a genetic correlation close to zero between NR56 and CM120 in NRF using a linear model. Other studies have reported negative correlations between similar traits. Pryce et al. (1998) and Kadarmideen et al. (2000) reported genetic correlations between mastitis and conception to first service of –0.58 and –0.21, respectively, for Holstein cattle in the United Kingdom. A negative genetic correlation is favorable in the sense that selection against mastitis would be expected to produce a positive correlated response in fertility (increased nonreturn rate) and vice versa.

Heringstad et al. (2004) investigated genetic correlations between liability to CM in 12 intervals of the first 3 lactations in NRF, and estimates ranged from 0.24 to 0.73. These values suggest that mastitis is not the same trait throughout lactation. Hence, it is possible that the genetic correlation between CM and NR56 varies throughout lactation as well. The objective of this study was to infer genetic correlations between CM in different intervals of lactation and NR56 in first-lactation NRF cows.

Mastitis and fertility data on the cows included in the study of Heringstad et al. (2006) were used. The data set had records on 620,492 first-lactation daughters of 3,064 NRF sires. First lactation was divided into 3 intervals: from 30 d before to 30 d after calving, from 31 to 150 d, and from 151 to 300 d. The second interval represents the period during which most first inseminations take place. Within each of these intervals, absence or presence of CM was scored as 0 or 1 based on whether the cow had at least one veterinary treatment of CM recorded in the interval. Mean frequency of CM was 11% in the first interval, 6% in the second interval, and 5% in the last interval. About 3% of the cows were culled before 31 d, and 8% were culled between 31 and 150 d; these cows had missing CM information for the second and third, or the third interval, respectively. A total of 475,270 cows (77%) had NR56 records. The NR56 was scored as 0 or 1 based on whether the cow had a second insemination (other than double insemination, defined as a new service within 5 d) within 56 d after the first one; mean NR56 was 0.68. The sire pedigree file had 3,756 males, including the 3,064 sires with daughter records in the data set.

A 4-variate threshold-liability model (e.g., Gianola, 1982; Foulley et al., 1987) was used. Similar models have been used for analyzing CM in different lactation intervals (Chang et al., 2004a; Heringstad et al., 2004). In matrix notation the model fitted can be written as:

$$\lambda = \mathbf{X}\beta + \mathbf{Z}_h\mathbf{h} + \mathbf{Z}_s\mathbf{s} + \mathbf{e}$$

where λ is a vector of unobserved liabilities for the 4 traits; β is a vector of trait-specific systematic effects,

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Table 1. Posterior means (SD) of heritability (on the diagonal) and genetic correlations (above the diagonal) of liability to nonreturn rate within 56 d (NR56) and clinical mastitis (CM) in the intervals –30 to 30, 31 to 150, and 151 to 300 d after first calving

	NR56	CM (–30 to 30)	CM (31 to 150)	CM (151 to 300)
NR56	0.02 (0.002)	–0.02 (0.05)	–0.03 (0.06)	–0.05 (0.06)
CM (–30 to 30)	—	0.09 (0.004)	0.57 (0.04)	0.46 (0.04)
CM (31 to 150)	—	—	0.05 (0.004)	0.85 (0.02)
CM (151 to 300)	—	—	—	0.05 (0.004)

including age at first calving (21 levels) and month $\times$ year of calving (288 levels) effects; $\mathbf{h}$ is a vector of herd-5-yr period of calving effects (51,808 levels); $\mathbf{s}$ is a vector of sire transmitting abilities (with $3,756 \times 4 = 15,024$ elements); $\mathbf{e}$ is a vector of residuals, and $\mathbf{X}$, $\mathbf{Z}_h$, and $\mathbf{Z}_s$ are the corresponding known incidence matrices. All residual variances were set equal to 1. Residuals were regarded as independent between cows but correlated within cows and assumed to follow the multivariate normal distribution: $\mathbf{e} \sim N(\mathbf{0}, \mathbf{R}_0 \otimes \mathbf{I})$, where $\mathbf{R}_0$ is the 4×4 residual (co)variance matrix, with all diagonals equal to 1, as stated earlier. Andersen-Ranberg et al. (2003) found that service sire accounted for a very small fraction of the variation of fertility in NRF. Hence, service sire was not included in the model for NR56 in the current study.

A Bayesian approach employing Markov chain Monte Carlo methods for sampling from marginal posterior distributions (Sorensen and Gianola, 2002), as applied by Heringstad et al. (2004), was used. Independent proper uniform priors were assigned to each of the elements of β . Multivariate normal prior distributions were assigned to the herd-5-yr effects, $\mathbf{h} \sim N(\mathbf{0}, \mathbf{H}_0 \otimes \mathbf{I})$, and to the sire transmitting abilities, $\mathbf{s} \sim N(\mathbf{0}, \mathbf{G}_0 \otimes \mathbf{A})$. Independent inverse Wishart prior distributions were used for the two 4×4 (co)variance matrices of herd-5-yr ($\mathbf{H}_0$) and sire effects ($\mathbf{G}_0$); off-diagonal elements of $\mathbf{R}_0$ were assigned uniform priors bounded between –1 and 1, covering the allowable space for residual correlations.

Draws from posterior distributions of the parameters, except for $\mathbf{R}_0$, were obtained using a Gibbs sampler, while a Metropolis algorithm was used to sample residual covariances, as described by Chang et al.

(2004a). Inferences were based on 90,000 samples, without thinning, after a burn-in of 10,000 iterations.

Posterior mean of heritability of liability to CM was 0.09 in the first interval and 0.05 in the second and third intervals, with posterior standard deviation equal to 0.004 (Table 1). A higher heritability of CM in early lactation than in later lactation is in agreement with Heringstad et al. (2004). Genetic correlations (Table 1) as well as residual correlations and correlations of herd-5-yr effects (Table 2) between CM in the 3 lactation intervals were within the range of previous estimates from multivariate threshold model analyses of CM (Chang et al., 2004a; Heringstad et al., 2004).

The posterior mean of heritability of liability to NR56 of 0.02 (Table 1) agrees with estimates from threshold model analyses of first-lactation NR56, ranging between 1.6 and 3.8% (Weigel and Rekaya, 2000; Andersen-Ranberg et al., 2005a). Although the heritability of liability to NR56 was low (0.02), it was twice as large as linear model estimates of heritability of NR56 in NRF (Andersen-Ranberg et al., 2005b). Other studies have reported a higher heritability of nonreturn rate (e.g., Jamrozik et al., 2005).

The posterior distributions of the genetic correlations between NR56 and CM in the 3 intervals are given in Figure 1. The 3 distributions had substantial overlap and they all include zero with high density; posterior means (standard deviation) ranged from –0.05 (0.06) to –0.02 (0.05) (Table 1). Posterior means of herd-5-yr and residual correlations between NR56 and the 3 CM traits ranged from –0.17 to –0.15 and from –0.01 to 0.02, respectively (Table 2). Negligible genetic correlation between NR56 and CM is in

Table 2. Posterior means (SD) of correlations between herd-5-yr effects (above the diagonal) and residual correlations (below the diagonal) for liability to nonreturn rate within 56 d (NR56) and clinical mastitis (CM) in the intervals –30 to 30, 31 to 150, and 151 to 300 d after first calving

	NR56	CM (–30 to 30)	CM (31 to 150)	CM (151 to 300)
NR56	—	–0.16 (0.02)	–0.17 (0.02)	–0.15 (0.02)
CM (–30 to 30)	0.02 (0.01)	—	0.65 (0.02)	0.61 (0.02)
CM (31 to 150)	–0.01 (0.01)	0.09 (0.01)	—	0.80 (0.02)
CM (151 to 300)	0.01 (0.01)	0.03 (0.01)	0.18 (0.01)	—

Figure 1. Posterior distributions of genetic correlations between nonreturn rate within 56 d (NR56) and clinical mastitis (CM) in the intervals -30 to 30, 31 to 150, and 151 to 300 d after first calving.

agreement with previous studies based on Norwegian data (Andersen-Ranberg and Heringstad, 2006) and an estimated genetic correlation of -0.05 between nonreturn rate and SCS (Kadarmideen, 2004), but it is in contrast with estimates of genetic correlation between mastitis and conception to first service of -0.58 and -0.21 for United Kingdom Holstein cattle (Pryce et al., 1998; Kadarmideen et al., 2000). For the same traits, Pryce et al. (1997) found that an estimate based on heifers (0.15) had an opposite sign to that based on all lactations (-0.19). The studies of Pryce et al. (1997, 1998) and Kadarmideen et al. (2000) were based on relatively small datasets.

Here, the longitudinal nature of the mastitis data was taken into account by using a multivariate model, defining mastitis in different lactation intervals as correlated traits. Using a longitudinal threshold model for CM (Heringstad et al., 2003; Chang et al., 2004b) and a binary threshold model for NR56 could be an alternative way to analyze the relationship between these traits. Censoring was not accounted for in the current model. However, because all traits were defined within relatively short time intervals, the problems caused by censoring are less severe than for traits based on information from a full lactation.

In conclusion, NR56 and CM were genetically uncorrelated traits in first-lactation NRF cows. Both NR56 and CM have antagonistic genetic relationships with

milk yield (Andersen-Ranberg and Heringstad, 2006), so the 2 traits should be included in a breeding objective to avoid genetic deterioration as a result of selection for increased milk yield.

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