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Genetic correlations between production and disease traits during first lactation in Holstein cows

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The aim of this study was to estimate genetic correlations between milk yield, somatic cell score (SCS), mastitis, and claw and leg disorders (CLDs) during first lactation in Holstein cows by using a threshold–linear random regression test-day model. We used daily records of milk, fat and protein yields; somatic cell count (SCC); and mastitis and CLD incidences from 46 771 first-lactation Holstein cows in Hokkaido, Japan, that calved between 2000 and 2009. A threshold animal model for binary records (mastitis and CLDs) and linear animal model for yield traits were applied in our multiple trait analysis. For both liabilities and yield traits, additive genetic effects were used as random regression on cubic Legendre polynomials of days on milk. The highest positive genetic correlations between yields and disease incidences (0.36 for milk and mastitis, 0.56 for fat and mastitis, 0.24 for protein and mastitis, 0.32 for milk and CLD, 0.44 for fat and CLD and 0.31 for protein and CLD) were estimated at about the time of peak milk yield (36 to 65 days in milk). Selection focused on early lactation yield may therefore increase the risk of mastitis and CLDs. The positive genetic correlations of SCS with mastitis or CLD incidence imply that selection to reduce SCS in the early stages of lactation would decrease the incidence of both mastitis and CLD.

Keywords: dairy cattle, genetic correlation, disease resistance, random regression, threshold

Implications

Mastitis and claw and leg disorders (CLDs) are common in dairy cows. Although increasing milk yield influences the incidence of these diseases, the relationships between disease incidence and milk yield at various lactation stages have not yet been thoroughly investigated. We used a threshold–linear random regression test-day model to estimate the genetic relationships between yield traits and mastitis or CLD incidence from first-lactation records of Holstein cows. The highest positive genetic correlations between yields and disease incidences appeared at about the time of peak milk yield. Selection focused on peak yield may therefore increase the risk of mastitis and CLDs.

Introduction

In commercial dairy populations, mastitis and claw and leg disorders (CLDs) reduce profitability in farm management because of reduced production, increased veterinary costs and involuntary culling (Enting *et al.*, 1997; Booth *et al.*, 2004; Shim *et al.*, 2004).

In general, the heritability of disease resistance is low, because genetic variance has been reduced through a long period of natural and artificial selection. In contrast, environmental variance is high owing to relatively large variations in management. Estimated heritabilities are less than 0.15 for mastitis (e.g. Harder *et al.*, 2006; Pérez-Cabal *et al.*, 2009; Hinrichs *et al.*, 2011) and 0.12 for CLDs (Koenig *et al.*, 2005; van der Waaij *et al.*, 2005; Onyiro *et al.*, 2008; Laursen *et al.*, 2009; Buch *et al.*, 2011). However, next to production and fertility traits, health traits are still important in national breeding schemes, because even a small increment in disease rates has a serious influence on farm management costs (Koenig *et al.*, 2005). Therefore, selection indices in many countries place positive weight on health traits or health-related traits (Miglior *et al.*, 2005). When health trait data are not available in routine genetic evaluations, genetic performance for disease resistance can be improved by indirect selection using health-related traits such as SCS or type traits (Lund *et al.*, 1999; Sewalem *et al.*, 2005).

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Antagonistic genetic correlations have been reported between lactation milk yield and disease resistance (e.g. Van Dorp *et al*., 1998; Carlén *et al*., 2004; Pritchard *et al*., 2013). Such correlations have the potential to cause serious problems in countries such as Japan, where breeders have been selecting Holstein cows mainly on the basis of milk yield. Several recent studies have suggested that the genetic correlations between production traits and disease resistance vary among lactation stages and that selection for lactation persistence could reduce disease incidence (Muir *et al*., 2004; Appuhamy *et al*., 2009; Yamazaki *et al*., 2013). Using a linear–linear model, Negussie *et al.* (2008) reported moderate positive genetic correlations between test-day (TD) milk yield and mastitis incidence in Ayrshire cows. They also suggested that selection for high milk yield in early lactation would increase the risk of mastitis. From this perspective, Togashi and Lin (2004) developed a selection procedure that emphasized the improvement of milk yield in the middle and late stages to improve total milk yield.

The relationships between disease incidence and milk yield in the various stages of lactation, however, have not yet been thoroughly investigated. Although disease incidence is generally recorded as a binary trait, the relationships between continuous values (production traits) and binary disease incidence have been estimated only by using a threshold–linear lactation model or a linear–linear random regression TD model (RRM; e.g. Negussie *et al*., 2008; Buch *et al*., 2011). The threshold–linear RRM should be the most appropriate model in theory and makes it easier to interpret the results of specific TD management rather than whole-lactation management.

Here, we aimed to elucidate the genetic relationships between continuous (TD milk yield or SCS) and binary (incidence of mastitis or CLDs) traits in different lactation stages by using a threshold–linear RRM.

Material and methods

Data

TD records of milk yields, SCCs, and mastitis incidence in Holstein cows that had calved for the first time between 2000 and 2009 were provided by the Hokkaido Dairy Milk Recording and Testing Association. The TD records were collected once a month as part of the Dairy Herd Improvement Program. SCCs were log-transformed into SCSs by using the formula $\mathrm{SCS} = \log2(\mathrm{SCC}/100\,000) + 3$ (Ali and Shook, 1980). Records of mastitis and CLDs were defined as binary traits of 0 or 1, where 1 indicated the presence of the disease in the past month, as reported monthly by the owner to the milk-recording supervisor. CLDs recorded included laminitis, sole ulcer, heel–horn erosion, digital dermatitis, inflammation of the bulb and other causes of lameness. Only TD records that were complete for all traits were used to analyse genetic correlations. Yield and disease records were from first-parity cows (calving age, 18 to 35 months), and TD records were limited to 305 days in milk (DIM). The total number of records was 402 332 from 46 771 cows. A pedigree file containing records of 80 132 animals that include up to three generations was obtained from the Holstein Cattle Association of Japan.

Model

Genetic correlations were estimated from the combined analysis of a threshold–linear bivariate animal model. A threshold model was applied to the binary records (mastitis and CLDs) and a linear model was applied to the other traits. In the preliminary study, we used a fourth-order RRM for the effects of additive genetic traits. However, it did not achieve good convergence for covariance. Therefore, a cubic RRM for both liabilities and continuous traits were used to examine the additive genetic effects in the first lactation, as modeled by Tsuruta *et al.* (2009). The threshold–linear RRM on DIM was as follows:

$$\begin{bmatrix} l \\ y \end{bmatrix} = \begin{bmatrix} X & 0 \\ 0 & X \end{bmatrix} \begin{bmatrix} \beta_1 \\ \beta_2 \end{bmatrix} + \begin{bmatrix} Z_1 & 0 \\ 0 & Z_1 \end{bmatrix} \begin{bmatrix} u_1 \\ u_2 \end{bmatrix} + \begin{bmatrix} Z_2 & 0 \\ 0 & Z_2 \end{bmatrix} \begin{bmatrix} p_1 \\ p_2 \end{bmatrix} + \begin{bmatrix} e_1 \\ e_2 \end{bmatrix},$$

where l is the unobserved liabilities in disease records; y is the vector of observation for TD yield or SCS; β_1 is the vector of fixed effect of herd × year (2608 subclasses); and β_2 is the vector of fixed effect of herd × TD (28 551 subclasses). Subclasses were determined by age at calving × calendar month at calving (15 calving age groups and 12 calendar months, giving a total of 180 subclasses), with fixed regressions on DIM using fourth-order Legendre polynomials with a coefficient of the exponential term of the Wilmink function (Wilmink, 1987) at DIM t, with $\phi(t) = [\,\phi_0(t) \quad \phi_1(t) \quad \phi_2(t) \quad \phi_3(t) \quad \phi_4(t) \quad \exp^{-0.05t}\,]$ (Shaeffer *et al*., 2000). Moreover, u_1 and u_2 are the vectors of additive genetic effects with random regressions on DIM using cubic Legendre polynomials; p_1 and p_2 are the effects of permanent environment of cows, with random regressions on DIM using cubic Legendre polynomials; e_1 and e_2 are the vectors of random residual effects; and X, Z_1, and Z_2 are the design matrices for β, u, and p, respectively. The covariance structure of random effects was defined as

$$\mathrm{var}\begin{bmatrix} u \\ p \\ e \end{bmatrix} = \begin{bmatrix} G \otimes A_u & 0 & 0 \\ 0 & P \otimes I_p & 0 \\ 0 & 0 & R \otimes I_n \end{bmatrix},$$

where G and P are 8×8 (co)variance matrices of the random regression coefficients for additive genetic and permanent environmental effects, respectively; R is a 2×2 residual (co) variance matrix; A_u is the matrix of additive genetic effect among animals; I_p is an 8×8 identity matrix for cows; I_n is a 2×2 identity matrix for records; and $\otimes$ is the Kronecker product.

The THRGIBBS1F90 program (Misztal *et al*., 2002) was used for Gibbs sampling for the threshold and linear models. Residual variances of binary traits were fixed at 1.0 on the liability scale. A flat prior was used for fixed effects, and an inverted Wishart distribution was used as the prior on the

random effects. For each analysis, 150 000 samples (saving every 10th sample after a burn-in of 50 000 iterations) were used to calculate the posterior means and standard deviations of the (co)variance components. Convergence was determined from a visual inspection of the plotting of Gibbs samples.

Random genetic effects were represented as a cubic Legendre polynomial at DIM t, with $\phi(t) = [\phi_0(t) \quad \phi_1(t) \quad \phi_2(t) \quad \phi_3(t)]$. The genetic correlation between yield or SCS at DIM t_1 and disease at DIM t_2 was estimated as

$$\hat{r}_{g,\text{Yield}(t_1)-\text{Disease}(t_2)} = \frac{\phi'(t_1)G_{\text{Yield}-\text{Disease}}\phi(t_2)}{\sqrt{\phi'(t_1)G_{\text{Yield}}\phi(t_1) \times \phi'(t_2)G_{\text{Disease}}\phi(t_2)}},$$

where $G_{\text{Yield–Disease}}$ is the additive genetic covariance between yield and disease incidence for random regression coefficients; G_{Yield} is the additive genetic (co)variance of yield for random regression coefficients; and G_{Disease} is the additive genetic (co)variance of disease incidence for random regression coefficients.

Results

Summary statistics

Mean values of daily milk, fat and protein yields and SCS are shown in Table 1. For milk and protein, TD production was higher from 36 to 125 DIM than from 6 to 35 DIM; thereafter it gradually decreased. Fat yield in first lactation decreased from 1.19 to 1.00 kg/day with increasing DIM. Mastitis frequency decreased with increasing DIM from 2.0% to 0.9%; CLD frequency also decreased with increasing DIM, from 0.5% to 0.2% (Table 2).

Phenotypic correlations

Daily phenotypic correlations between yield traits and disease incidence were negative and small (Table 3). Stronger correlations between mastitis or CLD incidence and milk or protein yield were found in the early stages of lactation than in the later stages; the correlations between milk yield and disease incidence ranged from –0.25 to –0.13 (mastitis) and from –0.26 to –0.17 (CLDs). The correlations between fat yield and mastitis incidence were small, negative and almost constant in the interval between calving and 215 DIM; they gradually became smaller toward the end of lactation (range throughout lactation: –0.15 to –0.07). The correlations between fat yield and CLD incidence ranged from –0.18 to –0.04. The phenotypic correlations between SCS and mastitis incidence ranged from 0.38 to 0.42, and showed no obvious pattern over the lactation period. The phenotypic correlations between SCSs and CLD incidence were low.

Heritability estimates

The heritability estimates of TD milk yield increased from 0.25 to 0.40 with increasing DIM (Table 4). For fat and protein yields, heritability estimates also generally increased steadily with increasing DIM (0.22 to 0.31 for fat and 0.19 to 0.33 for protein). The posterior means ± 1 SD of heritability over the entire 305-day lactation period were 0.42 ± 0.01 (milk), 0.33 ± 0.01 (fat) and 0.40 ± 0.01 (protein; data not shown). The posterior means of heritabilities of SCS were almost constant (between 0.06 and 0.09) during the lactation period. The heritability estimates of mastitis incidence

Table 2 *Diseased cows as percentages of the total number of cows for which data were recorded at various DIM*

DIM	*n*	Mastitis (%)	Claw and leg disorders (%)
6 to 35	38 694	2.0	0.5
36 to 65	42 094	1.1	0.5
66 to 95	42 200	1.0	0.4
96 to 125	41 612	1.0	0.3
126 to 155	41 459	1.1	0.3
156 to 185	41 115	0.9	0.2
186 to 215	40 749	0.9	0.2
216 to 245	40 338	0.9	0.2
246 to 275	39 569	0.8	0.2
276 to 305	34 501	0.9	0.2
Lactation[1]	46 771	6.1	2.0

DIM = days in milk.
[1]Percentage of cows with disease at any stage of first lactation.

Table 1 *Means (with SDs in parentheses) of milk, fat and protein yields and SCS in first-lactation cows at various DIM*

DIM	*n*	Milk (kg/day)	Fat (kg/day)	Protein (kg/day)	SCS
6 to 35	38 694	28.6 (6.3)	1.19 (0.31)	0.89 (0.19)	2.79 (1.64)
36 to 65	42 094	31.2 (6.3)	1.15 (0.27)	0.92 (0.19)	2.22 (1.57)
66 to 95	42 200	30.4 (6.3)	1.12 (0.25)	0.94 (0.20)	2.22 (1.59)
96 to 125	41 612	29.4 (6.3)	1.11 (0.25)	0.94 (0.20)	2.31 (1.59)
126 to 155	41 459	28.4 (6.2)	1.09 (0.24)	0.93 (0.20)	2.38 (1.61)
156 to 185	41 115	27.5 (6.2)	1.08 (0.24)	0.92 (0.20)	2.42 (1.62)
186 to 215	40 749	26.7 (6.1)	1.07 (0.24)	0.91 (0.20)	2.45 (1.61)
216 to 245	40 338	25.8 (6.0)	1.05 (0.24)	0.89 (0.20)	2.50 (1.60)
246 to 275	39 569	24.8 (5.9)	1.03 (0.24)	0.87 (0.20)	2.54 (1.60)
276 to 305	34 501	23.7 (6.0)	1.00 (0.24)	0.84 (0.20)	2.61 (1.60)

SCS = somatic cell score; DIM = days in milk.

Table 3 *Posterior means (with SDs in parentheses) of daily phenotypic correlations of disease incidences (mastitis and claw and leg disorders) with milk, fat and protein yields and SCS in first-lactation cows at various DIM*

DIM	Milk	Fat	Protein	SCS
Mastitis				
35	–0.25 (0.01)	–0.14 (0.01)	–0.23 (0.01)	0.41 (0.01)
65	–0.23 (0.01)	–0.13 (0.01)	–0.21 (0.01)	0.41 (0.01)
95	–0.22 (0.01)	–0.14 (0.01)	–0.21 (0.01)	0.40 (0.01)
125	–0.21 (0.01)	–0.15 (0.01)	–0.20 (0.01)	0.39 (0.01)
155	–0.20 (0.01)	–0.15 (0.01)	–0.19 (0.01)	0.39 (0.01)
185	–0.19 (0.02)	–0.15 (0.01)	–0.18 (0.02)	0.39 (0.01)
215	–0.18 (0.02)	–0.15 (0.01)	–0.17 (0.02)	0.39 (0.01)
245	–0.17 (0.02)	–0.13 (0.01)	–0.15 (0.02)	0.38 (0.01)
275	–0.14 (0.02)	–0.10 (0.02)	–0.13 (0.02)	0.38 (0.01)
305	–0.13 (0.04)	–0.07 (0.03)	–0.12 (0.03)	0.42 (0.03)
Claw and leg disorders				
35	–0.26 (0.02)	–0.08 (0.02)	–0.30 (0.02)	0.07 (0.02)
65	–0.25 (0.02)	–0.12 (0.02)	–0.31 (0.02)	0.04 (0.02)
95	–0.23 (0.02)	–0.15 (0.02)	–0.30 (0.02)	0.03 (0.02)
125	–0.22 (0.02)	–0.16 (0.02)	–0.28 (0.02)	0.04 (0.02)
155	–0.21 (0.02)	–0.17 (0.02)	–0.26 (0.02)	0.04 (0.02)
185	–0.21 (0.02)	–0.17 (0.02)	–0.25 (0.02)	0.03 (0.02)
215	–0.21 (0.03)	–0.18 (0.02)	–0.24 (0.03)	0.02 (0.02)
245	–0.21 (0.03)	–0.16 (0.02)	–0.24 (0.02)	0.01 (0.02)
275	–0.19 (0.03)	–0.12 (0.02)	–0.22 (0.02)	0.02 (0.02)
305	–0.17 (0.05)	–0.04 (0.04)	–0.21 (0.05)	0.10 (0.03)

SCS = somatic cell score; DIM = days in milk.

Table 4 *Posterior means (with SDs in parentheses) of heritabilities of milk, fat and protein yields, SCS, mastitis, and claw and leg disorders in first-lactation cows at various DIM*

DIM	Milk	Fat	Protein	SCS	Mastitis	Claw and leg disorders
35	0.25 (0.01)	0.28 (0.01)	0.21 (0.01)	0.08 (0.01)	0.10 (0.03)	0.14 (0.04)
65	0.26 (0.01)	0.23 (0.01)	0.19 (0.01)	0.07 (0.01)	0.13 (0.03)	0.14 (0.03)
95	0.30 (0.01)	0.22 (0.01)	0.21 (0.01)	0.06 (0.01)	0.15 (0.04)	0.16 (0.04)
125	0.33 (0.01)	0.23 (0.01)	0.24 (0.01)	0.06 (0.01)	0.14 (0.04)	0.17 (0.04)
155	0.35 (0.01)	0.24 (0.01)	0.26 (0.01)	0.07 (0.01)	0.13 (0.04)	0.18 (0.04)
185	0.36 (0.01)	0.26 (0.01)	0.28 (0.01)	0.07 (0.01)	0.12 (0.04)	0.17 (0.04)
215	0.37 (0.01)	0.27 (0.01)	0.29 (0.01)	0.08 (0.01)	0.11 (0.04)	0.16 (0.03)
245	0.38 (0.01)	0.28 (0.01)	0.31 (0.01)	0.08 (0.01)	0.11 (0.04)	0.13 (0.03)
275	0.40 (0.01)	0.29 (0.01)	0.33 (0.01)	0.08 (0.01)	0.11 (0.04)	0.09 (0.03)
305	0.39 (0.02)	0.31 (0.02)	0.33 (0.02)	0.09 (0.01)	0.21 (0.06)	0.09 (0.03)

SCS = somatic cell score; DIM = days in milk.

ranged between 0.10 and 0.21. For CLD incidence, the heritability estimates increased from 0.14 to 0.18 with increasing DIM but then gradually decreased after 155 DIM.

Genetic correlations

We calculated the posterior means and standard deviations of the daily genetic correlation estimates between yield traits and disease incidences (Figure 1). Daily genetic correlations between milk yield and mastitis incidence were highest (0.36) at about 35 DIM and then decreased with increasing DIM. Those between fat content and mastitis were highest (0.56) at 35 DIM. For the genetic correlation between protein and mastitis, the highest estimates were obtained at about 45 DIM (0.24) and again at the end of lactation (0.21); these estimates were smaller than those for milk and fat. The daily genetic correlations between milk or protein yield and CLD incidence were negative in the early stages of lactation and became positive after about 25 DIM. The graph of the correlation between fat and CLDs had a shape similar to the one between fat and mastitis, but the estimates were small or close to zero in the late stages of lactation. The highest estimated genetic correlations (0.32 for milk, 0.44 for fat, and 0.31 for protein) between TD yield and CLD incidence occurred near the time of peak milk yield (36 to 65 DIM).

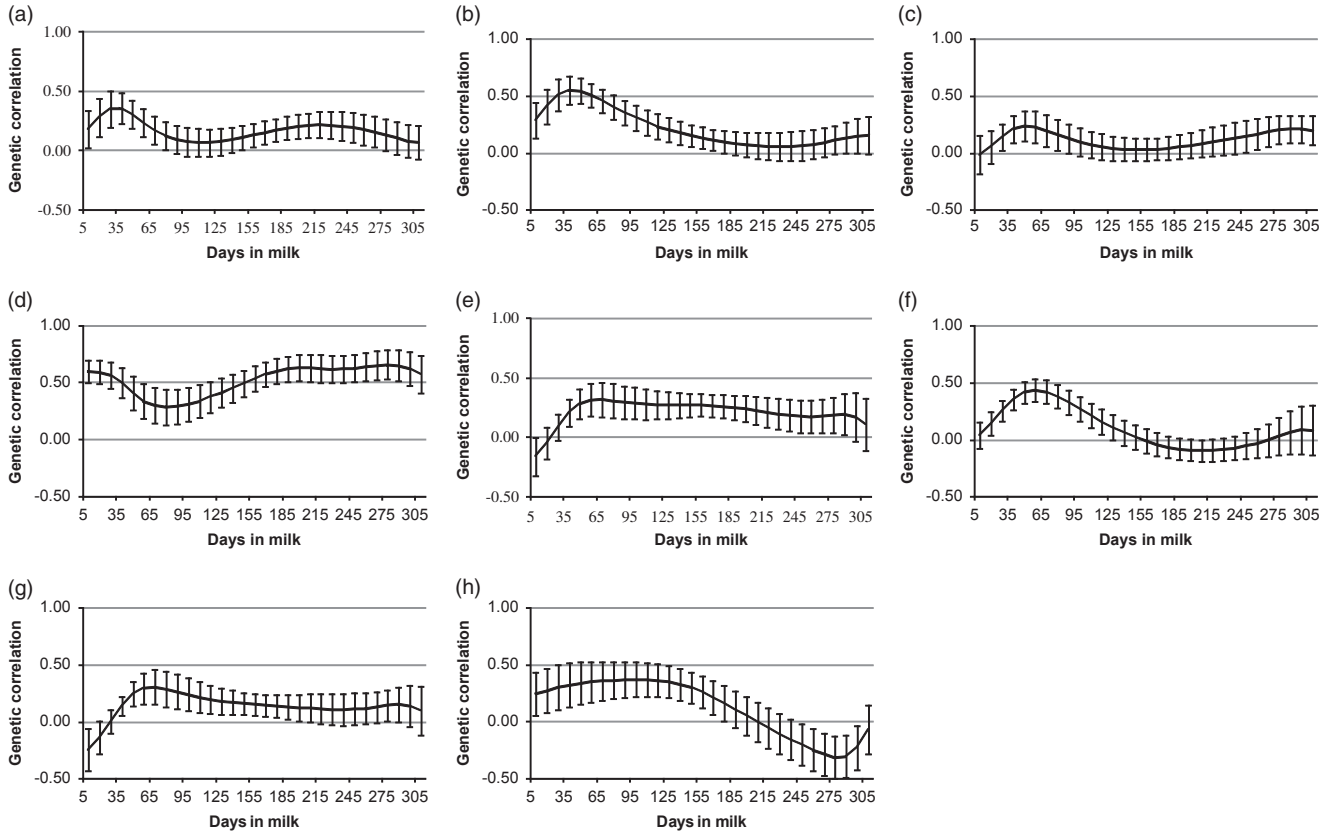

Figure 1 Posterior means and standard deviations (with bars) for daily genetic correlations (a) between milk yield and mastitis; (b) between fat yield and mastitis; (c) between protein yield and mastitis; (d) between somatic cell score and mastitis; (e) between milk yield and claw and leg disorders; (f) between fat yield and claw and leg disorders; (g) between protein yield and claw and leg disorders; (h) between somatic cell score and claw and leg disorders.

Table 5 *Posterior means (with SDs in parentheses) of daily genetic correlations between the same disease incidences (mastitis and claw and leg disorders) at 35, 275 and variable DIM in first-lactation cows*

	Mastitis		Claw and leg disorders	
DIM	35	275	35	275
35	–	0.25 (0.08)	–	0.05 (0.22)
65	0.81 (0.02)	0.29 (0.08)	0.91 (0.04)	–0.16 (0.22)
95	0.56 (0.04)	0.37 (0.08)	0.81 (0.08)	–0.19 (0.23)
125	0.37 (0.06)	0.51 (0.07)	0.73 (0.11)	–0.12 (0.23)
155	0.23 (0.08)	0.68 (0.05)	0.60 (0.16)	0.08 (0.24)
185	0.13 (0.10)	0.82 (0.03)	0.37 (0.24)	0.39 (0.21)
215	0.09 (0.10)	0.91 (0.02)	0.14 (0.27)	0.66 (0.17)
245	0.12 (0.09)	0.97 (0.01)	0.00 (0.25)	0.85 (0.10)
275	0.25 (0.08)	–	0.05 (0.22)	–
305	0.48 (0.11)	0.83 (0.03)	0.39 (0.28)	0.29 (0.27)

DIM = days in milk.

The daily genetic correlations between SCS and mastitis were positive, ranging from 0.28 to 0.66; they were high in the early and late stages of lactation. The genetic correlations between SCS and CLD decreased from 0.37 at about 95 DIM to –0.32 toward the end of lactation.

We also estimated the genetic correlation between the same disease traits at different days. The genetic correlation of mastitis at early (35 DIM) lactations decreased from 0.81 (65 DIM) to 0.09 (215 DIM) with increasing DIM; however, it increased slightly after 215 DIM toward the end of lactation (Table 5). Daily genetic correlations of mastitis at late (275 DIM) lactations gradually increased from 0.25 (35 DIM) to 0.97 (245 DIM) with increasing DIM. For CLD, trends of genetic correlation estimates were similar to those of mastitis but SDs were higher than those of mastitis (0.01 to 0.11 for mastitis and 0.04 to 0.27 for CLD).

Discussion

The overall frequency of mastitis in this study (6.1%) was lower than that in first-parity Holstein cows in the United States (9.5%; Appuhamy *et al.*, 2009) and in the United Kingdom (14%; Pritchard *et al.*, 2013). The overall frequency of CLD (2.0%) was much lower than that in Danish Holstein cows (6.3%; Laursen *et al.*, 2009). The Danish CLD data were recorded by veterinarians, whereas those in our study were reported by the cows' owners. Laursen *et al.* (2009) also suggested that using CLD records scored by claw trimmers would extend the CLD data available beyond those scored by classifiers. Therefore, the frequencies of CLDs in different evaluation systems cannot be compared directly.

The posterior means of the phenotypic correlations between yield traits and disease incidences were small and negative. Although our daily phenotypic correlations between TD milk yield and mastitis incidence were somewhat larger, in absolute terms, than the estimates (ranging from –0.06 to 0.02) of Negussie *et al.* (2008), who used a linear–linear model, negative correlations similar to ours (ranging from –0.13 to –0.11) between these traits have been reported from first-lactation records (Carlén *et al.*, 2004).

The posterior means of daily heritability of milk yield were in agreement with those reported using RRM in Holstein cows in Japan (Hagiya *et al.*, 2009). Our heritability estimates for mastitis were higher than those reported in Finnish Ayrshire cows by Negussie *et al.* (2008) using a linear model (0.03 in early DIM and 0.02 in the period from 31 to 300 DIM). Higher heritability (0.15) was estimated in German Holstein cows when the threshold RRM was used (Hinrichs *et al.*, 2011). Hinrichs *et al.* (2011) also reported that estimates using the threshold TD model were higher than those from linear or lactation models. Our posterior means of heritability of CLDs were higher than those obtained in Danish Holstein cows (0.01 for claw health and 0.01 for leg health; Laursen *et al.*, 2009); these researchers suggested that heritability estimates varied with the trait definition and the quality of trait recording. Although our disease data were reported by dairy farmers, they included medical treatment record in the past 1 month. Therefore, the quality of our records would be close to the record from veterinarian. As we applied the threshold–linear RRM, which should be the most appropriate model for disease traits analysis, quite reliable estimates were expected. However, SDs of posterior means of heritabilities for mastitis and CLD were still higher than those of production traits of SCS. Further studies are needed to obtain the reliable estimation for these disease traits.

The daily genetic correlation between yield traits and mastitis incidence was largest in the period of peak yield (35 to 45 DIM) (Figure 1). This antagonistic relationship agrees with that found by Negussie *et al.* (2008). Therefore, selection for increased production at peak yield may increase the risk of mastitis. The positive relationship between yield traits at about 65 DIM and daily CLD incidence suggested that high peak yield also increases CLD incidence.

Our positive genetic correlations between SCS and mastitis incidence agree with those (ranging from 0.34 to 0.77) of Negussie *et al.* (2008). Laursen *et al.* (2009) investigated claw health and leg health separately; they reported that the genetic correlation between claw health and leg health was low (0.35). Our definition of 'CLDs' included many kinds of disorders associated with lameness; it is therefore not easy to compare our results with those of previous studies. However, the positive genetic correlations of SCS with mastitis or CLD incidence imply that selection to reduce SCS in the early stages of lactation would decrease the incidence of both mastitis and CLD.

Zwald *et al.* (2006) observed that the genetic correlation between the incidences of mastitis in early and late lactation was low or moderate (ranging from 0.26 to 0.56 in first lactations). Therefore, Negussie *et al.* (2008) defined mastitis as two traits at different DIM; incidence of mastitis in either early or late lactation, and it was found that genetic correlations were high between mastitis and TD SCS during the same lactation stage. Our results for genetic correlations of mastitis in early and late lactation agree with those of Zwald *et al.* (2006). Although definition of several mastitis traits during lactation would result in high levels of modeling complexity, the threshold RRM for mastitis traits in terms of DIM – as we used here – would be suitable for obtaining genetic (co)variances on various TDs.

Conclusion

We investigated the genetic correlations between continuous traits (TD yields or SCS) and binary traits (mastitis or CLDs) at different lactation stages. The highest genetic correlation between yields and disease incidences (mastitis and CLD) was estimated to occur at about the time of peak milk yield. Selection focused on early lactation yield may therefore increase the risk of mastitis and CLD. We also found positive genetic correlations during the lactation period between SCS and mastitis incidence; therefore, reduction of SCS is likely to reduce the incidence of mastitis.

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References

Ali AKA and Shook GE 1980. An optimum transformation for somatic cell concentration in milk. Journal of Dairy Science 63, 487–490.

Appuhamy JADRN, Cassell BG and Cole JB 2009. Phenotypic and genetic relationships of common health disorders with milk and fat yield persistencies from producer-recorded health data and test-day yields. Journal of Dairy Science 92, 1785–1795.

Booth CJ, Warnick LD, Gröhn YT, Maizon DO, Guard CL and Janssen D 2004. Effect of lameness on culling in dairy cows. Journal of Dairy Science 87, 4115–4122.

Buch LH, Sørensen AC, Lassen J, Berg P, Eriksson J-Å, Jakobsen JH and Sørensen MK 2011. Hygiene-related and feed-related hoof diseases show different patterns of genetic correlations to clinical mastitis and female fertility. Journal of Dairy Science 94, 1540–1551.

Carlén E, Strandberg E and Roth A 2004. Genetic parameters for clinical mastitis, somatic cell score, and production in the first three lactations of Swedish Holstein cows. Journal of Dairy Science 87, 3062–3070.

Enting H, Kooij D, Dijkhuizen AA, Huirne RBM and Noordhuizen-Stassen EN 1997. Economic losses due to clinical lameness in dairy cattle. Livestock Production Science 49, 259–267.

Hagiya K, Togashi K, Takeda H, Yamasaki T, Shirai T, Saburi J, Masuda Y and Suzuki M 2009. Genetic correlation between persistency and calving interval of Holsteins in Japan. EAAP publication no. 126, 129–135. Wageningen Academic Publishers, Wageningen, the Netherlands.

Harder B, Bennewitz J, Hinrichs D and Kalm E 2006. Genetic parameters for health traits and their relationship to different persistency traits in German Holstein dairy cattle. Journal of Dairy Science 89, 3202–3212.

Hinrichs D, Bennewitz J, Stamer E, Junge W, Kalm E and Thaller G 2011. Genetic analysis of mastitis data with different models. Journal of Dairy Science 94, 471–478.

Koenig S, Sharifi AR, Wentrot H, Landmann D, Eise M and Simianer H 2005. Genetic parameters of claw and foot disorders estimated with logistic models. Journal of Dairy Science 88, 3316–3325.

Laursen MV, Boelling D and Mark T 2009. Genetic parameters for claw and leg health, foot and leg conformation, and locomotion in Danish Holsteins. Journal of Dairy Science 92, 1770–1777.

Lund MS, Jensen J and Petersen H 1999. Estimation of genetic and phenotypic parameters for clinical mastitis, somatic cell production deviance, and protein yield in dairy cattle using Gibbs sampling. Journal of Dairy Science 82, 1045–1051.

Miglior F, Muir BL and Van Doormaal BJ 2005. Selection indices in Holstein cattle of various countries. Journal of Dairy Science 88, 1255–1263.

Misztal I, Tsuruta S, Strabel T, Auvray B, Druet T and Lee DH 2002. BLUPF90 and Related Programs (BGF90). Proceedings of the 7th World Congress on Genetic Applied to Livestock Production, CD-ROM Communication no. 28, 07, Montpellier, France.

Muir BL, Fatehi J and Schaeffer LR 2004. Genetic relationships between persistency and reproductive performance in first-lactation Canadian Holsteins. Journal of Dairy Science 87, 3029–3037.

Negussie E, Strandén I and Mäntysaari EA 2008. Genetic analysis of liability to clinical mastitis, with somatic cell score and production traits using bivariate threshold–linear and linear–linear models. Livestock Science 117, 52–59.

Onyiro OM, Andrews LJ and Brotherstone S 2008. Genetic parameters for digital dermatitis and correlations with locomotion, production, fertility traits and longevity in Holstein–Friesian dairy cows. Journal of Dairy Science 91, 4037–4046.

Pérez-Cabal MA, de los Campos G, Vazquez AI, Gianola D, Rosa GJM, Weigel KA and Alenda R 2009. Genetic evaluation of susceptibility to clinical mastitis in Spanish Holstein cows. Journal of Dairy Science 92, 3472–3480.

Pritchard T, Coffey M, Mrode R and Wall E 2013. Genetic parameters for production, health, fertility and longevity traits in dairy cows. Animal 7, 34–46.

Sewalem A, Kistemaker GJ and Van Doormaal BJ 2005. Relationship between type traits and longevity in Canadian Jerseys and Ayrshires using a Weibull proportional hazards model. Journal of Dairy Science 88, 1552–1560.

Shaeffer LR, Jamrozik J, Kistemaker GJ and Van Doormaal BJ 2000. Experience with a Test-Day Model. Journal of Dairy Science 83, 1135–1144.

Shim EH, Shanks RD and Morin DE 2004. Milk loss and treatment costs associated with two treatment protocols for clinical mastitis in dairy cows. Journal of Dairy Science 87, 2702–2708.

Togashi K and Lin CY 2004. Development of an optimal index to improve lactation yield and persistency with the least selection intensity. Journal of Dairy Science 87, 3047–3052.

Tsuruta S, Misztal I, Huang C and Lawlor TJ 2009. Bivariate analysis of conception rates and test-day milk yields in Holsteins using threshold–linear model with random regressions. Journal of Dairy Science 92, 2922–2930.

van der Waaij EH, Holzhauer M, Ellen E, Kamphuis C and de Jong G 2005. Genetic parameters for claw disorders in Dutch dairy cattle and correlations with conformation traits. Journal of Dairy Science 88, 3672–3678.

Van Dorp TE, Dekkers JCM, Martin SW and Noordhuizen JPTM 1998. Genetic parameters of health disorders, and relationships with 305-day milk yield and conformation traits of registered Holstein cows. Journal of Dairy Science 81, 2264–2270.

Wilmink JBM 1987. Adjustment of test-day milk, fat and protein yield for age, season and stage of lactation. Livestock Production Science 16, 335–348.

Yamazaki T, Hagiya K, Takeda H, Sasaki O, Yamaguchi S, Sogabe M, Saito Y, Nakagawa S, Togashi K, Suzuki K and Nagamine Y 2013. Genetic correlations between lactation persistency and somatic cell scores on test day within and across first and second lactations in Holstein cows. Livestock Science 152, 120–126.

Zwald NR, Weigel KA, Chang YM, Welper RD and Clay JS 2006. Genetic analysis of clinical mastitis data from on-farm management software using threshold models. Journal of Dairy Science 89, 330–336.