Genetic relationships of clinical mastitis, cystic ovaries, and lameness with milk yield and somatic cell score in first-lactation Canadian Holsteins

A. Koeck,^{*1} S. Loker,^{*†} F. Miglior,^{*‡} D. F. Kelton,[§] J. Jamrozik,[‡] and F. S. Schenkel^{*}

^{*}Centre for Genetic Improvement of Livestock, University of Guelph, Guelph, Ontario, Canada, N1G 2W1

[†]Livestock Improvement Corporation, Newstead, Hamilton 3284, New Zealand

[‡]Canadian Dairy Network, Guelph, Ontario, Canada, N1K 1E5

[§]Department of Population Medicine, Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada, N1G 2W1

ABSTRACT

The objective of this study was to investigate the genetic relationships of the 3 most frequently reported dairy cattle diseases (clinical mastitis, cystic ovaries, and lameness) with test-day milk yield and somatic cell score (SCS) in first-lactation Canadian Holstein cows using random regression models. Health data recorded by producers were available from the National Dairy Cattle Health System in Canada. Disease traits were defined as binary traits (0 = healthy, 1 = affected) based on whether or not the cow had at least one disease case recorded within 305 d after calving. Mean frequencies of clinical mastitis, cystic ovaries, and lameness were 12.7, 8.2, and 9.1%, respectively. For genetic analyses, a Bayesian approach using Gibbs sampling was applied. Bivariate linear sire random regression model analyses were carried out between each of the 3 disease traits and test-day milk yield or SCS. Random regressions on second-degree Legendre polynomials were used to model the daily sire additive genetic and cow effects on test-day milk yield and SCS, whereas only the intercept term was fitted for disease traits. Estimated heritabilities were 0.03, 0.03, and 0.02 for clinical mastitis, cystic ovaries, and lameness, respectively. Average heritabilities for milk yield were between 0.41 and 0.49. Average heritabilities for SCS ranged from 0.10 to 0.12. The average genetic correlations between daily milk yield and clinical mastitis, cystic ovaries, and lameness were 0.40, 0.26, and 0.23, respectively; however, the last estimate was not statistically different from zero. Cows with a high genetic merit for milk yield during the lactation were more susceptible to clinical mastitis and cystic ovaries. Estimates of genetic correlations between daily milk yield and clinical mastitis were moderate throughout the lactation. The genetic correlations between daily milk yield and cystic ovaries were near zero at the beginning of lactation and were highest at

mid and end lactation. The average genetic correlation between daily SCS and clinical mastitis was 0.59 and was consistent throughout the lactation. The average genetic correlation between daily SCS and cystic ovaries was near zero (−0.01), whereas a moderate, but nonsignificant, correlation of 0.27 was observed between SCS and lameness. Unfavorable genetic associations between milk yield and diseases imply that production and health traits should be considered simultaneously in genetic selection.

Key words: disease, milk yield, somatic cell score, genetic correlation

INTRODUCTION

Breeding for disease resistance is becoming increasingly important for economic reasons, as well as animal welfare concerns and consumer demands for healthy and naturally produced products. In Canada, a national dairy cattle health and disease data recording system was started in 2007. Eight diseases that are known to affect herd profitability are recorded by producers on a voluntary basis: clinical mastitis, displaced abomasum, ketosis, milk fever, retained placenta, metritis, cystic ovaries, and lameness. The feasibility of using producer-recorded health data from Canada for genetic evaluations has been shown previously by Neuenschwander (2010) and Koeck et al. (2012b).

Milk yield, one of the main selection criteria for dairy cattle during the last few decades, is genetically unfavorably correlated with disease resistance (e.g., Hooijer et al., 2001; Carlén et al., 2004; König et al., 2008). Due to lack of disease recording systems, selection for disease resistance in many countries is based on genetically correlated traits, such as SCS, which is used as a mastitis indicator. Most literature estimates of genetic correlations between disease traits and milk yield or SCS are from lactation-average models. Recently, Negussie et al. (2008a, 2010) and Mrode et al. (2012) applied random regression models to investigate genetic relationships between mastitis and milk yield or SCS throughout lactation. The advantage of such an

Received November 30, 2013.

Accepted May 16, 2014.

¹Corresponding author: akoeck@uoguelph.ca

Table 1. Summary statistics of analyzed data

Item	Clinical mastitis	Cystic ovaries	Lameness
Cows (no.)	61,086	45,641	36,001
Test-day records (no.)	453,829	346,579	266,082
Sires (no.)	3,080	2,530	2,431
Pedigree (no.)	4,106	3,518	3,376
Disease frequency (%)	12.7	8.2	9.1
Mean test-day milk yield (kg)	29.5	29.4	29.6
Mean test-day SCS ($\log_2$)	2.2	2.1	2.1

analysis is that all information in the data are utilized, as SCS and milk production traits vary with stage of lactation.

In previous studies of Canadian Holsteins, the genetic correlation between clinical mastitis and lactation average SCS was estimated to be in the range of 0.63 to 0.69 (Koeck et al., 2012a,c). Genetic associations between diseases and milk yield throughout the lactation have never been studied in this population. Therefore, the objective of this study was to investigate the genetic relationships of the 3 most frequently reported dairy cattle diseases (clinical mastitis, cystic ovaries, and lameness) with test-day milk yield and SCS in first-lactation Canadian Holstein cows using random regression models.

MATERIALS AND METHODS

Data

Data for clinical mastitis, cystic ovaries, and lameness recorded by dairy producers from April 1, 2007, to April 20, 2011, as well as monthly test-day records of milk yield and SCC, were obtained from the Canadian Dairy Network (Guelph, ON, Canada). To ensure that all cows were from herds with reliable disease recording, several editing criteria were applied separately for each disease. Only herds having at least 2 records of the disease being analyzed were considered. The first and last record in a herd had to be at least 180 d distant. This criterion would remove herds that had recorded for only a short time. In addition, minimum disease frequencies were applied to ensure continuous data recording within individual herds. Minimum frequencies (reported cases per herd and year) were 5% for clinical mastitis and 1% for cystic ovaries and lameness.

Because not all herds record all diseases (Koeck et al., 2012b), 3 data sets, one for each disease, were created by matching test-day data to health records. In all data sets, only records from first-lactation Holstein cows with an age at calving between 19 and 43 mo and at least 1 test-day observation were considered. For each of the 3 data sets, a sire pedigree file was gen-

erated by tracing the pedigrees of sires and maternal grandsires back as far as possible. Summary statistics for the analyzed data sets are given in Table 1.

Trait Definitions

In Canada, cases of clinical mastitis, cystic ovaries, and lameness are recorded by producers according to the disease definitions described by Kelton et al. (1998). Clinical mastitis, cystic ovaries, and lameness were defined as binary traits (0 = healthy, 1 = affected) based on whether or not the cow had at least one disease case recorded within 305 d after calving. Test-day records between 5 and 305 DIM were considered for milk yield and SCC. Test-day SCC was transformed to SCS, to achieve an approximately normal distribution, as follows: $SCS = \log_2 (SCC/100,000) + 3$.

Models

For genetic analyses, a Bayesian approach using Gibbs sampling was applied. Bivariate linear sire random regression model analyses were carried out between the 3 diseases (clinical mastitis, cystic ovaries, and lameness) and test-day milk yield or SCS. Legendre polynomials of the second degree were used to describe regression curves for milk yield and SCS, whereas only the intercept term was fitted for disease traits. As disease frequencies were low, the disease traits were defined on a lactation basis. An alternative approach would be to define disease traits as longitudinal traits. However, as shown by Carlén et al. (2009), the application of random regression models was unstable and sensitive for estimation of genetic parameters for clinical mastitis.

Linear models were applied for disease traits, even though these traits are binary. Although threshold models are, at least in theory, more appropriate to analyze binary traits, the literature indicates they have little effect on the ranking of animals (e.g., Ramirez-Valverde et al., 2001). Moreover, Negussie et al. (2008b) analyzed clinical mastitis in bivariate analyses with production traits (SCS, milk, protein, and fat yields) by using both a threshold-linear and linear-linear model and found

similar estimates of genetic correlations with no underestimation with the linear model analysis.

For milk yield and SCS, the following model was used:

$$\mathbf{y} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}_{\text{htd}}\mathbf{h}_{\text{td}} + \mathbf{Z}_{\text{s}}\mathbf{s} + \mathbf{Z}_{\text{c}}\mathbf{c} + \mathbf{e},$$

where $\mathbf{y}$ is a vector of observations for milk yield or SCS; $\boldsymbol{\beta}$ is a vector of systematic effects, including fixed regression coefficients of age-year-season of calving; $\mathbf{h}_{\text{td}}$ is a vector of random herd-test-day effects; $\mathbf{s}$ is a vector of random regression coefficients of additive genetic sire effects; $\mathbf{c}$ is a vector of random regression coefficients of cow effects; $\mathbf{e}$ is a vector of random residuals; and $\mathbf{X}$, $\mathbf{Z}_{\text{htd}}$, $\mathbf{Z}_{\text{s}}$, and $\mathbf{Z}_{\text{c}}$ are the corresponding incidence matrices. Age at first calving had 16 classes, in which <22 and >35 mo were the first and last classes, respectively, and other classes were single months. Season of calving classes were formed by combining 3 mo (January–March, April–June, July–September, and October–December).

For clinical mastitis, cystic ovaries, and lameness, the following model was applied:

$$\mathbf{y} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}_{\text{h}}\mathbf{h} + \mathbf{Z}_{\text{s}}\mathbf{s} + \mathbf{Z}_{\text{c}}\mathbf{c} + \mathbf{e},$$

where $\mathbf{y}$ is a vector of observations for clinical mastitis, cystic ovaries, or lameness; $\boldsymbol{\beta}$ is a vector of systematic effects, including fixed effects of age at calving and year-season of calving; $\mathbf{h}$ is a vector of random herd-year of calving effects; $\mathbf{s}$ is a vector of random additive genetic sire effects; $\mathbf{c}$ is a vector of cow effects; $\mathbf{e}$ is a vector of random residuals; and $\mathbf{X}$, $\mathbf{Z}_{\text{h}}$, $\mathbf{Z}_{\text{s}}$, and $\mathbf{Z}_{\text{c}}$ are the corresponding incidence matrices. Age at first calving and season of calving were defined as described above.

To facilitate variance component estimation, the residual covariance between the test-day and disease trait was set to zero, and the animal covariance between traits was modeled by the cow effect (e.g., Negussie et al., 2008a). As a result, the residual variance for the disease trait was small. In addition, for computational reasons, a homogeneous residual variance structure was assumed for the test-day traits.

Expectations and covariance structures for the random effects were

$$\begin{aligned} E(\mathbf{y}) &= \mathbf{X}\boldsymbol{\beta}, E(\mathbf{h}) = 0, E(\mathbf{h}_{\text{td}}) = 0, E(\mathbf{s}) = 0, \\ E(\mathbf{c}) &= 0, E(\mathbf{e}) = 0 \end{aligned}$$

and

$$\begin{aligned} V(\mathbf{h}) &= \mathbf{H}, V(\mathbf{h}_{\text{td}}) = \mathbf{T}, V(\mathbf{s}) = \mathbf{A} \otimes \mathbf{G}, \\ V(\mathbf{c}) &= \mathbf{I} \otimes \mathbf{C}, V(\mathbf{e}) = \mathbf{I} \otimes \mathbf{R}, \end{aligned}$$

where $\mathbf{I}$ represents an identity matrix, $\mathbf{H}$ is a diagonal matrix of the form $\mathbf{I}\sigma_h^2$, and σ_h^2 is the variance of the random herd-year of calving effect, $\mathbf{T}$ is a diagonal matrix of the form $\mathbf{I}\sigma_{\text{htd}}^2$, and σ_{htd}^2 is the variance of the random herd-test-day effect, $\mathbf{A}$ is the matrix of additive genetic relationships among sires, $\otimes$ is the Kronecker product, $\mathbf{G}$ and $\mathbf{C}$ are covariance matrices for additive genetic sire effects and cow effects, respectively, and $\mathbf{R}$ is the residual covariance matrix, which was assumed to be equal to $\mathbf{I}\sigma_e^2$, where σ_e^2 is the error variance. All random effects were assumed to be normally distributed.

Bayesian analyses were run using the RJMC routine implemented in the DMU software (Madsen and Jensen, 2008). The sample size and length of burn-in were decided based on visual inspections of trace plots. Inference for all bivariate analyses was based on a chain length of 100,000 samples, with samples stored at every fifth round. The burn-in was 20,000 rounds for all analyses.

Daily heritabilities for test-day traits were calculated as the ratio of additive genetic variance to phenotypic variance on a given DIM. Average daily heritabilities for test-day traits and genetic correlations were obtained by summing daily heritabilities or genetic correlations from 5 to 305 DIM and dividing by 301.

RESULTS AND DISCUSSION

Disease Frequencies

Mean frequencies of clinical mastitis, cystic ovaries, and lameness in first-lactation Holstein cows were 12.7, 8.2, and 9.1%, respectively (Table 1). In US Holstein cows, Zwald et al. (2004) found similar frequencies for cystic ovaries (8%) and lameness (10%), whereas masti-

Figure 1. First occurrence of each disease in 30-d intervals after calving (% of disease cases in each interval).

Table 2. Heritability (h^2) for disease traits (clinical mastitis, cystic ovaries, lameness), average heritability for daily milk yield, and average genetic correlations (r_g) between disease traits and daily milk yield from the 3 bivariate analyses (posterior SD in parentheses)

Disease trait	h^2_{Disease}	$h^2_{\text{Milk yield}}$	r_g
Clinical mastitis	0.03 (0.006)	0.48 (0.021)	0.40 (0.09)
Cystic ovaries	0.03 (0.007)	0.41 (0.022)	0.26 (0.10)
Lameness	0.02 (0.004)	0.49 (0.027)	0.23 (0.17)

tis frequency was higher (20%). In agreement with our study, Mrode et al. (2012) reported a mastitis frequency of 13.5% in first-lactation UK Holstein cows.

Figure 1 shows the distribution of first occurrence of each disease according to days after calving. A high proportion (35%) of clinical mastitis cases occurred in the first month of lactation, whereas the remaining cases were equally distributed across lactation. Cases of cystic ovaries were reported mainly after the first month of lactation. Cases of lameness were more evenly distributed throughout the lactation, with slightly more cases in early lactation.

Phenotypic Associations

The mean milk yield from healthy and diseased cows throughout lactation is shown in Figure 2. Cows with cystic ovaries had a higher milk yield throughout lactation compared with healthy cows. For the other 2 diseases, clinical mastitis and lameness, no visible differences were observed between healthy and diseased cows. Figure 3 presents the mean milk yield of diseased cows from 30 d before to 30 d after the recording of the first disease case. Cows with clinical mastitis or lameness had a decrease in milk yield during the occurrence, whereas cows with cystic ovaries showed no decrease in milk yield. Numerous studies have shown that milk yield is reduced during the occurrence of a case of clinical mastitis or lameness (e.g., Green et al., 2002; Gröhn et al., 2004; Hagness et al., 2007).

The lactation curve for mean SCC from cows with clinical mastitis showed a similar shape as for healthy cows (Figure 4a), but SCC from infected cows was higher throughout the lactation. As expected, cows with cystic ovaries (Figure 4b) or lameness (Figure 4c) had a mean SCC throughout the lactation similar to that

of healthy cows. These findings are in agreement with Figure 5, which shows the mean SCC of diseased cows before and after the disease case. Cows with clinical mastitis showed a considerable increase in SCC during infection. In addition, the mean SCC before the first occurrence of clinical mastitis was already at a higher level, at around 250,000 cells/mL, and remained high after the clinical mastitis case. Hence, treatment does not seem to decrease SCC to the level of healthy cows. For cows with cystic ovaries or lameness, no alteration in SCC was observed. de Haas et al. (2002) showed that mastitis caused by different pathogens affects the lactation curve for SCC differently. For example, SCC was low before a case of *Escherichia coli* mastitis and decreased rapidly after the infection to a level only slightly higher than that before infection. In contrast, an increase in SCC was observed before a clinical case of *Staphylococcus aureus* mastitis, which suggests that the pathogen is subclinically present before clinical symptoms are observed. In addition, SCC remained high after the case of *Staphylococcus aureus* mastitis.

Heritabilities

Heritabilities for disease traits were similar between analyses (Tables 2 and 3). Estimated heritabilities were 0.03, 0.03, and 0.02 for clinical mastitis, cystic ovaries, and lameness, respectively. As disease traits have a categorical outcome, heritability estimates on the linear scale are frequency dependent, and estimates from different studies are, therefore, not straightforwardly comparable. In UK Holstein cows, Kadarmideen et al. (2000) obtained heritabilities of 0.04 and 0.02 for mastitis and lameness, respectively. Dechow et al. (2004) reported heritability estimates of 0.02 for mastitis and cystic ovaries. In a more recent study by Mrode et al.

Table 3. Heritability (h^2) for disease traits (clinical mastitis, cystic ovaries, lameness), average heritability for daily SCS, and average genetic correlations (r_g) between disease traits and daily SCS from the 3 bivariate analyses (posterior SD in parentheses)

Disease trait	h^2_{Disease}	h^2_{SCS}	r_g
Clinical mastitis	0.03 (0.005)	0.12 (0.012)	0.59 (0.11)
Cystic ovaries	0.03 (0.007)	0.11 (0.013)	-0.01 (0.15)
Lameness	0.02 (0.005)	0.10 (0.014)	0.27 (0.17)

Table 4. Estimates of genetic correlations between disease traits (clinical mastitis, cystic ovaries, and lameness) and milk yield at selected DIM (posterior SD in parentheses)

Milk yield at DIM	Clinical mastitis	Cystic ovaries	Lameness
5	0.34 (0.10)	0.06 (0.12)	0.23 (0.14)
50	0.42 (0.09)	0.18 (0.11)	0.24 (0.15)
100	0.43 (0.09)	0.26 (0.10)	0.23 (0.17)
150	0.41 (0.10)	0.28 (0.10)	0.21 (0.17)
200	0.39 (0.10)	0.29 (0.10)	0.22 (0.18)
250	0.36 (0.10)	0.30 (0.11)	0.23 (0.18)
305	0.30 (0.10)	0.29 (0.11)	0.26 (0.19)

(2012), a heritability of 0.05 was obtained for mastitis in first-lactation UK Holstein cows.

Average heritabilities for daily milk yield were between 0.41 and 0.49 (Table 2) and in agreement with previous analyses in Canadian Holsteins. In first-lactation Canadian Holstein cows, Miglior et al. (2007) reported a heritability of 0.52 for milk yield, whereas Loker et al. (2012) found a slightly lower heritability of 0.38.

Average heritabilities for daily SCS ranged from 0.10 to 0.12 (Table 3). Slightly higher heritability estimates of 0.19 and 0.15 were obtained by Miglior et al. (2007) and Loker et al. (2012), respectively, based on data from the same population. Differences in results could be because of differences in data editing or modeling.

Genetic Correlations Between Diseases and Milk Yield

Genetic correlations between disease traits and milk yield were positive and unfavorable (Tables 2 and 4). The strongest unfavorable genetic correlation was found between clinical mastitis and milk yield, with an average estimate of 0.40. Similarly, Carlén et al. (2004) obtained a genetic correlation of 0.32 between clinical mastitis and 305-d milk yield in first-lactation Swedish Holstein cows. Other studies (e.g., Heringstad et al., 2005; Norberg et al., 2009) have presented genetic correlations between protein yield and clinical mastitis that were similar to the estimates found in the current study.

Estimates of genetic correlations between daily milk yield and clinical mastitis were moderate throughout the lactation. Although we observed a tendency for correlations to be higher around the peak of the lactation at 50 to 100 DIM, these estimates were not significantly different compared with estimates from the beginning and end of lactation. In contrast, Negussie et al. (2008a) obtained the strongest genetic correlations between milk yield and clinical mastitis at 30 DIM, and the unfavorable genetic correlation between these traits became weaker as lactation progressed.

The average genetic correlation between cystic ovaries and daily milk yield was 0.26. Estimates of genetic correlations between cystic ovaries and daily milk yield were near zero at the beginning of lactation and were highest at mid and end lactation. Slightly stronger unfavorable genetic correlations were reported by Pösö and Mäntysaari (1996) and Hooijer et al. (2001). Pösö and Mäntysaari (1996) found a genetic correlation of 0.42 between 305-d milk yield and ovulatory disorders, whereas Hooijer et al. (2001) estimated a genetic correlation of 0.34 between 305-d milk yield and cystic ovaries.

The average genetic correlation between lameness and daily milk yield was 0.23 and was consistent throughout the lactation. Although this result was not statistically different from zero, similar estimates were found in previous studies. Kadarmideen et al. (2000) obtained a genetic correlation of 0.27 between 305-d milk yield and lameness. König et al. (2008) investigated the genetic relationships between 4 different claw

Table 5. Estimates of genetic correlations between disease traits (clinical mastitis, cystic ovaries, and lameness) and SCS at selected DIM (posterior SD in parentheses)

SCS at DIM	Clinical mastitis	Cystic ovaries	Lameness
5	0.59 (0.10)	0.15 (0.15)	0.17 (0.18)
50	0.62 (0.08)	0.09 (0.15)	0.24 (0.17)
100	0.60 (0.08)	0.02 (0.15)	0.27 (0.17)
150	0.58 (0.09)	−0.02 (0.16)	0.28 (0.17)
200	0.57 (0.09)	−0.05 (0.16)	0.28 (0.17)
250	0.58 (0.09)	−0.06 (0.16)	0.28 (0.18)
305	0.60 (0.09)	−0.07 (0.17)	0.26 (0.18)

Figure 2. Mean milk yield by DIM for healthy cows (no disease case of the respective disease) and cows with at least one case of (a) clinical mastitis, (b) cystic ovaries, and (c) lameness, within the first 305 d after calving.

Figure 3. The pattern of mean test-day milk yield before and after the first case of clinical mastitis, cystic ovaries, or lameness.

disorders (digital dermatitis, sole ulcer, wall disorder, and interdigital hyperplasia) and milk yield before and after the diagnosis and found all correlations to be positive, with estimates ranging from 0.08 to 0.44.

Genetic Correlations Between Diseases and SCS

Genetic correlations between disease traits and SCS are shown in Tables 3 and 5. The average genetic correlation between clinical mastitis and daily SCS was moderate (0.59) and consistent throughout the lactation. This is in agreement with an earlier study in Canadian Holsteins (Koeck et al., 2012a), where genetic correlations between clinical mastitis and SCS in early and late lactation were almost identical (0.69 and 0.68, respectively). Negussie et al. (2010) found a wider range of genetic correlations of 0.46 to 0.65 between test-day SCS and clinical mastitis in first-lactation Danish Holstein cows. Similarly, Mrode et al. (2012) reported genetic correlations between SCS and mastitis that ranged from 0.37 to 0.55 in first-lactation UK Holstein cows.

Cystic ovaries and daily SCS seem to be genetically independent traits as the average genetic correlation was -0.01 . A moderate, but nonsignificant, average genetic correlation of 0.27 was obtained between daily SCS and lameness. Estimates of genetic correlations between daily SCS and cystic ovaries or lameness were not different from zero.

CONCLUSIONS

Milk yield was unfavorably correlated with clinical mastitis and cystic ovaries, which confirms the antagonistic relationship between disease resistance and milk production found in previous studies. As expected, SCS

Figure 4. Mean SCC by DIM for healthy cows (no disease case of the respective disease) and cows with at least one case of (a) clinical mastitis, (b) cystic ovaries, and (c) lameness, within the first 305 d after calving.

Figure 5. The pattern of mean test-day SCC before and after the first case of clinical mastitis, cystic ovaries, or lameness.

was highly correlated with clinical mastitis and the magnitude of association was constant over the lactation. The unfavorable relationships of health traits with milk yield suggest that health traits should be included in genetic selection.

ACKNOWLEDGMENTS

All dairy producers recording health data are gratefully acknowledged. This study was funded by the DairyGen Council of Canadian Dairy Network (Guelph, ON, Canada) and the Natural Sciences and Engineering Research Council of Canada (Ottawa, ON, Canada).

REFERENCES

- Carlén, E., K. Grandinson, U. Emanuelson, and E. Strandberg. 2009. Random regression models for genetic evaluation of clinical mastitis in dairy cattle. *Animal* 3:1100–1108.
- Carlén, E., E. Strandberg, and A. Roth. 2004. Genetic parameters for clinical mastitis, somatic cell score, and production in the first three lactations of Swedish Holstein cows. *J. Dairy Sci.* 87:3062–3070.
- de Haas, Y., H. W. Barkema, and R. F. Veerkamp. 2002. The effect of pathogen-specific clinical mastitis on the lactation curve for somatic cell count. *J. Dairy Sci.* 85:1314–1323.
- Dechow, C. D., G. W. Rogers, U. Sander-Nielsen, L. Kiel, T. J. Lawlor, J. S. Clay, A. E. Freeman, G. Abdel-Azim, A. Kuck, and S. Schnell. 2004. Correlations among body conditions scores from various sources, dairy form, and cow health from the United States and Denmark. *J. Dairy Sci.* 87:3526–3533.
- Green, L. E., V. J. Hedges, Y. H. Schukken, R. W. Blowey, and A. J. Packington. 2002. The impact of clinical lameness on the milk yield of dairy cows. *J. Dairy Sci.* 85:2250–2256.
- Gröhn, Y. T., D. J. Wilson, R. N. González, J. A. Hertl, H. Schulte, G. Bennett, and Y. H. Schukken. 2004. Effect of pathogen-specific clinical mastitis on milk yield in dairy cows. *J. Dairy Sci.* 87:3358–3374.
- Hagnestam, C., U. Emanuelson, and B. Berglund. 2007. Yield losses associated with clinical mastitis occurring in different weeks of lactation. *J. Dairy Sci.* 90:2260–2270.
- Heringstad, B., Y. M. Chang, D. Gianola, and G. Klemetsdal. 2005. Genetic association between susceptibility to clinical mastitis and

- protein yield in Norwegian dairy cattle. *J. Dairy Sci.* 88:1509–1514.
- Hooijer, G. A., R. B. F. Lubbers, B. J. Ducro, J. A. M. van Arendonk, L. M. T. E. Kaal-Lansbergen, and T. van der Lende. 2001. Genetic parameters for cystic ovarian disease in Dutch Black and White dairy cattle. *J. Dairy Sci.* 84:286–291.
- Kadarmideen, H. N., R. Thompson, and G. Simm. 2000. Linear and threshold model genetic parameters for disease, fertility and milk production in dairy cattle. *Anim. Sci.* 71:411–419.
- Kelton, D. F., K. D. Lissemore, and R. E. Martin. 1998. Recommendations for recording and calculating the incidence of selected clinical diseases of dairy cattle. *J. Dairy Sci.* 81:2502–2509.
- Koeck, A., F. Miglior, D. F. Kelton, and F. S. Schenkel. 2012a. Alternative somatic cell count traits to improve mastitis resistance in Canadian Holsteins. *J. Dairy Sci.* 95:432–439.
- Koeck, A., F. Miglior, D. F. Kelton, and F. S. Schenkel. 2012b. Health recording in Canadian Holsteins: Data and genetic parameters. *J. Dairy Sci.* 95:4099–4108.
- Koeck, A., F. Miglior, D. F. Kelton, and F. S. Schenkel. 2012c. Short communication: Genetic parameters for mastitis and its predictors in Canadian Holsteins. *J. Dairy Sci.* 95:7363–7366.
- König, S., X. L. Wu, D. Gianola, B. Heringstad, and H. Simianer. 2008. Exploration of relationships between claw disorders and milk yield in Holstein cows via recursive linear and threshold models. *J. Dairy Sci.* 91:395–406.
- Loker, S., C. Bastin, F. Miglior, A. Sewalem, L. R. Schaeffer, J. Jamrozik, A. Ali, and V. Osborne. 2012. Genetic and environmental relationships between body condition score and milk production traits in Canadian Holsteins. *J. Dairy Sci.* 95:410–419.
- Madsen, P., and J. Jensen. 2008. A user's guide to DMU: A package for analyzing multivariate mixed models. Version 6, release 4.7. Danish Institute of Agricultural Sciences, Tjele, Denmark.
- Miglior, F., A. Sewalem, J. Jamrozik, J. Bohmanova, D. M. Lefebvre, and R. K. Moore. 2007. Genetic analysis of milk urea nitrogen and lactose and their relationships with other production traits in Canadian Holstein cattle. *J. Dairy Sci.* 90:2468–2479.
- Mrode, R., T. Pritchard, M. Coffey, and E. Wall. 2012. Joint estimation of genetic parameters for test-day somatic cell count and mastitis in the United Kingdom. *J. Dairy Sci.* 95:4618–4628.
- Negussie, E., M. Lidauer, E. A. Mäntysaari, I. Strandén, J. Pösö, U. S. Nielsen, K. Johansson, J.-Å. Eriksson, and G. P. Aamand. 2010. Combining test day SCS with clinical mastitis and udder type traits: A random regression model for joint genetic evaluation of udder health in Denmark, Finland and Sweden. Pages 25–32 in *Interbull Bulletin 42*: Riga, Latvia. Interbull, Uppsala, Sweden.
- Negussie, E., I. Strandén, and E. A. Mäntysaari. 2008a. Genetic association of clinical mastitis with test-day somatic cell score and milk yield during first lactation of Finnish Ayrshire cows. *J. Dairy Sci.* 91:1189–1197.
- Negussie, E., I. Strandén, and E. A. Mäntysaari. 2008b. Genetic analysis of liability to clinical mastitis, with somatic cell score and production traits using bivariate threshold-linear and linear-linear models. *Livest. Sci.* 117:52–59.
- Neuenschwander, T. F.-O. 2010. Studies on disease resistance based on producer-recorded data in Canadian Holsteins. PhD Thesis. University of Guelph, Guelph, ON, Canada.
- Norberg, E., P. Madsen, and J. Pedersen. 2009. A multi-trait genetic analysis of protein yield, udder health, and fertility in first lactation Danish Holstein, Danish Red, and Danish Jersey using an animal model. *Acta Agric. Scand. A Anim. Sci.* 59:197–203.
- Pösö, J., and E. A. Mäntysaari. 1996. Genetic relationships between reproductive disorders, operational days open and milk yield. *Livest. Prod. Sci.* 46:41–48.
- Ramirez-Valverde, R., I. Misztal, and J. K. Bertrand. 2001. Comparison of threshold vs. linear and animal vs sire models for predicting direct and maternal genetic effects on calving difficulty in beef cattle. *J. Anim. Sci.* 79:333–338.
- Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004. Genetic selection for health traits using producer-recorded data. I. Incidence rates, heritability estimates, and sire breeding values. *J. Dairy Sci.* 87:4287–4294.