Short communication: Genetic parameters for mastitis and its predictors in Canadian Holsteins

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

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

[†]Guelph Food Research Centre, Agriculture and Agri-Food Canada, Guelph, Ontario, Canada, N1G 5C9

[‡]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 estimate genetic parameters for mastitis and its predictors [mean somatic cell score (SCS) in early lactation, standard deviation of SCS, excessive test-day somatic cell count (SCC), udder depth (UD), fore udder attachment (FUA), and body condition score (BCS)]. Mastitis data recorded by producers were available from the national dairy cattle health system in Canada. Mastitis was defined as a binary variable based on whether or not the cow had at least 1 mastitis case in the period from calving to 305 d after calving. A Bayesian analysis using Gibbs sampling was applied. Threshold liability models were applied for binary traits (mastitis and excessive test-day SCC), and linear models were used for other normally distributed traits. For mastitis, a heritability of 0.07 was obtained. Heritability estimates for mean SCS in early lactation, standard deviation of SCS, excessive test-day SCC, UD, FUA, and BCS were 0.10, 0.04, 0.06, 0.41, 0.21, and 0.18, respectively. Mastitis was highly correlated with mean SCS in early lactation (0.63), standard deviation of SCS (0.74), and excessive test-day SCC (0.76). Moderate genetic correlations of -0.36 , -0.24 , and -0.28 were found between mastitis and UD, FUA, and BCS, respectively. As much as 72% of the genetic variation in mastitis resistance was explained by all the indirect predictor traits, whereas the most commonly used indirect measures of mastitis resistance (SCS in early lactation, UD, and FUA) explained together only 46% of the genetic variation in mastitis resistance. A combination of mean and standard deviation of SCS seem to be more successful in improving udder health than the traditional indirect measures. The results of the present study highlight that although routine cow SCC is the best measurement to monitor udder health, it cannot explain all the genetic variation in mastitis

resistance and, therefore, direct information on mastitis resistance can be expected to yield to a more accurate genetic evaluation for this trait.

Key words: mastitis, predictor, genetic correlation

Short Communication

Mastitis, an inflammation of the mammary gland caused by bacteria, is one of the most frequent and costly diseases in dairy cows. As disease-recording systems are not widely implemented, selection for mastitis resistance is in many countries based on genetically correlated traits, such as SCC, udder depth (UD), and fore udder attachment (FUA; Interbull, 2012). Recently, other traits that are derived from SCC have been suggested as alternatives to improve genetic evaluations for mastitis resistance (de Haas et al., 2008; Koeck et al., 2012; Urioste et al., 2012). Several studies have also reported favorable associations between mastitis and BCS (Lassen et al., 2003; Dechow et al., 2004). In Canada, a disease-recording system was implemented in 2007. Eight diseases, including mastitis, are recorded by producers voluntarily. The objectives of this study were to estimate genetic parameters for mastitis and its predictors (SCC, UD, FUA, and BCS) and to determine how much genetic variation in mastitis resistance can be explained by these traits.

Mastitis data from April 2007 to August 2011, as well as monthly test-day records of SCC between 5 and 305 DIM and type records for UD, FUA, and BCS were obtained from the Canadian Dairy Network (Guelph, ON, Canada). Mastitis data were recorded by producers on a voluntary basis. To ensure that all cows were from herds with reliable mastitis recording, only herds having at least 2 recorded mastitis cases within the study period were considered. The first and last mastitis case had to be at least 180 d apart for the herd to be included in the analysis; otherwise, it would be considered to have health recording for just a short period of time. In addition, a minimum mastitis frequency of 5% per herd and year was applied to ensure continuous

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¹Corresponding author: akoeck@uoguelph.ca

data recording. Mastitis was defined as a binary trait (0 = no mastitis and 1 = mastitis) based on whether or not the cow had at least 1 mastitis case in the period from calving to 305 d after calving. Three traits derived from SCC were considered as mastitis indicators based on a previous study in Canadian Holstein cows (Koeck et al., 2012). Mean SCS in early lactation (SCS_{150}) was the mean of monthly test-day SCS from 5 to 150 DIM. Somatic cell scores were calculated from SCC as follows: $\text{SCS} = \log_2(\text{SCC}/100,000) + 3$. The standard deviation of SCS (SCS_{SD}) was the standard deviation of monthly test-day SCS from 5 to 305 DIM. Excessive test-day SCC ($\text{TD}_{>500}$) was scored as 1 or 0, based on whether or not the cow had at least 1 SCC test-day above 500,000 cells/mL within 305 DIM. The type traits UD, FUA, and BCS were routinely recorded by professional type classifiers. Only first classifications within 365 DIM were analyzed; reclassification records were not considered. In this study, only first-lactation Holstein cows with an age between 19 and 43 mo were included. A summary of statistics for the analyzed data is given in Table 1.

The sire pedigree file was generated by tracing the pedigrees of sires and maternal grandsires back as far as possible. The sire pedigree file had 4,448 males, including the 3,389 sires with daughters in the data set.

For genetic analyses, a Bayesian approach using Gibbs sampling was applied. A 7-variate sire model was fitted, with probit (liability threshold) models for the 2 binary traits (mastitis and $\text{TD}_{>500}$) and Gaussian models for SCS_{150} , SCS_{SD} , UD, FUA, and BCS. In matrix notation, the model was

$$\mathbf{Y} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}_h\mathbf{h} + \mathbf{Z}_s\mathbf{s} + \mathbf{e},$$

where $\mathbf{Y}$ is a vector of liabilities for the binary traits (mastitis and $\text{TD}_{>500}$) and phenotypic observations for the continuous traits (SCS_{150} , SCS_{SD} , UD, FUA, and BCS); $\boldsymbol{\beta}$ is a vector of systematic effects, including fixed effects of herd-round-classifier for UD, FUA, and BCS, age at calving for mastitis, SCS_{150} , SCS_{SD} , and $\text{TD}_{>500}$, age at calving-stage of lactation for UD, FUA, and BCS, and year-season of calving for mastitis, SCS_{150} , SCS_{SD} , and $\text{TD}_{>500}$; $\mathbf{h}$ is a vector of random herd-year of calving effects for mastitis, SCS_{150} , SCS_{SD} , and $\text{TD}_{>500}$; $\mathbf{s}$ is a vector of random sire effects; $\mathbf{e}$ is a vector of random residuals; and $\mathbf{X}$, $\mathbf{Z}_h$, and $\mathbf{Z}_s$ are the corresponding incidence matrices. Age at first calving had 16 classes, in which <22 and >35 mo were the first and last class, respectively, and other classes were single months. Stage of lactation was coded in approximately 30-d intervals (1 = 0 to 30 DIM, 2 = 31 to 60 DIM, . . . , 10 = 271 to 300 DIM, and 11 = 301 to 365 DIM).

Four seasons of calving were defined from January to March, April to June, July to September, and October to December.

Random effects were assumed to be normally distributed with means equals to zero and covariance structure equal to

$$\text{Var} \begin{bmatrix} \mathbf{h} \\ \mathbf{s} \\ \mathbf{e} \end{bmatrix} = \begin{bmatrix} \mathbf{H}_0 \otimes \mathbf{I} & 0 & 0 \\ & \mathbf{G}_0 \otimes \mathbf{A} & 0 \\ \text{symm.} & & \mathbf{R}_0 \otimes \mathbf{I} \end{bmatrix},$$

$$\mathbf{H}_0 = \begin{bmatrix} \sigma_{h1}^2 & \sigma_{h1h2} & \sigma_{h1h3} & \sigma_{h1h4} \\ & \sigma_{h2}^2 & \sigma_{h2h3} & \sigma_{h2h4} \\ & & \sigma_{h3}^2 & \sigma_{h3h4} \\ \text{symm.} & & & \sigma_{h4}^2 \end{bmatrix}$$

is the (co)variance matrix between traits due to herd-year of calving effects; $\mathbf{I}$ is an identity matrix;

$$\mathbf{G}_0 = \begin{bmatrix} \sigma_{s1}^2 & \sigma_{s1s2} & \sigma_{s1s3} & \sigma_{s1s4} & \sigma_{s1s5} & \sigma_{s1s6} & \sigma_{s1s7} \\ & \sigma_{s2}^2 & \sigma_{s2s3} & \sigma_{s2s4} & \sigma_{s2s5} & \sigma_{s2s6} & \sigma_{s2s7} \\ & & \sigma_{s3}^2 & \sigma_{s3s4} & \sigma_{s3s5} & \sigma_{s3s6} & \sigma_{s3s7} \\ & & & \sigma_{s4}^2 & \sigma_{s4s5} & \sigma_{s4s6} & \sigma_{s4s7} \\ & & & & \sigma_{s5}^2 & \sigma_{s5s6} & \sigma_{s5s7} \\ & & & & & \sigma_{s6}^2 & \sigma_{s6s7} \\ \text{symm.} & & & & & & \sigma_{s7}^2 \end{bmatrix}$$

is the (co)variance matrix between traits due to sire transmitting abilities; $\mathbf{A}$ is the additive relationship matrix between sires; and

$$\mathbf{R}_0 = \begin{bmatrix} 1 & \sigma_{e1e2} & \sigma_{e1e3} & \sigma_{e1e4} & \sigma_{e1e5} & \sigma_{e1e6} & \sigma_{e1e7} \\ & \sigma_{e2}^2 & \sigma_{e2e3} & \sigma_{e2e4} & \sigma_{e2e5} & \sigma_{e2e6} & \sigma_{e2e7} \\ & & \sigma_{e3}^2 & \sigma_{e3e4} & \sigma_{e3e5} & \sigma_{e3e6} & \sigma_{e3e7} \\ & & & 1 & \sigma_{e4e5} & \sigma_{e4e6} & \sigma_{e4e7} \\ & & & & \sigma_{e5}^2 & \sigma_{e5e6} & \sigma_{e5e7} \\ & & & & & \sigma_{e6}^2 & \sigma_{e6e7} \\ \text{symm.} & & & & & & \sigma_{e7}^2 \end{bmatrix}$$

is the (co)variance matrix between traits due to residual effects. Residual variances of the binary traits (mastitis and $\text{TD}_{>500}$) were set to 1. Flat priors were assumed for herd-year, sire, and residual (co)variances.

Table 1. Summary of statistics for the data set used in the multiple trait evaluations for mastitis and its predictors

Trait	Description of trait	Number of records	Mean
Mastitis, %	Cows with at least 1 mastitis case within 305 d after calving	70,851	12.7
SCS ₁₅₀ , log ₂	Mean SCS in early lactation (5 to 150 DIM)	69,799	2.2
SCS _{SD} , log ₂	Standard deviation of SCS during lactation	67,238	1.0
TD _{>500} , %	Cows with at least 1 SCC test day above 500,000 cells/mL within 305 DIM	69,886	21.8
UD, cm	Udder depth (measurement from hock to floor of the udder)	62,923	10.25
FUA	Fore udder attachment (1 = extremely weak to 9 = extremely strong)	62,923	5.05
BCS	1 = very thin to 5 = very fat	62,923	2.81

Additionally a 4-variate sire model was fitted, with a probit (liability threshold) model for the binary trait, mastitis, and Gaussian models for SCS₁₅₀, UD, and FUA. Fixed and random effects were as described previously.

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 the convergence diagnostics of Raftery and Lewis (1992) and on visual inspections of trace plots using the Bayesian Output Analysis (BOA) software package (Smith, 2007). For both analyses, inference was based on a chain length of 300,000 samples, with samples stored at every round. The burn-in was 5,000 rounds.

The relative amount of genetic variation in mastitis resistance that can be explained by SCS₁₅₀, SCS_{SD}, TD_{>500}, UD, FUA, and BCS was examined through Cholesky decomposition of the genetic correlation matrix. The proportion of genetic variation in mastitis explained by these traits is the square of the elements in the last row of the resulting matrix $\mathbf{L}_{77}$ with mastitis as the last trait. The order of traits in the genetic correlation matrix used to obtain the proportion of genetic variation in mastitis resistance that was explained by its predictors was SCS₁₅₀, UD, and FUA (traits in the traditional model), and then SCS_{SD}, TD_{>500}, and BCS (additional traits in the full model), with mastitis being the last trait.

Mastitis frequency was 12.7% in first-lactation Holstein cows (Table 1). Genetic parameters for mastitis

and its predictors from the 7-variate analysis are given in Table 2. Results for the traits included in the 4-variate analysis were almost identical (data not shown). All posterior distributions of heritabilities and genetic correlations were sharp and quite symmetric (data not shown). For mastitis, a heritability of 0.07 was obtained, which is in agreement with literature results applying threshold models (Heringstad et al., 2000; Urioste et al., 2012). When transformed to the observable scale using the classical formula of Dempster and Lerner (1950), the heritability estimate of mastitis was 0.03 and, thus, fairly similar to a previous estimate in Canadian Holstein cows using a linear model (Koeck et al., 2012). Heritability estimates for the mastitis predictors SCS₁₅₀, SCS_{SD}, TD_{>500}, UD, FUA, and BCS were 0.10, 0.04, 0.06, 0.41, 0.21, and 0.18, respectively. Urioste et al. (2012) obtained higher heritability estimates of 0.14, 0.14, and 0.12 for mean SCC in early lactation, standard deviation of SCC, and TD_{>500} in first-parity Swedish Holsteins, respectively. Estimates of the remaining traits were in agreement with previous studies. In first-lactation Danish Holstein cows, Sørensen et al. (2000) reported heritabilities of 0.36 and 0.26 for UD and FUA, respectively. In the same population, Lassen et al. (2003) found a heritability of 0.25 for BCS.

Mastitis was highly correlated with SCS₁₅₀, SCS_{SD}, and TD_{>500}, with estimates of 0.63, 0.74, and 0.76, respectively. In a previous study in Canadian Holstein cows, SCS_{SD} and TD_{>500} tended also to show stronger genetic correlations with mastitis than SCS₁₅₀ (Koeck et al., 2012). Moderate genetic correlations of -0.36 ,

Table 2. Posterior means (SD) of heritabilities (on the diagonal) and genetic correlations (above the diagonal) for mastitis, mean SCS in early lactation (SCS₁₅₀), standard deviation of SCS (SCS_{SD}), presence of at least 1 test-day SCC above 500,000 cells/mL (TD_{>500}), udder depth (UD), fore udder attachment (FUA), and BCS from 7-variate analysis

Trait	Mastitis	SCS ₁₅₀	SCS _{SD}	TD _{>500}	UD	FUA	BCS
Mastitis	0.07 (0.01)	0.63 (0.08)	0.74 (0.09)	0.76 (0.08)	-0.36 (0.09)	-0.24 (0.10)	-0.28 (0.11)
SCS ₁₅₀		0.10 (0.01)	0.35 (0.09)	0.90 (0.03)	-0.26 (0.07)	-0.22 (0.07)	-0.08 (0.08)
SCS _{SD}			0.04 (0.01)	0.62 (0.08)	-0.31 (0.09)	-0.15 (0.10)	-0.27 (0.11)
TD _{>500}				0.06 (0.01)	-0.30 (0.09)	-0.25 (0.09)	-0.23 (0.10)
UD					0.41 (0.03)	0.77 (0.03)	0.03 (0.07)
FUA						0.21 (0.02)	0.19 (0.07)
BCS							0.18 (0.02)

Table 3. Genetic variation in mastitis resistance sequentially explained by traits in the traditional evaluation model [mean SCS in early lactation (SCS_{150}), udder depth (UD), and fore udder attachment (FUA)], and by traits in a full model, which also included standard deviation of SCS (SCS_{SD}), presence of at least 1 SCC test-day above 500,000 cells/mL ($TD_{>500}$), and BCS

Trait	Explained genetic variation in mastitis resistance, %	
	Traditional model (4-variate analysis)	Full model (7-variate analysis)
SCS_{150}	39.7	39.7
UD	5.5	4.1
FUA	1.1	0.6
SCS_{SD}	—	26.6
$TD_{>500}$	—	0.2
BCS	—	0.8
Total	46.3	72.0

−0.24, and −0.28 were found between mastitis and UD, FUA, and BCS, respectively. In Danish dairy cattle, Sørensen et al. (2000) reported similar correlations, with estimates ranging from −0.27 to −0.56 between mastitis and UD and from −0.28 to −0.47 between mastitis and FUA. Lassen et al. (2003) obtained a weaker genetic correlation of −0.16 between BCS and mastitis in first-lactation Danish Holstein cows. The estimated genetic correlations among the mastitis predictors were mostly in agreement with previous studies (Sørensen et al., 2000; Kadarmideen, 2004). Urioste et al. (2012), however, obtained a much higher genetic correlation of 0.97 between mean SCC in early lactation and standard deviation of SCC, which is mainly due to differences in the trait definition. In the study of Urioste et al. (2012), mean and standard deviation were calculated from log SCC. Therefore, both traits are strongly influenced by peak values and a high genetic correlation between these traits can be expected.

As much as 72% of the genetic variation in mastitis resistance was explained by all the indirect predictor traits in a full model (SCS_{150} , UD, FUA, SCS_{SD} , $TD_{>500}$, and BCS; Table 3). The trait SCS_{SD} , which is strongly correlated to mastitis, but less strongly correlated to mean SCS, added significant new information. In comparison, SCS_{150} , UD, and FUA, which are often used as mastitis indicators, explained together only 46% of the genetic variation in mastitis resistance. Therefore, a combination of SCS_{150} and SCS_{SD} may be more successful in improving udder health than the commonly used traits mean SCS, UD, and FUA.

The results of the present study highlight that although routine cow SCC is the best measurement to monitor udder health, SCC cannot explain all the genetic variation in mastitis resistance. Therefore, dairy

producers should be encouraged to keep accurate and complete mastitis data, which will provide direct information on mastitis resistance and will be expected to yield to a more accurate genetic evaluation for this trait.

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