Genetic correlations between pathogen-specific mastitis and somatic cell count in Danish Holsteins

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ABSTRACT

The aim of this study was to estimate genetic correlations (r_a) between 2 lactation average somatic cell count (LASCC) traits and 6 different mastitis traits in 226,482 first-parity Danish Holstein cows that calved between 1998 and 2008. The LASCC traits were defined from 5 to either 170 d (LASCC_170) or 300 d (LASCC_300) after calving, and the mastitis traits were unspecific mastitis (all mastitis treatments, both clinical and subclinical, regardless of the causative pathogen) and mastitis caused by either *Streptococcus dysgalactiae*, *Escherichia coli*, coagulase-negative staphylococci (CNS), *Staphylococcus aureus*, or *Streptococcus uberis*. Variance components were estimated using bivariate threshold-Gaussian models via Gibbs sampling. The posterior means of r_a between LASCC_170 and the mastitis traits were greatest for unspecific mastitis ($r_a = 0.71$), followed by CNS, *Strep. dysgalactiae*, *Strep. uberis*, and *E. coli* ($r_a = 0.54$ to 0.69) and were lowest for *Staph. aureus* mastitis ($r_a = 0.44$). The genetic correlation between LASCC_300 and the mastitis traits were generally smaller ($r_a = 0.47$ to 0.69). Caution should be taken when interpreting the results, however, because some posterior density intervals for r_a were large (between 0.14 and 0.47 units). Phenotypically, *Staph. aureus* is known to be associated with high SCC and especially with subclinical mastitis through chronic infections, so the low r_a between *Staph. aureus* mastitis and LASCC, compared with r_a for the other pathogens, was not expected. Subclinical cases are usually submitted to dry cow therapy (not included in the present study), not treated at all, or wrongly recorded as clinical cases. Thus, the incidence of *Staph. aureus* mastitis is likely too low, and the genetic correlation between *Staph. aureus* mastitis and LASCC may therefore be underestimated in the present study. The results for the remaining pathogens were as expected, smallest for

E. coli and larger but similar for *Strep. dysgalactiae*, *Strep. uberis*, and CNS. Selection for lower LASCC is expected to decrease the incidence of pathogen-specific mastitis, especially for *Strep. uberis*, *Strep. dysgalactiae*, and CNS and, to a lesser extent, for *Staph. aureus* and *E. coli*. Data recording should preferably be improved, and economic weights for the pathogen-specific mastitis traits should be estimated before implementing an udder health index that includes pathogen-specific mastitis traits.

Key words: mastitis, somatic cell count, pathogen specific, genetic parameter

INTRODUCTION

In many dairy-producing countries, genetic selection for improved mastitis resistance is performed indirectly by using traits genetically correlated to mastitis (e.g., SCC and udder conformation traits). In the Nordic countries (Denmark, Finland, Norway, and Sweden), records of mastitis treatments are available and used for direct selection for mastitis resistance. Moreover, in Denmark, Finland, and Sweden, SCC and udder conformation are used as additional correlated information. Somatic cell count is widely considered to be the most useful indirect measure of mastitis resistance (De Jong and Lansbergen, 1996). Heringstad et al. (2000) found (based on literature values) that heritability estimates for SCC ranged from 0.08 to 0.19 when using a lactation average model for SCC. The heritability for SCC derived from a test-day model for SCC ranged from 0.09 in the beginning of the lactation to approximately 0.20 at the end of the lactation (e.g., Liu et al., 2000). Genetic correlations (r_a) between mastitis and SCC were moderate to high, with an average of 0.60 for Nordic field data as summarized by Heringstad et al. (2000).

In the Nordic cattle, genetic evaluation (Denmark, Finland, and Sweden) SCC traits are defined as lactation average SCC (**LASCC**) in the period from 5 to 170 d after the first, second, and third calving. The relatively short collection period (5 to 170 d) is used

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because most cases of mastitis occur early in the lactation, the information is available earlier, and it reduces bias that may otherwise occur because of culling of cows. The magnitude of LASCC can be thought of as a function of a base level SCC, the number of clinical mastitis (CM) and subclinical mastitis (SCM) cases, the causative pathogen, and time from calving until mastitis is observed. However, LASCC largely ignores test-day variation caused by short-term environmental variation (Reents et al., 1995).

De Haas et al. (2002) showed that mastitis caused by different pathogens affected the lactation curve for SCC differently on the phenotypic level. For example, an increase in SCC (from a baseline SCC curve) was observed before a clinical case of *Staphylococcus aureus* mastitis, suggesting a prior subclinical state, and SCC remained high after the case of *Staph. aureus* mastitis was observed. In contrast, SCC was low before a case of *Escherichia coli* mastitis and decreased rapidly after the infection to a level only slightly larger than the level before infection. Evidently, mastitis caused by *Staph. aureus* and other bacteria responsible for elevated SCC for a long period of time is more likely to influence LASCC than mastitis caused by, for example, *E. coli*, which causes short-term variation in SCC. In addition, mastitis caused by pathogens such as *Staph. aureus* that occurs early in lactation will have a greater impact on LASCC than cases occurring in late lactation because SCC usually stays high after infections caused by *Staph. aureus* owing to establishment of chronic infections. At the genetic level, associations between pathogen-specific mastitis and SCC patterns were less clear (De Haas et al., 2003). For example, the genetic correlation (SE) between *E. coli* mastitis and the “quick recovery pattern” was 0.92 (0.25), whereas it was 0.66 (0.27) for *Staph. aureus* mastitis. For the “slow recovery pattern,” no difference was found between mastitis caused by *E. coli* and *Staph. aureus* because the genetic correlation in both cases was close to unity. The reason for these unexpected results as seen from a biological point of view could be an overlap between definitions of patterns or that SCC patterns are unsuitable in a genetic context because most pathogens, phenotypically, can be associated with several patterns.

Sørensen et al. (2009) demonstrated that different pathogen-specific mastitis traits can be considered as different traits genetically ($r_a = 0.45$ to 0.77). They showed that the genetic correlation between mastitis caused by pathogens with similar pathogenesis and induction of mammary immune response, such as *Streptococcus uberis* and *Streptococcus dysgalactiae*, was larger than the genetic correlation between pathogens such as *Staph. aureus* and *E. coli*. Because genetic correlations among pathogen-specific traits differ and are

less than unity, they may be considered different traits in a breeding goal. Therefore, the genetic association between mastitis and SCC may also be pathogen-specific, and SCC may be helpful in determining true breeding goals for different pathogens causing mastitis in dairy cattle. A pathogen-specific breeding program for udder health may be beneficial because pathogen information provides direct measures of udder infections, in contrast to mastitis treatments. At present, a large number of cows, in which bacteriological culturing of milk samples revealed no pathogens, are treated for mastitis and these records are included in the breeding value estimation. However, it is questionable whether these records indicate good or poor mastitis resistance, and they should preferably be disregarded or treated differently from mastitis cases in which pathogens are present. In addition, the economic values of pathogen-specific mastitis traits (environmental vs. contagious pathogens) differ (Østergaard et al., 2005), and the selection index should preferably account for this.

To consider pathogen-specific mastitis in a breeding goal, the genetic correlations with all or the most important traits in the selection index need to be known. In this study, LASCC was used to define the SCC trait because LASCC is currently used in the genetic evaluation of udder health in the Nordic countries. The genetic correlation between mastitis caused by *E. coli* and LASCC was expected to be small because this pathogen has a short-term effect on SCC, whereas the genetic correlation between mastitis caused by pathogens that have a long-term effect on SCC was expected to be greater. The objective of this study was to estimate genetic correlations between pathogen-specific mastitis traits and LASCC by using a large field data set from Danish Holstein cows.

MATERIALS AND METHODS

Data and Edits

Records of CM and SCM treatments, test-day SCC, and pathogen information (results of bacteriological culturing of milk samples from mastitic cows) were extracted from the Danish National Cattle Database. In the present study, no distinction was made between CM and SCM. In Denmark, CM is defined as visible changes in milk composition or visible clinical changes to the udder, or both. Subclinical mastitis is defined as an infection of the udder without clinical symptoms and is usually discovered using the California Mastitis Test, indicator paper, or examination of SCC or electric conductivity (International Dairy Federation, 1987). The initial data set contained records from first-parity Holstein cows that had calved between January

1998 and January 2008. However, lactations that began in March 2007 (300 d before the end of the collection period) or later were removed to reduce bias caused by censoring. Additionally, age at first calving was required to be between 19 and 36 mo. Furthermore, only records from -15 to 300 d after calving (period at risk) were included. Finally, participating herds had to have at least 30 first calvings per year; that is, if a herd had only 29 calvings in a particular year, records from that herd in that year were excluded from the analysis. This number was chosen arbitrarily to 1) avoid extreme category problems during analysis (e.g., Moreno et al., 1997), 2) make genetic parameters represent presumed future herd sizes, and 3) reduce the amount of data to a manageable size for analyses.

In Denmark, cows with CM or SCM are treated by veterinarians or farmers with a special health agreement only. Approximately 12% of dairy farmers have signed up for the special health agreement. The same persons, together with veterinarians, are also responsible for reporting treatments of mastitis to the national database. However, not all veterinarians and farmers do this on a regular basis, which implies that not all herds can be considered as having reliable disease recording. Requirements for active participation in disease recording were the following (Nielsen et al., 2000). Considering a (moving) window of 9 mo, each herd had to have at least 10 calvings and at least 0.3 disease treatments (all diseases) per calving in each 9 mo interval. These summaries were done separately for each month of calving. Treatments were combined in 4 main categories: udder diseases, reproductive diseases, digestive diseases, and foot and leg diseases. For example, if the requirements were fulfilled in the interval from January to September, but not February to October, only records from January were included. If the requirements were fulfilled again in the interval from March to November, all records from February were excluded. These requirements are currently used for prediction of breeding values for disease traits in Denmark, Finland, and Sweden and are assumed to reduce bias caused by inadequate reporting of disease treatments.

The database contains information about both mastitis treatments and pathogen recordings. Collecting milk samples from cows with a mastitis infection is voluntary in Denmark, except for farms with a special health agreement. The dairy farmer decides when to collect a milk sample for bacteriological testing. On some farms, milk samples are collected from all cows that are treated for mastitis, and on other farms, no samples are collected at all or the samples have not been registered in the database. An attempt was made to minimize the resulting sampling bias by data editing.

The date entered in the database for the pathogen may have been either the day of sampling or the day of recording of pathogen information into the national database. These dates may not be the same and may not be the date the cow actually was treated for mastitis. In the present study, 76.9% of all bacteriological samples were recorded -3 to 4 d after recordings of mastitis treatments (56.5% on the same day) when requirements for calving age, data censoring, and period at risk had been fulfilled. Hence, we chose this interval for assigning pathogen information to a mastitis incidence. The remaining 23.1% of the samples were excluded from the data set.

In a few cases (13%), more than 1 pathogen from the same cow was recorded on the same date. Observations of multiple pathogens in the same quarter are rarely seen compared with multiple pathogens in several quarters (C. M. Røntved, University of Aarhus, Foulum, Denmark, personal communication). However, because we are working at the cow level and there was no quarter-level information in the present study, it was assumed that a cow was treated for all recorded pathogens in the defined interval around the recorded mastitis treatment. Thus, a cow may have been "treated" for multiple pathogen-specific mastitis traits. Finally, to guarantee a certain amount of pathogen information, only herds that had pathogen information for at least 50% of the registered mastitis cases every year of disease recording were considered, and animals with pathogen information without a corresponding mastitis treatment were removed. Effects of major editing procedures are shown in Table 1. The cows were distributed in 17 calving age subclasses (1 for each mo) with an average calving age of 27 mo, 112 year-month subclasses, and with cows descended from 6,756 sires with an average of 33.5 daughters per sire, ranging from 1 to 16,011.

Definition of Traits

Mastitis was defined as an all-or-none trait in the period -15 to 300 d after first calving, that is, 1 if a cow was treated and 0 otherwise. Only the first observed mastitis treatment for each cow was used. The 5 most common pathogens (*Strep. dysgalactiae*, *E. coli*, CNS, *Staph. aureus*, and *Strep. uberis*) in the Danish Holstein herds were chosen to represent the pathogen-specific mastitis traits. The pathogen-specific traits were differentiated from unspecific mastitis by distinguishing between mastitis with pathogen information and mastitis without. The trait unspecific mastitis contained all recorded treatments of mastitis (i.e., both treatments with and without the growth of pathogens).

The LASCC was calculated as

Table 1. Numbers of animals and herds after major editing steps for the data set containing information on mastitis treatments and pathogen information and the data sets with addition of lactation average SCC (LASCC) from 5 to 170 d and 5 to 300 d after calving

Editing procedure	Animals, n	Herds, n
Initial data set	1,676,680	12,523
Calving age (19 to 36 mo)	1,564,768	11,677
Data censoring (January 1998 to January 2008)	1,450,266	11,586
Active disease recording	885,121	6,272
Minimum 50% pathogen data per herd	347,410	3,119
Minimum 30 first calvings per herd per year	247,080	1,748
Only pathogen information with matching mastitis treatments	237,356	1,748
Only known sires	226,482	1,747
With LASCC 5 to 170 d (Data_170)	226,482	1,747
With LASCC 5 to 300 d (Data_300)	226,482	1,747

$$\text{LASCC} = \frac{1}{m} \sum_{i=1}^m \ln \left(\frac{\text{SCC}_i / 1,000}{\text{mL}} \right),$$

where SCC_i is the number of somatic cells per milliliter of milk on test-day i if the test day is between 5 and 170 d (**LASCC_170**) or between 5 and 300 d (**LASCC_300**) after calving, and m is the number of test days.

From the final data set containing mastitis treatments and pathogen information, 2 data sets were created: The first (**Data_170**) contained LASCC_170 as currently used in the breeding value evaluation for udder health in Denmark, Finland, and Sweden. In contrast to the current breeding value estimation, a minimum of 3 test-day records of SCC were required for inclusion of LASCC in this data set to ensure a reasonable estimate of LASCC. For cows with fewer than 3 test-day records, LASCC_170 was set to missing. Variations in SCC caused by infections observed after d 170 are not captured when using LASCC_170. Thus, a second data set (**Data_300**) was created, containing LASCC_300. For this data set, a minimum of 5 test-day records of SCC were required, and for cows with fewer than 5 test-day records, LASCC_300 was set to missing.

Statistical Models and Analysis

The LASCC, unspecific, and pathogen-specific mastitis data were analyzed using a bivariate sire model with a threshold-liability (Wright, 1934) and a linear Gaussian trait for mastitis and LASCC, respectively. In addition, LASCC_170 and LASCC_300 were analyzed mutually using a bivariate linear model.

Threshold Model. In the threshold model, it is assumed that an underlying continuous variable exists, liability (λ), such that the observed binary variable takes the value 1 if λ is larger than a fixed threshold (τ), and takes the value 0 otherwise. With binary data,

τ and the residual variance (σ_e^2) are not identifiable; therefore, these parameters were set to the arbitrary values, $\tau = 0$ and $\sigma_e^2 = 1$, such that

$$\lambda \mid \mu \sim N(\mu, \mathbf{I}).$$

The probability (π_i) that observation i is scored as 1, given the model parameter vector (θ), is

$$\begin{aligned} \pi_i &= \Pr(y_i = 1 \mid \theta) \\ &= \Pr(\lambda_i > 0 \mid \theta) \\ &= 1 - \Pr(\lambda_i \leq 0 \mid \theta) \\ &= \Phi(\mu_i), \end{aligned}$$

where $\Phi(\cdot)$ is the standard normal cumulative distribution.

The following threshold-liability sire model was used for analysis of both unspecific and pathogen-specific mastitis traits:

$$\lambda_{ijklm} = YM_i + AGE_j + b_1 t_{ijklm} + hys_k + sire_l + e_{ijklm},$$

where λ_{ijklm} is the liability to mastitis of daughter m of sire l calving in year-month class i at calving age class j and herd-year-season class k , YM_i is the “fixed” effect of year-month of calving, AGE_j is the “fixed” effect of calving age, hys_k is the random effect of herd-year-season (season is the year divided into quarters) $sire_l$ is the transmitting ability of sire l , b_1 is the “fixed” regression coefficient of λ on the length of the period at risk, t_{ijklm} is the period at risk for daughter m of sire l , defined as the number of days from 15 d before calving to the date of culling or to the end of the risk period. It was assumed that all cows with mastitis had a full risk period. Following Heringstad et al. (2001), this reduces the recursive relationship between mastitis

Figure 1. Showing values of t and t^* for 4 different scenarios. In (a), the first line represents a cow with no occurrence of mastitis and a full lactation, and the second line represents a cow that gets culled at d 150 without any occurrence of mastitis. In (b), the first line represents a cow with mastitis on d 50, and the second line represents a cow that gets culled on d 150 and has mastitis on d 50.

and the length of the risk period; e_{ijklm} is the residual $\sim N(0,1)$ and independent.

Gaussian Model. The following linear sire model was used for analysis of LASCC:

$$y^* = YM_i + AGE_j + b_1 t_{ijklm} + b_2 t^*_{ijklm} + hys_k + sire_l + e_{ijklm},$$

where y^* is the LASCC_170 or LASCC_300, b_2 is the “fixed” regression coefficient of y^* on the length of the lactation after a case of mastitis has been observed, and t^*_{ijklm} is the length of the lactation (d) after a case of mastitis has been observed.

The remaining parameters are as described previously for the threshold model. An example of the concepts behind t and t^* in the models is given in Figure 1.

In matrix notation, the bivariate models can be expressed as

$$\Pr(\mathbf{y} \mid \mathbf{b}, \mathbf{h}, \mathbf{s}) = \begin{bmatrix} \boldsymbol{\lambda} \\ \mathbf{y}^* \end{bmatrix} = \begin{bmatrix} \mathbf{X}_{b1} & \mathbf{0} \\ \mathbf{0} & \mathbf{X}_{b2} \end{bmatrix} \begin{bmatrix} \mathbf{b}_1 \\ \mathbf{b}_2 \end{bmatrix} + \begin{bmatrix} \mathbf{X}_{h1} & \mathbf{0} \\ \mathbf{0} & \mathbf{X}_{h2} \end{bmatrix} \begin{bmatrix} \mathbf{h}_1 \\ \mathbf{h}_2 \end{bmatrix} + \begin{bmatrix} \mathbf{Z}_1 & \mathbf{0} \\ \mathbf{0} & \mathbf{Z}_2 \end{bmatrix} \begin{bmatrix} \mathbf{s}_1 \\ \mathbf{s}_2 \end{bmatrix} + \begin{bmatrix} \mathbf{e}_1 \\ \mathbf{e}_2 \end{bmatrix},$$

where $\boldsymbol{\lambda}$ is an $n_1 \times 1$ vector of the underlying liabilities of mastitis, $\mathbf{y}^*$ is an $n_2 \times 1$ vector of LASCC_170 or an $n_3 \times 1$ vector of LASCC_300, n_i is the number of records for each trait, $\mathbf{b}$ is a vector of “fixed” effects of order p as described previously, $\mathbf{h}$ is a vector of random herd-year-season effects of order q , $\mathbf{s}$ is a vector of random sire effects of order r , and $\mathbf{e}$ is a vector of residual effects. $\mathbf{X}_b$, $\mathbf{X}_h$, and $\mathbf{Z}$ are corresponding incidence matrices. The variance components of this model were

$$\text{Var} \begin{bmatrix} \mathbf{h}_1 \\ \mathbf{h}_2 \end{bmatrix} = \mathbf{H} = \mathbf{H}_0 \otimes \mathbf{I}_n,$$

where

$$\mathbf{H}_0 = \begin{bmatrix} \sigma_{h1}^2 & \sigma_{h1h2} \\ \sigma_{h1h2} & \sigma_{h2}^2 \end{bmatrix},$$

$\mathbf{I}_n$ is an identity matrix of an order (n) equal to the number of records for both traits, $\otimes$ denotes the Kronecker product, and σ_h^2 is the herd-year-season variance,

$$\text{Var} \begin{bmatrix} \mathbf{s}_1 \\ \mathbf{s}_2 \end{bmatrix} = \mathbf{G} = \mathbf{G}_0 \otimes \mathbf{A},$$

where

$$\mathbf{G}_0 = \begin{bmatrix} \sigma_{s1}^2 & \sigma_{s1s2} \\ \sigma_{s1s2} & \sigma_{s2}^2 \end{bmatrix},$$

$\mathbf{A}$ is the additive genetic relationship matrix containing the relation between sires, and σ_s^2 is the sire variance. Relationships among sires were traced as far back as possible (1 to 8 generations),

$$\text{Var} \begin{bmatrix} \mathbf{e}_1 \\ \mathbf{e}_2 \end{bmatrix} = \mathbf{R} = \mathbf{R}_0 \otimes \mathbf{I}_n,$$

where

$$\mathbf{R}_0 = \begin{bmatrix} 1 & \sigma_{e1e2} \\ \sigma_{e1e2} & \sigma_{e2}^2 \end{bmatrix}$$

and σ_e^2 is the residual variance. Thus, the residual correlation is $\sigma_{e1e2} / \sigma_{e1}$.

A full Bayesian approach via Gibbs sampling and implemented in the DMU package (Madsen and Jensen, 2006) was used to fit the models and analyze data. Conditional on $\boldsymbol{\theta}$, the distribution of the observed binary mastitis data was assumed to be the product of n independent Bernoulli trials with conditional probability distribution:

$$\Pr(\mathbf{y} | \boldsymbol{\theta}) = \prod_{i=1}^n \pi_i^{y_i} (1 - \pi_i)^{(1-y_i)}, \quad [1]$$

where $\pi_i = \Phi(\mathbf{x}'_{bi}\mathbf{b} + \mathbf{x}'_{hi}\mathbf{h} + \mathbf{z}'_i\mathbf{s})$ and $\mathbf{x}'_b$, $\mathbf{x}'_h$, and $\mathbf{z}'_i$ are the i th row vector of $\mathbf{X}_b$, $\mathbf{X}_h$, and $\mathbf{Z}$.

Prior Distributions. Adopted prior distributions of the parameters of the bivariate threshold-liability model were:

$$\Pr(\mathbf{b}) \propto \text{constant}, \quad [2]$$

$$\Pr(\mathbf{h} | \mathbf{H}_0) \sim MVN[\mathbf{0}, (\mathbf{H}_0 \otimes \mathbf{I}_n)], \quad [3]$$

$$\Pr(\mathbf{s} | \mathbf{G}_0, \mathbf{A}) \sim MVN[\mathbf{0}, (\mathbf{G}_0 \otimes \mathbf{A})], \quad [4]$$

$$\Pr(\mathbf{H}_0 | \mathbf{S}_h, \boldsymbol{\nu}_h) \sim IW_j[\mathbf{S}_h \boldsymbol{\nu}_h, \boldsymbol{\nu}_h], \quad [5]$$

$$\Pr(\mathbf{G}_0 | \mathbf{S}_g, \boldsymbol{\nu}_g) \sim IW_j[\mathbf{S}_g \boldsymbol{\nu}_g, \boldsymbol{\nu}_g], \text{ and} \quad [6]$$

$$\Pr(\mathbf{R}_0 | \mathbf{S}_R, \boldsymbol{\nu}_R) \sim CIW_j[\mathbf{S}_R \boldsymbol{\nu}_R, \boldsymbol{\nu}_R]. \quad [7]$$

In equations [2] to [7] $MVN[.,.]$ is the multivariate normal distribution. $IW_j[.,.]$ is the $j \times j$ scaled inverse Wishart distribution, where $j = 2$ is the dimension of the herd and genetic covariance matrices $\mathbf{H}_0$ and $\mathbf{G}_0$, respectively. CIW_j is the conditional scaled inverse Wishart distribution of dimension $j = 2$ equal to the dimension of $\mathbf{R}_0$ given the residual variance for the liability to mastitis. $\mathbf{S}_h$, $\boldsymbol{\nu}_h$, $\mathbf{S}_g$, $\boldsymbol{\nu}_g$, $\mathbf{S}_R$, $\boldsymbol{\nu}_R$ are known scale parameters (prior values) and degrees of freedom for IW (degrees of belief) for herd-year-season and additive genetic and residual covariances, respectively. Prior values were set arbitrarily to 0.026 for the sire variance and 0.065 for the herd-year-season variance for both

the LASCC and the mastitis traits. The prior values for the residual variance were set arbitrarily to 0.01 and 1.0 for the LASCC and the mastitis traits, respectively. Degrees of belief were chosen to represent vague a priori information ($\nu_h = \nu_g = \nu_R = 4$). The residual covariance matrix was sampled using an alternative normalization and a decomposition of the inverted Wishart density as described in González (2003) and Korsgaard et al. (2005).

The joint posterior density of all unknowns in the model was given as the product of densities in equations [1] to [6]:

$$\begin{aligned} & \Pr(\mathbf{b}, \mathbf{h}, \mathbf{s}, \mathbf{H}_0, \mathbf{G}_0 | \mathbf{y}) \propto \\ & \Pr(\mathbf{y} | \mathbf{b}, \mathbf{h}, \mathbf{s}) \Pr(\mathbf{b}) \Pr(\mathbf{h} | \mathbf{H}_0) \\ & \Pr(\mathbf{s} | \mathbf{G}_0 \otimes \mathbf{A}) \Pr(\mathbf{H}_0) \Pr(\mathbf{G}_0). \end{aligned} \quad [8]$$

The joint posterior density in equation [8] was augmented with the unknown liabilities λ_i (Tanner and Wong, 1987). The conditional posterior distributions of the model parameters were normal for the location parameters, scaled inverse Wishart for the sire and herd-year-season variance, and truncated normal for the liabilities.

Convergence Diagnostics. Convergence of the Gibbs chain was determined by using a standardized time-series method of batch means (Glynn and Iglehart, 1990; Geyer, 1992). The chain of length α was divided into β batches of equal size. Then the batch means

$$S_{\alpha, \delta} = \frac{\beta}{\alpha} \sum_{q=(\delta-1)\alpha/\beta+1}^{\delta\alpha/\beta} g(Q_i), \quad \delta = 1, \dots, \beta,$$

converge to independent, identically distributed random variables. Convergence of the batch means was then checked by standard one-way ANOVA and by estimating the lag correlation between batch means. The effective sample size (**ESS**) was calculated as $\text{ESS} = V_w/V_b$, where V_w is the variance within batches and V_b is the variance between batches. The information criterion was set arbitrarily to $\text{ESS} \geq 100$. The Gibbs sampler was run as a single long chain and every 10th sample was saved for post-Gibbs analyses. A chain length of 300,000 samples and a burn-in of 50,000 samples were used for all bivariate analyses.

RESULTS

The mean incidence rate of unspecific mastitis was 23 cases per 100 cows for both data sets (Data_170 and Data_300), and pathogen information was avail-

Table 2. Frequency of different pathogens in the original pathogen data and incidence proportions (%) of pathogen-specific mastitis cases from the 2 data sets (Data_170 and Data_300) used in this study

Pathogen	Frequency in original pathogen data, %	Data_170 incidence proportions, ¹ %	Data_300 incidence proportions, ¹ %
<i>Streptococcus dysgalactiae</i>	9.4	15.0	14.9
<i>Escherichia coli</i>	10.4	13.2	13.1
CNS	13.6	14.8	14.8
<i>Staphylococcus aureus</i>	15.7	15.5	15.4
<i>Streptococcus uberis</i>	13.9	18.7	18.6
Others	15.9	13.5	13.4
Culture negative	21.1	23.4	23.6
Sum	100	114.1 ²	113.8 ²

¹In the edited data set used for genetic inferences. Incidence of mastitis cases caused by the different pathogens.

²The sum of the pathogen-specific frequencies does not add to 100% in the final data set because, in several cases, more than 1 pathogen was found for the same case of mastitis.

able for 73% of the mastitis cases for both Data_170 and Data_300 following the restriction on the amount of pathogen information (pathogen information on a minimum of 50% of mastitis treatments within a herd). The occurrences of the different groups of pathogens in the original pathogen data and the 2 final data sets are shown in Table 2. The number of missing LASSC records was 10.9 and 13.5% for LASSC_170 and LASSC_300, respectively. Cows without pathogen-specific mastitis had on average lower LASSC_170 (Table 3) than cows with pathogen-specific mastitis (e.g., 73,700 and 109,900 cells/mL for cows without and with *Staph. aureus* mastitis, respectively), and LASSC_300 was greater than with LASSC_170 (e.g., 79,000 and 117,900 cells/mL for cows without and with *Staph. aureus* mastitis, respectively). For cows without pathogen-specific mastitis, only minor differences in LASSC_170 were found (with 2 digits after the decimal sign, the values were similar) in contrast to LASSC_300.

Posterior means, standard deviations, 95% highest posterior density intervals (HPDI), and ESS for the additive genetic correlations, r_a , among the LASSC traits and the unspecific and pathogen-specific mastitis traits are given in Table 4. The posterior distributions of the genetic correlations were all symmetric (not shown). Mixing of the Gibbs chains for the genetic covariances was slow, and the autocorrelations between sampled values for the genetic correlations were large (Figure 2). Consequently, the ESS for the genetic correlations was low (Table 4). Tests of equality between batch means were, in all cases, nonsignificant ($P > 0.05$; results not shown); therefore, it was assumed that all Gibbs chains had reached convergence with the applied chain length and burn-in.

The posterior means of the genetic correlations were all moderate to large and were largest for LASSC_170 and unspecific mastitis, $r_a = 0.71$, and smallest for LASSC_170 and *Staph. aureus* mastitis, $r_a = 0.44$.

Table 3. Lactation average SCC for 5 to 170 d (LASSC_170) and 5 to 300 d (LASSC_300) after calving for healthy and mastitic cows (unspecific mastitis) and cows with and without pathogen-specific¹ mastitis (SD in parentheses)

Pathogen	Cows with mastitis		Cows without mastitis	
	LASSC_170	LASSC_300	LASSC_170	LASSC_300
Unspecific mastitis ²	4.59 (0.84)	4.64 (0.78)	4.22 (0.72)	4.30 (0.69)
<i>Streptococcus dysgalactiae</i>	4.57 (0.81)	4.62 (0.75)	4.30 (0.76)	4.38 (0.72)
<i>Escherichia coli</i>	4.55 (0.81)	4.60 (0.73)	4.30 (0.76)	4.38 (0.72)
CNS	4.57 (0.84)	4.38 (0.72)	4.30 (0.76)	4.62 (0.78)
<i>Staphylococcus aureus</i>	4.70 (0.88)	4.77 (0.83)	4.30 (0.76)	4.37 (0.72)
<i>Streptococcus uberis</i>	4.64 (0.85)	4.68 (0.77)	4.30 (0.76)	4.37 (0.72)

¹Consider the 5 different pathogen-specific groups as 5 different data sets. The same number of records (same animals) is in each data set within Data_170 and Data_300. Cows with and without mastitis are defined with 1 and 0, respectively. For example, in the *E. coli* data set, there are several records with 1 (cows with *E. coli* mastitis). The remaining records received 0. These records contain animals with mastitis (*E. coli* mastitis excluded) and animals without mastitis. In this way, calculation of LASSC for cows with and without *E. coli* mastitis is possible.

²Strictly cows with and without mastitis.

Considering the pathogen-specific traits only, the genetic correlations with LASCC_300 were smaller than with LASCC_170. In addition, the ranking of the genetic correlations were different for LASCC_170 and LASCC_300 because *Strep. uberis* and *Strep. dysgalactiae* ranked differently for LASCC_170 and LASCC_300. For the remaining 3 pathogens, ranking was similar. However, the differences between the pathogen-specific mastitis traits could be due to chance, considering the rather large 95% HPDI for the genetic correlations.

The posterior means of the phenotypic correlations, r_p , between pathogen-specific mastitis and LASCC (Table 5) were close to zero for all pathogens except for *Staph. aureus* ($r_p = 0.11$ for both LASCC_170 and LASCC_300). In addition, the phenotypic correlations between *Strep. uberis* and the LASCC traits ($r_p = 0.042$ and $r_p = 0.043$ for LASCC_170 and LASCC_300, respectively) were slightly higher than for the remaining pathogens. The phenotypic correlations between unspecific mastitis and the LASCC traits were higher than among the LASCC traits and the pathogen-specific mastitis traits, $r_p = 0.24$ and $r_p = 0.26$ for LASCC_170 and LASCC_300, respectively.

Summary statistics of variance components and heritabilities from the bivariate analyses of LASCC_170 and LASCC_300 with unspecific mastitis are given in Table 6. The posterior mean estimates of the variance components for LASCC_170 and LASCC_300 were of similar magnitude. The posterior means of the corresponding heritabilities were moderate, with $h^2 = 0.20$ and $h^2 = 0.23$ for LASCC_170 and LASCC_300, respectively. The corresponding HPDI were [0.17;0.22] and [0.21;0.26], respectively. The genetic and phenotypic correlations (SD) between LASCC_170 and LASCC_300 were close to unity, $r_a = 0.997$ (4×10^{-4}) and $r_p = 0.479$ (7.7×10^{-3}), respectively. Variance components for the mastitis traits from bivariate analyses with LASCC_170 are presented in Table 7. The heritability of unspecific mastitis was greater than that of pathogen-specific mastitis, $h^2 = 0.142$ and $h^2 = 0.039$ to 0.079, respectively. The largest heritability of the pathogen-specific traits was found for *Strep. uberis* ($h^2 = 0.079$), whereas the smallest was found for *Staph. aureus* ($h^2 = 0.039$).

Liability of pathogen-specific mastitis was greater with an increasing period at risk (Table 7; $b_1 = 0.026 \pm 0.006$ to 0.030 ± 0.013), and LASCC was smaller with an increasing period at risk ($b_1 = -0.002 \pm 5.2 \times 10^{-5}$). Both LASCC_170 and LASCC_300 increased with increasing length of the lactation after observing a case of mastitis ($b_2 = 0.001 \pm 1.6 \times 10^{-5}$). All regression coefficients were different from zero when considering $1.96 \times$ standard deviation for LASCC_170 and LASCC_300.

Table 4. Posterior means of genetic correlations between different mastitis traits (pathogen-specific and unspecific) and lactation average SCC (LASCC) traits (either d 5–170 or 5–300), posterior standard deviations, 95% highest posterior density intervals (HPDI), and estimated sample size (ESS)

Trait	Item	Unspecific mastitis	<i>Streptococcus dysgalactiae</i>	<i>Escherichia coli</i>	CNS	<i>Staphylococcus aureus</i>	<i>Streptococcus uberis</i>
LASCC_170	Mean (SD), 95% HPDI, ESS	0.71 (0.04), [0.64;0.78], 660	0.66 (0.08), [0.47;0.80], 166	0.54 (0.09), [0.34;0.71], 220	0.64 (0.09), [0.45;0.79], 173	0.44 (0.12), [0.19;0.66], 110	0.69 (0.07), [0.54;0.80], 234
LASCC_300	Mean (SD), 95% HPDI, ESS	0.69 (0.04), [0.61;0.76], 342	0.66 (0.09), [0.46;0.80], 91	0.60 (0.10), [0.35;0.73], 108	0.62 (0.10), [0.41;0.78], 86	0.47 (0.12), [0.22;0.68], 96	0.65 (0.07), [0.49;0.78], 117

DISCUSSION

Genetic and Phenotypic Correlations

Unspecific Mastitis. The estimated positive genetic correlation (Table 4) between unspecific mastitis and LASCC_170 were in agreement with other studies of Holstein populations (De Haas et al., 2003; Carlén et al., 2004; Johansson et al., 2006). The genetic correlation between LASCC_300 and unspecific mastitis was of a similar magnitude. Luttinen and Juga (1997) and De Haas et al. (2003) estimated lower values (SE), $r_a = 0.31$ (0.09) and 0.50 (0.13), respectively. However, Luttinen and Juga (1997) collected mastitis data from the first 150 d after calving only. This resulted in an overall low mastitis incidence of 0.109 and could have an effect on the estimated genetic correlation between mastitis and LASCC because data were analyzed with a linear model (Gianola, 1982). In Finnish Ayrshires, Negussie et al. (2006) estimated a genetic correlation (SE) between unspecific mastitis and LASCC (whole lactation) of $r_a = 0.70$ (0.06), although this result may not be comparable with results from Holsteins. Genetically, a mastitis case will have an effect on SCC through induction of the mammary immune response. Over time, this effect will decrease as the invading pathogen is killed and production of somatic cells returns to “normal” level, meaning that a mastitis case will have a larger effect on SCC on test days close to the time of the mastitis infection and less effect on SCC from more distant test days. Because most cases of mastitis happen early in lactation, we would expect the genetic correlation between unspecific mastitis and LASCC_300 to be smaller than between unspecific mastitis and LASCC_170. However, in the present study a part-whole relationship exists between LASCC_170 and LASCC_300 because the majority of the LASCC_170 records were included in the LASCC_300 data set. If only records of SCC from d 171 to d 300 after calving were used, the genetic correlation with unspecific mastitis would most likely have been smaller because of the low incidence of mastitis in this period (17% of the first mastitis cases in the first lactation were recorded after d 170). Emanuelson et al. (1988) separated test-day records of SCC before and after d 150 in Swedish Friesians, resulting in a small difference in the genetic correlation between mastitis and LASCC_150 and between mastitis and LASCC (whole lactation), $r_a = 0.79$ and 0.73 , respectively.

The phenotypic correlations (Table 5) between the LASCC_170 and unspecific mastitis were positive but smaller than the genetic correlations. These estimates agree with the estimates given by De Haas et al. (2003), who found a phenotypic correlation (SE) of 0.26 (0.01) between clinical mastitis and 2 LASCC traits,

Figure 2. Autocorrelations between sampled values for the genetic correlation between lactation average SCC from 5 to 170 d after calving and *Staphylococcus aureus* mastitis associated with lags 1 to 30. Autocorrelations were similar for the remaining pathogens.

LASCC_150 and LASCC_305, respectively. This indicates slightly higher LASCC in lactations with mastitis than in lactations without mastitis, which also was observed in the present study (Table 3). In the present study the phenotypic correlation between LASCC_300 and unspecific mastitis ($r_p = 0.26$) was slightly higher than between LASCC_170 and unspecific mastitis ($r_p = 0.24$).

Pathogen-Specific Mastitis. The genetic correlations between the pathogen-specific mastitis traits and the 2 LASCC traits were all moderate to high (Table 4), suggesting that genetic selection for lower LASCC improves resistance to mastitis regardless of the causative pathogen. The genetic correlations between the pathogen-specific mastitis traits and LASCC_170 were slightly larger (but of the same magnitude when considering HPDI) than the genetic correlations between the pathogen-specific mastitis traits and LASCC_300. The genetic correlations between LASCC and pathogen-specific mastitis tended to differ for different pathogens, although the results should be interpreted with caution when considering the large HPDI in this study. The lowest genetic correlation ($r_a = 0.45$) was found between *Staph. aureus* mastitis and LASCC_170. The result was consistent with the genetic correlation ($r_a = 0.53$) between *Staph. aureus* mastitis and LASCC_150 given by De Haas et al. (2003), considering the large standard errors in that study. Phenotypically, cases of *Staph. aureus* mastitis are usually of long duration

Table 5. Posterior means of phenotypic correlations between different mastitis traits (pathogen-specific and unspecific) and lactation average SCC (LASCC) traits (either d 5–170 or 5–300), posterior standard deviations, 95% highest posterior density intervals (HPDI), and estimated sample size (ESS)

Trait	Item	Unspecific mastitis	<i>Streptococcus dysgalactiae</i>	<i>Escherichia coli</i>	CNS	<i>Staphylococcus aureus</i>	<i>Streptococcus uberis</i>
LASCC_170	Mean (SD), 95% HPDI, ESS	0.160 (0.004), [0.153;0.168], 2,792	0.019 (0.005), [0.009;0.028], 6,440	0.021 (0.005), [0.011;0.030], 6,123	0.018 (0.005), [0.008;0.028], 5,356	0.106 (0.005), [0.096;0.116], 2,930	0.042 (0.005), [0.042;0.050], 4,968
LASCC_300	Mean (SD), 95% HPDI, ESS	0.264 (0.005), [0.253;0.274], 1,102	0.021 (0.005), [0.010;0.032], 3,007	0.021 (0.006), [0.010;0.032], 2,953	0.021 (0.006), [0.008;0.028], 3,530	0.113 (0.005), [0.103;0.124], 2,625	0.043 (0.005), [0.033;0.052], 2,958

and are associated with high SCC (Daley et al., 1991). This result was also observed in the present study because the phenotypic correlation ($r_p = 0.11$) between *Staph. aureus* mastitis and LASCC was high compared with the remaining pathogens. *Staphylococcus aureus* is known to establish chronic mammary infections, as indicated by prolonged periods of elevated SCC (Wilson and Richards, 1980). In such cases, the mammary immune system is attempting to control the pathogen, as indicated by the elevated SCC, but is unable to annihilate the infection because *Staph. aureus* avoids killing by macrophages and so forth, for example, by invasion of the mammary epithelial cells (Almeida et al., 1996) or production of virulence factors (Baselga et al., 1994). These circumstances are reflected by a smaller genetic correlation between LASCC and *Staph. aureus* compared with other mastitis pathogens. A common feature of *Staph. aureus* and, to a lesser degree, other pathogens is the ability to establish subclinical infections, which are related to increased SCC through cyclic shedding of bacteria (Daley et al., 1991). This fact may explain why a lower SCC does not improve genetic resistance as much for *Staph. aureus* mastitis as for the remaining pathogens. Usually, subclinical infections are discovered when pathogens are found in milk samples from cows in which the test-day SCC exceeds a given threshold (e.g., 400,000 cells/mL, depending on parity, etc.). In Denmark, culturing of milk from cows with elevated SCC is a common practice at the time of drying off because detection of a pathogen is required before dry cow therapy can be carried out. These treatments are recorded as “dry cow therapy” and are therefore not included in the present study. It is possible to record a mastitis treatment as a subclinical case. However, this option is rarely used; in the present study, approximately 1% of mastitis treatments were recorded as SCM, which is very likely to be lower than the true number of SCM cases in first-parity cows. For example, Sloth et al. (2003) found an incidence for SCM of approximately 0.22 across parities. Instead, cases of SCM might not have been treated and recorded at all, or they might have been recorded wrongly as “mastitis (unspecified).”

The genetic correlation between *E. coli* mastitis and LASCC was higher than the genetic correlation between *Staph. aureus* mastitis and LASCC. These results agree with the estimates presented by De Haas et al. (2003), who found estimates (SE) of $r_a = 0.68$ (0.17) and 0.54 (0.20) for LASCC_150 and LASCC_305, respectively. In addition, the magnitude of the genetic correlation between *E. coli* mastitis and LASCC seems reasonable when compared with *Strep. dysgalactiae*, CNS, and *Strep. uberis* because cows with mastitis caused by these pathogens have slightly higher LASCC (Table 3) and

Table 6. Mean, standard deviation, and effective sample size (ESS) of the posterior distributions of sire (σ_{sire}^2), herd-year-season (σ_{hys}^2), residual (σ_{e}^2) variances, and within-herd heritability¹ (h^2) of lactation average SCC (LASCC_170 and LASCC_300)¹

Trait	Item	σ_{sire}^2	σ_{hys}^2	σ_{e}^2	h^2	HPDI
LASCC_170	Mean	0.025	0.063	0.476	0.196	[0.174;0.220]
	SD	0.002	0.001	0.002	0.012	
	ESS	1,013	12,362	28,012	1,008	
LASCC_300	Mean	0.030	0.066	0.447	0.234	[0.210;0.259]
	SD	0.002	0.001	0.002	0.013	
	ESS	1,842	35,970	8,862	2,109	

¹In brackets, 95% highest posterior density intervals (HPDI) of the heritabilities. $h^2 = 4 \times \sigma_{\text{sire}}^2 / (\sigma_{\text{sire}}^2 + \sigma_{\text{e}}^2)$.

affect LASCC for a longer period (De Haas et al., 2002) than cows with mastitis caused by *E. coli*. *Staphylococcus aureus* may be considered a special case because of the more frequent ability of this pathogen to establish SCM compared with the other mastitis pathogens in the present study.

The genetic correlations between the LASCC traits and *Strep. dysgalactiae*, CNS, and *Strep. uberis* were large and of the same magnitude (between 0.62 and 0.69). These results suggest that genetic selection for decreased SCC will have the greatest impact on the incidence of mastitis from these pathogens.

Heritability

Posterior means of heritabilities for LASCC_170 and LASCC_300 (Table 6) were moderate and were larger than the estimates from most other studies of similar

populations. Lund et al. (1994) estimated a heritability of 0.18 for LASCC_180 in Danish Holsteins, Luttinen and Juga (1997) estimated a heritability of 0.23 for LASCC_305 (entire lactation) in Finnish Holsteins, and Rupp and Boichard (1999) estimated a heritability of 0.17 for LASCC_180 in French Holsteins. Most other studies have estimated heritabilities of 0.11 to 0.14 (e.g., Nielsen et al., 2000; Carlén et al., 2004; Johansson et al., 2006). Several plausible reasons can be proposed for the higher heritability; for example, multiple requirements were placed on data in the present study, and some of the editing procedures might affect the outcome of the genetic analyses. Compared with other studies, we placed a requirement on the minimum number of test-day records for calculating LASCC. We used only data from herds with a minimum of 30 first calvings per year, which may have created some selection bias. This may have increased the sire variance and decreased the

Table 7. Mean, standard deviation, and effective sample size (ESS) of the posterior distributions of sire (σ_{sire}^2) and herd-year-season variances (σ_{hys}^2) and within-herd heritability¹ (h^2) of liability to unspecific and pathogen-specific mastitis from bivariate analyses with lactation average SCC (5 to 170 d)¹

Pathogen	Item	σ_{sire}^2	σ_{hys}^2	h^2	HPDI	b
Unspecific mastitis	Mean	0.037	0.138	0.142	[0.119;0.168]	0.071
	SD	0.003	0.004	0.013		0.013
	ESS	738	1,449	739		
<i>Streptococcus dysgalactiae</i>	Mean	0.011	0.128	0.045	[0.029;0.068]	0.028
	SD	0.003	0.009	0.010		0.006
	ESS	112	1,037	112		
<i>Escherichia coli</i>	Mean	0.013	0.065	0.051	[0.033;0.078]	0.026
	SD	0.003	0.008	0.012		0.006
	ESS	108	498	108		
CNS	Mean	0.015	0.176	0.057	[0.038;0.084]	0.028
	SD	0.003	0.010	0.012		0.006
	ESS	117	1,070	117		
<i>Staphylococcus aureus</i>	Mean	0.010	0.137	0.039	[0.023;0.062]	0.028
	SD	0.003	0.010	0.010		0.005
	ESS	132	1,957	132		
<i>Streptococcus uberis</i>	Mean	0.020	0.183	0.079	[0.055;0.110]	0.030
	SD	0.004	0.009	0.014		0.006
	ESS	158	1,409	158		

¹Also, 95% highest posterior density intervals (HPDI) for h^2 in brackets, and regression coefficient (b) of the period at risk. $h^2 = 4 \times \sigma_{\text{sire}}^2 / (\sigma_{\text{sire}}^2 + \sigma_{\text{e}}^2)$.

environmental variance, resulting in a larger heritability. Finally, only herds with pathogen information on at least 50% of the recorded mastitis treatments were included. Data editing had a small effect on the magnitude of LASCC; for example, LASCC_300 was 4.86 in the unedited data set for cows with *Staph. aureus* mastitis, whereas it was 4.77 in the edited data set. In general, LASCC values were smaller in the edited data set (Table 3) than in the unedited data set (results not shown).

Variance components (Table 7) for the mastitis traits, both unspecific and pathogen specific, were similar to those published by Sørensen et al. (2009). They presented heritabilities of 0.034 to 0.074 for the pathogen-specific mastitis traits and $h^2 = 0.114$ for unspecific mastitis. In Sørensen et al. (2009), between-herd heritabilities were presented, but this did not have large effects on the heritabilities when compared with results from the present study, especially not when considering the HPDI.

Implications

An udder health index with different economic weights for different pathogen-specific mastitis traits may be desirable. For such an index, genetic correlations with LASCC, or other SCC traits, and the breeding goal traits are useful because the coheritability ($r_a \times h^2$) of LASCC is higher than the heritability of the breeding goal traits. The results from the present study suggest that the use of LASCC as an indicator trait in an udder health index including pathogen-specific mastitis traits will have a positive effect on the incidence of pathogen-specific mastitis. The pathogen-specific traits used in the present study could be divided into 3 groups based on the magnitude of the genetic correlations among these traits and the LASCC traits: 1) *E. coli*, 2) *Staph. aureus*, and 3) *Strep. dysgalactiae*, CNS, and *Strep. uberis*. *Escherichia coli* is purely of environmental origin (Smith and Hogan, 1993), whereas *Staph. aureus* is a contagious pathogen (Fox and Gay, 1993). For the remaining 3 pathogens, grouping is less clear because their reservoir can be of both environmental and contagious origin. Before it can be determined whether a pathogen-specific udder health index will be superior to the current index, economic weights of the pathogen-specific traits need to be estimated and estimated genetic-economic gain should be compared between the 2 indices. One advantage of a pathogen-specific index is that only true cases of mastitis would be included in the genetic evaluation, avoiding inclusion of questionable information, as seen with the current index.

Data quality is a major drawback for establishment of a pathogen-specific udder health index. For example, a great amount of data was lost through the editing

procedure in the present study. We do not have pathogen information on all mastitis treatments; in some herds no pathogen information is recorded at all, for example, because registration was not done correctly. These are challenges that have to be dealt with before a pathogen-specific udder health program can be initiated. However, because breeding values for udder health are predicted for bulls only, some loss of data can be accepted. In addition, future changes that may occur in animal evaluation systems may also affect the data needed. For example, fewer phenotypic records may be needed with genomic selection once initial relationships between genotypes and phenotypes are established (Meuwissen et al., 2001; Goddard, 2008).

De Haas et al. (2004) suggested the use of pathogen-specific SCC patterns as indicator traits in an udder health index. This may be a better option than LASCC for 2 reasons: 1) fluctuations in SCC through lactations are captured when test-day records for SCC are used, and 2) SCC patterns are specific for at least some pathogens. However, construction of SCC patterns is sensitive to missing test-day records of SCC around the time of infection with a pathogen. For example, if a cow is undergoing antibiotic treatment on the day of herd testing, it is likely that this animal will not be sampled for milk analysis. Thus, SCC patterns around the time of infection for such cows will be imprecise because the high peaks on the SCC curve around the time of infection will disappear, thereby defining an incorrect pattern. However, studies have to be conducted to prove or disprove this statement. In our study, it was not considered beneficial to take the shape of the SCC curve into account. This approach would have required additional restrictions (a minimum number of test-day records to ascertain reliable patterns) on the number of test-day records per lactation and thus loss of data. In addition to LASCC and SCC patterns, De Haas et al. (2008) presented an alternative SCC trait, excessive test-day SCC, in which each test day was treated as a binary variable: 1 if test-day SCC >150,000 cells/mL and 0 otherwise. The concept behind this trait is the relationship to risk of a mammary infection. This trait proved excellent in capturing SCM with a genetic correlation to SCM of $r_a = 0.73$ to 0.98, whereas the genetic correlation to CM was $r_a = 0.62$ to 0.75. In the context of pathogen-specific mastitis, this trait definition of SCC may be especially good for selection against *Staph. aureus* mastitis. It is not known, but would be interesting to investigate, how a trait such as excessive test-day SCC will behave in a Nordic field setting, where mastitis cases are recorded and cases of SCM are most often unknown.

In Lund et al. (1999), the use of variable somatic cell production deviance was introduced as the lactation

mean of test-day deviance from a normal SCC curve describing the expectation for a healthy cow at any particular stage of lactation. Because SCC is measured per unit volume of milk, test-day SCC will fluctuate with the milk yield because of a dilution effect. Therefore, somatic cell production deviance may be a more exact measure of LASCC. Moreover, the heritability of SCDP ($h^2 = 0.17$) was somewhat greater than the heritability of LASCC in most other studies but was smaller than the heritabilities found in the present study. Moreover, the genetic correlation to clinical mastitis was high ($r_a = 0.80$).

A test-day model for SCC (similar to SCC patterns) may also be a future option. Despite the disadvantages of missing test-day records, which also have an effect on LASCC, this model takes into consideration variation in SCC across time. A single-trait test-day model has, for example, been presented by Liu et al. (2000) and Ødegård et al. (2003). Liu et al. (2000) found increasing heritability for test-day SCC with increasing DIM from approximately 0.09 in early lactation to 0.19 in late lactation. Ødegård et al. (2003) found lower heritabilities from approximately 0.07 in early lactation to 0.10 in late lactation. In the Nordic countries (Denmark, Sweden, and Finland) a multitrait model is used for udder health, including records of mastitis treatments. Considering test-day records for SCC simultaneously with pathogen-specific mastitis as defined in this study is not straightforward because the residual correlation between test-day SCC and mastitis would depend on the time of mastitis infection, which is not modeled. Negussie et al. (2007) assumed zero residual covariances between mastitis and test-day SCC in this case. In addition, because no permanent environmental variance could be estimated for the mastitis trait, restrictions were put on the residual variance for this trait to facilitate estimation of permanent environmental correlation between mastitis and test-day SCC. They found decreasing genetic correlations ($r_a = 0.77$ to 0.41) between mastitis (-7 to 30 d after calving) and test-day SCC with increasing DIM and increasing genetic correlations ($r_a = 0.34$ to 0.71) between mastitis (31 to 300 d after calving) and test-day SCC with increasing DIM.

In the Nordic countries, breeding for improved udder health is primarily based on direct measures of mastitis treatments. The LASCC is used as an indicator trait (Denmark, Finland, and Sweden) and has a smaller effect on the incidence of CM than the direct measures of mastitis treatments. The LASCC has a correlated effect on both CM and SCM, which, in similarity to CM, gives rise to elevated SCC and affects yield negatively. Few studies have investigated genetic parameters for SCM. In Madsen et al. (1987), 2 SCM traits were

defined based on increased (from a baseline level) SCC, BSA, or both and the presence of either minor or major pathogens. The heritability estimates (based on a linear model) were rather high compared with the heritability of clinical mastitis ($h^2 = 0.034$) because of the close relationship to SCC. Thus, the heritability for SCM caused by major pathogens was $h^2 = 0.125$, and the heritability of SCM caused by minor pathogens was $h^2 = 0.108$. These results were based on data from 67 dairy farms. Other estimates have been presented by De Haas et al. (2008), $h^2 = 0.03$ to 0.04 , and Svendsen and Heringstad (2006) estimated larger heritabilities, $h^2 = 0.045$ to 0.086 for SCM, depending on the definition of the trait. In practice, it is difficult to obtain recordings of SCM on a national level because many cases of SCM remain unobserved or are treated and recorded as clinical cases. Therefore, before genetic analyses of SCM treatments can be carried out, veterinarians and farmers have to be encouraged to perform proper recording of subclinical treatments. If recording of SCM is successful, it may prove beneficial to include treatments of SCM specifically in an udder health index. Definition of SCC is usually not consistent across countries because it may depend on quality criteria (SCC limits) for produced milk, costs of mastitis treatments, and so forth. Instead of using only SCC to assess udder health status, a variety of traits could be used, as suggested by Sloth et al. (2003). These authors used 8 milk parameters, including SCC and electrical conductivity, and combined the description of the adjusted variance of the milk parameters through a principal components analysis. They proved, via cluster analysis based on the first principal component, that the occurrence of SCM was strongly associated with increasing cluster number. How this kind of trait definition will behave in a genetic setting remains to be investigated.

CONCLUSIONS

The posterior means of the genetic correlations between LASCC traits and pathogen-specific mastitis traits were moderate to high ($r_a = 0.44$ to 0.69) and were slightly smaller than between LASCC and unspecific mastitis ($r_a = 0.71$ and 0.69 for LASCC_170 and LASCC_300, respectively). In addition, the genetic correlations were slightly larger between LASCC_170 and the mastitis traits compared with LASCC_300 and the mastitis traits. The results from this study show that it may be possible to decrease the incidence of mastitis regardless of the pathogen by selecting for lower LASCC. Despite large HPDI of the genetic correlations, there may be differences between the investigated pathogens: genetic correlations are pathogen specific. The smallest genetic correlation was found between LASCC_170

and *Staph. aureus* mastitis ($r_a = 0.44$), and the largest genetic correlations ($r_a = 0.64$ to 0.69) were found between LASCC_170 and *Strep. dysgalactiae*, CNS, and *Strep. uberis*. The results from the present study suggest that the use of LASCC as an indicator trait in an udder health index including pathogen-specific mastitis traits will have a positive effect on the incidence of pathogen-specific mastitis where magnitude depends on the pathogen. However, data quality has to be improved and economic weights for the different mastitis traits have to be estimated before a pathogen-specific udder health index may prove beneficial.

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