Genetic Analysis of Clinical Mastitis Data from On-Farm Management Software Using Threshold Models

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ABSTRACT

Producer-recorded clinical mastitis data from 77,791 cows in 418 herds were used to determine the potential for genetic improvement of mastitis resistance using data from on-farm management software programs. The following threshold sire models were applied: 1) a single-trait lactation model, where mastitis was recorded as 0 or 1 in first lactation only; 2) a 3-trait lactation model, where mastitis was recorded as 0 or 1 in each of the first 3 lactations, and 3) a 12-trait, lactation-segment model, where mastitis was recorded as 0 or 1 in each of 4 segments (0 to 50, 51 to 155, 156 to 260, and 261 to 365 d postpartum) in each of the first 3 lactations. Lactation incidence rates were 0.16, 0.20, and 0.24 in first, second, and third lactation, respectively, and incidence rates within various segments of these lactations ranged from 0.036 in late first lactation to 0.093 in early third lactation. Estimated heritability of liability to clinical mastitis ranged from 0.07 to 0.15, depending on the model and stage of lactation. Heritability estimates were higher in first lactation than in subsequent lactations, but estimates were generally similar for different segments of the same lactation. Genetic correlations between lactations from the 3-trait model ranged from 0.42 to 0.49, while correlations between segments within lactation from the 12-trait model ranged from 0.26 to 0.64. Based on the results presented herein, it appears that at least 2 segments are needed per lactation, because mastitis in early lactation is lowly correlated with mastitis in mid or late lactation. Predicted transmitting abilities of sires ranged from 0.77 to 0.89 for probability of no mastitis during the first lactation and from 0.36 to 0.59 for probability of no mastitis during the first 3 lactations. Overall, this study shows that farmer-recorded clinical mastitis data can make a valuable contribution to genetic selection programs, but additional systems for gather-

ing and storing this information must be developed, and more extensive data recording in progeny test herds should be encouraged.

Key words: clinical mastitis, genetic selection, threshold model, breeding value prediction

INTRODUCTION

Mastitis is the most costly disease facing dairy producers because of expenses associated with veterinary treatment, discarded milk, reduced milk production, impaired reproduction, and increased risk of culling (Fetrow, 2000). Furthermore, selection for increased milk yield without simultaneous selection for mastitis resistance will lead to increased susceptibility to mastitis (Rupp and Boichard, 1999). In the United States, direct selection for resistance to clinical mastitis is currently impossible, because no national recording and genetic evaluation system exists for this trait. However, indirect selection for mastitis resistance has been possible since 1994, when national genetic evaluations for SCS became available (<http://www.aipl.arsusda.gov>). Because SCS data are available for the majority of milk-recorded cows in the United States, sire PTA for this trait can be a useful selection tool (Shook and Schutz, 1994). However, an udder health index including both SCS and clinical mastitis would provide greater accuracy than selection for SCS alone (Boettcher et al., 1998).

The lack of a national recording system for mastitis and other diseases has been a major impediment with respect to genetic improvement of the health, fertility, and fitness of US dairy cattle. However, many commercial dairy producers routinely record clinical cases of mastitis and other important diseases in on-farm computer software for herd management purposes. Although these data are not well standardized between farms or between software providers, such data could still be useful in genetic selection programs. In addition, systems for routine collection and storage of these data should be developed, and this could be accomplished on a national, regional (in specific states), or proprietary

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(by specific breeding companies or specific software providers) basis.

Statistical analysis of clinical mastitis data can pose several challenges because this trait is typically recorded in a binary (0, 1) manner. Furthermore, multiple cases of mastitis can occur within or across lactations for a given animal, and non-unity genetic and residual correlations exist between different time periods. Lastly, if ≥ 2 clinical episodes are reported for a particular animal, one must determine whether these represent multiple, independent infections or a single, chronic infection. Recent developments in methodology for longitudinal binary analysis by Heringstad et al. (2003a, 2004) have addressed many of these concerns.

The objectives of this paper were 1) to assess the usefulness of producer-recorded mastitis incidence data from progeny test herds for developing tools that would allow direct selection for resistance to clinical mastitis and 2) to compare sire PTA from 3 alternative threshold models: a single-trait, single-lactation model; a multiple-trait, 3-lactation model, and a multiple-trait, 12-lactation segment model.

MATERIALS AND METHODS

Data corresponding to clinical mastitis cases that occurred between January 1, 2001 and December 31, 2003 among cows that calved between January 1, 2001 and August 31, 2003 were available for 2 groups of herds: Alta Genetics (Watertown, WI) Advantage Progeny Test Program cooperators and Dairy Records Management Systems (DRMS, Raleigh, NC) customers. Data from the Alta Genetics progeny test herds were downloaded directly from on-farm computers in herds that used the Dairy Comp 305 (Valley Ag Software, Tulare, CA), PCDART (DRMS, Raleigh, NC), or DHI-Plus (DHI-Provo, Provo, UT) software during monthly visits by Alta Genetics employees. Data from DRMS were uploaded electronically to the processing center monthly, along with other data routinely reported in the DHI milk recording system, from farms that used the PCDART software program and provided access to the health records of their cattle. Standardization of diagnosis and recording practices between farms for traits such as clinical mastitis is lacking, so various recording methods must be accommodated. A detailed list of the acceptable recording practices for health traits in herds participating in the present study was provided by Zwald et al. (2004).

Incidence data regarding clinical mastitis were available for 77,711 cows in 418 herds, and a summary of these is provided in Table 1. Data from some herds were not available for the duration of this study because these herds joined the progeny test program late, exited

Table 1. Summary of the clinical mastitis data used in the 3-trait lactation model

	Lactation 1	Lactation 2	Lactation 3
Cows, no.	77,711	49,909	29,086
Herds, no.	418	394	371
Sires, no.	4,505	3,487	2,975
Progeny per sire, no.	17	14	10
Incidence rate ¹	0.155	0.200	0.238

¹Percentage of cows with at least one reported case of clinical mastitis during that period.

from farming, or discontinued milk recording or the use of farm management software prior to the end of the study. Therefore, data for mastitis in all lactations were unavailable for some herds. Animals that were culled within a given lactation or had not completed their current lactation by the end of data collection were coded as missing liabilities in an augmented posterior distribution. Genetic merit of dairy sires for their daughters' liability to clinical mastitis was evaluated using 3 alternative threshold sire models: 1) a single-trait lactation model, where mastitis was recorded as 0 or 1 in first lactation only; 2) a 3-trait lactation model, where mastitis was recorded as 0 or 1 in each of the first 3 lactations, and 3) a 12-trait lactation segment model, where mastitis recorded was 0 or 1 in each of 4 segments of the first 3 lactations: 0 to 50, 51 to 155, 156 to 260, and 261 to 365 d postpartum. All animals were required to have a first lactation (or lactation segment) record. As shown in Table 1, lactation incidence rates were 0.16, 0.20, and 0.24 in first, second, and third lactation, respectively; incidence rates within various segments of these lactations ranged from 0.036 in late first lactation to 0.093 in early third lactation, as shown in Table 2.

Data regarding mastitis incidence after 365 d postpartum were limited because few animals had extended lactations, and incidence rates were extremely low; therefore, cases occurring >365 d postpartum were excluded from the analysis. Similarly, mastitis cases that occurred during the dry period were not used in the present study because of the manner in which data regarding the incidence of mastitis during the dry period are stored in the Dairy Comp 305 program. However, the heritability of clinical mastitis may be higher during the dry period than during late lactation (Heringstad et al., 2003a), so it would be desirable to incorporate such data in future studies.

Statistical analyses used Bayesian threshold models, assuming that mastitis (presence vs. absence) was a different trait in each lactation or segment. Markov chain Monte Carlo methods were used to draw samples from the posterior distributions of interest. The threshold model postulates a multivariate or univariate mixed

Table 2. Summary of the clinical mastitis data used in the 12-trait lactation segment model

	Lactation 1				Lactation 2				Lactation 3			
	0–50 d	51–155 d	156–260 d	261–365 d	0–50 d	51–155 d	156–260 d	261–365 d	0–50 d	51–155 d	156–260 d	261–365 d
Cows, no.	77,711	73,042	70,230	68,174	49,909	41,467	38,645	35,671	29,086	23,481	22,002	20,603
Herds, no.	418	418	417	416	394	393	391	387	371	369	366	365
Sires, no.	4,505	4,398	4,317	4,293	3,487	3,373	3,317	3,107	2,975	2,862	2,795	2,701
Progeny per sire, no.	17	17	16	16	14	12	12	11	10	8	8	8
Incidence rate ¹	0.074	0.037	0.036	0.037	0.078	0.076	0.061	0.044	0.093	0.093	0.070	0.050

¹Percentage of cows with at least one reported case of clinical mastitis during that period.

effects model at the scale of the unobserved liabilities. Normal priors were used for herd-year-season and sire effects; a scaled inverse χ^2 prior was used for variance components in the univariate model, and scaled inverse Wishart priors were assumed for (co)variance matrices in the multivariate models. Noninformative priors were used in all cases.

The threshold model applied herein assumes an unobserved latent variable, l , which reflects the underlying liability to disease, and a conceptual threshold, T , which represents the liability at or above which clinical disease is observed. This threshold is not an identifiable parameter for binary data (Harville and Mee, 1984), and its value is set to zero, defining the origin of the liability scale. Hence, the binary response variable, y , takes the value of 1 if the cow is diseased or 0 if the cow is healthy, according to the following rule:

$$y = 1, \text{ if } l \geq 0, \text{ and } y = 0, \text{ if } l < 0.$$

The threshold model describing liability to clinical mastitis for trait i , where i refers to a specific lactation or lactation segment, for cow l had the following form:

$$l_{ijkl} = \mu_i + \text{hys}_{ij} + s_{ik} + e_{ijkl}$$

where

- μ_i = liability of mastitis during period i for an average cow;
- hys_{ij} = random herd-year-season of calving effect (6-mo seasons, January to June and July to December) for period i distributed as $N(\mathbf{0}, \mathbf{H}_0 \otimes \mathbf{I})$, where $\mathbf{H}_0$ is a covariance matrix of herd-year-season effects (scalar, 3×3 , or 12×12 , depending on the model);
- s_{ik} = random sire effect for period i distributed as $N(\mathbf{0}, \mathbf{G}_0 \otimes \mathbf{I})$, where $\mathbf{A}$ is the numerator relationship matrix between sires and $\mathbf{G}_0$ is a covariance matrix of sire transmitting abilities (scalar, 3×3 , or 12×12 , depending on the model);
- e_{ijkl} = random residual for period i distributed as $N(\mathbf{0}, \mathbf{R}_0 \otimes \mathbf{I})$, where $\mathbf{R}_0$ is scalar for the univariate model, diagonal for the 3-trait model, and block diagonal for the 12-trait model (i.e., residuals were assumed to be correlated within a lactation but independent between lactations).

Posterior means of sire transmitting abilities were transformed from the underlying liability scale to probabilities of disease using the following function:

$$P_{ik} = \Phi(\mu_i + \hat{s}_{ik})$$

Table 3. Posterior means of heritability (on diagonal) and genetic correlations (above diagonal), along with posterior standard deviations of these estimates (in parentheses), from the 3-trait lactation model

	Lactation 1	Lactation 2	Lactation 3
Lactation 1	0.12 (0.01)	0.46 (0.08)	0.42 (0.07)
Lactation 2		0.10 (0.02)	0.49 (0.08)
Lactation 3			0.09 (0.03)

where

P_{ik} = probability of mastitis in period i for daughters (infinite number) of sire k ,

Φ = standard normal cumulative density function,

μ_i = probit function corresponding to mean liability of mastitis in period i , and

$\hat{s}_{ik}$ = posterior mean of liability to mastitis in period i for daughters of sire k .

A FORTRAN 90 program was developed for the statistical computations, and the same model was used for all 3 analyses; however, the number of traits considered was one (single-trait analysis of liability in first lactation), 3 (multiple-trait analysis of liability in first, second, or third lactation), or 12 (multiple-trait analysis of liability at 0 to 50, 51 to 155, 156 to 260, or 261 to 365 d postpartum in first, second, or third lactation).

Although an animal model might have been theoretically preferable, the existence of extreme category problems (i.e., subclasses that contain either all failures or all successes) precluded its application in the present study (Sorensen and Gianola, 2000). Application of a sire-maternal grandsire model would have also been inappropriate because only 18% of the cows had maternal grandsires with valid National Association of Animal Breeders (Columbia, MO) identification numbers (because many of these herds had recently expanded).

RESULTS AND DISCUSSION

Estimated heritability of liability to clinical mastitis was 0.09 in the univariate model that used first lactation data only, and this was similar to the heritability used for SCS in the United States (<http://www.aipl.arsusda.gov>). This estimate was also similar to those previously reported from threshold model analyses (Heringstad et al., 2003a,b), but greater than those reported from linear model analyses (Poso and Mantysaari, 1996; Hansen et al., 2002). Heritability estimates from the multivariate model that considered mastitis as a different trait in first, second, and third lactation were 0.12, 0.10, and 0.09, respectively, as shown in Table 3. Corresponding estimates of genetic

correlations were 0.46 between first and second lactation, 0.49 between second and third lactation, and 0.42 between first and third lactation. As in the present study, Heringstad et al. (2004) reported higher heritability estimates in first lactation than in later lactations.

Heritability estimates from the 12-trait lactation segment model were relatively consistent across segments and lactations, as shown in Table 4. With the exception of an estimate of 0.15 in the fourth segment of first lactation, all other estimates ranged from 0.08 to 0.11. The trend toward increasing heritability estimates as each lactation progressed seemed to contradict most previously published research (Heringstad et al., 2003a, 2004), but this trend agreed with results from a genetic analysis of SCS by Ødegard et al. (2003).

Estimated genetic correlations between lactation segments from the 12-trait model are shown in Table 4. Genetic correlations between different segments of first lactation ranged from 0.26 to 0.56, while corresponding ranges in second and third lactation were 0.33 to 0.64 and 0.38 to 0.55, respectively. As expected, estimates tended to be highest between consecutive segments of the same lactation. Between lactations, estimated correlations were generally highest between corresponding time periods. For example, correlations between the first segment of first lactation and the first segments of second and third lactation were 0.33 and 0.31, respectively; correlations between the first segment of first lactation and other segments of later lactations ranged from 0.19 to 0.27. Genetic correlations within and between lactations tended to be slightly lower than estimates reported previously by Heringstad et al. (2004). Although posterior standard deviations of the estimated genetic correlations were relatively large in the present study, ranging from 0.07 to 0.14, these results may indicate that clinical mastitis in early lactation should be treated as a separate trait from clinical mastitis in mid or late lactation. Residual correlations ranged from 0.24 to 0.30 between adjacent periods within the lactation and from 0.08 to 0.18 between nonadjacent periods within the lactation.

Animals were considered either diseased or free of disease in each trait or period. Therefore, a cow was considered diseased in a given period regardless of whether it had been diagnosed (and recorded) with 1, 2, 3, or numerous cases of mastitis during that period. However, if liability to mastitis in early lactation is a different trait, genetically speaking, from liability to mastitis in late lactation, then a single-trait lactation model may be too simplistic. Conversely, if liability to mastitis is the same trait, genetically speaking, throughout the entire lactation, a multiple-trait lactation segment model may lead to overparameterization.

Table 4. Posterior means of heritability (on diagonal), genetic correlations (above diagonal), and residual correlations (below diagonal), along with posterior standard deviations of these estimates (in parentheses), from the 12-trait lactation segment model

	Lactation 1				Lactation 2				Lactation 3			
	0–50 d	51–155 d	156–260 d	261–365 d	0–50 d	51–155 d	156–260 d	261–365 d	0–50 d	51–155 d	156–260 d	261–365 d
Lactation 1												
0–50 d	0.08 (0.01)	0.33 (0.11)	0.26 (0.12)	0.31 (0.12)	0.33 (0.12)	0.27 (0.12)	0.26 (0.12)	0.19 (0.13)	0.31 (0.13)	0.22 (0.13)	0.22 (0.14)	0.28 (0.14)
51–155 d	0.28 (0.02)	0.10 (0.02)	0.56 (0.09)	0.41 (0.12)	0.39 (0.12)	0.51 (0.10)	0.51 (0.10)	0.34 (0.13)	0.38 (0.13)	0.40 (0.12)	0.47 (0.12)	0.50 (0.12)
156–260 d	0.18 (0.02)	0.24 (0.02)	0.11 (0.02)	0.54 (0.09)	0.40 (0.11)	0.57 (0.09)	0.59 (0.08)	0.50 (0.11)	0.47 (0.11)	0.50 (0.11)	0.59 (0.09)	0.53 (0.12)
261–365 d	0.08 (0.02)	0.13 (0.02)	0.25 (0.02)	0.15 (0.03)	0.40 (0.11)	0.59 (0.09)	0.63 (0.08)	0.58 (0.09)	0.55 (0.10)	0.59 (0.08)	0.57 (0.11)	0.51 (0.13)
Lactation 2												
0–50 d					0.08 (0.02)	0.45 (0.10)	0.44 (0.11)	0.33 (0.12)	0.38 (0.12)	0.32 (0.13)	0.37 (0.13)	0.38 (0.13)
51–155 d					0.29 (0.02)	0.10 (0.02)	0.64 (0.07)	0.51 (0.11)	0.46 (0.11)	0.47 (0.11)	0.56 (0.10)	0.52 (0.10)
156–260 d					0.18 (0.02)	0.25 (0.02)	0.11 (0.02)	0.57 (0.09)	0.52 (0.10)	0.53 (0.10)	0.58 (0.09)	0.55 (0.12)
261–365 d					0.11 (0.02)	0.15 (0.02)	0.26 (0.02)	0.11 (0.02)	0.44 (0.11)	0.47 (0.11)	0.54 (0.11)	0.48 (0.13)
Lactation 3												
0–50 d									0.08 (0.02)	0.46 (0.11)	0.47 (0.11)	0.38 (0.14)
51–155 d									0.29 (0.02)	0.08 (0.02)	0.55 (0.10)	0.45 (0.13)
156–260 d									0.15 (0.02)	0.25 (0.02)	0.11 (0.02)	0.55 (0.11)
261–365 d									0.14 (0.02)	0.15 (0.02)	0.30 (0.02)	0.10 (0.02)

Figure 1. Frequency distributions of sires' PTA for probability of no mastitis in first lactation using the single-trait lactation model, the 3-trait lactation model, or the 12-trait lactation segment model.

Probabilities of no clinical mastitis during first lactation for daughters of each sire were estimated using each of the aforementioned threshold models, and the corresponding frequency distributions of sire PTA are shown in Figure 1. As shown in these graphs, the probability of no mastitis during first lactation ranged from 0.79 to 0.89 in the single-trait model, from 0.78 to 87 in the 3-trait model, and from 0.77 to 0.87 in the 12-trait model. These ranges in sire PTA correspond reasonably well with the range of approximately 1 unit of SCS among active AI sires, coupled with literature estimates of an increase of approximately 13% in mastitis incidence per 1-unit increase in SCS (Nash et al., 2000). However, it should be noted that the average number of progeny per sire was limited in the present study, decreasing the possible maximum range of PTA.

Figure 2. Frequency distributions of sires' PTA for probability of no mastitis in first, second, or third lactation using the 3-trait lactation model or the 12-trait lactation segment model.

Sire PTA for probability of no mastitis during any of the first 3 lactations were also computed for the 3-trait lactation model and the 12-trait lactation-segment model. The corresponding frequency distributions of sire PTA are shown in Figure 2. As shown in these graphs, probabilities of no mastitis ranged from 0.40 to 0.59 in the 3-trait model and from 0.36 to 0.55 in the 12-trait model. In general, sire PTA for probability of no mastitis tended to be lower in the more complicated models because models that allowed repeated cases of mastitis in subsequent lactations or lactation segments had higher overall incidence rates (i.e., a given cow could have >1 clinical case per lactation). Furthermore, the multiple-trait models allowed reported cases of mastitis in second or third lactation to contribute to sire PTA for resistance to mastitis in first lactation through genetic correlations, even for daughters that had no mastitis during first lactation. Thus, it appears that models based on multiple lactations or lactation segments may be preferable in practice.

Although estimated genetic correlations between lactations and lactation segments were always positive, ranging from 0.26 to 0.64, these estimates were far less than unity. Therefore, it appears that resistance to mastitis in different lactations may be genetically different traits, and the same can be said for resistance in different stages of the same lactation. Genetic evaluation with a multiple-trait model that can accommodate such differences should lead to more useful information for dairy producers and greater accuracy and stability of sire PTA. For example, one might wish to consider first and later parities as separate traits, with repeated records for the latter. Furthermore, one might wish to treat early lactation mastitis as a separate trait from mid or late lactation mastitis. Such an approach would lead to a 4-trait model, including 1) resistance to mastitis early in first lactation, 2) resistance to mastitis later in first lactation, 3) resistance to mastitis early in subsequent lactations, and 4) resistance to mastitis later in subsequent lactations. Future research should consider whether mastitis during the dry period can be considered as one of the aforementioned traits or if it should be considered separately, because the present study was not able to address this question.

The present study considered herd-year-season effects as random, and this could have resulted in biased sire PTA or biased heritability estimates if sires were not used randomly across contemporary groups. Because the present study used data from a relatively short period (2001 to 2003), bias attributable to management trends is unlikely. However, some herds might have been more aggressive in selecting sires with low genetic evaluations for SCS, even though that trait receives low weight in the total merit index.

An important consideration is the relationship between clinical mastitis and SCS evaluations, because selection programs will almost certainly use an index that combines sire PTA for SCS and clinical mastitis. At the present time, however, genetic evaluations for SCS in this country are based on lactation average SCS, rather than individual test-day observations. If a multiple-trait model were used for clinical mastitis, to differentiate between resistance to mastitis pathogens at different times during the lactation, it would seem desirable to use an equally sophisticated model (e.g., a random-regression, test-day model) for SCS as well. Reents et al. (1995) has shown that test-day models for SCS tend to provide slightly higher heritability estimates than lactation average models, and adoption of a more advanced genetic evaluation model for SCS appears warranted.

Development of systems to routinely collect clinical mastitis data is almost certainly possible, because US dairy herds continue to expand at a rapid pace, and

these herds are becoming increasingly reliant on farm-based herd management software programs. In the near future, direct selection among US dairy sires for resistance to clinical mastitis may be challenging early in life (i.e., based on first-crop progeny), because some cooperator herds do not yet record mastitis incidence and because progeny groups are often too small for accurate evaluation of lowly heritable, binary disease traits. With some effort, however, an individual breeding company may be able to gather mastitis data from a subset of large herds that have the appropriate software and tend to record clinical cases diligently. These data can then be combined with PTA for SCS and relevant udder conformation traits to create a useful udder health index for producers to use as a selection tool.

CONCLUSIONS

In summary, the present study demonstrates the potential usefulness of farmer-recorded clinical mastitis data in genetic selection programs. Significant genetic variation exists between sires, and despite differences between farms in diagnosis and recording of mastitis, heritability estimates were within the range reported by previous studies that used more tightly controlled data collection strategies (Heringstad et al., 2003c). Estimated genetic correlations between mastitis resistance in different lactations or lactation segments were moderately high, but far less than unity, indicating that mastitis resistance may be a genetically different trait in first lactation vs. later lactations or in early lactation vs. mid and late lactation. Therefore, application of a multiple-trait model that can account for genetic differences in susceptibility to infection by mastitis pathogens at different points during a cow's life seems warranted. Regardless of the statistical model chosen for computation of sire PTA, further development and refinement of systems for collection, validation, and storage of information about producer-recorded cases of clinical mastitis will be critical.

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