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Heritability of Clinical Mastitis Incidence and Relationships with Sire Transmitting Abilities for Somatic Cell Score, Udder Type Traits, Productive Life, and Protein Yield

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ABSTRACT

The objective of this study was to determine the relationships among daughter clinical mastitis during first and second lactations and sire transmitting abilities for somatic cell score, udder type traits, productive life, and protein yield. Data on clinical mastitis during first lactation were available for 1795 daughters (in six Pennsylvania herds, one Minnesota herd, and one Nebraska herd) of 283 Holstein sires. Data on clinical mastitis during second lactation were available for 1055 of these daughters. A total of 479 cows had 864 clinical episodes during first lactation, and 230 cows had 384 clinical episodes during second lactation. Clinical mastitis incidence and the total number of clinical episodes during each lactation were regressed on herd-season of calving (a classification variable), age at first calving, lactation length, and sire transmitting abilities taken one at a time. Linear effects, nonlinear effects, and odds ratios were estimated for sire transmitting abilities. Separate analyses were conducted on dependent variables that considered clinical mastitis from: all organisms, coagulase-negative staphylococci, coliform species, streptococci other than *Streptococcus agalactiae*, and the most common environmental organisms (coliform species and streptococci other than *Streptococcus agalactiae*). Heritability of clinical mastitis ranged from 0.01 to 0.42. Daughters of sires that transmit the lowest somatic cell score had the lowest incidence of clinical mastitis and the fewest clinical episodes during first and second lactations. Daughters of sires that transmit longer productive life, shallower udders, deeper udder cleft, and strongly attached fore udders had either fewer clinical episodes or lower clinical mastitis incidence during first and second lactations. The incidence of clinical mastitis and the number of clinical episodes per lactation may be reduced by selection for lower somatic cell score, longer productive life, shallower udders, deeper udder cleft, or strongly attached fore udders.
(**Key words:** clinical mastitis, somatic cell score, productive life, udder type traits)

Abbreviation key: CMI = clinical mastitis incidence, **CNS** = coagulase-negative staphylococci, **PL** = productive life, **SCS** = somatic cell score, **SNA** = streptococci other than *Streptococcus agalactiae*, **STA** = standardized transmitting abilities, **TCE** = total number of clinical episodes.

INTRODUCTION

Annual losses due to mastitis have been estimated to be nearly $2 billion dollars or approximately 10% of the total value of milk sales made by US dairy farms (10). Despite improvements in management, which have reduced mastitis from contagious organisms (especially *Streptococcus agalactiae*), economic losses due to mastitis will continue because environmental organisms cannot be eradicated (10). Furthermore, increased economic losses may result from the unfavorable (positive) genetic correlation between mastitis and milk yield (3, 13, 19, 20). The increased susceptibility to mastitis accompanying selection for milk yield suggests that selection for resistance to mastitis is needed. Under current economic conditions, optimal selection for resistance to mastitis would slow, rather than halt, the rate of increase in genetic susceptibility to mastitis (21).

Selection for resistance to mastitis could also address food safety, food quality, and animal welfare issues. Improved genetic resistance to mastitis could enhance the safety of dairy products by reducing the need for antibiotic treatment. Decreased SCC, which should result from selection for resistance to mastitis, has been shown to improve dairy product quality, shelf life, and cheese yield (10). In addition, selection criteria that include resistance to mastitis may be perceived by con-

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sumers to be more humane than selection criteria that exclude resistance to mastitis.

Because records of clinical mastitis occurrence are not readily available for most US dairy cattle, other selection criteria are needed to improve resistance to mastitis. Possible criteria for selection include somatic cell score (**SCS**), udder type traits, and productive life (**PL**).

Approximately 80% of the cows in the national DHIA milk recording program, representing 40% of all US dairy cows, have milk SCC recorded monthly (20). Increases in SCC are primarily due to the presence of mastitis-causing organisms in the udder (4). In addition, the genetic correlation between SCS (a logarithmic transformation of SCC) and clinical mastitis is positive; six estimates averaged 0.71 and ranged from 0.37 to 0.98 (3, 7, 11, 12, 13, 24). Estimates by Coffey et al. (1) and Weller et al. (23) were lower (–0.09 to 0.30). However, their estimates of the genetic correlation between IMI and SCS ranged from 0.36 to 0.99.

Because of these findings, it has been suggested that selection for lower SCS may improve resistance to mastitis (1, 3, 13, 19, 20, 24). However, no study has examined the relationships among clinical mastitis from particular organisms (or groups of organisms) and SCS. Because the etiology of each mastitis-causing organism is different (10), it is possible that selection for lower SCS may not improve resistance to clinical mastitis from every organism (20). Therefore, the objectives of this study were to estimate the heritability of clinical mastitis (during first and second lactations) caused by the most prevalent organism groups and determine the relationships among daughter clinical mastitis caused by these organisms and sire transmitting abilities for SCS, udder type traits, PL, and protein yield.

MATERIALS AND METHODS

Data

Recording of clinical episodes began at first parturition for 1860 cows in eight herds (six in Pennsylvania, one in Minnesota, and one in Nebraska) and continued into second lactation for 1098 of these cows in six of the eight herds. Clinical episodes were recorded from first parturition onward because the health history of multiparous cows may be unknown, and multiparous daughters of a sire are not a random sample of daughters from that sire because it is likely that some have been culled.

Herdsmen collected milk samples from all quarters that had clinical mastitis. Quarter samples were collected when clinical mastitis was first observed. Quarter samples were frozen and transported weekly to diagnostic laboratories in Minnesota, Nebraska, or Pennsylvania for culturing following procedures described previously (9).

Research technicians taught herdsmen the techniques to use to avoid contamination when collecting milk samples. They also taught herdsmen how to visually identify and classify clinical mastitis. In addition, research technicians made weekly visits to the Pennsylvania herds to ensure that the study protocol was being followed.

Clinical mastitis data from 41 cows with unknown sires were not included in the analyses. Data from 24 additional cows that were not 22 to 33 mo of age at first parturition were not considered because these animals may have experienced an atypical environment. A total of 599 of the remaining cows were removed from six herds before their second calving. Furthermore, clinical episodes that occurred during second lactation were not recorded for 141 cows in two herds (one herd in Pennsylvania was sold and the herd in Minnesota recorded clinical episodes during first lactation only). After edits, data on clinical mastitis during first lactation were available for 1795 cows. Data on clinical mastitis during second lactation were available for 1055 of these cows.

Only those clinical episodes that occurred before 365 DIM had elapsed are considered here. When a cow had clinical mastitis in multiple quarters on the same day, each quarter that had clinical mastitis was considered to be a separate clinical episode. Each quarter that had clinical mastitis on the same day was considered to be a clinical episode because mastitis-causing organisms entered each quarter separately. Clinical mastitis that occurred at least 30 d after a prior clinical episode in a quarter was considered a separate clinical episode.

Analyses

Estimates of the heritability of clinical mastitis incidence (**CMI**), a binary variable, and the total number of clinical episodes (**TCE**) during a lactation were calculated from sire variances estimated using the MIXED procedure of SAS (16). The model used included sire, herd, age at first calving, and lactation length. Only data from daughters of sires that had three or more progeny among the cows contributed by the cooperating herds were used to obtain heritability estimates. A normal linear model was assumed. The model was a mixed model; sire was a random effect, all other independent variables were fixed effects. Sire and herd were classification variables. The model did not consider relationships among sires because an algorithm that could handle relationships and account for distributional assumptions for binary data was not readily available. Estimates of the heritability of CMI and TCE during

first lactation were obtained with data from 1577 daughters of 115 sires. Data from 910 daughters of 58 sires were used to obtain estimates of the heritability of CMI and TCE during second lactation.

Both CMI and TCE were regressed on age at first calving, lactation length, herd-season of calving (a classification variable), and sire transmitting abilities for SCS, udder type traits, PL, and protein yield taken one at a time. Lactation length provided a measure of time at risk for clinical mastitis. Because clinical episodes that occurred after 365 DIM were not considered, the maximum time at risk for clinical mastitis was 365 d. Therefore, lactation length was truncated to 365 d for cows that were in milk more than a year. When TCE was the dependent variable, linear and quadratic effects were estimated for each of the transmitting abilities using the GLM procedure of SAS (16). Cubic effects were also estimated for PTA for SCS. When CMI was the dependent variable, odds ratios were estimated for each of the transmitting abilities by using the PROBIT procedure of SAS (16) to perform logistic regression.

The relationships among CMI and sire transmitting abilities were investigated using logistic regression because linear regression is intended for analysis of data derived from a normal, rather than a binomial, distribution (2). Furthermore, the predicted values obtained via linear regression are not constrained to lie between zero and one (2). The logistic transformation (the natural logarithm of the odds of a success) ensures that the predicted values lie between zero and one (2).

Separate analyses were conducted on dependent variables that considered clinical mastitis from: all organisms, coagulase-negative staphylococci (**CNS**), coliform species, streptococci other than *Streptococcus agalactiae* (**SNA**), and the most common environmental organisms (coliform species and SNA). Clinical mastitis from all organisms contained all observed clinical episodes, including those that had missing or contaminated quarter samples or no detectable organism growth. Two organisms were detected in 57 quarters (8% of those that had clinical mastitis) during first lactation and 30 quarters (9%) during second lactation. These quarters were considered to have a clinical episode from each organism when clinical mastitis from different organism groups were analyzed separately.

Incidence of clinical mastitis from CNS during second lactation was too low to analyze separately. Furthermore, only TCE from all organisms during first and second lactations were analyzed. The TCE from the other organism groups were not analyzed separately because few cows experienced more than one or two clinical episodes from these organism groups.

Before analyses were conducted on TCE, cows that experienced six or more clinical episodes during first lactation were classified as having five episodes. In addition, cows that experienced five or more clinical episodes during second lactation were classified as having four episodes. These classifications were made because only nine cows experienced more than five clinical episodes during first lactation and only nine cows had over four clinical episodes during second lactation.

When CMI or TCE were regressed on standardized transmitting abilities (**STA**) for udder type traits, data from 20 first-lactation cows and eight second-lactation cows were excluded because STA for udder type traits were not available for their sires. When CMI or TCE were regressed on STA for teat length, data from five more first-lactation cows and four more second-lactation cows were excluded because STA for teat length were not available for their sires.

RESULTS AND DISCUSSION

Data

A total of 479 of 1795 cows (27%) had 864 clinical episodes in 724 of 7180 quarters (10%) during first lactation. In addition, 230 of 1055 cows (22%) had 384 clinical episodes in 324 of 4220 quarters (8%) during second lactation. Total episodes per cow ranged from one to eight during first lactation and from one to six during second lactation. A total of 426 first-lactation cows and 208 second-lactation cows had three or fewer episodes.

Five of the cooperating herds were commercial herds (all in Pennsylvania), and three were university research herds. Table 1 presents the total number of first- and second-lactation cows contributed by each herd, the number that had clinical mastitis, and the frequency of mastitis by lactation in each herd.

Lactation average SCS was available from DHIA records for 810 first-lactation cows in six herds and 348 second-lactation cows in four herds. Mean lactation average SCS was 2.73 for first lactation (range was 0.00 to 9.00) and 2.85 for second lactation (range was 0.00 to 8.40).

Variations in mastitis incidence and lactation average SCS between herds were likely due to differences in management, not selection for resistance to mastitis. When sires of the cows in this study were selected, producers had little ability to select for resistance to mastitis. The PTA for SCS and PL were not available at the time.

Table 2 summarizes CMI and TCE by lactation for all herds. Most clinical mastitis during first or second lactation was caused by SNA or coliform species (*Escherichia coli* and *Klebsiella* species). Few clinical episodes were caused by contagious organisms. Approximately 2% of the cows had clinical mastitis caused by *Staphylo-*

Table 1. Total first- and second-lactation cows per herd, the number that had clinical mastitis, and the frequency of mastitis in each herd.

	First-lactation cows			Second-lactation cows		
Herd	Total	Had mastitis	Mastitis frequency	Total	Had mastitis	Mastitis frequency
1	170	90	0.53	110	43	0.39
2	49	7	0.14	0[1]		
3	136	54	0.40	73	23	0.23
4	226	101	0.45	98	45	0.46
5	753	80	0.11	614	75	0.12
6	211	63	0.30	100	28	0.28
7	159	61	0.38	60	16	0.27
8	91	23	0.25	0[2]		
Totals	1795	479	0.27	1055	230	0.22

[1]Herd 2 was sold before clinical episodes during second lactation could be recorded.

[2]Herd 8 recorded clinical episodes during first lactation only.

coccus aureus during first or second lactation. Fewer than 1% of the cows had clinical mastitis caused by *Streptococcus agalactiae* during first or second lactation.

No organism growth was detected in clinical episodes experienced by 8% of the cows during first lactation and 6% of the cows during second lactation. Milk samples were missing (not collected) for clinical episodes experienced by 5% of the cows during first lactation and 4% of the cows during second lactation. Milk samples were classified as contaminated for clinical episodes experienced by 2% of the cows during first lactation and 1% of the cows during second lactation. Nash (9) determined that the incidence of clinical mastitis during first and second lactations and the types and relative proportions of organisms detected in quarters that had clinical mastitis were similar to those in other studies.

The majority of clinical episodes occurred early in each lactation. The mean DIM at detection was 86 for all clinical episodes that occurred during first lactation. The first clinical episode was detected within 1 mo after calving for 261 of the 479 cows (54%) that had clinical mastitis during first lactation. One hundred and eighty-seven of these cows had their first clinical episode within 1 wk of calving. During second lactation, the mean DIM at detection was 104 for all clinical episodes. The first clinical episode was detected within 3 mo of calving for 134 of the 230 cows (58%) that had clinical mastitis during second lactation. Eighty-four of these cows had their first clinical episode within 1 mo of calving. Nash (9) determined that the DIM when clinical episodes were detected were similar to those in other studies.

Mean lengths of first and second lactations were 296 and 271 d, respectively. Approximately 77% of the first-

Table 2. Clinical mastitis incidence and the total number of clinical episodes during first and second lactation by organism group.

	Clinical mastitis incidence			Total number of clinical episodes		
Organism group	Mean	SD	Range	Mean	SD	Range
First lactation (N = 1795)						
All organisms	0.27	0.44	0 to 1	0.48	1.03	0 to 8
CNS[1]	0.05	0.22	0 to 1	0.07	0.31	0 to 4
SNA[2]	0.09	0.29	0 to 1	0.12	0.40	0 to 4
Coliform species	0.06	0.24	0 to 1	0.08	0.36	0 to 6
Environmental organisms[3]	0.14	0.35	0 to 1	0.19	0.56	0 to 6
Second lactation (N = 1055)						
All organisms	0.22	0.41	0 to 1	0.36	0.86	0 to 6
CNS[1]	0.02	0.12	0 to 1	0.02	0.14	0 to 2
SNA[2]	0.09	0.29	0 to 1	0.12	0.42	0 to 5
Coliform species	0.05	0.21	0 to 1	0.05	0.23	0 to 2
Environmental organisms[3]	0.12	0.33	0 to 1	0.16	0.49	0 to 5

[1]CNS = Coagulase-negative staphylococci.

[2]SNA = Streptococci other than *Streptococcus agalactiae*.

[3]Coliform species and streptococci other than *Streptococcus agalactiae*.

Table 3. Predicted and standardized transmitting abilities for sires of Holstein cows that were observed for clinical mastitis during first lactation.

Transmitting abilities	N	Mean	SD	Minimum	Maximum
Somatic cell score (log_2)	283	3.23	0.210	2.72	3.88
Protein (kg)	283	7.30	9.14	–33.1	29.9
Productive life	283	0.295	1.25	–3.80	3.30
Fore udder attachment	272[1]	0.0597	1.23	–4.06	3.20
Rear udder height	272	0.315	1.13	–3.58	3.18
Rear udder width	272	0.312	1.09	–3.17	3.25
Udder cleft	272	0.159	1.18	–3.59	3.54
Udder depth	272	–0.182	1.47	–4.42	3.41
Front teat placement	272	0.123	1.37	–4.25	3.50
Teat length	270[2]	–0.0211	1.31	–3.85	4.31

[1]The standardized transmitting abilities for udder type traits were not available for 11 sires.

[2]The standardized transmitting abilities for teat length were not available for 13 sires.

lactation cows and 86% of the second-lactation cows were in milk for less than 365 d.

The actual or projected 305-d milk yield was available from DHIA records for 1746 first-lactation cows and 1040 second-lactation cows. Mean 305-d milk yield was 8459 kg for first lactation (range was 2257 to 12,351 kg) and 9825 kg for second lactation (range was 1194 to 15,246 kg).

The mean age at first calving was 25 mo. Fifteen seasons were defined for first calving, beginning in May 1991 and ending in December 1995. Twelve seasons were defined for second calving, beginning in June 1992 and ending in November 1995. All seasons of calving were 4 mo in duration (July through October, November through February, and March through June) except for the seasons at the beginning and end of both calving periods.

The 1795 first-lactation cows were sired by 283 Holstein bulls. The 1055 second-lactation cows were sired by 166 of these bulls. The mean number of first- or second-lactation daughters per sire was 6.3. Together, two bulls had 343 first-lactation daughters (19% of the first-lactation cows) and 291 second-lactation daughters (28% of the second-lactation cows) in the cooperating herds. All other sires had no more than 65 first-lactation daughters or 51 second-lactation daughters. A total of 199 sires had three or fewer first lactation daughters and 124 sires had three or fewer second lactation daughters. A total of 174 sires had one or more daughters that had clinical mastitis during first lactation. One hundred sires had one or more daughters that had clinical mastitis during second lactation. The PTA and STA from the USDA Sire Summary of November, 1997 (22) and Holstein Association USA (5) for sires of second-lactation cows (not shown) were similar to those summarized for sires of first-lactation cows in Table 3.

Heritability of Clinical Mastitis During First and Second Lactations

Table 4 contains estimates of the heritability of CMI and TCE by lactation and organism group. For the most part, CMI was more heritable when the environmental organisms were considered separately or together (compared to mastitis from all organisms or CNS). Estimates of the heritability of CMI from the environmental organism groups during first and second lactations ranged from 0.11 to 0.25 and from 0.12 to 0.19, respectively. Estimates of the heritability of CMI from all organisms during first and second lactations were 0.14 and 0.01, respectively. Estimates of the heritability of TCE during first and second lactations were 0.42 and 0.15, respectively.

In general, these heritability estimates are higher than those in the literature for clinical mastitis. Most estimates of the heritability of clinical mastitis have been less than 0.10 (3, 7, 12, 23, 24). Most estimates of the heritability of SCS have ranged from 0.10 to 0.20 (3, 7, 12, 14, 18, 23). The low estimates of the heritability of clinical mastitis in some studies may have been the result of failing to detect or report all clinical episodes (12, 23). Improving detection and reporting of clinical episodes could result in heritability estimates of 0.10 or greater (12).

It is hypothesized that resistance to mastitis from a related group of organisms may be controlled by fewer genes than resistance to mastitis from all organisms. This theory may explain why CMI from related groups of organisms (e.g., SNA) were generally more heritable than CMI from all organisms in the current study.

Relationships among Clinical Mastitis and Sire Transmitting Abilities

Tables 5 and 6 contain the odds ratios, regression coefficients, and standard errors obtained when logistic

regression was used to regress CMI on PTA and STA. The regression coefficients and standard errors from regressing TCE on the linear effects of PTA and STA are in Table 7. Table 8 contains the regression coefficients from regressing TCE on both the linear and quadratic effects of PTA and STA.

PTA for SCS. The PTA for SCS was a significant ($P \leq 0.10$) predictor of CMI during first lactation when all organisms, CNS, or the most common environmental organisms were considered (Table 5). The PTA for SCS was also a significant predictor of CMI during second lactation when SNA were considered (Table 6). Odds ratios calculated from logistic regression coefficients for PTA for SCS were >1 except when coliform species during second lactation were considered (Tables 5 and 6).

The ratio of odds for a one-unit change in an explanatory variable is obtained by exponentiating the parameter estimates for a logistic regression model (15). Odds ratios can be calculated only if the explanatory variable does not interact with any other variable and is represented by one term in the model (15). Odds ratios that are >1 (<1) indicate that the odds of an event increase (decrease) as the explanatory variable increases by one unit (15). For example, the odds ratio for PTA for SCS was 1.75 when CMI from all organisms during first lactation were considered (Table 5). The interpretation of this value is that the odds of a daughter having clinical mastitis during first lactation are 1.75 times higher for a one-unit increase in sire PTA for SCS. Similar interpretations apply to the other odds ratios in Tables 5 and 6.

If the odds ratio for a change in an independent variable of other than one unit is of interest, it can be calculated by exponentiating the product of the regression coefficient and the number of units of interest (15). For example, the range in PTA for SCS in this study was 1.16 (see Table 3) and the regression coefficient was 0.560 when CMI from all organisms during first lactation were considered (Table 5). Therefore, daughters of the sire that transmits the highest SCS would be $e^{(1.16 * 0.560)} = 1.91$ times more likely than daughters of the sire that transmits the lowest SCS to have clinical mastitis during first lactation.

The linear effect of PTA for SCS was a significant predictor of TCE during first lactation (Table 7). Regressions of TCE during second lactation on the linear effect of PTA for SCS (Table 7) were not significant ($P > 0.10$). Regression coefficients for the linear effect of PTA for SCS were positive when TCE during first or second lactation was the dependent variable. Positive regression coefficients indicate that daughters of sires that transmit higher SCS had higher TCE during first and second lactations. Regression coefficients were of the same magnitude when TCE during first or second lactation were regressed on the linear effect of PTA for SCS. However, the regression coefficient may not have been statistically significant when TCE during second lactation was the dependent variable because there were fewer second-lactation cows.

The quadratic effect of PTA for SCS was a significant predictor of TCE during first and second lactations (Table 8). The cubic effect of PTA for SCS (regression coefficient not shown) was not a significant predictor of TCE during first or second lactation. As shown in Figure 1, the quadratic regression lines (includes both linear and quadratic effects) are concave with the peak occurring between the mean and maximum PTA for SCS. The shape of the quadratic regression lines indicates that daughters of sires that transmit the lowest SCS had the lowest TCE during first and second lactations.

Table 4. Heritability of clinical mastitis incidence and the total number of clinical episodes during first and second lactation.

	Clinical mastitis incidence		Total number of clinical episodes	
	h^2	SE	h^2	SE
First lactation				
All organisms	0.14	0.02	0.42	0.12
CNS[1]	0.03	0.002		
SNA[2]	0.25	0.01		
Coliform species	0.19	0.005		
Environmental organisms[3]	0.11	0.01		
Second lactation				
All organisms	0.01	0.01	0.15	0.08
SNA[2]	0.12	0.007		
Coliform species	0.17	0.005		
Environmental organisms[3]	0.19	0.01		

[1]CNS = Coagulase-negative staphylococci.

[2]SNA = Streptococci other than *Streptococcus agalactiae*.

[3]Coliform species and streptococci other than *Streptococcus agalactiae*.

Table 5. Logistic regression of daughter clinical mastitis incidence (binary variable) during first lactation on sire transmitting abilities by organism group.

	All organisms			CNS[1]			Coliform species			SNA[2]			Environmental organisms[3]		
Transmitting abilities	OR[4]	b-value	SE	OR	b-value	SE	OR	b-value	SE	OR	b-value	SE	OR	b-value	SE
Somatic cell score	1.75†	0.560	0.300	5.09**	1.63	0.569	2.09	0.739	0.519	1.90	0.640	0.440	1.84†	0.610	0.369
Protein (kg)	1.00	0.00217	0.00827	1.00	–0.00105	0.0150	0.99	–0.0100	0.0141	1.02†	0.0190	0.0114	1.01	0.0102	0.00975
Productive life	0.91†	–0.0962	0.0518	0.86	–0.145	0.0945	0.92	–0.0793	0.0894	0.94	–0.0612	0.0710	0.96	–0.0358	0.0617
Fore udder attachment	0.92	–0.0821	0.0567	0.88	–0.123	0.104	0.89	–0.116	0.105	1.06	0.0546	0.0784	1.01	0.00572	0.0684
Rear udder height	0.98	–0.0220	0.0609	0.91	–0.0928	0.108	0.93	–0.0757	0.105	1.14	0.133	0.0861	1.08	0.0807	0.0731
Rear udder width	0.98	–0.0231	0.0624	0.88	–0.131	0.113	0.91	–0.0903	0.113	1.12	0.115	0.0877	1.06	0.0606	0.0755
Udder cleft	0.91	–0.0959	0.0597	0.93	–0.0725	0.110	0.93	–0.0717	0.109	0.92	–0.0798	0.0819	0.94	–0.0669	0.0714
Udder depth	0.90*	–0.104	0.0468	0.89	–0.121	0.0882	0.90	–0.107	0.0827	0.98	–0.0246	0.0657	0.95	–0.0536	0.0564
Front teat placement	0.97	–0.0283	0.0458	0.97	–0.0315	0.0871	0.96	–0.0396	0.0894	1.06	0.0618	0.0674	1.02	0.0246	0.0577
Teat length	1.04	0.0347	0.0462	1.10	0.0917	0.0886	1.01	0.00680	0.0839	1.08	0.0776	0.0654	1.06	0.0570	0.0561

[1]CNS = Coagulase-negative staphylococci.
[2]SNA = Streptococci other than *Streptococcus agalactiae*.
[3]Coliform species and streptococci other than *Streptococcus agalactiae*.
[4]OR = odds ratio; b-value = regression coefficient.
†$P \leq 0.10$.
*$P \leq 0.05$.
**$P \leq 0.01$.

Figure 1. Quadratic regression of the total number of clinical episodes during first and second lactation on sire predicted transmitting abilities (PTA) for somatic cell score (SCS). Lines represent first (–) and second (…) lactation.

The shape of the quadratic regression lines also indicates that the marginal advantage increases as PTA for SCS decreases.

Rogers et al. (13) detected a similar quadratic effect, which indicated that sires with the lowest genetic evaluations for SCS also had the most favorable evaluations for clinical mastitis. However, Philipsson et al. (11) regressed genetic evaluations for clinical mastitis on SCS evaluations and found no evidence of a nonlinear effect.

The results of the current study and the studies by Philipsson et al. (11) and Rogers et al. (13), with regard to a nonlinear association between clinical mastitis and SCS, do not support the theory that selection for the lowest SCS will result in dairy cattle that are unable to respond to infection. If such were the case, the lowest SCS would be associated with a higher number of clinical episodes, and an intermediate SCS would provide optimal resistance to mastitis. This theory stems from the results of experimental challenge studies, which indicated that elevated SCC prior to infusion protects against infection by mastitis causing organisms (6).

McDaniel et al. (8) also regressed CMI and TCE data (from 1231 primiparous cows in three herds) on sire PTA for SCS. As in the current study, daughters of

Table 6. Logistic regression of daughter clinical mastitis incidence (binary variable) during second lactation on sire transmitting abilities by organism group.

	All organisms			Coliform species			SNA[1]			Environmental organisms[2]		
Transmitting abilities	OR[3]	b-value	SE	OR	b-value	SE	OR	b-value	SE	OR	b-value	SE
Somatic cell score	1.76	0.567	0.409	0.58	–0.547	0.776	2.99†	1.09	0.597	1.51	0.410	0.507
Protein (kg)	1.00	0.00276	0.0109	1.01	0.0110	0.0200	0.99	–0.0126	0.0150	0.99	–0.00969	0.0131
Productive life	0.89†	–0.118	0.0694	0.85	–0.159	0.125	0.76**	–0.269	0.0959	0.79**	–0.234	0.0841
Fore udder attachment	0.93	–0.0708	0.0749	1.11	0.102	0.143	0.87	–0.139	0.107	0.91	–0.0976	0.0930
Rear udder height	1.04	0.0347	0.0859	1.33†	0.286	0.167	0.87	–0.140	0.117	0.96	–0.0443	0.103
Rear udder width	1.03	0.0264	0.0912	1.38†	0.325	0.175	0.92	–0.0849	0.128	1.00	0.00102	0.111
Udder cleft	0.88	–0.130	0.0816	0.77†	–0.265	0.159	0.88	–0.125	0.115	0.81*	–0.207	0.100
Udder depth	0.98	–0.0218	0.0644	0.99	–0.00722	0.123	0.88	–0.123	0.0926	0.89	–0.116	0.0801
Front teat placement	0.96	–0.0440	0.0583	0.94	–0.0638	0.116	0.94	–0.0667	0.0823	0.93	–0.0687	0.0716
Teat length	0.98	–0.0196	0.0623	1.06	0.0603	0.117	1.05	0.0502	0.0843	1.02	0.0229	0.0748

[1]SNA = Streptococci other than *Streptococcus agalactiae*.

[2]Coliform species and streptococci other than *Streptococcus agalactiae*.

[3]OR = Odds ratio; b-value = regression coefficient.

†$P \leq 0.10$.

*$P \leq 0.05$.

**$P \leq 0.01$.

sires that transmit higher SCS had higher CMI and TCE during first lactation.

Mastitis from environmental organisms is generally of shorter duration than mastitis from contagious organisms (10). Therefore, Shook (20) hypothesized that selection for lower SCS may not improve resistance to mastitis from environmental organisms because monthly SCC measurement (the current practice in the United States) may not detect elevated SCC due to mastitis that is of short duration. However, in the current study, daughters of sires that transmit higher SCS had higher CMI during first and second lactations from all organism groups considered. These findings indicate that selection for lower SCS may improve resistance to clinical mastitis caused by environmental organisms or CNS. The effect of selection for lower SCS on CMI from other organisms (including contagious) could not be predicted because clinical episodes caused by these organisms were not prevalent enough.

The daughters of sires that transmit higher SCS may have had higher CMI from environmental organisms because exposure to these organisms occurs daily and the less resistant cows may become infected more often or have longer lasting clinical or subclinical episodes. As a result, the less resistant cows may be more likely to have elevated SCC on the day it is measured despite the relatively short duration of mastitis caused by environmental organisms.

Even if the relatively short duration of mastitis from environmental organisms decreases the likelihood of detecting a concomitant increase in SCC, a biological interpretation is lacking for an organism-dependent re-

Table 7. Linear regression of the total number of clinical episodes during first and second lactation on sire transmitting abilities.

	First lactation		Second lactation	
Transmitting abilities	b-Value[1]	SE	b-value	SE
Somatic cell score	0.259*	0.102	0.175	0.114
Protein (kg)	–0.000979	0.00298	0.00245	0.00326
Productive life	–0.0572**	0.0182	–0.0250	0.0207
Fore udder attachment	–0.0398*	0.0196	–0.0296	0.0220
Rear udder height	–0.0258	0.0224	–0.00375	0.0267
Rear udder width	–0.0225	0.0226	–0.00991	0.0278
Udder cleft	–0.0263	0.0209	–0.0497*	0.0242
Udder depth	–0.0447**	0.0165	–0.0234	0.0191
Front teat placement	–0.0118	0.0152	–0.00632	0.0165
Teat length	0.00882	0.0154	–0.00149	0.0176

[1]b-Value = regression coefficient.

*$P \leq 0.05$.

**$P \leq 0.01$.

Table 8. Quadratic regression of the total number of clinical episodes during first and second lactation on sire transmitting abilities.

	First lactation		Second lactation	
Transmitting abilities	Linear[1]	Quadratic	Linear	Quadratic
Somatic cell score	10.2***	–1.49***	8.01**	–1.17**
Protein (kg)	–0.00247	9.14E-05	0.00291	–2.61E-05
Productive life	–0.0724***	0.0212†	–0.0378	0.0128
Fore udder attachment	–0.0406*	0.00768	–0.0196	–0.0279*
Rear udder height	–0.0422†	0.0457**	0.00973	–0.0320
Rear udder width	–0.0370	0.0263†	0.0184	–0.0374†
Udder cleft	–0.0468†	0.0248	–0.0688*	0.0184
Udder depth	–0.0445*	0.000200	–0.0251	–0.00237
Front teat placement	–0.0171	0.0152	0.00330	–0.0203†
Teat length	0.0114	–0.00264	–0.00658	0.00592

[1]Linear = regression coefficient for the linear effect, Quadratic = regression coefficient for the quadratic effect.

†$P \leq 0.10$.

*$P \leq 0.05$.

**$P \leq 0.01$.

***$P \leq 0.001$.

sponse to selection for lower SCS. The manner in which the cow's defense mechanisms respond to infections from environmental and contagious organisms would have to differ considerably to prevent selection for lower SCS from improving resistance to mastitis from environmental organisms. However, the manner in which the cow's defense mechanisms respond to infection is generally the same regardless of whether a contagious or environmental organism is involved (10).

STA for udder type traits. The linear effect of the STA for fore udder attachment was a significant ($P \leq 0.10$) predictor of TCE during first lactation (Table 7), and the quadratic effect was a significant predictor of TCE during second lactation (Table 8). The regression coefficient for the linear effect was negative, indicating that daughters of sires that transmit strongly attached fore udders had lower TCE during first lactation. The regression coefficient for the quadratic effect was also negative, indicating that TCE decreased at an increasing rate during second lactation among daughters of sires that transmit strongly attached fore udders. These findings are supported by other studies that found that more strongly attached fore udders were associated with less clinical mastitis or lower SCS (13, 14, 17).

The STA for rear udder height was a significant ($P \leq 0.10$) predictor of CMI during second lactation when coliform species were considered (Table 6). In addition, the quadratic effect of the STA for rear udder height was a significant predictor of TCE during first lactation (Table 8). The odds ratio was >1, indicating that daughters of sires that transmit higher rear udders had higher CMI during second lactation when coliform species were considered. However, the quadratic regression line indicated that TCE during first lactation decreased as rear udder height increased to intermediate values. At higher values, TCE during first lactation increased as rear udder height increased. The relationship between udder health and rear udder height has not been consistent across other studies either. Some studies have found that higher rear udders are associated with less clinical mastitis or lower SCS (7, 17), while others found no effect or an effect that is inconsistent across lactations or data sets (13, 14). The lack of a consistent relationship between measures of udder health (such as CMI and TCE) and rear udder height suggests that there may not be a biological basis for an association between the two.

The STA for rear udder width was a significant ($P \leq 0.10$) predictor of CMI during second lactation when coliform species were considered (Table 6). Furthermore, the quadratic effect of the STA for rear udder width was a significant predictor of TCE during first and second lactations (Table 8). The odds ratio was >1, indicating that daughters of sires that transmit wider rear udders had higher CMI during second lactation when coliform species were considered. However, the significant regression coefficients for the quadratic effect lacked a consistent interpretation because they had different signs depending on which lactation was considered. Other studies have not found a consistent relationship between udder health and rear udder width either. Both wider and narrower rear udders have been associated with less clinical mastitis or lower SCS (13, 14, 17). This indicates that associations between measures of udder health (such as CMI and TCE) and rear udder width may not have a biological basis.

The STA for udder cleft was a significant predictor of CMI during second lactation when coliform species

or the most common environmental organisms were considered (Table 6). In addition, the linear effect of the STA for udder cleft was a significant predictor of TCE during second lactation (Table 7). All odds ratios, regardless of the statistical significance of the underlying logistic regression coefficients, were <1, and all regression coefficients for the linear effect, regardless of their statistical significance, were negative. These findings suggest that daughters of sires that transmit deeper udder cleft had lower CMI and TCE during first and second lactations. Deeper udder cleft was associated with less clinical mastitis or lower SCS in other studies as well (13, 17, 18).

The STA for udder depth was a significant predictor of CMI during first lactation when all organisms were considered (Table 5). Furthermore, the linear effect of the STA for udder depth was a significant predictor of TCE during first lactation (Table 7). All odds ratios, regardless of the statistical significance of the underlying logistic regression coefficients, were <1, and all regression coefficients for the linear effect, regardless of their statistical significance, were negative. These results indicate that daughters of sires that transmit shallower udders had lower CMI and TCE during first and second lactations. Several other studies have also found that shallower udders were associated with less clinical mastitis or lower SCS (7, 13, 14, 17, 18, 24).

The range in STA for udder depth in this study was 7.83 (see Table 3) and the logistic regression coefficient was –0.104 when CMI from all organisms during first lactation was the dependent variable (Table 5). Therefore, daughters of the sire that transmits the shallowest udders would be $e^{(7.83 * -0.104)} = 0.44$ times less likely than daughters of the sire that transmits the deepest udders to have clinical mastitis during first lactation.

The quadratic effect of the STA for front teat placement was a significant predictor of TCE during second lactation (Table 8). The significant regression coefficient was negative, indicating that TCE decreased at an increasing rate during second lactation among daughters of sires that transmit closely spaced front teats. Closely spaced front teats have been associated with less clinical mastitis or lower SCS in other studies as well (7, 13, 14, 17, 18).

PTA for PL. The PTA for PL was a significant predictor of CMI during first and second lactations when all organisms were considered (Tables 5 and 6). The PTA for PL was also a significant predictor of CMI during second lactation when SNA or the most common environmental organisms were considered (Table 6). In addition, both the linear and quadratic effects of PTA for PL were significant predictors of TCE during first lactation (Tables 7 and 8). All odds ratios, regardless of the statistical significance of the underlying logistic regression coefficients, were <1, and all regression coefficients for the linear effect, regardless of their statistical significance, were negative. The quadratic regression line indicated that TCE during first lactation increased at an increasing rate among daughters of sires that transmit shorter PL. These findings suggest that daughters of sires that transmit longer PL had lower CMI and TCE during first and second lactations. Previous research found that longer PL was associated with both decreased clinical mastitis and lower SCS (13).

PTA for protein yield. The PTA for protein was a significant predictor of CMI during first lactation when SNA were considered (Table 5). The odds ratio was >1, indicating that daughters of sires that transmit higher protein yield had higher CMI during first lactation when SNA were considered. Higher yield has been associated with more clinical mastitis or higher SCS in other studies as well (3, 12, 13, 18, 23).

CONCLUSIONS

Estimates of the heritability of clinical mastitis were generally higher compared to other studies. Estimates of the heritability of the total number of clinical episodes during first and second lactations were 0.42 and 0.15, respectively. Estimates of the heritability of clinical mastitis incidence during first and second lactations from all organism groups considered ranged from 0.03 to 0.25 and from 0.01 to 0.19, respectively. Incidence of clinical mastitis caused by environmental organisms tended to be the most heritable.

Incidence of clinical mastitis from all organism groups considered, including incidence of clinical mastitis from all organisms, was higher during first and second lactations among daughters of sires that transmit higher SCS. Daughters of these sires also experienced more clinical episodes during first and second lactations. The total number of clinical episodes per lactation were nonlinearly associated with PTA for SCS. However, daughters of sires that transmit the lowest SCS had the fewest clinical episodes during first and second lactations. Therefore, selection for lower SCS may reduce both the incidence of clinical mastitis and the number of clinical episodes per lactation without diminishing the ability to respond to infection. In particular, these findings indicate that the incidence of clinical mastitis caused by CNS or environmental organisms may be reduced by selection for lower SCS. The effect of selection for lower SCS on the incidence of clinical mastitis from other organisms (including contagious) could not be predicted because clinical episodes caused by these organisms were not prevalent in this study.

Daughters of sires that transmit longer productive life, stronger fore udder attachment, shallower udders,

and deeper udder cleft had either fewer clinical episodes or lower clinical mastitis incidence during first and second lactations. As a result, selection for longer productive life, stronger fore udder attachment, shallower udders, or deeper udder cleft may reduce both the incidence of clinical mastitis and number of clinical episodes per lactation.

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