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Relationships Among Severity and Duration of Clinical Mastitis and 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 severity and duration of clinical mastitis during first and second lactation and sire transmitting abilities for somatic cell score, udder type traits, productive life, and protein yield. Recording of clinical episodes began at first parturition for 1704 Holstein cows (in six Pennsylvania herds and one Nebraska herd) and continued into second lactation for 1055 of these cows. A total of 456 cows (sired by 168 bulls) had at least one clinical episode during first lactation, and 230 cows (sired by 100 bulls) had at least one clinical episode during second lactation. A severity code from 1 (normal milk) to 5 (acute systemic mastitis) was assigned daily (for up to 30 d after detection) to all quarters that had clinical mastitis. Only the severity codes for the first clinical episode to occur during first and second lactation are considered here. The initial and maximum severity codes, as well as the natural logarithms of both the sum of severity codes that were above normal (> 1) and the total days severity codes were above normal were regressed on herd (a classification variable), age at first calving, days in milk at clinical detection, and sire transmitting abilities taken one at a time. Linear and nonlinear effects were estimated for sire transmitting abilities. Separate analyses were conducted on dependent variables that considered severity and duration of 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*). Daughters of sires that transmit the lowest somatic cell score had the least severe and shortest clinical episodes from environmental organisms during first lactation. Selection for lower somatic cell score may reduce the severity and duration of clinical episodes from environmental organisms during first lactation. (**Key words:** severity and duration of clinical mastitis, somatic cell score, productive life, udder type traits)

Abbreviation key: CNS = coagulase-negative staphylococci, **ISC** = initial severity code, **LOGDAYS** = natural logarithm of the total days severity codes were above normal in the 30 d after detection, **LOGSUM** = natural logarithm of the sum of severity codes that were above normal in the 30 d following detection, **MSC** = maximum severity code in the 30 d after detection, **PL** = productive life, **SNA** = streptococci other than *Streptococcus agalactiae*, **STA** = standardized transmitting abilities.

INTRODUCTION

Approximately 10% of the total value of milk sales made by US dairy farms (nearly $2 billion dollars) is lost to mastitis each year (National Mastitis Council, 1996). Although mastitis from contagious organisms (especially *Streptococcus agalactiae*) has been reduced by improvements in management, economic losses due to mastitis will continue because the causative organisms in the dairy cow's environment cannot be eradicated (National Mastitis Council, 1996). In addition, an unfavorable (positive) genetic correlation exists between mastitis and milk yield (Schmidt and Van Vleck, 1965; Emanuelson et al., 1988; Shook, 1993; Rogers et al., 1998), indicating that economic losses may increase. The increased susceptibility to mastitis accompanying selection for milk yield suggests that selection for resistance to mastitis is needed. Optimal selection for resistance to mastitis (under current economic conditions) would slow, rather than halt, the rate of increase in genetic susceptibility to mastitis (Strandberg and Shook, 1989; Rogers, 1993).

Direct selection for resistance to mastitis is not possible because records of clinical mastitis occurrence are not readily available for most US dairy cattle. Traits being considered for indirect selection for resistance to

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mastitis include 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 once a month (Shook, 1993). The primary cause of elevated SCC is the presence of mastitis causing organisms in the udder (Harmon, 1994). Furthermore, higher SCS (a logarithmic transformation of SCC) is genetically associated with higher occurrence of clinical mastitis (Coffey et al., 1986; Emanuelson et al., 1988; Philipsson et al., 1995; Rogers et al., 1998; Nash et al., 2000). These findings indicate that selection for lower SCS may reduce the incidence of clinical mastitis. However, the impact of selection for lower SCS on the severity and duration of clinical episodes is unknown. Therefore, the objective of this study was to determine the relationships among severity and duration of daughter clinical mastitis during first and second lactation 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 1704 cows in seven herds (six in Pennsylvania, one in Nebraska) and continued into second lactation for 1055 of these cows in six of the seven herds. Clinical episodes were recorded from May, 1991, through December, 1995. Recording of clinical episodes began at first parturition 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. All quarter samples were frozen and transported weekly to diagnostic laboratories (one in Pennsylvania and one in Nebraska) for culturing following procedures described previously (Wanner et al., 1998).

Herdsmen also assigned a severity code daily to all quarters that had clinical mastitis. Daily assignment of a severity code began when clinical mastitis was first detected and continued until the milk and cow returned to normal or 30 d had elapsed. If a quarter returned to normal and subsequently became abnormal within 30 d after clinical mastitis was first detected, daily assignment of a severity code resumed.

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 order to avoid inconsistent assignment of severity codes across herds. In addition, research technicians made weekly visits to the Pennsylvania herds to ensure that the study protocol was being followed.

One of five severity codes could be assigned by herdsmen. A severity code of 1 indicated normal milk and quarter, which allowed recording that a previously clinical quarter had returned to normal. A severity code of 2 indicated a normal quarter with questionably normal milk. A severity code of 3 indicated abnormal milk (definite clots or flakes) but little or no swelling of the quarter (subacute clinical mastitis). A severity code of 4 indicated abnormal milk and a swollen quarter (acute clinical mastitis). A severity code of 5 indicated abnormal milk, swollen quarter, and a physically ill cow (acute systemic mastitis). A cow was considered to have clinical mastitis if a severity code of 2 or higher was recorded.

Four measures of the severity and duration of clinical mastitis were developed from these severity codes. The initial severity code (**ISC**) and the maximum severity code in the 30 d following detection (**MSC**) were two of the measures. The third measure was the natural logarithm of the sum of severity codes that were above normal (> 1) in the 30 d following detection (**LOGSUM**). The fourth measure was the natural logarithm of the total days severity codes were above normal in the 30 d following detection (**LOGDAYS**). The logarithmic transformation was used to normalize the last two measures.

Only the severity and duration of the first clinical episode to occur during first and second lactation are considered here. To be considered, the first clinical episode of a lactation had to occur before 365 DIM had elapsed.

Analyses

The four measures of severity and duration of clinical mastitis (ISC, MSC, LOGSUM, and LOGDAYS) were regressed on herd (a classification variable), age at first calving, DIM at clinical detection, and sire transmitting abilities for SCS, udder type traits, PL, and protein yield taken one at a time. Linear and quadratic effects were estimated for each of the transmitting abilities using the GLM procedure of SAS (SAS Inst., Inc., 1995). Cubic effects were also estimated for PTA for SCS.

First and second lactation data were analyzed separately. Furthermore, separate analyses were conducted on dependent variables that considered severity and duration of clinical mastitis from: all organisms, coagulase-negative staphylococci (**CNS**), coliform species, streptococci other than *Streptococcus agalactiae* (**SNA**), and the most common environmental organisms (coli-

form species and SNA). Clinical mastitis from all organisms contained all initial clinical episodes during a lactation, including those that had missing or contaminated quarter samples or no detectable organism growth. Organism (CNS, coliform species, SNA, *Staphylococcus aureus*, other organisms, no growth, missing quarter sample, or contaminated quarter sample) was included as an explanatory variable in the regression model when the dependent variables considered severity and duration of clinical mastitis from all organisms. Severity and duration of clinical mastitis from CNS during second lactation were not analyzed separately because its incidence was too low.

When a cow had clinical mastitis in multiple quarters on the same day, each quarter that had clinical mastitis was considered a separate episode. Each quarter that had mastitis on the same day was considered a clinical episode because mastitis causing organisms entered each quarter separately. If a cow had clinical mastitis from a particular organism in multiple quarters on the same day, the quarter that had the highest sum of severity codes that were above normal was considered to be the first clinical episode of the lactation. If a cow had clinical mastitis from different organisms in multiple quarters on the same day, each quarter infected with a different organism was considered to be the first clinical episode of the lactation from that organism. This was done only when the severity and duration of clinical mastitis from different organism groups were analyzed separately.

Two organisms were detected in 32 quarters (7% of those that had the first clinical episode) during first lactation and 15 quarters (7%) during second lactation. These quarters were considered to have a clinical episode from each organism when the severity and duration of clinical mastitis from different organism groups were analyzed separately.

When the dependent variables were regressed on STA for udder type traits, data from 11 first-lactation cows and four second-lactation cows were excluded because STA for udder type traits were not available for their sires. When the dependent variables were regressed on STA for teat length, data from 12 first-lactation cows and five second-lactation cows were excluded because STA for teat length were not available for their sires.

RESULTS AND DISCUSSION

Data

A total of 456 of 1704 cows (27%) had at least one clinical episode during first lactation. In addition, 230 of 1055 cows (22%) had at least one clinical episode during second lactation. Eighty-four of these cows had clinical mastitis during first lactation. The relationships among clinical mastitis incidence during first and second lactation in the cooperating herds and sire transmitting abilities for SCS, udder type traits, PL, and protein yield are presented elsewhere (Nash et al., 2000).

Five of the cooperating herds were commercial herds (all in Pennsylvania) and two were university research herds. 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 are in Table 1. Herd 5 had the lowest incidence of clinical mastitis during first and second lactation. Herd 5 was the only herd to routinely give all heifers an intramammary antibiotic infusion in each quarter 30 d before the expected calving date. All herds routinely postdipped and administered dry cow therapy.

Table 1 also contains the number of cows in each herd that had clinical mastitis caused by the most prevalent organism groups. Although there was considerable variation in clinical mastitis incidence between herds, the proportion of clinical episodes caused by each organism group was similar.

The efficacy of therapies used to treat the initial clinical episodes in five of the seven herds (all in Pennsylvania) has been studied (Sischo et al., 1995). For this study, therapies were categorized as follows: no antibiotics used, treatment with intramammary beta-lactams, treatment with intramammary ceftiofur, and miscellaneous therapy. Intramammary ceftiofur was the most commonly used mastitis therapy (used in 39% of cases). No antibiotics were used in 14% of cases. Only one farm administered some form of antibiotic to all cows that had clinical mastitis. Mastitis therapy had no effect on the number of days until milk and quarter returned to normal (severity code = 1).

Mastitis was the second and fourth most common reason for culling cows during first and second lactation, respectively. However, fewer than 3% of first- and second-lactation cows were culled due to mastitis.

The measures of severity and duration of the first clinical episode during first and second lactation are summarized in Table 2 for all herds by organism group. Approximately 37, 39, 20, and 4% of the severity codes assigned to the initial clinical episode during first lactation were severity codes 2, 3, 4, and 5, respectively. Approximately 34, 41, 19, and 6% of the severity codes assigned to the initial clinical episode during second lactation were severity codes 2, 3, 4, and 5, respectively.

Table 2 also contains the number of cows that had clinical mastitis caused by the most prevalent organism groups. Most of the initial clinical episodes during first or second lactation were caused by SNA or coliform

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

	Herd							
	1	2	3	4	5	6	7	Totals
First lactation								
Total cows	170	49	136	226	753	211	159	1704
Cows that had mastitis	90	7	54	101	80	63	61	456
Frequency	0.53	0.14	0.40	0.45	0.11	0.30	0.38	0.27
Cows that had mastitis by organism group								
SNA[1]	29	2	20	26	25	18	25	145
Frequency	0.32	0.29	0.37	0.26	0.31	0.29	0.41	0.32
Coliform species	19	1	16	17	7	15	6	81
Frequency	0.21	0.14	0.30	0.17	0.09	0.24	0.10	0.18
CNS[2]	15	1	11	24	22	11	1	85
Frequency	0.17	0.14	0.20	0.24	0.28	0.17	0.02	0.19
Environmental organisms[3]	45	2	36	39	32	33	31	218
Frequency	0.50	0.29	0.67	0.39	0.40	0.52	0.51	0.48
Second lactation								
Total cows	110	0[4]	73	98	614	100	60	1055
Cows that had mastitis	43		23	45	75	28	16	230
Mastitis frequency	0.39		0.32	0.46	0.12	0.28	0.27	0.22
Cows that had mastitis by organism group								
SNA[1]	9		10	18	28	6	5	76
Frequency	0.21		0.43	0.40	0.37	0.21	0.31	0.33
Coliform species	5		3	8	17	6	0	39
Frequency	0.12		0.13	0.18	0.23	0.21	0.00	0.17
CNS[2]	2		0	3	5	1	1	12
Frequency	0.05		0.00	0.7	0.07	0.04	0.06	0.05
Environmental organisms[3]	14		13	25	44	11	5	112
Frequency	0.33		0.57	0.56	0.59	0.39	0.31	0.49

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

[2]CNS = Coagulase-negative staphylococci.

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

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

species (*Escherichia coli* and *Klebsiella* species). Few of the initial clinical episodes were caused by contagious organisms. *Staphylococcus aureus* was detected in the first clinical episode experienced by 4 and 6% of the cows that had clinical mastitis during first and second lactation, respectively. *Streptococcus agalactiae* was detected in the first clinical episode experienced by approximately 1% of the cows that had clinical mastitis during first or second lactation.

No organism growth was detected in the milk samples collected from the first clinical episode experienced by 19% of the cows that had clinical mastitis during first or second lactation. Milk samples were missing (not collected) for the first clinical episode experienced by 10 and 13% of the cows that had clinical mastitis during first and second lactation, respectively. Milk samples were classified as contaminated for the first clinical episode experienced by 6 and 3% of the cows that had clinical mastitis during first and second lactations, respectively. Severity codes were recorded for cows with missing or contaminated quarter samples. Therefore, data from these cows were used in the analyses. Nash (1999) concluded 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 first clinical episode usually occurred early in each lactation. The mean DIM at detection was 65 d for the first clinical episode during first lactation. The first clinical episode was detected within 1 mo after calving for 250 of the 456 cows (55%) that had clinical mastitis during first lactation. One hundred and seventy-seven of these cows had their first clinical episode within 1 wk after calving. During second lactation, the mean DIM at detection was 89 d for the first clinical episode. The first clinical episode was detected within 3 mo after 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 after calving.

Mean length of lactation was 283 and 264 d for cows that had clinical mastitis during first and second lactations, respectively. Approximately 78% of the cows that had clinical mastitis during first lactation and 85% of the cows that had clinical mastitis during second lactation were milked fewer than 365 d. Mean length of lactation was 301 d for first-lactation cows and 273

Table 2. Measures of severity and duration of the first clinical mastitis episode during first and second lactations by organism group.

Severity and duration measure by organism group	First lactation					Second lactation				
	Cows	Mean	SD	Minimum	Maximum	Cows	Mean	SD	Minimum	Maximum
ISC[1]										
All organisms	456	3.14	0.76	2	5	230	3.33	0.79	2	5
SNA[2]	145	3.23	0.71	2	5	76	3.20	0.73	2	5
Coliform species	81	3.40	0.75	2	5	39	3.72	0.79	2	5
CNS[3]	85	2.96	0.63	2	5	12	3.33	0.65	2	4
Environmental organisms[4]	218	3.27	0.72	2	5	112	3.39	0.79	2	5
MSC[1]										
All organisms		3.24	0.79	2	5		3.42	0.80	2	5
SNA[2]		3.34	0.75	2	5		3.34	0.70	2	5
Coliform species		3.51	0.79	2	5		3.77	0.81	2	5
CNS[3]		3.04	0.70	2	5		3.33	0.65	2	4
Environmental organisms[4]		3.39	0.76	2	5		3.50	0.76	2	5
Sum of severity codes that were above normal										
All organisms		17.75	18.84	2	128		19.30	18.72	2	117
SNA[2]		22.08	24.02	2	128		19.45	15.37	2	90
Coliform species		17.52	15.78	2	79		24.41	18.16	5	82
CNS[3]		10.99	10.25	2	58		11.33	6.79	2	30
Environmental organisms[4]		20.65	21.84	2	128		20.88	16.19	2	90
LOGSUM[1]										
All organisms		2.45	0.93	0.69	4.85		2.58	0.89	0.69	4.76
SNA[2]		2.68	0.91	0.69	4.85		2.70	0.76	0.69	4.50
Coliform species		2.53	0.82	0.69	4.37		2.92	0.77	1.61	4.41
CNS[3]		2.08	0.80	0.69	4.06		2.26	0.65	0.69	3.40
Environmental organisms[4]		2.63	0.89	0.69	4.85		2.77	0.77	0.69	4.50
Total days severity codes were above normal										
All organisms		6.11	5.70	1	30		6.50	5.51	1	30
SNA[2]		7.49	6.92	1	30		7.01	5.00	1	30
Coliform species		5.70	4.72	1	30		7.85	5.24	2	25
CNS[3]		4.08	3.76	1	27		4.00	2.22	1	10
Environmental organisms[4]		6.92	6.36	1	30		7.19	4.97	1	30
LOGDAYS[1]										
All organisms		1.47	0.83	0	3.40		1.56	0.80	0	3.40
SNA[2]		1.67	0.83	0	3.40		1.73	0.69	0	3.40
Coliform species		1.47	0.73	0	3.40		1.84	0.69	0.69	3.22
CNS[3]		1.13	0.73	0	3.30		1.25	0.55	0	2.30
Environmental organisms[4]		1.61	0.81	0	3.40		1.75	0.69	0	3.40

[1]ISC = Initial severity code, LOGDAYS = natural logarithm of the total days severity codes were above normal in the 30 d following detection, LOGSUM = natural logarithm of the sum of severity codes that were above normal in the 30 d following detection, MSC = maximum severity code in the 30 d following detection.

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

[3]CNS = Coagulase-negative staphylococci.

[4]Combines all cows that had mastitis from environmental organisms: coliform species and streptococci other than *Streptococcus agalactiae*.

d for second-lactation cows that did not have clinical mastitis. Approximately 76% of the cows that did not have clinical mastitis during first lactation and 86% of the cows that did not have clinical mastitis during second lactation were milked fewer than 365 d.

Actual or projected 305-d milk yield was available from DHIA records for 441 cows that had clinical mastitis during first lactation and 230 cows that had clinical mastitis during second lactation. Mean 305-d milk yield was 8074 kg for first lactation (range was 3215 to 11,629 kg) and 9357 kg for second lactation (range was 3669 to 13,023 kg). Actual or projected 305-d milk yield was available from DHIA records for 1223 first-lactation cows and 810 second-lactation cows that did not have clinical mastitis. Mean 305-d milk yield was 8575 kg for first lactation (range: 2257 to 12,351 kg) and 9958 kg for second lactation (range: 1194 to 15,246 kg).

Lactation average SCS was available from DHIA records for 286 cows (in five herds) that had clinical mastitis during first lactation and 125 cows (in four herds) that had clinical mastitis during second lactation. Mean lactation average SCS was 3.29 for first lactation (range was 0.30 to 8.50) and 3.39 for second lactation (range was 0.20 to 8.40). Lactation average SCS was available from DHIA records for 445 first-lactation cows (in five herds) and 223 second-lactation cows (in four herds) that did not have clinical mastitis. Mean lactation average SCS was 2.41 for first lactation (range was 0.00

Table 3. Predicted and standardized transmitting abilities for sires of Holstein cows that had at least one clinical mastitis episode during first lactation and sires that did not have any daughters that had clinical mastitis during first lactation.

Transmitting abilities	N	Mean	SD	Minimum	Maximum
Sires of cows that had clinical mastitis					
SCS (log_2)	168	3.26	0.206	2.74	3.80
Protein (kg)	168	6.98	9.66	–33.1	27.2
Productive life	168	0.226	1.31	–3.80	3.20
Fore udder attachment	160[1]	–0.0489	1.25	–3.13	2.83
Rear udder height	160	0.279	1.19	–3.58	3.15
Rear udder width	160	0.266	1.13	–3.17	3.25
Udder cleft	160	0.113	1.22	–3.59	3.13
Udder depth	160	–0.240	1.49	–4.36	3.41
Front teat placement	160	0.103	1.43	–4.25	3.50
Teat length	159[2]	–0.0446	1.42	–3.85	4.31
Sires that did not have any daughters that had clinical mastitis					
SCS (log_2)	106	3.19	0.204	2.76	3.88
Protein (kg)	106	7.36	8.49	–18.6	29.9
Productive life	106	0.416	1.16	–3.30	3.30
Fore udder attachment	103[3]	0.316	1.09	–2.91	3.20
Rear udder height	103	0.463	0.953	–1.62	3.18
Rear udder width	103	0.460	0.899	–1.58	2.48
Udder cleft	103	0.284	1.05	–1.77	3.54
Udder depth	103	–0.0111	1.34	–3.56	2.85
Front teat placement	103	0.210	1.23	–2.46	3.40
Teat length	102[4]	0.0626	1.14	–2.39	3.61

[1]The standardized transmitting abilities for udder type were not available for eight sires.
[2]The standardized transmitting abilities for teat length were not available for nine sires.
[3]The standardized transmitting abilities for udder type were not available for three sires.
[4]The standardized transmitting abilities for teat length were not available for four sires.

to 9.00) and 2.56 for second lactation (range was 0.00 to 7.90).

The mean age at first calving for all cows that had clinical mastitis during first or second lactation was 26 mo. The range was 22 to 33 mo. The mean age at first calving for all first- and second-lactation cows that did not have clinical mastitis was 25 mo. The range was 22 to 33 mo.

The 456 cows that had clinical mastitis during first lactation were sired by 168 Holstein bulls. The 230 cows that had clinical mastitis during second lactation were sired by 100 Holstein bulls. Fifty-seven of these sires had at least one daughter that had clinical mastitis during first lactation. The mean numbers of first- and second-lactation daughters per sire were 2.7 and 2.3., respectively. One bull had 27 first-lactation daughters and 19 second-lactation daughters in the cooperating herds. All other sires had no more than 17 first-lactation daughters or 12 second-lactation daughters. A total of 136 sires had three or fewer first-lactation daughters and 83 sires had three or fewer second-lactation daughters. The PTA and standardized transmitting abilities (**STA**) from the USDA Sire Summary of November, 1997 (1997) and Holstein Association USA (1997) are summarized in Tables 3 and 4 for sires of first and second lactation cows that had clinical mastitis. Tables 3 and 4 also summarize PTA and STA for the sires that did not have any daughters that had clinical mastitis during first or second lactation.

Relationships Among Severity and Duration of Clinical Mastitis and Sire Transmitting Abilities

Table 5 contains the coefficients and standard errors for the linear regression of ISC, MSC, LOGSUM, and LOGDAYS on PTA for SCS. Table 6 contains the regression coefficients for the linear and quadratic effects of PTA and STA that were significant ($P \leq 0.10$) predictors of ISC, MSC, LOGSUM, or LOGDAYS. The regression coefficients and standard errors for the linear effects of transmitting abilities for traits other than SCS that were significant ($P \leq 0.10$) predictors of ISC, MSC, LOGSUM, or LOGDAYS are in Table 7.

PTA for SCS. The linear effect of PTA for SCS was a significant ($P \leq 0.10$) predictor of ISC and MSC for clinical episodes from coliform species and the most common environmental organisms during first lactation (Table 5). The significant regression coefficients were positive, indicating that daughters of sires that transmit higher SCS had higher ISC and MSC for clinical episodes from coliform species and the most common environmental organisms during first lactation.

The linear effect of PTA for SCS was not a significant ($P > 0.10$) predictor of LOGSUM or LOGDAYS for clini-

Table 4. Predicted and standardized transmitting abilities for sires of Holstein cows that had at least one clinical mastitis episode during second lactation and sires that did not have any daughters that had clinical mastitis during second lactation.

Transmitting abilities	N	Mean	SD	Minimum	Maximum
Sires of cows that had clinical mastitis					
SCS (log_2)	100	3.24	0.204	2.76	3.72
Protein (kg)	100	7.11	9.14	–24.0	27.2
Productive life	100	0.369	1.24	–3.00	2.70
Fore udder attachment	96[1]	0.0116	1.13	–2.53	3.20
Rear udder height	96	0.438	1.04	–2.01	3.15
Rear udder width	96	0.370	0.98	–1.61	2.84
Udder cleft	96	0.188	1.10	–3.59	2.21
Udder depth	96	–0.0689	1.30	–4.16	2.85
Front teat placement	96	0.113	1.27	–3.15	3.38
Teat length	95[2]	0.0889	1.35	–3.30	4.31
Sires that did not have any daughters that had clinical mastitis					
SCS (log_2)	66	3.21	0.225	2.81	3.88
Protein (kg)	66	7.00	10.3	–33.1	29.9
Productive life	66	0.320	1.29	–3.80	3.20
Fore udder attachment	63[3]	–0.0598	1.27	–3.13	2.66
Rear udder height	63	0.0254	1.12	–2.65	2.66
Rear udder width	63	0.0717	1.08	–2.69	2.48
Udder cleft	63	0.0344	1.26	–2.83	3.54
Udder depth	63	–0.337	1.54	–4.36	2.93
Front teat placement	63	–0.0160	1.44	–3.29	3.40
Teat length	63	0.0956	1.44	–3.85	3.61

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

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

[3]The standardized transmitting abilities for udder type were not available for three sires.

cal episodes during first lactation (Table 5). In addition, the linear effect was not a significant predictor of ISC, MSC, LOGSUM, or LOGDAYS for clinical episodes during second lactation (Table 5).

The quadratic effect of PTA for SCS was a significant ($P \leq 0.10$) predictor of LOGSUM for clinical episodes from SNA during first lactation (Table 6). As shown in Figure 1, the quadratic regression line (includes linear and quadratic effects) was concave, with the peak occurring near the mean PTA for SCS. The cubic effect of PTA for SCS (regression coefficients and standard errors not shown) was a significant predictor of ISC for

Table 5. Linear regression of measures of severity and duration of the first clinical mastitis episode during first and second lactation on sire PTA for SCS by organism group.

Severity and duration measure[4]	Lactation	All organisms		CNS[1]		Coliform species		SNA[2]		Environmental organisms[3]	
		b-value[5]	SE	b-value	SE	b-value	SE	b-value	SE	b-value	SE
ISC	1	0.196	0.171	0.177	0.386	0.818†	0.417	0.447	0.330	0.502*	0.251
	2	–0.0707	0.264			0.639	0.713	–0.537	0.456	–0.208	0.396
MSC	1	0.139	0.179	–0.0759	0.427	0.995*	0.437	0.306	0.346	0.454†	0.266
	2	–0.00710	0.273			0.716	0.731	–0.285	0.475	0.00410	0.392
LOGSUM	1	–0.0306	0.192	–0.521	0.410	0.711	0.456	0.0306	0.383	0.131	0.288
	2	–0.329	0.261			–0.111	0.677	–0.468	0.460	–0.290	0.364
LOGDAYS	1	–0.0535	0.171	–0.568	0.371	0.451	0.401	–0.0365	0.351	0.0177	0.260
	2	–0.304	0.233			–0.265	0.592	–0.375	0.413	–0.249	0.324

[1]CNS = Coagulase-negative staphylococci.

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

[3]Environmental organisms = coliform species and streptococci other than *Streptococcus agalactiae*.

[4]ISC = Initial severity code, LOGDAYS = natural logarithm of the total days severity codes were above normal in the 30 d after detection, LOGSUM = natural logarithm of the sum of severity codes that were above normal in the 30 d after detection, MSC = maximum severity code in the 30 d following detection.

[5]b-value = Regression coefficient.

†$P \leq 0.10$.

*$P \leq 0.05$.

Table 6. Quadratic regression of measures of severity and duration of the first clinical mastitis episode during first and second lactation on sire transmitting abilities by organism group.[1]

Transmitting abilities	Linear[2]	Quadratic[3]	Organism group[4]	Severity and duration measure[5]	Lactation
SCS	17.3†	–2.64†	SNA	LOGSUM	1
Productive life	0.0718	–0.0731†	Coliform species	LOGSUM	1
	0.0540	–0.0678*	Coliform species	LOGDAYS	1
Udder depth	–0.0272	–0.0497*	Coliform species	LOGDAYS	1
Front teat placement	0.0729†	–0.0459†	All organisms	LOGSUM	2
Teat length	–0.0996*	0.0438*	SNA	ISC	1
	–0.101*	0.0449*	SNA	MSC	1

[1]Results shown for only those sire transmitting abilities that were significant ($P \leq 0.10$) predictors. The quadratic effect of each transmitting ability was tested for significance the same number of times (36) as presented in Table 5 for PTA for SCS.

[2]Linear = Regression coefficient for the linear effect.

[3]Quadratic = Regression coefficient for the quadratic effect.

[4]CNS = Coagulase-negative staphylococci, environmental organisms = coliform species and streptococci other than *Streptococcus agalactiae*, SNA = Streptococci other than *Streptococcus agalactiae*.

[5]ISC = Initial severity code, LOGDAYS = natural logarithm of the total days severity codes were above normal in the 30 d after detection, LOGSUM = natural logarithm of the sum of severity codes that were above normal in the 30 d after detection, MSC = maximum severity code in the 30 d after detection.

†$P \leq 0.10$.

*$P \leq 0.05$.

Table 7. Linear regression of measures of severity and duration of the first clinical mastitis episode during first and second lactations on sire transmitting abilities by organism group.[1]

Transmitting abilities	b-value[2]	SE	Organism group[3]	Severity and duration measure[4]	Lactation
Protein	–0.0320†	0.0179	Coliform species	LOGDAYS	2
Fore udder attachment	–0.105*	0.0486	All organisms	ISC	2
	–0.225†	0.133	Coliform species	ISC	2
	0.0999†	0.0512	SNA	MSC	1
	–0.122*	0.0499	All organisms	MSC	2
	–0.128†	0.0763	Environmental organisms	MSC	2
	0.100†	0.0574	CNS	LOGDAYS	1
Udder depth	0.0989*	0.0404	SNA	MSC	1
	–0.0721†	0.0425	All organisms	MSC	2
	0.104†	0.0581	CNS	LOGSUM	1
	0.102†	0.0526	CNS	LOGDAYS	1
Front teat placement	0.0984*	0.0482	SNA	MSC	1
	0.143†	0.0718	Coliform species	LOGSUM	1
	0.0862*	0.0420	Environmental organisms	LOGSUM	1
	0.128*	0.0621	Coliform species	LOGDAYS	1
	0.0705†	0.0381	Environmental organisms	LOGDAYS	1
Teat length	–0.145†	0.0803	Coliform species	MSC	1

[1]Results shown for only those sire transmitting abilities that were significant ($P \leq 0.10$) predictors. Transmitting abilities for traits other than SCS were each tested for significance the same number of times (36) as presented in Table 5 for PTA for SCS.

[2]b-value = Regression coefficient.

[3]CNS = Coagulase-negative staphylococci, environmental organisms = coliform species and streptococci other than *Streptococcus agalactiae*, SNA = Streptococci other than *Streptococcus agalactiae*.

[4]ISC = Initial severity code, LOGDAYS = natural logarithm of the total days severity codes were above normal in the 30 d following detection, LOGSUM = natural logarithm of the sum of severity codes that were above normal in the 30 d following detection, MSC = maximum severity code in the 30 d following detection.

†$P \leq 0.10$.

*$P \leq 0.05$.

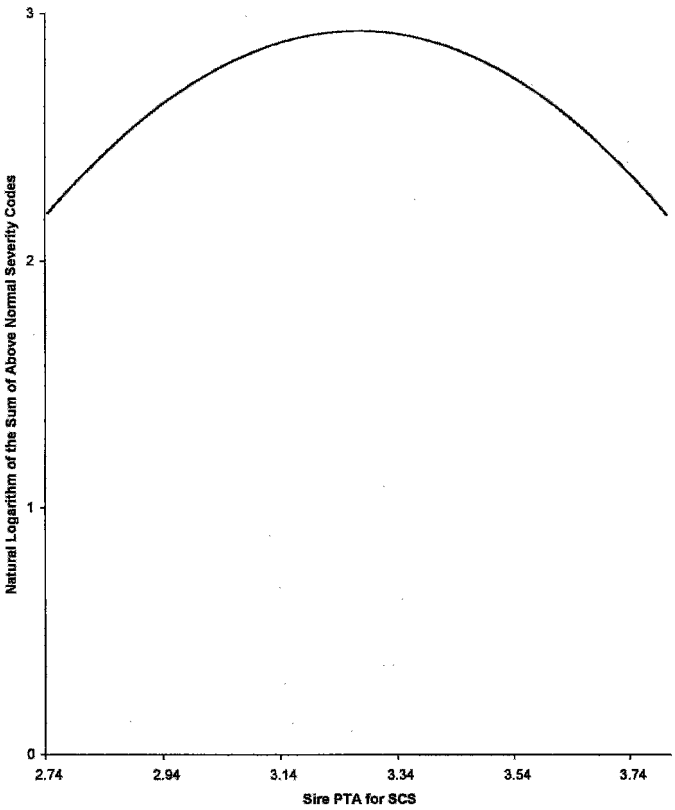

Figure 1. Quadratic regression of the natural logarithm of the sum of severity codes that were above normal (> 1) in the 30 d after detection of the first clinical episode caused by streptococci other than *Streptococcus agalactiae* during first lactation on sire PTA for SCS.

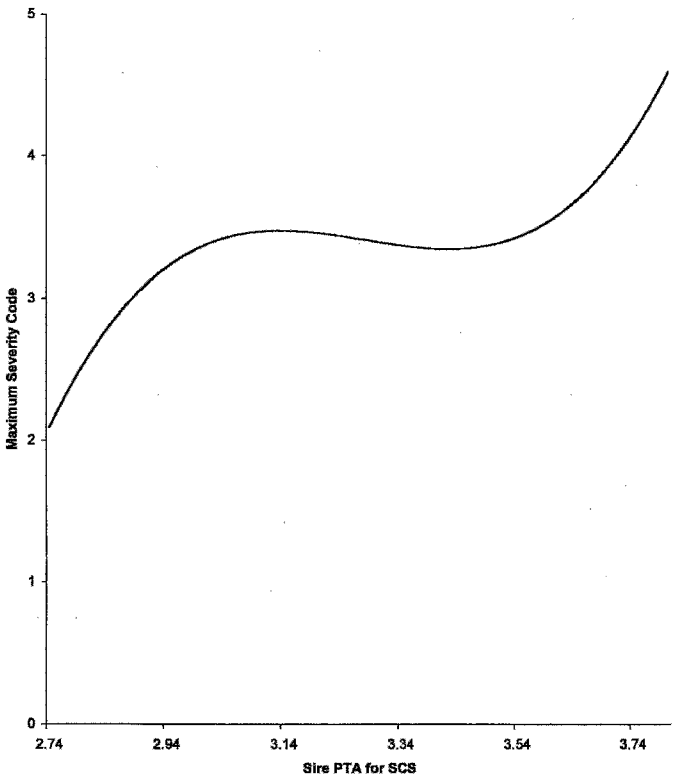

Figure 2. Cubic regression of the maximum severity code in the 30 d after detection of the first clinical episode caused by the most common environmental organisms during first lactation on sire PTA for SCS.

clinical episodes from all organisms, CNS, and the most common environmental organisms during first lactation. In addition, the cubic effect was a significant predictor of MSC for clinical episodes from all organisms, SNA, and the most common environmental organisms during first lactation. The cubic effect of PTA for SCS was also a significant predictor of LOGSUM for clinical episodes from SNA and the most common environmental organisms during first lactation. Finally, the cubic effect of PTA for SCS was a significant predictor of LOGDAYS for clinical episodes from the most common environmental organisms during first lactation. Figure 2 presents the cubic regression line for MSC for clinical episodes from the most common environmental organisms during first lactation. As shown in Figure 2, the cubic regression line was sigmoid, with the nadir occurring at the lowest PTA for SCS. The cubic regression lines for all other measures of severity and duration were similarly shaped. The shape of the quadratic and cubic regression lines indicates that daughters of sires that transmit the lowest SCS had the lowest ISC, MSC, LOGSUM, and LOGDAYS for clinical episodes during first lactation.

These findings 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 more severe, longer lasting clinical episodes, and an intermediate SCS would provide optimal resistance to mastitis. This theory stems from the results of experimental challenge studies that indicated that elevated SCC before infusion protects against infection by mastitis-causing organisms (Kehrli and Shuster, 1994; Schukken et al., 1994).

Results of studies that examined the association between the occurrence of mastitis and SCS also refute the theory that selection for the lowest SCS will result in dairy cattle that are unable to respond to infection. Philipsson et al. (1995) regressed genetic evaluations for the occurrence of clinical mastitis on SCS evaluations and found no evidence of a nonlinear effect. Rogers et al. (1998) detected a quadratic effect, which indicated that sires with the lowest genetic evaluations for SCS also had the most favorable evaluations for the occurrence of clinical mastitis. Research conducted on a population that included the cows used in the current study

concluded that daughters of sires that transmit the lowest SCS had the least IMI at first parturition and the lowest number of clinical episodes per lactation (Nash, 1999; Nash et al., 2000).

Elevated SCC due to mastitis from environmental organisms may not be detected by monthly SCC measurement (the current practice in the US) because mastitis from these organisms is generally of shorter duration than mastitis from contagious organisms (National Mastitis Council, 1996). Therefore, it has been hypothesized that selection for lower SCS may not improve resistance to mastitis from environmental organisms (Shook, 1993). In the current study though, daughters of sires that transmit higher SCS had higher ISC, MSC, LOGSUM, and LOGDAYS for clinical episodes from environmental organisms during first lactation. These results indicate that selection for lower SCS may lessen the severity and shorten the duration of clinical mastitis caused by environmental organisms during first lactation. Research conducted on a population which included some of the cows used in the current study suggests that selection for lower SCS may also reduce the incidence of both IMI at first parturition and clinical mastitis caused by environmental organisms (Nash, 1999; Nash et al., 2000).

The effect of selection for lower SCS on the severity and duration 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. Furthermore, the severity and duration of clinical mastitis from environmental organisms (as measured here) may have been more variable than the severity and duration of clinical mastitis from other organisms. If this were the case, the severity and duration of clinical mastitis from environmental organisms may have been more likely to be associated with sire transmitting abilities for SCS.

It is hypothesized that daughters of sires that transmit higher SCS may have more severe, longer lasting clinical episodes from environmental organisms because their immune systems are dysfunctional and therefore incapable of preventing mastitis-causing organisms from surviving or multiplying. Daughters of sires that transmit higher SCS may also have more severe, longer lasting clinical episodes from environmental organisms because their udder conformation increases exposure to mastitis causing organisms or fails to limit the number that gain entry to the mammary gland.

STA for udder type traits. The linear effect of the STA for fore udder attachment was a significant ($P \leq 0.10$) predictor of ISC for clinical episodes from all organisms and coliform species during second lactation (Table 7). The significant regression coefficients were negative, indicating that daughters of sires that transmit strongly attached fore udders had lower ISC for clinical episodes from all organisms and coliform species during second lactation.

The linear effect of the STA for fore udder attachment was also a significant predictor of MSC for clinical episodes from SNA during first lactation, all organisms during second lactation, and the most common environmental organisms during second lactation (Table 7). The significant regression coefficients were positive when MSC for clinical episodes during first lactation were considered, and negative when MSC for clinical episodes during second lactation were considered. These results indicate that daughters of sires that transmit strongly attached fore udders had higher MSC for clinical episodes from SNA during first lactation and lower MSC for clinical episodes from all organisms and the most common environmental organisms during second lactation.

The linear effect of the STA for fore udder attachment was also a significant predictor of LOGDAYS for clinical episodes from CNS during first lactation (Table 7). The regression coefficient was positive, indicating that daughters of sires that transmit strongly attached fore udders had higher LOGDAYS for clinical episodes from CNS during first lactation.

The linear effect of the STA for udder depth was a significant predictor of MSC for clinical episodes from SNA during first lactation and all organisms during second lactation (Table 7). The significant regression coefficients were positive when MSC for clinical episodes during first lactation were considered, and negative when MSC for clinical episodes during second lactation were considered. These results indicate that daughters of sires that transmit shallower udders had higher MSC for clinical episodes from SNA during first lactation and lower MSC for clinical episodes from all organisms during second lactation.

The linear effect of the STA for udder depth was also a significant predictor of LOGSUM and LOGDAYS for clinical episodes from CNS during first lactation (Table 7). The significant regression coefficients were positive, indicating that daughters of sires that transmit shallower udders had higher LOGSUM and LOGDAYS for clinical episodes from CNS during first lactation.

The quadratic effect of the STA for udder depth was a significant predictor of LOGDAYS for clinical episodes from coliform species during first lactation (Table 6). The regression coefficient was negative, indicating that LOGDAYS for clinical episodes from coliform species decreased at an increasing rate during first lactation among daughters of sires that transmit shallower udders.

The linear effect of the STA for front teat placement was a significant predictor of MSC for clinical episodes from SNA during first lactation (Table 7). The regression coefficient was positive, indicating that daughters of sires that transmit closely spaced front teats had higher MSC for clinical episodes from SNA during first lactation.

The linear effect of the STA for front teat placement was also a significant predictor of LOGSUM and LOGDAYS for clinical episodes from coliform species and the most common environmental organisms during first lactation (Table 7). In addition, the quadratic effect of the STA for front teat placement was a significant predictor of LOGSUM for clinical episodes from all organisms during second lactation (Table 6). The significant regression coefficients for the linear effect were positive, indicating that daughters of sires that transmit closely spaced front teats had higher LOGSUM and LOGDAYS for clinical episodes from coliform species and the most common environmental organisms during first lactation. The quadratic regression line indicated that LOGSUM for clinical episodes during second lactation increased at a decreasing rate among daughters of sires that transmit closely spaced front teats.

The linear effect of the STA for teat length was a significant predictor of MSC for clinical episodes from coliform species during first lactation (Table 7). In addition, the quadratic effect of the STA for teat length was a significant predictor of ISC and MSC for clinical episodes from SNA during first lactation (Table 6). The regression coefficient for the linear effect was negative, indicating that daughters of sires that transmit longer teats had lower MSC for clinical episodes from coliform species during first lactation. The quadratic regression lines indicated that ISC and MSC for clinical episodes from SNA during first lactation increased at an increasing rate among daughters of sires that transmit shorter teats.

In general, variations in the severity and duration of daughter clinical mastitis were not consistently associated with variation in sire transmitting abilities for udder type traits. However, strongly attached fore udders, shallower udders, closely spaced front teats, and shorter teats have been associated with lower SCS and reduced incidence of clinical mastitis (Seykora and McDaniel, 1986; Schutz et al., 1993; Lund et al., 1994; Rogers et al., 1998; Nash et al., 2000). These findings indicate that variation in udder conformation is associated with variation in exposure to mastitis causing organisms and their entry into the mammary gland, rather than variation in severity and duration of clinical episodes.

PTA for PL. The linear effect of PTA for PL was not a significant ($P > 0.10$) predictor of ISC, MSC, LOGSUM, or LOGDAYS for clinical episodes during first or second lactation. However, the quadratic effect of PTA for PL was a significant predictor of LOGSUM and LOGDAYS for clinical episodes from coliform species during first lactation (Table 6). The significant regression coefficients for the quadratic effect were negative, indicating that LOGSUM and LOGDAYS for clinical episodes from coliform species decreased at a increasing rate during first lactation among daughters of sires that transmit longer PL. These results indicate that daughters of sires that transmit long PL had lower LOGSUM and LOGDAYS for clinical episodes from coliform species during first lactation. Lower SCS, decreased clinical mastitis, and less IMI at first parturition have also been associated with longer PL (Weigel et al., 1997; Rogers et al., 1998; Nash, 1999; Nash et al., 2000).

PTA for protein yield. The linear effect of the PTA for protein was a significant ($P \leq 0.10$) predictor of LOGDAYS for clinical episodes from coliform species during second lactation (Table 7). The regression coefficient was negative, indicating that daughters of sires that transmit higher protein yield had lower LOGDAYS for clinical episodes from coliform species during second lactation. However, lower, not higher, yield has been associated with reduced incidence of clinical mastitis and lower SCS (Schmidt and Van Vleck, 1965; Wilton et al., 1972; Emanuelson et al., 1988; Weller et al., 1992; Rogers et al., 1998).

CONCLUSIONS

Daughters of sires that transmit higher SCS had more severe, longer lasting clinical episodes from environmental organisms during first lactation (only the first clinical episode during first and second lactations were considered in this study). In addition, the severity and duration of clinical episodes caused by environmental organisms during first lactation were nonlinearly associated with PTA for SCS. However, daughters of sires that transmit the lowest SCS had the least severe, shortest lasting clinical episodes from environmental organisms during first lactation. Therefore, selection for lower SCS may reduce the severity and duration of clinical episodes caused by environmental organisms during first lactation without diminishing the ability to respond to infection.

Variation in the severity and duration of daughter clinical episodes were not consistently associated with variation in sire transmitting abilities for udder type traits. However, daughters of sires that transmit longer productive life had less severe, shorter lasting clinical episodes from coliform species during first lactation. This indicates that selection for longer productive life

may reduce the severity and duration of clinical episodes from coliform species during first lactation.

The number of significant ($P \leq 0.10$) regressions of measures of severity and duration on sire transmitting abilities were low (e.g., 4 of 36 linear regressions on PTA for SCS were significant). Therefore, these results should be interpreted with caution.

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