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Genetic Correlations Among Somatic Cell Scores, Productive Life, and Type Traits from the United States and Udder Health Measures from Denmark and Sweden

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ABSTRACT

Sire genetic evaluations for protein yield, somatic cell score (SCS), productive life, and udder type traits from the US were correlated with sire evaluations for udder health from Denmark and Sweden and then the correlations were adjusted for accuracies to approximate genetic correlations. Traits from Denmark and Sweden included somatic cell count (SCC) and clinical mastitis from single-trait analyses. In addition, evaluations for clinical mastitis from Denmark and Sweden were regressed on US traits to test for quadratic relationships. Information from 85 bulls with US and Danish evaluations (77 with US type) and from 80 bulls with US and Swedish evaluations (79 with US type) was used to calculate correlations. Genetic correlations of US protein yield with Danish and Swedish SCC and clinical mastitis were all unfavorable (–0.09 to –0.32). Genetic correlations of US productive life with Danish and Swedish SCC and clinical mastitis were all favorable (0.06 to 0.59). Genetic correlations between US SCS and Danish SCC and between US SCS and Swedish SCC were –0.87 and –0.99, respectively (favorable). Genetic correlations between US SCS and Danish clinical mastitis and between US SCS and Swedish clinical mastitis were –0.66 and –0.49, respectively (favorable). The US type traits that had the largest correlations with clinical mastitis from Denmark and Sweden, respectively, were udder composite (0.26, 0.47), udder depth (0.45, 0.52), and fore udder attachment (0.31, 0.34). In general, quadratic regressions indicated little nonlinearity between clinical mastitis and the US traits. Specifically, the US bulls with the lowest predicted transmitting abilities for SCS had the most favorable rates of daughter clinical mastitis in Denmark and Sweden. Selection for increased productive life, lower SCS, and more shallow udders should improve mastitis resistance.

(**Key words**: mastitis, somatic cell score, type traits, longevity)

Abbreviation key: **PL** = productive life.

INTRODUCTION

Intense selection of dairy cattle for yield is clearly justified, and breeding programs around the world have been successful at improving yield. However, improvement in udder health through selection has been moderate (in Scandinavia) or almost nonexistent (North America) because of the major emphasis on yield traits. Countries such as Norway and Sweden place considerable emphasis on direct selection for mastitis resistance, but many other countries, including the US, use only indirect measures of udder health and place much less emphasis on these indirect measures than on yield traits (3).

Udder diseases can have large direct costs and can ultimately lead to involuntary culling. Heritabilities for clinical mastitis in most dairy cattle populations—when recorded accurately, consistently, and completely—are probably 0.10 or higher (4, 5). However, in most countries, the lack of accurate and standardized recording prohibits direct selection for mastitis resistance (3). Udder type traits, teat structure, and SCC or SCS (SCC converted to logarithmic form) have been identified as traits that are potentially useful for indirect selection to improve udder health (2, 6, 7, 8, 9, 10, 11, 12, 13).

Genetic relationships among SCS, udder traits, and mastitis have an impact on the value of these

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traits for breeding programs. To utilize these traits properly for breeding programs, detailed information is needed about their interrelationships. In addition, these genetic relationships are needed to facilitate international genetic evaluations. Multiple-trait methods for genetic evaluation require knowledge of these genetic correlations. In the future, multiple-trait evaluations across multiple countries might be feasible; then, sire evaluations for mastitis would be available on all bulls even though direct recording of mastitis takes place in only a few countries. For example, clinical mastitis, SCC, and possibly some udder type traits from Scandinavian countries could be combined with SCC and some udder type traits from other countries to obtain evaluations for clinical mastitis that include multiple traits from multiple countries. The major benefit of this approach would be the availability of genetic evaluations for clinical mastitis (from correlated traits and direct information on relatives) on newly evaluated bulls in countries where clinical mastitis is not recorded. Genetic correlations among SCC, udder type traits, and mastitis would be required to calculate the most accurate sire evaluations possible from the most commonly recorded data.

The objectives of this paper are to estimate genetic relationships between SCS, productive life (**PL**), and type traits from the US and udder health measures from Denmark and Sweden. The objectives include the exploration of possible quadratic genetic relationships between clinical mastitis and potential indicator traits measured in the US.

MATERIALS AND METHODS

Official sire evaluations from the US (July 1995) for production traits, PL, SCS, and type traits (from USDA-DHIA and Holstein Association) were used in estimating the genetic correlations. Details of the US genetic evaluations can be found elsewhere (14, 15, 17). Heritabilities for US SCS, PL, and production traits were 0.10, 0.085, and 0.25, respectively. All traits are summarized using animal models.

The official sire evaluations from Denmark and from Sweden that were used in the analyses were also from July 1995. Unofficial evaluations for clinical mastitis and SCC from Denmark were calculated from the exact data used to calculate the official sire evaluations for Denmark; however, a single-trait model was used that included only clinical mastitis data (or SCC data in the case of SCC evaluations). Official sire evaluations from Denmark are calculated from a multiple-trait model that includes SCC and clinical mastitis. The SCC data are included to increase the accuracy of the sire evaluations for clinical mastitis. Denmark uses a sire model with relationships through sire paths. To construct the relationship matrix, a minimum of three generations of sires was utilized. Denmark assumes heritabilities for clinical mastitis and SCC of 0.04 and 0.11, respectively. For the official multiple-trait model, the genetic correlation between clinical mastitis and SCC was 0.63. Mastitis is recorded between 10 d before calving and 180 d after calving for the first lactation in Denmark. The records are a combination of data collected through the milk recording system and through veterinarians. Mastitis is defined as a binomial trait (0 or 1); no occurrence is represented by 0, and occurrence of one or more cases during this period is represented by 1. Somatic cell counts are collected during this same period through the milk recording system (3).

Genetic evaluations from Sweden are from single-trait analyses using a sire model with relationships calculated using sire paths. Heritabilities assumed for clinical mastitis and SCC in Sweden are 0.02 and 0.08, respectively. Occurrence of mastitis in Sweden, which is used in the genetic evaluations, is recorded between 10 d before calving and 150 d after calving during the first lactation. Records are from veterinary reports of clinical treatments and culling reported specifically for mastitis during this 160-d period. Culling records are handled through the milk recording program. Mastitis in Sweden is also defined as a binomial trait for analysis. Treatments scored 0 are in the untreated category, and one or more treatments are combined into the treated category (for purposes of analyses, culling specifically for mastitis with no veterinarian treatment recorded during this period is handled as a veterinary treated mastitis event). Somatic cell counts are collected for the entire first lactation through the milk recording system. The selection index for mastitis and SCC from Sweden is described by Philipsson et al. (6). Further details of the genetic evaluation procedures and data collection have been researched (3, 6).

Genetic evaluations for sires from the US were merged with genetic evaluations for sires from Denmark to establish a file that included bulls with evaluations and daughters in both countries. An international cross-reference file established by the INTERBULL Centre (Uppsala, Sweden) was used to facilitate file merging. The same procedure was used to establish a file that included bulls with evaluations and daughters in Sweden and the US. Genetic correlations, which should be interpreted as approximate genetic correlations, were estimated by adjustment of product-moment correlations among sire evaluations

for reliabilities (1). The correlation between daughter deviations on two traits, when one trait is measured on one group of daughters and the second trait is measured on a second group of daughters sired by the same bulls, is an estimate of the genetic correlation. The method of Calo et al. (1) utilizes this concept and involves adjusting correlations among genetic evaluations based on independent sources of data. This adjustment eliminates the impact of regressing genetic evaluations toward the mean when the amount of information in the deviations is insufficient to provide accuracy approaching 1.0. Adjustment of the correlation between genetic evaluations for reliabilities attempts to undo the impact of regressing genetic evaluations toward the mean. After adjustment for birth year of the bull to eliminate the effect of genetic trend on correlation estimates, residual correlations were also calculated but are not reported because they were similar to the product-moment correlations. Also, residual correlations among traits after adjustment for PTA milk yield, PTA SCS, or both were also calculated but are not presented. However, some residual correlations are discussed. In addition, clinical mastitis evaluations (the single-trait evaluations) from Denmark and Sweden were regressed in separate models on US traits to test for linear and quadratic relationships.

Genetic evaluations from the US and from Denmark or Sweden are from independent daughter groups; thus, only genetic covariance should be responsible for the correlations among progeny group performance. For the US-Denmark matching file, edits were made to include only sires with 50 daughter equivalents in Denmark and reliability for SCS from the US of 0.60 or greater or, in the case of matches with type, reliability for linear type of 0.70 or greater. For the US-Sweden matching file, edits were made to include only sires with 50 daughter equivalents in Sweden and reliability for SCS from the US of 0.60 or greater or, in the case of matches with type, reliability for linear type of 0.70 or greater. Genetic correlations calculated from evaluations on sires with 125 daughter equivalents or more in Denmark or Sweden were made but not reported because those genetic correlations were similar to those calculated from evaluations on sires with 50 daughter equivalents or more. One undesirable characteristic of the estimation procedure is the potential to obtain estimates outside the parameter space because of the adjustment for reliabilities, which can accentuate sampling effects. This problem diminishes as reliabilities of the evaluations increase. At the limit for reliabilities, the correlations among genetic evaluations represent the estimated genetic correlation. Confidence in the estimated genetic correlations increases if the estimated genetic correlations are similar when various edits are utilized. The undesirable characteristics of the method utilized here are at least partially offset by the flexibility of the method, which allows one to approximate partial genetic correlations (genetic correlations after removal of the genetic contribution of another trait).

RESULTS AND DISCUSSION

Means, standard deviations, and descriptions for the sire evaluations used in the study are given in Table 1. For the Danish and Swedish traits, higher sire evaluations are more desirable. Mean reliabilities for the US traits were all 0.95 or above. Mean reliabilities for the Danish evaluations were from 0.72 to 0.88, depending on the trait and data subset (match with US data for yield or type). Mean reliabilities for the Swedish evaluations were 0.52 for mastitis and 0.77 for SCC. Adjustments to product-moment correlations are dependent on these reliabilities. In the US-Denmark file, genetic correlations were calculated by dividing the product-moment correlations among sire evaluations by a factor that ranged from 0.93 to 0.83, depending on the pair of traits involved. In the US-Sweden file, genetic correlations were calculated by dividing the product-moment correlations among sire evaluations by a factor that ranged from 0.87 to 0.71, depending on the pair of traits involved. In all cases, the adjustment was primarily a reflection of the mean reliabilities of the Danish and Swedish evaluations because the mean reliabilities for all US evaluations were high.

It is difficult to know whether genetic correlations calculated primarily from information on daughters of selected bulls are representative of the true genetic correlations within the population. However, the genetic correlations calculated by using information from daughters of selected bulls are likely representative of the genetic correlations for the contemporary breeding population and the genes segregating in the contemporary breeding population. It should be noted that genetic correlations used to calculate international evaluations and international conversions come primarily from information on selected bulls and their close relatives (essentially the same subset or type of subset of sires that was used in this study). As a reference point and to help understand the bulls represented in these data, the July 1996 US average PTA for 584 active US AI Holstein bulls for protein yield, PL, and SCS were 20 kg, 1.4 mo, and 3.18 SCS, respectively. Standard deviations for the PTA on these same active AI bulls for protein yield, PL, and

SCS were 6.8 kg, 0.9 mo, and 0.15 SCS, respectively. When compared with the current active AI population, the subset of bulls used in this study had larger standard deviations for their PTA (from higher reliabilities because they were older bulls with more daughters) and slightly smaller PTA for protein yield and PL. Means for PTA SCS were nearly identical between the subset of bulls in this study and the active population in the US during a similar time period. The subset of bulls in this study were somewhat less selected for protein yield and PL than were more recent active AI bulls in the US. Mean genetic evaluations for SCC and clinical mastitis from Denmark (Table 1) were similar to the genetic base used in Denmark, which is bulls born 6 to 7 yr prior to the evaluation. Mean genetic evaluations for SCC and clinical mastitis from Sweden (Table 1) were similar to the Swedish genetic base, which is the average of tested bulls born in the 3 yr prior to the evaluation (the mean of the base population is set at 100).

Genetic correlations among SCS, PL, and protein yield from the US and udder health measures from Denmark and Sweden are given in Table 2. Genetic correlations between protein yield and the udder health measures from Denmark and Sweden were unfavorable and ranged from –0.09 to –0.28. Correlations with milk and fat yield are not reported but were similar to the correlations with protein yield. Results are in agreement with most other studies of Holsteins using within-country data (2, 11). Genetic correlations between PL and udder health measures were favorable, especially between PL and clinical mastitis. The correlation between PL and clinical mastitis in Sweden (0.59) was higher than between PL and clinical mastitis in Denmark (0.28). The differing results could be due to sampling. Of the 80 and 85 bulls in the US-Denmark and US-Sweden files, only 37 were common to both the US-Denmark and US-Sweden files. Note that PL evaluations from

TABLE 1. Means, standard deviations, and descriptions for genetic evaluations for US, Danish, and Swedish sires.[1]

Traits	$\bar{X}$	SD	Higher values for the trait correspond to
US			
Protein yield, kg	3.61	10.3	Higher yield
Productive life, mo	0.62	1.41	Longer life
SCS, $\log_2$	3.18	0.171	Higher SCS
Final score	0.078	0.905	Higher final scores
Udder composite[2]	–0.087	0.917	Higher composite scores
Udder cleft[2]	0.099	1.08	Deeper cleft
Rear udder height[2]	0.218	1.19	Higher attachment
Rear udder width[2]	0.343	1.10	Wider attachment
Udder depth[2]	–0.536	1.33	Higher udder
Fore udder attachment[2]	–0.248	1.18	Tighter attachment
Teat placement[2]	0.173	1.24	Closer teats
Teat length[2]	–0.042	1.17	Longer teats
Danish[3]			
SCC (Single-trait analysis), $\log_{10}$	–0.026	0.110	Lower SCC
Clinical mastitis (single-trait analysis)[4]	–0.019	0.030	Lower rates of clinical mastitis
Clinical mastitis (multiple-trait analysis of SCC and clinical mastitis)[4]	–0.020	0.031	Lower rates of clinical mastitis
Swedish			
SCC (Single-trait analysis)[2]	99.5	6.85	Lower SCC
Clinical mastitis (single-trait analysis)[2]	100.3	6.11	Lower rates of clinical mastitis
Index for mastitis[5]	99.8	6.37	Lower rates of clinical mastitis

[1]Data on US evaluations on protein, productive life, and SCS and Danish evaluations on udder health are from 85 sires. Data on US type and Swedish evaluations on udder health are from 79 sires.

[2]Standardized.

[3]Danish evaluations are not standardized, but they are routinely standardized before official publication in Denmark.

[4]Binomial trait scored 0 or 1.

[5]Index of two single traits, SCC and clinical mastitis.

TABLE 2. Approximate genetic correlations (correlations among sire genetic evaluations adjusted for reliabilities) among SCS, productive life, and protein yield from the US and udder health measures from Denmark and Sweden.[1]

	Denmark			Sweden		
US Trait	SCC[2]	Clinical mastitis	Multiple-trait mastitis	SCC	Clinical mastitis	Index for mastitis
Protein yield	–0.18	–0.28*	–0.28*	–0.32*	–0.09	–0.20
Productive life	0.06	0.28*	0.26*	0.30*	0.59*	0.65*
SCS	–0.87*	–0.66*	–0.75*	–0.99*	–0.49*	–0.87*

[1]Correlations are based on 85 bulls with US and Danish genetic evaluations and 80 bulls with US and Swedish genetic evaluations. Edits were made to include only bulls with approximate minimum of 50 daughter equivalents in SCC and clinical mastitis evaluations and reliabilities for US SCS of 0.60 or greater.

[2]Both SCC and clinical mastitis are from single-trait analyses. Multiple-trait mastitis is from a multiple-trait analysis including clinical mastitis and SCC, and selection index for mastitis is from single-trait evaluation of clinical mastitis and single-trait evaluation for SCC. Because of scaling, higher values are desirable for the Danish and Swedish traits.

*Correlations among sire evaluations were different from 0 ($P < 0.05$).

the US had high reliabilities (mean >0.95); thus, essentially all of the information in the PL evaluations would have been direct information on daughter PL (PL evaluations included would have little influence from the correlated traits used in calculating PL evaluations). Correlations between PL on bulls with early first-crop progeny and mastitis might not be as favorable as those reported here because PL evaluations on bulls with early first-crop progeny were affected by traits other than the actual PL of relatives (16, 17). The genetic correlation between US PL and US SCS is probably near –0.25 (16). The correlation between US PL and US SCS in the largest subset of these data was –0.30.

Genetic correlations between clinical mastitis in Denmark and US PL and between clinical mastitis in Sweden and US PL that were calculated from residual correlations after adjustment for PTA milk yield were 0.39 and 0.68, respectively. These genetic correlations were higher than the genetic correlations calculated from the product-moment correlations unadjusted for PTA milk yield (0.28 and 0.59, respectively). The component of PL that does not reflect the influence of milk yield has a higher correlation with clinical mastitis than does unadjusted PL.

The quadratic regressions of clinical mastitis from Denmark and from Sweden on US PL were not significant ($P > 0.10$), indicating that the genetic relationship between clinical mastitis and PL is linear (linear regressions were significant at $P < 0.05$). The linear regression of Danish clinical mastitis on US PL had an intercept of –0.0226 and a linear coefficient of 0.00493 (SE = 0.00230). The linear regression of Swedish clinical mastitis on US PL had an intercept of 99.8 and a linear coefficient of 1.75 (SE = 0.430).

Genetic correlations between SCC from Denmark and SCS from the US and between SCC from Sweden and SCS from the US were –0.87 and –0.99, respectively. The negative sign reflects the scaling of Danish and Swedish sire evaluations (higher values represent lower SCC). Clearly, genetic correlations between US SCS and SCC in Denmark and Sweden are very large, indicating that the measures are genetically similar traits.

Genetic correlations between clinical mastitis from Denmark and SCS from the US and between clinical mastitis from Sweden and SCS from the US were –0.66 and –0.49, respectively. These estimates are within the range reported in the literature (2, 6, 18). The correlation between US SCS and Danish clinical mastitis is in close agreement with the within-Denmark estimates between these two traits (3). The genetic correlation between the US SCS and clinical mastitis from Sweden is lower than the estimate within Sweden of 0.70 (6). The correlation is also lower than the estimate between the US and Denmark, which could partly be due to sampling. The correlation between Swedish sire evaluations for SCC and clinical mastitis in the matching 80 bulls was 0.40, which is slightly lower than expected based on the reliability of the proofs and the estimated genetic correlation within the population. The results could also indicate that the true genetic correlation is lower between the US and Sweden than between the US and Denmark. Clinical mastitis in Denmark and Sweden may be slightly different traits because of slightly different management conditions and the manner in which clinical mastitis is recorded and

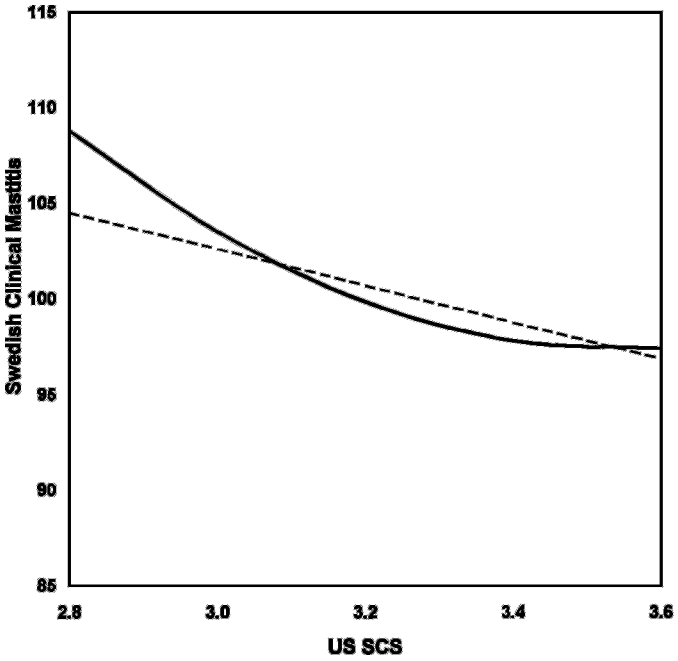

Figure 1. Regression of Swedish sire evaluations for clinical mastitis on US sire evaluations for SCS. Lines represent the linear (. . .) and quadratic (———) regressions and are based on 80 bulls with Swedish and US genetic evaluations. Edits were made to include only bulls with a minimum of approximately 50 daughter equivalents in Swedish clinical mastitis evaluations and reliabilities for US SCS of 0.60 or greater. Swedish sire evaluations are standardized to a mean of 100 and a standard deviation of approximately 5. The range for these 80 bulls was from 81 to 113. The US PTA for SCS are on a $\log_2$ scale and range from 2.78 to 4.07 for these 80 bulls.

summarized in each country. The frequency in first lactation of recorded clinical mastitis (trait scored 0 or 1; 1 represents 1 or more episodes) differs between Denmark and Sweden by approximately 15%. The genetic correlation between Danish clinical mastitis and Swedish Clinical mastitis calculated from the 37 bulls with evaluations in both countries was 0.72.

The quadratic regression of clinical mastitis from Denmark on US SCS was not significant ($P > 0.10$), indicating that the genetic relationship between clinical mastitis from Denmark and SCS is linear (linear regression was significant at $P < 0.01$). The linear regression of clinical mastitis from Denmark on US SCS has an intercept of 0.289 and a linear coefficient of –0.0972 (SE = 0.0163). The quadratic regression of clinical mastitis from Sweden on US SCS was significant ($P < 0.05$). The linear and quadratic equations are plotted in Figure 1. The linear equation has an intercept of 131.3 and a linear coefficient of –9.581 (SE = 2.95); the quadratic equation has an intercept of 353.5, a linear coefficient of –144.3 (SE = 51.0), and a quadratic coefficient of 20.32 (SE = 7.68). The quadratic relationship could be because of sampling or could be an indication of a true nonlinear relationship. The quadratic relationship contrasts with the results of Philipsson et al. (6) who found no significant quadratic relationship between clinical mastitis and SCC in Swedish data. The shape of the curve in Figure 1 indicates that the linear component of the relationship between clinical mastitis and SCS is most apparent for bulls that have PTA SCS below the mean of 3.18. The horizontal axis in Figure 1 covers only the range in which most of the sires occur. Extrapolation beyond this range should be done cautiously. Bulls with very low PTA for SCS clearly have daughters with a mastitis frequency that is lower than that of daughters of bulls with average or above average PTA for SCS, which is in direct contrast to theories that question the mastitis resistance of daughters of bulls that have very low SCS. Indeed, bulls with daughters that have the lowest SCS are also the bulls with daughters that have the lowest frequency of clinical mastitis in Sweden. No results indicate that any concern is warranted over the use of bulls with low PTA SCS.

The genetic correlation between US SCS and Danish clinical mastitis was lower than the genetic correlation between US SCS and Danish multiple-trait evaluation for mastitis (–0.66 versus –0.75). This result seems reasonable because the genetic correlation between US SCS and Danish SCC was larger than the genetic correlation between US SCS and Danish clinical mastitis. Trends were similar for the US SCS and the Swedish index of SCC and clinical mastitis.

Genetic correlations between selected type traits from the US and the measures of udder health from Denmark and Sweden are presented in Table 3. In general, the results agree with those of other studies that have analyzed these relationships [(9, 11); J. A. Eriksson 1995, personal communication]. However, those previous studies do not include a measure of clinical mastitis (9, 11) and could be affected by environmental correlations [(11); J. A. Eriksson, 1995, personal communication). Genetic correlations between final score and SCC and between final score and clinical mastitis were all positive and ranged from 0.07 to 0.32. These positive correlations were likely due to the impact of udder conformation on final score. Genetic correlations between US udder composite and SCC and between udder composite and clinical mastitis ranged from 0.26 to 0.47. Higher values for udder composite were genetically associated with less mastitis. Udder composite is calcu-

lated by the Holstein Association, USA and is a composite of six linear type traits. The six traits and their relative weights are udder depth, 0.30; fore udder attachment, 0.16; front teat placement, 0.16; rear udder height, 0.16; rear udder width, 0.12; and udder cleft, 0.10. The weights were derived from the relationships among the traits and measures of longevity in the US Holstein population. Selection for higher udder composite should improve mastitis resistance.

The genetic correlations between udder cleft and the Danish measures of udder health were small; however, higher scores for udder cleft tended to be associated with reduced mastitis and SCC in the US-Sweden file. The rear udder traits (height and width) had small correlations with the measures of udder health.

The genetic correlations between teat placement and the measures of udder health were all positive except for between teat placement and clinical mastitis from Denmark. Selection for closer teat placement may have a small desirable impact on udder health. The genetic correlations between teat length and the measures of udder health were all negative but small. These results agree with those of Rogers et al. (9) and suggest a small genetic association between teat length and udder disease (shorter teats may be favorable).

The genetic correlations between udder depth and SCC were moderate and positive (0.37, 0.52). Both magnitude and direction agree closely with previous results [(9, 11); J. A. Eriksson, 1995, personal communication]. Similarly, the genetic correlations between udder depth and clinical mastitis were positive (0.45, 0.52). These genetic correlations indicate that more shallow udders (those higher in relation to the hock) are genetically associated with reduced mastitis. The genetic correlations between udder depth and clinical mastitis approach the size of the correlations between SCS and clinical mastitis. The correlation between udder depth and clinical mastitis in the US-Sweden file is numerically larger than the correlation between SCS and clinical mastitis. The results indicate that udder depth may be useful for selection to improve udder health, especially where clinical mastitis is not routinely recorded. The results support those of Rogers (8), who reported that an index of SCS and some udder traits (especially udder depth) substantially improved selection for mastitis resistance compared with selection using only SCS. The genetic correlations between fore udder attachment and the measures of udder health were, in general, slightly smaller than the genetic correlations between udder depth and the measures of udder health. The similarity is expected because fore udder attachment and udder depth are highly correlated. The associations between some type traits such as fore udder attachment or udder composite and mastitis are likely mediated primarily through udder depth.

Genetic correlations between clinical mastitis in Denmark and udder depth and between clinical mastitis in Sweden and udder depth calculated from residual correlations after adjustment for PTA milk yield were 0.43 and 0.50, respectively. Mastitis, milk

TABLE 3. Approximate genetic correlations (correlations among sire genetic evaluations adjusted for reliabilities) among selected type traits from the US and udder health measures from Denmark and Sweden.[1]

	Denmark			Sweden		
US Trait	SCC[2]	Clinical mastitis	Multiple-trait mastitis	SCC	Clinical mastitis	Index for mastitis
Final score	0.18	0.07	0.09	0.25	0.32*	0.34*
Udder composite	0.34*	0.26	0.31*	0.40*	0.47*	0.46*
Udder cleft	0.09	–0.01	0.01	0.21	0.23	0.29
Rear udder height	0.01	–0.02	–0.02	0.09	0.09	0.13
Rear udder width	0.11	–0.06	–0.05	0.10	–0.07	0.01
Udder depth	0.37*	0.45*	0.49*	0.52*	0.52*	0.63*
Fore udder attachment	0.38*	0.34*	0.39*	0.39*	0.31*	0.41*
Teat placement	0.24	–0.01	0.04	0.18	0.19	0.21
Teat length	–0.05	–0.09	–0.09	–0.02	–0.09	–0.05

[1]Correlations are based on 77 bulls with US and Danish genetic evaluations and 79 bulls with US and Swedish genetic evaluations. Edits were made to include only bulls with approximate minimum of 50 daughter equivalents in SCC and clinical mastitis evaluations and reliabilities for US type of 0.70 or greater.

[2]Both SCC and clinical mastitis are from single-trait analyses. Multiple-trait mastitis is from a multiple-trait analysis including clinical mastitis and SCC, and selection index for mastitis is from single-trait evaluation of clinical mastitis and single-trait evaluation for SCC. Because of scaling, higher values are desirable for the Danish and Swedish traits.

*Correlations among sire evaluations were different ($P < 0.05$).

yield, and udder depth are all correlated (9, 11, 13), and the relationship between clinical mastitis and udder depth could be partially explained by the antagonism between milk yield and udder depth and the antagonism between clinical mastitis and milk yield. Bulls that transmit more milk yield have daughters with more mastitis and a tendency toward deeper udders; so, one could speculate that the relationship between udder depth and mastitis possibly is primarily a result of the relationship between milk yield and the two traits (i.e., deep udders and more clinical mastitis are essentially a result of stress from heavy milking). However, these correlations indicate that udder depth has a significant association with clinical mastitis that is independent of milk yield. These results support the theory that udder depth and clinical mastitis are genetically correlated because lower udders are likely to have increased exposure to pathogenic bacteria and are more likely to be injured.

Genetic correlations between clinical mastitis in Denmark and udder depth and between clinical mastitis in Sweden and udder depth calculated from residual correlations after adjustment for PTA SCS were 0.21 and 0.36, respectively. Genetic correlations between clinical mastitis in Denmark and udder depth and between clinical mastitis in Sweden and udder depth calculated from residual correlations after adjustment for PTA milk yield and PTA SCS were 0.19 and 0.35, respectively. Apparently, udder depth may contain some information on clinical mastitis that cannot be ascertained from SCS alone or from SCS and milk yield.

The quadratic regressions of clinical mastitis from Sweden on each udder linear type trait taken one at a time were all nonsignificant ($P > 0.10$). The quadratic regressions of clinical mastitis from Denmark on each udder linear type trait taken one at a time were nonsignificant ($P > 0.10$), except for the regression of clinical mastitis on udder cleft. This result could be due to sampling, especially given the large number of regressions that were performed. The trend for udder cleft from the quadratic model involving clinical mastitis from Denmark indicated that extreme values for udder cleft (shallow cleft and deep cleft) tended to be associated with lower incidence of clinical mastitis.

CONCLUSIONS

Sire evaluations for SCS from the US are a good indicator of clinical mastitis in Danish and Swedish daughter groups. Also, the genetic correlations between US SCS and SCC in Denmark and Sweden are large, indicating that those measures are similar.

Bulls that transmit more shallow (higher relative to the hock) udders that are more tightly attached have daughters with lower rates of clinical mastitis. Individual type traits other than udder depth and fore udder attachment have low or negligible genetic correlations with measures of udder health. Selection for lower SCS and more shallow udders should reduce the incidence of clinical mastitis.

Quadratic genetic relationships (genetic regressions) among clinical mastitis and the indicator traits considered in this study were not important in most cases. If any nonlinear relationships between clinical mastitis and SCS exist at the genetic level, indications are that extremely low PTA for SCS are more favorable than moderate or high PTA for SCS.

Correlations among the various measures of udder health and udder-related type traits indicate that multiple-trait and multiple-country evaluations for clinical mastitis would have moderate accuracies for bulls sampled in countries where clinical mastitis data are not available. Procedures to calculate these multiple-trait and multiple-country evaluations should be pursued. As a precursor to multiple-trait and multiple-country evaluations for clinical mastitis, the US and possibly other countries should consider development of multiple-trait predictions for clinical mastitis in which both SCS and udder depth are included.

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