Selection on Somatic Cell Score to Improve Resistance to Mastitis in the United States

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ABSTRACT

Increased genetic susceptibility to mastitis has accompanied the rapid genetic increase in milk yield, and genetic selection for mastitis resistance should be considered. Somatic cell score is recommended as an indicator trait to achieve genetic improvement for mastitis resistance. Heritability of somatic cell score is around 10%, and genetic correlation between somatic cell score and clinical mastitis is around .6 to .8. Selection for lower somatic cell score is consistent with the goal of maximizing genetic improvement for total economic merit and should be included in breeding programs. National genetic evaluations for somatic cells scores will use the same animal models and methods as are currently used for milk yield traits. Reliabilities of PTA for somatic cell scores will be smaller than for yield traits because of lower heritability and availability of records from fewer cows. Several forms are proposed for reporting genetic evaluations of somatic cells to producers, and advantages and disadvantages are discussed. Using somatic cell scores for breeding decisions would marginally decrease genetic gain for milk yield and increase total economic merit. Optimal selection indexes would slow the rate of increase in mastitis, rather than decrease its incidence.

(**Key words**: mastitis, somatic cell count, genetic improvement, selection)

Abbreviation key: **SCS** = somatic cell score.

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INTRODUCTION

Somatic cell testing programs began in DHI programs in the US during the late 1970s and early 1980s. Currently, nearly 80% of all cows in the DHI program are on somatic cell testing. Research with these data has indicated that the somatic cell records would be useful for genetic improvement of mastitis resistance. The objective of this paper is to discuss why mastitis should be used for breeding decisions, why genetic improvement of resistance to mastitis should be based upon somatic cell scores (**SCS**), how genetic evaluations for SCS are derived, how genetic evaluations for SCS may be reported, and what the consequences are of including SCS in breeding programs.

IMPORTANCE OF GENETIC IMPROVEMENT FOR MASTITIS RESISTANCE

The overall reason for genetic improvement is to increase the total economic merit of animals, and the reasons for including mastitis in breeding programs are no different from those for including any other trait: reducing the cost of producing milk, reducing premature culling of cows, improving the quality of milk and dairy products, and improving the health and well-being of dairy cows.

Mastitis is one of the leading disease and management problems of dairy cattle. A recent review (33) showed that mastitis was the third leading reason for premature culling of dairy cows, following low yield and reproduction as the primary reasons for disposal. Among the five studies reviewed, mastitis accounted for 6 to 17% of culling. Studies (18, 30) have shown that approximately 50% of total health care costs are attributable to mammary function and that the vast majority of the mammary health care costs are due to mastitis. Study (3) of an experimental herd in Michigan found that

mastitis was the second most important trait for determining profit per year of herd life. Milk yield accounted for 37%, and mastitis accounted for 14%, of the variance of profit. The number of live calvings per year and herd life accounted for 9 and 5% of the total variance (3).

Genetic improvement has increased milk yield dramatically during the past 25 yr. One of the great success stories of agricultural research, genetic gain for milk yield has increased at an increasing rate since the late 1960s. Estimates indicate that genetic improvement for milk yield during recent years is about 139 kg/yr (26), and the rate of improvement is increasing. Empirical evidence is clear that genetic improvement for milk yield is accompanied by a slow rate of increase in health problems and costs. Selection experiments in Iowa (31) and Minnesota (17) found that differences between lines selected for high and average milk yield were from 600 to 850 kg. Income over feed cost was $60 to $95 higher for the high milk yield line than for the average milk yield line. Health costs for the high lines ranged from 12 to 20% of the value of the income over feed cost. Although health care costs were increased, the added milk yield more than compensated for the added health care costs; that is, milk income over feed cost of the higher lines was from five to eight times the value of the increased health care costs.

This empirical evidence is consistent with estimates of the genetic correlation of milk yield and mastitis. A review of six studies (33) found estimates of the genetic correlation of milk yield and mastitis ranged from −.07 to +.33 and averaged around .20. This positive genetic correlation implies that cattle with higher genetic values for milk yield tend to have higher genetic values for infection rate and clinical mastitis. In the absence of selection on mastitis, genetic increase in mastitis incidence is proportional to the genetic improvement of milk yield. Therefore, as genetic merit for milk yield increases at ever increasing rates, so does the genetic level of mastitis incidence.

Three simulation studies have predicted genetic trends for mastitis. Wilton et al. (41) estimated an increase of .013 more infections per year per cow as a result of genetic increases in yield. Philipsson et al. (24) estimated an annual increase in the probability of mastitis of .4%/yr per cow. Strandberg and Shook (35) estimated the number of mastitis cases would increase by .02/yr per cow as a correlated response to genetic improvement of milk yield. The rates of increase in milk yield assumed in those studies were less than half the rate of increase in yield that is currently taking place. Therefore, current rates of increase in mastitis are perhaps more than twice the rates of increase predicted in those studies. Although the trends for increased mastitis incidence appear to be low, if they are projected over a span of 50 yr, the amount of increase in mastitis that may accompany genetic improvement for milk yield is indeed alarming. Apparently, improvements in management have been offsetting these genetic increases in susceptibility. Can these trends be allowed to continue at the current rates? Can the industry afford to provide increasingly sanitary environments to negate genetic increases in mastitis susceptibility? Genetic approaches would be useful to moderate the genetic trend for increased mastitis incidence.

Two Scandinavian studies (25, 34) have shown large differences among sires for disease incidence among their daughters. A Norwegian study reported that the breeding value for mastitis averaged 18.7% for the 10 worst bulls and 3.7% for the 10 best bulls (34). In Sweden, the frequency of all veterinary treatments differed by 10% for the highest and lowest among 13 progeny groups. In a larger number of progeny groups, difference was expected to be 14 to 17% (25). In a recent review (33), it was reasoned that mastitis incidence for the daughters of the worst 5% of bulls would be 10 to 15% higher than for the daughters of the best 5% of bulls in the same environment. This substantial difference in mastitis incidence of daughters of sires is perhaps the most compelling reason to include mastitis in breeding programs.

In summary, disease is economically more important than some traits now under selection. Genetic improvement for milk yield should result in increased mastitis susceptibility. The cost of increased disease incidence that accompanies genetic improvement for milk yield accounts for 10 to 20% of the increased value of milk yield. About half of this increased disease cost is attributable to

mastitis. These rates of change in disease are alarming when projected over the long term.

IMPROVED MASTITIS RESISTANCE THROUGH SELECTION ON SCS

Interpretation of SCS

Individual SCC are converted by DHI Dairy Records Processing Centers to scores using a base 2 logarithm. An SCC of 100 cells/μl converts to a score of 3. Each 1-unit increase (or decrease) in score is associated with a doubling (or halving) of cell count. For example, score 2 is equivalent to a cell count of 50, and scores of 4 and 5 correspond to 200 and 400 cells/μl. This scoring method has been used since 1983 at all Dairy Records Processing Centers in the US. The reasons for using this scoring method are detailed elsewhere (2, 32), but the principal reasons are to achieve properties required for use of conventional statistical methods: 1) mean equal to median, 2) normal distribution, and 3) uniform variance among samples within lactation, among cows within herd, or among daughters within sire.

The frequency distribution of SCC is highly skewed; the mean is much higher than the median, and the median is higher than the mode. The SCS is normally distributed so that the mean, median, and mode have the same value. Thus, the mean divides the top 50% from the bottom 50% of the data, and the mean is within a short range of most observations. The advantage of uniform variance may be difficult to appreciate, but it is among the most important reasons. Briefly, if variances differed among groups, both mean and variance would have to be estimated to characterize the groups. When data are normally distributed and have a uniform subclass variance, the group, whether it is cow lactation average, herd average, or sire daughter average, is characterized by the mean, which varies among groups and variance, which is similar for all groups. Without this property, a variance would have to be computed for each group. Variances computed from small numbers of observations are unreliable and have large sampling errors. These properties allow averaging of monthly SCS into a lactation average score. The lactation average score is the basic measure used for the proposed genetic evaluation procedures.

Somatic cells, primarily polymorphonuclear leukocytes, are attracted to the mammary gland in large numbers during infection. The cow's somatic cell response differs with bacterial species (36). The major pathogens, such as *Streptococcus agalactiae* and *Staphylococcus aureus*, elicit a stronger response than do the minor pathogens. Somatic cell score is widely used as a measure of udder health and as a means of managing control of mastitis (27). Elevated SCS are indicative of clinical or subclinical infections.

The DHI tests for somatic cells are based on bucket milk samples taken at approximately monthly intervals. Each sample characterizes the general health of all four quarters on the day the sample is taken. Monthly scores are accumulated into a lactation average score. We shall refer to this lactation average as SCS. The SCS roughly characterizes the udder health of the cow throughout the lactation and may be thought to reflect the net effect of presence or absence of infection, number and duration of infections, severity of infection, and number of quarters infected. A low SCS indicates a low level or absence of infection throughout the lactation. The continuum of scores indicates degrees of mastitis that cows experience during a lactation. Increases in SCS between lactations and between months within lactation are attributable to increases in clinical mastitis (12). In the absence of infection, SCS remains low with advances in either age or stage of lactation. Also, low SCS are associated with a low probability of clinical mastitis, and, specifically, the lowest scores are associated with lowest probability (9, 11), which is contrary to the widely held view, based on early research, that elevated SCC is necessary to prevent mastitis. A great deal of practical experience suggests that SCC can be maintained at low levels without risk of major outbreaks of mastitis. The major determinant of variation in SCS is intramammary infection (19, 27).

Selection on SCS

Indirect selection is a well-established concept in quantitative genetics (15). Indirect selection involves selection based on an indicator trait as a means of making genetic improvement for an economically important trait.

For mastitis, SCS is the trait on which selection will be based while genetic improvement is sought for mastitis resistance. Indirect selection, in order to be effective, must have a high genetic correlation between the indicator trait and the economically important trait. In addition, the indicator trait must have one or more of the following advantages over the economically important trait: higher heritability, lower recording costs, measurable earlier in life, and measurable in both sexes.

Genetic correlation between clinical mastitis and SCS is a measure of the extent to which genetic merit of an animal for SCS measures that animal's genetic merit for clinical mastitis. Genetic lines (e.g., sire's daughter groups) that are more susceptible (resistant) to infection exhibit higher (lower) SCS and rates of clinical mastitis. Three major studies have estimated genetic correlation between clinical mastitis and SCS. Young et al. (42) estimated the value at .80 and .98 by two methods. Afifi (1) found a genetic correlation of .83. Emanuelson et al. (13) estimated a correlation of .46 for Swedish black and white cattle and .78 for Swedish red and white cattle. The average of the estimates in this study was .62. The more recent study is perhaps the most reliable because it is based on the most data (field data), and it utilizes the most recent statistical methods. Weller et al. (37) estimated the genetic correlation between clinical mastitis and SCS to be .3. However, they found that the genetic correlation between bacterial infection and SCS was near unity and pointed out that the low estimate of .3 may have been due to inaccurate recording of clinical mastitis. In conclusion, the genetic correlation between clinical mastitis and SCS is around 60 to 80%, which indicates a strong genetic association between these two traits: selection of SCS should produce a response in clinical mastitis.

Heritability indicates the relative contribution of genetics and environment to phenotypic differences among animals. Heritability is the portion of the phenotypic difference among animals that, on average, is attributable to the genetic difference. A higher heritability indicates a relatively greater genetic importance and a lesser importance of management and environment as a cause of variability among phenotypes. Higher heritability also indicates an opportunity to make more rapid genetic improvement and attainment of a higher reliability of genetic evaluations on individual animals. Although heritability estimates for mastitis vary widely, the more recent studies based on field data for large numbers of herds indicate that heritability of mastitis is around 2 to 6% (13, 20). Monardes et al. (22) found that heritability of SCC was around 6%, and heritability of SCS was 12%. Thus, higher heritability is another principal advantage for the use of SCS rather than SCC for computing genetic evaluations. Other recent studies of SCS using DHI data indicate that heritability of SCS is around 10 to 12% (4, 6). Furthermore, heritability is similar for herds with low or high average SCS (4). Although heritability tended to be higher in low SCS herds, the differences were not statistically significant. A common heritability can be assumed for all herd levels of SCS.

Other advantages for basing selection on SCS are that it serves as an indicator of both clinical and subclinical mastitis. Also, SCS is an objective measure, but clinical mastitis is subjective. Perhaps it is the lack of objectivity that causes clinical mastitis to have a lower heritability than SCS and results in only moderate genetic correlation between SCS and clinical mastitis. Furthermore, SCS is much less expensive to obtain in the laboratory than are bacteriological tests. Although clinical mastitis would be less costly to record than SCS, producers appear to be unwilling to record treatments for clinical mastitis. Producers are far more willing to submit milk samples and to pay the cost of laboratory tests for SCS. The DHI program throughout the US involves more than 4.8 million cows. Over 3.8 million of those are currently involved in the somatic cell testing program of DHI (Phil Dukas, 1992, personal communication). A substantial data base has accumulated on SCS that can be used for genetic evaluation of dairy cattle, which makes SCS the only trait closely related to mastitis for which genetic evaluation is feasible.

In conclusion, genetic correlation is reasonably high between SCS and clinical mastitis, heritability is somewhat higher for SCS than SCC or clinical mastitis, and SCS are relatively easy and inexpensive to obtain. Therefore, SCS is a useful trait for indirect selection for genetic improvement of mastitis resistance.

A PROPOSAL FOR GENETIC EVALUATION OF SCS IN THE US

Seven of nine Dairy Records Processing Centers have contributed lactation measures of somatic cells. The trait reported is the arithmetic mean of test day SCS in the first 305 d of lactation. The practicality of conducting genetic evaluations of SCS nationally has been demonstrated by Boettcher et al. (6). They (6) used a sire model, but proposed national genetic evaluations would use the animal model currently used by USDA for evaluation of yield traits (38):

$$y_{ijkl} = m_{ij} + c_{ik} + p_{kl} + a_{kl} + e_{ijkl}$$

where y_{ijkl} = lactation average SCS, standardized for age and month of calving and length of lactation of cow kl (daughter l of sire k) in herd i and year-season, parity, and registry group j. Terms in the model are fixed management group (m), random herd-sire interaction (c), permanent environment (p), animal (a), and residual (e) (38).

Standardizing lactation measures of SCS for age and month of calving has been recommended by several studies (6, 21, 29). Adjustment of test day SCS for stage of lactation is also recommended (14, 29, 39), but Boettcher et al. (6) developed a procedure to account for length of lactation when only a single value for the entire lactation is available.

Management groups (m_{ij}) combine records of cows in a given herd-year-season of calving (2-mo seasons), registration status (registered or grade), and lactation (first or later). Thereby, records of each cow are compared with records of other cows in environments that are as similar as possible. Further details about management group definitions are given by Wiggans et al. (38). A challenge for future research is to determine whether management groups for SCS should be defined differently from those for yield traits. The herd-sire (c_{ik}) effect is an effect common to paternal half-sibs in a single herd. This effect is part of a cow's performance but is not part of a sire's across-herd evaluation. Therefore, the impact of a single herd on a sire's evaluation is limited (7). Permanent environment (p_{kl}) is an effect on all records of a cow that is not transmissible to offspring. For example, a cow injuring a teat during first lactation may have higher SCS for every subsequent lactation.

Animal effect (a_{kl}) represents the breeding value of the cow producing the record. Animal models allow estimation of breeding values to include all known relationships, including sire, dam, offspring, sisters, cousins, nieces, and aunts. Contributions from offspring take into account the genetic contribution of the other parent, which is usually termed merit of mates. Further details about definitions of animal effects can be found elsewhere (38).

Wiggans et al. (38) and Cassell (7) described how genetic evaluations are calculated with the animal model. Briefly, estimates of all effects in the model are obtained by iterative procedures, which allow estimation of effects from records adjusted for all other effects. For example, genetic differences between management groups are taken into account as management group effects are estimated; conversely, adjustments for management group are made as animal (genetic) effects are estimated. Processing of evaluations begins by herd, and management group effects are estimated first and are adjusted for the other effects estimated during the previous round of iteration. Next, processing by sire within herd, permanent environment, and herd-sire effects are estimated. Special techniques to include cows with records from more than one herd are described by Wiggans and VanRaden (40). Records adjusted for management group, herd-sire, and permanent environment effects are accumulated; animal effects, or breeding values, are computed across herds after all data has been processed. Iteration allows influence of an animal to pass through the breeding values of all relatives so that all are affected after a number of rounds of iteration.

Breeding values predicted by animal model procedures are divided by 2 and expressed as PTA. Ranks of PTA and differences among PTA are important. Actual PTA are not directly applicable to a given herd, but the difference in PTA for two animals predicts expected difference in average performance of their progeny.

Previous work has characterized the frequency distribution of PTA for SCS (4, 6). Boettcher et al. (6) computed genetic evaluations for average SCS from first lactation adjusted for length of lactation and age and

month of calving. They used records from 5 of the regional Dairy Records Processing Centers initiated from January 1987 to October 1989, conducted evaluations for each region, and combined all five into a single national evaluation. Mean sire PTA was −.01 for the national evaluation. The range of sire PTA was from −.50 to .56, and PTA was approximately normally distributed. The standard deviation of PTA was .15, meaning that about 95% of sire PTA fell between −.30 and .30. Regional evaluations in their study gave similar results, despite having smaller numbers of sires. Similar results were reported also by Banos and Shook (4).

Reliability is the measure of accuracy or degree of confidence with PTA. As discussed by Hansen (16), the reliabilities of PTA for SCS are smaller than reliabilities for milk yield with a given number of daughters and records per sire (Table 1). Reliability is related to heritability; that is, the less genetic differences contribute to performance, the lower is the confidence in a PTA based on the same number of records. Because the heritability of SCS is much lower than for milk yield (approximately .12 versus .25), the same number of daughters and records results in lower reliability of PTA for SCS than for milk.

Coffey et al. (8) expressed concern that SCS may be a genetically different trait for first lactation versus later lactations. This concern was also expressed by Da et al. (10), who found that genetic and phenotypic correlations for SCS were lower between lactations 1 and 2 and 1 and 3 than between 2 and 3, implying that first and later lactations could be separate traits. Similarly, Banos and Shook (4) reported that genetic correlations between first and later lactations were about .75. Genetic correlation between second and third lactation was near 1.0. However, as mentioned previously, only about 80% of cows on test have SCS information recorded. This limits the amount of information available, which is especially noteworthy for a trait that has lower reliability even for the same quantity of information. Thus, there is a trade-off between additional accuracy for including later lactations and the moderate genetic correlation between first and later lactations for SCS. Obviously, more information is desirable for overcoming lower accuracy for estimating PTA of SCS. Also, if SCS is genetically different for parities and the overall goal is to increase mastitis resistance throughout the productive life, SCS records from all parities should be included in evaluations. Ultimately, treating SCS from first and later lactations as separate but correlated traits in multiple-trait animal evaluations could circumvent these problems. Such evaluations are not yet computationally practical on a large scale.

TABLE 1. Approximate reliabilities of PTA for milk yield and SCS by number of daughters.[1]

	Reliability	
Daughters	Milk	SCS
(no.)	(%)	
20	67	40
30	73	48
50	81	59
100	89	73
200	94	84
500	97	93
1000	99	96
2500	99	99

[1]From Hansen (16).

Interpreting PTA for SCS

Only one study has examined the genotype by environment interaction for SCS (4). Herds were divided into quartiles based on herd average SCS. Sire evaluations were computed within each of the four quartiles of herd average. Estimates of genetic correlations of sire PTA calculated in the different herd quartiles are shown in Table 2. The results are presented as approximate upper and lower limits of the genetic correlation between herd levels. The lower limit assumed perfect information on

TABLE 2. Limits of genetic correlations between sire PTA from different herd levels for SCS during first parity.[1]

Herd level	Herd level		
	1	2	3
2	.96–1.04		
3	1.12–1.27	1.02–1.15	
4	.88–1.02	1.02–1.16	1.13–1.32

[1]From Banos and Shook (4).

male relatives, and the upper limit ignored information on male relatives of the sires. Genetic evaluations calculated for any one herd level were very highly correlated with genetic evaluations calculated for any other herd level; all correlations were near 1.0, which implies that the genes that are beneficial or detrimental to SCS are similar for high and low SCS herds. This result is fortuitous, and it greatly simplifies the calculation of genetic evaluations and their application for genetic improvement. First, SCS differences of bulls are likely similar for herds with low or high SCS; second, records from both low and high SCS herds can be used in the genetic evaluation process without regard for herd level; third, low SCS herds will benefit from sire selection to the same extent as high SCS herds; finally, sire recommendations can be the same for low and high SCS herds.

Selection on SCS involves some shortcomings and risks. The environmental pathogens (e.g., *Escherichia coli*) are becoming increasingly prevalent causes of mastitis. These organisms tend to produce extraordinarily high responses of somatic cell for relatively short duration. The monthly sampling scheme in DHI will detect a small fraction, perhaps 10 to 20%, of these infections through SCS. The SCS, which is based on monthly sampling, reflects much more accurately the effects of chronic pathogens that exhibit a more sustained duration of somatic cell response. The shift toward higher prevalence of environmental pathogens relative to contagious pathogens over time may reduce the genetic correlation between SCS and clinical mastitis. Currently, there are no studies of the effect of types of pathogens on the genetic correlation between SCS and clinical mastitis. Another risk is that selection on low SCS favors animals with no infections as well as animals with a low somatic cell response to the presence of infection. The great majority of the variation in SCS is thought to be associated with presence of intramammary infection. To the extent that variance of SCS is associated with degree of response to infection, selection for low SCS would result in an increasing frequency of animals that have a low response to infection as well as a low rate of intramammary infection. A weak intensity of selection would mitigate against this undesirable result. Most of the selection emphasis will continue to be placed on the yield traits because of their greater economic importance. The intensity of selection of SCS will not be great, nor will genetic change in SCS be rapid. Research should continue to seek a better trait than SCS on which to select for improving mastitis resistance. In the meantime, the best tools now available should be used. They are credible, and they can be effective if used properly.

Genetic selection to decrease SCS has direct economic benefits in addition to decreased susceptibility to mastitis. The National Conference on Interstate Milk Shipments reduced the limit for Grade A milk sold in the commercial marketplace from 1000 to 750 cells/μl of milk. Recommendations to reduce the limit further to 600 cells/μl seem likely (5). Many dairy plants pay premiums for milk low in somatic cells. This lower premium is related to decreased cheese yield and storage life of milk with high SCC. Such adjustment to milk price for SCC have recently been proposed for inclusion with multiple component pricing for several federal Milk Marketing Orders (23). Industry sales of US semen to certain foreign countries could increase if PTA for SCS were routinely reported, because breeders in several European countries are accustomed to evaluations for several health traits.

Mastitis and SCS have much greater economic importance than many of the traits that are already measured and evaluated for breeding programs. To maximize genetic improvement of total economic merit of dairy cattle, SCS should be used in breeding programs.

Reporting Genetic Evaluations

Several concerns about reporting genetic evaluations for SCS must be resolved. First, producers may be confused over whether high or low SCS is desirable. For yield traits, producers are accustomed to selecting for higher values, but, for SCS, selection should be toward low values to decrease mastitis incidence. Second, many producers are not yet familiar with SCS but prefer SCC as reported by milk plants and on which quality premium payments are based. Third, any scale to report genetic evaluations for SCS must not encourage overemphasis in breeding programs; SCS does not economically warrant nearly as

much selection emphasis as the yield traits, yet many would apply independent selection thresholds and avoid selecting any bull worse than the threshold no matter how desirable his profile may be for other traits.

Table 3 lists 7 proposed prototypes for reporting genetic evaluations for SCS. The names of the indexes are arbitrary, and some of the names could appropriately be assigned to more than one of the indexes. These prototypes are representative of a larger array of possibilities. The first index is the genetic evaluation for SCS relative to a base group of animals. For milk yield, the base is the average genetic evaluation of cows born in 1985. This base sets their average evaluation to zero. The advantages of prototype 1 are that SCS is used by DHI throughout the country, and, as described previously, SCS allows use of familiar statistical techniques. The PTA would parallel those for yield traits. The range of values is quite narrow and may avoid overemphasis on this trait as producers select sires. The primary disadvantages of prototype 1 are that, despite being used by DHI, SCS is not familiar to all producers, and the narrow range of values could downplay real differences among sires. Confusion may exist with regard to the direction to select (negative values are favorable), but could be overcome by replacing plus and minus signs with H for high and L for low. Perhaps the biggest disadvantage, however, is that the mean evaluation near zero would encourage some to use zero as a natural culling level.

For somatic cell index (prototype 2), the difference between two evaluations is 10 times the difference expected between daughter averages for SCS. Prototype 2 has a wider range of differences than prototype 1. Also, no natural selection threshold exists because all values are positive. Perhaps no index can eliminate overemphasizing SCS evaluations, because producers may impose as a selection threshold any value that becomes accepted as a rule of thumb. Nevertheless, natural thresholds near the mean should be avoided. The disadvantages of somatic cell index are that the units are not the same as SCS, and the numbers could be misinterpreted as expected SCS of daughters instead of 10 times the average difference between progeny groups. Mastitis resistance index (prototype 3) expands the somatic cell index by a factor of 10 and inverts the ranking. Positive values are favored with this index, yet the wide range of values could overemphasize small differences among animals. Standardized transmitting ability (prototype 4) is directly related to SCS and is familiar as the method used by Holstein Association of America and several AI organizations to report linear type trait evaluations. Not all producers are familiar with this scale, and zero could be misinterpreted as a natural threshold.

Prototypes 5 and 6 may overcome the confusion related to SCS versus counts by transforming SCS evaluations back to a SCC basis. The constant 2 is used in these equations because SCS is a base 2 logarithm of SCC. The constant 1.0 in the SCC index (prototype 5) sets the base equal to 200. For this index, differences among sires provide a realistic indication of expected daughter performance,

TABLE 3. Prototypes for reporting genetic evaluations for somatic cell scores.

Index name	Equation	Range of values for 95% of sires
1. Somatic cell score	SCS[1] – Base	–.30–.30
2. Somatic cell index	SCI = (SCS–Base) × 10 + 5	2–8
3. Mastitis resistance index	MRI = (SCS–Base) × (–100) + 50	80–20
4. Standardized transmitting ability	STA = (SCS–Base) + SD of SCS	–2.0–2.0
5. Somatic cell count index	SCCI = 100 × $(2^{(SCS-Base + 1.0)})$	160–260
6. Percentage change index	PCI = $2^{(SCS-Base)}$	.80–1.25
7. Mastitis susceptibility index	MSI = $100(\frac{e^y}{1 + e_y})$ where y = (SCS–Base) × 1.00–1.10	20–31%

[1]SCS is PTA for SCS, and base is the average PTA for all cows born in a particular year.

and evaluations are expressed in the same units on which quality premiums are paid. The drawbacks are that the values have a nonlinear relationship with SCS and could be misinterpreted as the expected average SCC of daughters in a specific herd. The percentage change index (prototype 6) provides a ratio of the SCC of one bull's daughters to daughters of a bull for which PTA is equal to the genetic base. This index could be easily explained to producers, and the ratio applies to expected daughter performance in any herd. This index has a mean of 1, which could be misused as a natural selection threshold.

Mastitis susceptibility index (prototype 7) relates genetic evaluations to clinical mastitis. This index is based on a logistic regression of clinical mastitis on SCS (11). Additional research is needed to verify these regression coefficients. The constant 1.10 sets the mastitis susceptibility index at 25% when SCS is equal to the base. The index is an estimate of the percentage of daughters that will become infected in a herd with average incidence of 25%. This index is easy to describe and use; the direction for selection is obvious; and there is no natural selection threshold. However, there are several important disadvantages. The values could be mistaken for expected daughter performance in any herd; the relationship with SCS is nonlinear; and the index implies that the only economic value of selection against SCS is decreased clinical mastitis, which ignores the direct value of decreased SCS in milk.

Prototypes 2, 3, and 4 all have a linear relationship with SCS, and, therefore, possess all the desirable statistical properties of SCS. Prototypes 5, 6, and 7 all have a nonlinear relationship with SCS. Because PTA for SCS occur over a narrow range, the degree of nonlinearity is not great. Linear approximations to these indexes would obtain the statistical properties of SCS without seriously impairing their intended interpretation as SCC or clinical mastitis probabilities. Any method of reporting evaluations has advantages and disadvantages. Regardless of the method chosen to report SCS evaluations, eventual release of evaluations requires a large educational effort by extension, veterinary, and AI personnel to ensure proper understanding and use by producers.

The best way to encourage proper emphasis of genetic evaluations in selection programs is to provide producers with an index that combines important traits using appropriate economic weights. This index could be easily accomplished by combining PTA for SCS with PTA for milk, fat, and protein in an economic index as is currently done for the milk, fat, and protein dollar index routinely reported by USDA. Somatic cell scores have several major economic impacts. Increased value of milk with low SCC; clinical mastitis costs, including discarded milk, labor, and treatment costs; and subclinical milk loss are important. Subclinical milk loss, perhaps the largest cost, is already taken into account if genetic evaluation for milk yield is included in the index. Premature replacement of cows is another major component of the cost of disease. Many studies of disease economics have ignored this cost. Total economic merit of dairy cows should be evaluated on the basis of life cycle rather than lactation cycle to account for replacement costs. Further work is necessary to estimate the expected economic value through decreased clinical mastitis and increased milk quality per unit decrease in SCS.

CONSEQUENCES OF INCLUDING SCS IN BREEDING PROGRAMS

Table 4 shows the responses to selection indexes that include mastitis (35). As discussed earlier, that study (35) shows that selection for milk yields alone results in increased mastitis incidence. Including clinical mastitis with milk yield in a selection index slightly diminishes the rate of increase in milk yield but slows the rate of increase in mastitis by 20%. That study (35) also found that a selection index that includes SCS was nearly as effective as an index that included clinical mastitis. The rate

TABLE 4. Responses in milk yield, mastitis, and total merit from selection indexes with different index traits.[1]

Index traits[2]	Response		
	Milk	Mastitis	Merit
	(kg)	(cases/yr)	($)
None	53.5	.020	98.2
SCS	52.7	.016	98.6
Mastitis	52.4	.015	99.1

[1]Adapted from Strandberg and Shook (35).

[2]All indexes also included milk and fat yield.

of increase in mastitis was diminished by 80% of the reduction accomplished by selection on clinical mastitis. Selection indexes are designed to maximize the rate of improvement in total economic merit. That study (35) indicates that simultaneous improvement of both milk yield and mastitis resistance appears to be economically undesirable because of the positive correlation between milk yield and mastitis and the relatively greater economic importance of yield. Rogers (28) obtained similar results. Using a selection index approach, Rogers (28) found that response in total merit, defined by milk yield, clinical mastitis, milking labor, and laminitis, was increased by 1 to 4% when selection included SCS and udder depth compared with selection on milk yield alone. Undesirable response to mastitis was reduced but not eliminated. Rogers (28) also pointed out that the most important factor for determining the optimal selection emphasis to be placed on evaluations for SCS is the genetic correlation between SCS and clinical mastitis. If the genetic correlation is as high as .80, then SCS is more useful than any trait other than yield. In useful indexes, SCS received about 5 to 8% as much emphasis as milk yield.

Clearly, inclusion of SCS in breeding programs will not reduce mastitis substantially. Continued attention to good sanitation and proper milking practices will continue to be needed as the primary approach to mastitis prevention. Genetic improvement will not reduce the need for these practices but is one more tool that may be used for mastitis prevention. Used effectively, it can reduce the need for antibiotic therapy and culling of cows because of mastitis.

CONCLUSIONS

The primary approach to genetic improvement based on SCS will be to select bulls with low PTA for SCS or arbitrarily to select against some bulls with high PTA for SCS. Selection of cows may also be possible; however, the low reliability of cow PTA and the narrow range expected in cow PTA would indicate little opportunity for genetic improvement through cow selection. Including SCS with proper economic weights in selection indexes will increase genetic improvement for overall merit. Including SCS in selection indexes is not expected to improve mastitis resistance but will slow the rate of increase in mastitis susceptibility. Addition of SCS to the tools available for genetic improvement will improve the economic efficiency of dairy cattle. Selection is an important new practice for prevention of mastitis.

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