Genetic Evaluation of Somatic Cell Scores for United States Dairy Cattle

M. M. SCHUTZ
Animal Improvement Programs Laboratory
Agricultural Research Service, USDA
Beltsville, MD 20705-2350

ABSTRACT

Increases in milk yield from genetic selection may be accompanied by correlated increases in genetic susceptibility to clinical mastitis and somatic cells. Unlike clinical mastitis, somatic cell scores can be easily determined and recorded and are related to milk loss from subclinical mastitis. Selection against high somatic cell scores should decrease incidence of clinical mastitis and provide direct economic benefits through higher milk quality premiums. Genetic evaluation for lactation means of linear somatic cell scores has been implemented by USDA and parallels that for yield traits. Because additive genetics accounts for only about 10% of differences in somatic cell scores among cows, more information is needed for the same degree of confidence in genetic estimates as for yield. Only 80% of DHIA cows currently have somatic cell records. Thus, reliabilities of somatic cell evaluations are smaller than those for yield traits. Most progress in selection for lower somatic cell scores will come through sires of cows considered as bull dams. Somatic cell evaluations may best be reported through an economic index with a small amount of emphasis on somatic cell score relative to yield traits. Greater emphasis on somatic cell scores would decrease genetic gain in yield traits, which are economically more important.
(**Key words**: somatic cells, mastitis, genetic evaluations)

Abbreviation key: DE = daughter equivalents, **LSCS** = lactation mean of SCS, **MFP$** = economic index that includes PTA for milk, fat, and protein yields, **REL** = reliability, **SCS** = somatic cell score, the sample day $\log_2$ SCC.

Received June 16, 1993.
Accepted December 7, 1993.

INTRODUCTION

Mastitis is the most costly health problem of dairy cows (18, 46). Expenses and revenue losses arise from veterinary treatment, discarded milk, increased labor and milking time, milk loss from subclinical mastitis, decreased yield following serious infection, premature culling of cows, and poorer milk quality. Estimates of the economic loss from mastitis generally range from $100 to $200 per cow per lactation (70). Dobbins (13) documented annual losses from $35 to $60 per cow but suggested a more reasonable range was $90 to $250 because some costs were ignored. Jasper et al. (21) estimated annual costs at $182 per cow, which projected an annual cost in excess of $2 billion to the US dairy industry. A Michigan study (3) identified mastitis as the second most important trait for determining profit per year of life; only milk yield was more important. The economic impact of mastitis on dairying is enormous.

Management practices have been and will continue to be the most effective way to prevent mastitis infections. There is no replacement for milking clean and dry udders, ensuring proper function of milking equipment, dipping teats after milking, and providing sanitary housing conditions for cows. Eradication would be the best choice for control of mastitis (48) but is not feasible because of the variety of pathogens responsible for mastitis, the ubiquity of those pathogens in the environment, and the expense and difficulty of completely eliminating even one species of pathogen. Vaccination of cattle against some pathogens shows promise, but success has been limited and has occurred only for certain pathogens, such as *Staphylococcus aureus* (26, 31, 59).

Treatment of clinical mastitis and culling of cows may ease the impact of mastitis in a herd but are expensive in terms of lost productivity, labor and treatment costs, discarded milk, and cost of raising replacement heifers. Dairy producers are increasingly pressed to limit use of drug therapy by consumers who are aware of drug residues in milk and meat and are concerned for the welfare of cows.

Genetic improvement of dairy cattle for reduced mastitis incidence by selection for fewer somatic cells in milk is possible (48, 49). Rates of improvement through genetic selection are likely to be slow (37, 54), but the cost involved in genetic enhancement of disease resistance is small compared with the large cost of treating clinical mastitis and the milk yield lost from subclinical mastitis. The feasibility of conducting genetic evaluations for lactation mean of sample day $\log_2$ somatic cell scores (**LSCS**) on a national basis has been shown (7). Indeed, genetic evaluations for LSCS are conducted in several Scandinavian countries (6) and were implemented in the US in 1994 by USDA.

Genetic susceptibility to mastitis increases slowly as a correlated response to selection for milk yield; if projected over a long period, the increase in cases of mastitis and associated costs is noteworthy (50). Mastitis incidence for daughters of the 5% of bulls with highest genetic merit for mastitis resistance has been suggested to be 10 to 15% lower than for daughters of the 10% of bulls with lowest merit (48, 49). Such differences in progeny groups demonstrate the possibility of increasing mastitis resistance or at least slowing the rate of increase in susceptibility. Because of growing consumer opposition to drug contamination of food products, a genetic approach to decreasing mastitis incidence (although slow) would help to maintain the wholesome image of dairy products. The objectives of this paper were to review briefly the genetics of mastitis and somatic cells in milk, to outline a method for calculating genetic evaluations for LSCS, and to describe how evaluation results are reported.

GENETICS OF MASTITIS

Genetic Terminology

Many questions are likely to be raised by dairy producers when genetic evaluations for LSCS are released to the industry. Traditionally, questions about genetic evaluations have been addressed to extension specialists, genetics researchers, and AI industry personnel. However, questions on evaluations for LSCS also will be directed to veterinarians, immunologists, and mastitis researchers, who may not feel entirely comfortable in all cases with terminology and methodology related to genetic evaluations. Descriptions of several terms for the basic concepts behind genetic evaluation for resistance to mastitis follow. Further clarification of definitions is presented by Wilcox (68), Van Vleck et al. (57), and Falconer (16).

Breeding Value. Breeding value refers to the value for a particular trait of an animal in a breeding program. An animal's breeding value is estimated to be twice the expected performance of its progeny relative to a population mean when mated at random. The reason for doubling the expected progeny performance is that only half of the genes from the individual are transmitted to any offspring (the remaining half come from the other parent randomly from the population). The expected progeny performance as a deviation from the population is called transmitting ability and is, therefore, half of the breeding value. In other words, transmitting ability is the genetic advantage an individual transmits to its offspring.

In practice, breeders want to know the expected performance from progeny of certain individuals. True breeding values of individuals are not known because 1) most traits of interest are influenced by many genes, 2) only a sample half of the genes are transmitted at random to the offspring, 3) the number of possible combinations of genes in the offspring is large, and 4) performance of individuals is affected by environment. However, breeding values can be estimated based on the animal's own records and the performance of known relatives. These estimated breeding values divided by 2 may be used to predict the performance of future offspring relative to the population mean and are termed PTA. For example, the daughters of a bull with a PTA of 1000 kg for milk yield would be expected to produce, on average, 200 kg more milk per mature lactation than the daughters of a bull with a PTA of 800 kg for milk yield if their dams have equal genetic merit. The actual difference

TABLE 1. Approximate heritabilities for common traits of dairy cattle.[1]

Trait	h^2	Trait	h^2
Milk yield	.25	Stature	.50
Fat yield	.25	Body weight	.50
Protein yield	.25	Overall type	.20
Fat percentage	.50	Reproductive efficiency	.05
Protein percentage	.50	Mastitis resistance	.10

[1]From data of Wilcox (68).

will not exactly compare individual daughters because no two daughters would get exactly the same combination of genes or would be exposed to exactly the same environment. Thus, daughters of the same sire may have widely varying performance.

Heritability. Heritability is the extent to which genetics influences a trait or characteristic. Unlike breeding values and transmitting abilities, which are estimated for individuals, heritability is a population parameter. Strictly defined, heritability is the ratio of additive genetic variance to phenotypic variance. Additive genetic variance is the true variance among breeding values of animals in a population. Hence, heritability is a ratio of the variance of breeding values to the variance of phenotypes. The possible range of values for heritability is from 0 to 1.0, because additive genetic variance is a part of phenotypic variance. Phenotypes are what is observed or measured about a particular trait; phenotypes are influenced by genetic and environmental effects. For heritability measurement, phenotypic variances are taken to be the total of random sources of variation after adjustment for systematic sources of variability, such as herd-year, age, month of calving, or stage of lactation.

The extent of genetic control is different for each trait. Approximate heritabilities for several common traits of dairy cattle are in Table 1. The higher the heritability, the greater is the genetic control on the trait and the more rapidly selection results in genetic progress. In general, yield traits and overall type tend to be moderately heritable; fat and protein percentages, stature, and size have higher heritabilities; and reproductive efficiency has lower heritability. Mastitis resistance has a heritability of about .10. In other words, genetics accounts for 10% of the variation in the capacity of cows to resist mastitis infection, and environment accounts for the remaining 90%.

Genetic Correlation. The correlation between breeding values for two traits is genetic correlation and indicates the extent to which two traits are influenced by the same genes. For example, the genetic correlation of milk and protein yields (.9) is high (68). Many of the same genes that influence milk yield also influence protein yield, and a bull with daughters that have a high mean for milk yield almost always sires daughters that have a high mean for protein yield. However, the genetic correlation of milk yield and fat percentage is −.3 (68); therefore, bulls with daughters that have high milk yield often have daughters with low fat percentage. As with any correlation, the larger the magnitude (i.e., the farther from 0), the greater is the relationship between traits. For a heritable trait, selection of genetically superior animals as parents (i.e., genetic selection) produces offspring that are genetically better, on average, for that trait. This result is termed response to selection. Genetic selection on such a trait also affects all genetically correlated characteristics; this effect is termed correlated response to selection.

Reliability. The measure of accuracy or degree of confidence in a PTA is called reliability (**REL**), the squared correlation of an animal's true transmitting ability and PTA. In practice, this value is often approximated rather than calculated directly. Further details are given by VanRaden and Wiggans (55). Essentially, REL for PTA of a trait is a function of the heritability of that trait and the amount of information available. That information may come from the animal's own performance, from the performance of offspring, or from information for parents. As heritability and amount of information increase, REL also increases. Thus, an animal has a higher REL

for milk yield than for reproductive efficiency (even if the same number of records are available from the animal and its relatives) because milk yield is under greater genetic control. Also, a bull with many daughters has a more reliable PTA for any given trait than a bull with few daughters.

Heritability of Mastitis

For dairy cows, mastitis long has been known to be influenced to some extent by genetics. In 1938, Ward (58) reported preliminary work showing inheritance of susceptibility to mastitis for New Zealand dairy cows. Lush (25) further analyzed the same data and reached similar conclusions. Clinical mastitis is most often diagnosed by detection of abnormal appearance or consistency of milk or by observation of visible inflammation of the udder. Most often, clinical mastitis is recorded as the presence or absence of mastitis during a lactation. Some studies have used scoring systems to account for multiple cases during the same lactation, the number of quarters infected, and the relative severity of each case (15, 24, 61, 69). In a review of literature, Miller (28) summarized previous heritability estimates for clinical mastitis. Estimates ranged from about 0 to .50; mean was .12. More recently, Emanuelson et al. (15) reported a much smaller estimate (.01 to .02) for the heritability of number of treated mastitis cases per lactation reported by veterinarians for Swedish cattle. Also, Weller et al. (61) estimated the heritability of clinical mastitis to be .01 in an Israeli field study. They attributed their low estimate to the use of field data, which may have been inaccurately reported. Simianer et al. (50) found a heritability of .05 for incidence of mastitis requiring veterinary treatment.

Some studies have also examined genetic control of infection status as detected by bacteriological cultures. An advantage of this measure is that it takes subclinical infections into account as well as clinical cases not detected by milkers. Young et al. (71) found a heritability of .18 for bacterial infection scores of cows in four state-owned herds. However, their estimate of the genetic correlation of bacterial infection status and clinical mastitis was low (.26), which suggests that the two traits are affected by different genes. Miller (28) reviewed three studies; mean heritability for bacteriological status was .11. Weller et al. (61), using data from 31 Israeli herds, found a heritability of .04.

Other studies have looked at subjective scores for rating producers' interpretation of cows' resistance to mastitis (32, 34) and found heritabilities of 0 to .07. Lawstuen et al. (23) studied genetic effects on mastitis recorded retrospectively by producers at the time of type classification. Cows were rated for resistance to mastitis on a 50-point scale (1 = least resistant; 50 = most resistant). The estimated heritability for this measure of mastitis was .03. Overall, the heritability of mastitis appears to be about .02 to .04 (49) from field data but may be nearer to .10 if it is more accurately measured and consistently reported as in experimental conditions.

Genetic Correlation with Milk Yield

Genetic correlations provide information about expected changes in one trait as a correlated response to selection on another trait. Most selection of dairy cattle has been for milk yield, but, more recently, emphasis has begun to shift toward selection for protein yield. A recent estimate of the genetic gain in milk yield was 139 kg of milk/yr per cow (35). With such rapid progress for a trait, changes in genetically correlated traits would be expected, too.

The genetic correlation of mastitis and milk yield or protein yield has been estimated in several studies. Shook (48) reviewed six papers and found that mean genetic correlation of clinical mastitis and milk yield was .20. A more recent study (61) found the correlation of bacterial infection status with milk yield to be .22. Simianer et al. (50) found higher correlations of .51 for mastitis and milk yield. Thus, a slow but steady increase in mastitis incidence is expected to accompany genetic gain for milk yield. Further evidence for the relationship between milk yield and mastitis comes from selection experiments. Hansen et al. (18) reported differences between a line of cattle selected for higher milk yield and a control line. The mean PTA for milk yield of cows from the two lines differed by 653 kg. Total health costs and mammary costs were higher

for the selected line by $7.74 and $4.99 per lactation, respectively. In an Iowa study (14), a line of cattle was selected for milk yield beginning in 1968. By 1989, PTA for milk of cows from that line averaged 612 kg higher than for the control line. During the experiment, the selection line averaged $19.66, $3.37, and $9.76 higher than the control line for total health costs, mammary costs, and discarded milk loss, respectively. The differences would likely have been larger if only cows in the later years of the experiment had been compared. Increased milk yield appears to be genetically associated with increased susceptibility to mastitis and corresponding health costs.

SOMATIC CELL SCORES AS A SUBSTITUTE TRAIT

For a trait to be considered in a breeding program, it must be under a reasonable amount of genetic control, have an important economic value, and be easily measured at an acceptable cost. Clinical mastitis and bacterial infection status meet the first two criteria. Heritability of these traits is about .02 to .10 compared with .25 for milk yield. Rapid genetic progress has been achieved for milk yield, and it seems logical that a small gain could certainly be achieved in controlling mastitis if some selection emphasis were placed on it. The economic importance of mastitis is obvious. However, measurement of mastitis incidence is very inconsistent. Dairy producers in the US are reluctant to record all cases of mastitis, and bacteriological tests are too expensive to use routinely. In some Scandinavian countries, only veterinarians are allowed to treat mastitis cases with drugs and are required to report incidences. Of course, the need for veterinary treatment may be interpreted differently depending on the cow or herd. Difficulty and expense of recording clinical mastitis or bacterial infection status on a large scale make it unlikely that these records would be successfully used for US genetic programs.

Fortunately, SCC in milk may serve as a useful substitute for mastitis in breeding programs. One trait is allowed to substitute for another if it is genetically correlated with the other trait, if recording is less expensive or easier, if measurement is earlier in life, or if heritability is higher.

The genetic correlation between SCC and clinical mastitis or bacterial status is moderately high. Using records of cows in four state-owned herds, Young et al. (71) estimated the correlation of SCC and clinical mastitis to be .80 or .98 from two methods. Afifi (1) reported a correlation of .83. More recently and using more appropriate statistical techniques, Emanuelson et al. (15) found a genetic correlation of .46 for Swedish Black and White cattle and .78 for Swedish Red and White cattle from a field study. Weller et al. (61) found a smaller genetic correlation between somatic cells and clinical mastitis of .30 but attributed the smaller correlation to inaccurate recording of field data. Although there is a range in estimates, the genetic correlation of SCC and clinical mastitis appears to be about .60 (49), which is adequate to achieve genetic progress by selection for a substitute trait. Weller et al. (61) also estimated the genetic correlation of somatic cells with bacterial infection status to be near 1. Thus, genetic selection for lower SCC apparently would reduce subclinical as well as clinical mastitis.

Currently, SCC are recorded for about 80% of cows on DHIA programs (64), and SCC serve as an indicator of mastitis and as a management tool to control mastitis (36). The presence of intramammary infection or inflammation is the major factor affecting SCC of cows (19). Dairy records processing centers transform SCC to a sample day $\log_2$ somatic cell score (**SCS**) (44). These SCS have several statistical advantages over SCC, including normal distribution and uniform variance among samples. Further advantages are explained in other reports (2, 47, 49). An SCS of 3 is equal to an SCC of 100,000 cells/ml. Because a $\log_2$ transformation is used, each doubling or halving of SCC corresponds to a change in SCS of +1 or −1, respectively. Thus, SCS of 1 and 4 correspond to SCC of 25,000 and 200,000 cells/ml. Lowest SCS is associated with lowest rates of mastitis infection (10), which contradicts a popularly held view that elevated SCC is necessary to prevent mastitis.

Recording of SCS has primarily been for management purposes, but records could be used for genetic evaluations, too. The cost of using the data for genetic evaluations would be small because records could accompany records for yield traits, which are already used

in evaluation systems; small costs would be incurred for relatively minor changes in programming. Another advantage of SCS over incidence of clinical mastitis or bacterial infection status is that SCS can be recorded at each sample day for all cows. Incidence traits often may not be recorded until the end of lactation. Records in progress (incomplete lactations) are important for evaluation of sires, especially if accurate and timely evaluations are desired from a limited number of daughters.

The LSCS often has been used as the lactational measure for genetic studies (4, 7, 41, 43). Recent heritability estimates for LSCS range from .05 to .27 (4, 7, 11, 15, 41, 43, 61, 62), and the mean is about .12, which is higher than most estimates of heritability for clinical mastitis or bacterial infection status. Emanuelson et al. (15) reported heritability estimates for LSCS of .11 and .05 compared with estimates for clinical mastitis of .02 and .01, respectively, for two breeds of Swedish cattle from a field study. Weller et al. (61) found that heritabilities were .08 and .04 for LSCS and bacterial infection status for Israeli cattle. Heritability of LSCS appears to be nearly double that of direct measures of mastitis.

Other traits have been suggested as substitute traits or markers for mastitis. Detilleux (12) examined several measures of cell-mediated immunity for their effect and usefulness as indicators of mastitis. Significant sire effects existed for all assays studied, and Detilleux concluded that genetic control existed for general immune function and its relationship to mastitis. Heritabilities of specific measures of cell-mediated immunity ranged widely, and standard errors of estimates were large because of small sample sizes. Heritabilities were high for percentage of neutrophils in blood leukocyte counts, chemiluminescence, cytochrome reduction, and serum protein and immunoglobulin assays. Heritabilities for measures of clinical and subclinical mastitis were about .10. Several alleles of the gene complex for bovine lymphocyte antigen have been associated with clinical mastitis (26, 53, 60). Measures of actual immune function or specific genes or gene markers may assist in measuring the ability of cows to resist mastitis infection. At present, however, measurement of cell function remains too difficult and too expensive for routine evaluation of cattle. Also, the complexity of mastitis and regulation by many genes may downplay the effectiveness of selection for specific alleles of one or a few genes. Young et al. (71) reported negative correlations of udder height with clinical mastitis (–.28) and with bacteriological status (–.38); higher udders were associated with lower incidences of mastitis. Other physiological measures, such as teat shape characteristics (45), certainly play a role in defense systems but would be difficult to measure on a large scale.

The LSCS fits criteria for inclusion in breeding programs as a relatively inexpensive substitute for clinical mastitis or bacterial infection status.

Correlation of SCS with Other Traits

Many studies have reported genetic correlations of LSCS and milk yield (4, 7, 15, 22, 29, 41, 61, 62). In those studies, estimated correlations ranged from –.20 to .48, but most values were closer to the mean of .12. As with mastitis, SCS would be expected to increase slowly as a correlated response to genetic improvement for milk yield. In studies (22, 29, 41) that examined the genetic relationship of SCS and milk yield, mean correlations were .28 for first lactation, –.15 for second lactation, and .05 for third and later lactations. Schutz et al. (41) suggested that mastitis, as indicated by SCS, is more common during first lactations of cows with sires that transmit higher milk yield, perhaps because of the stress from producing more milk. Clinical or subclinical mastitis may limit the potential for milk yield during subsequent lactations. Culling of first lactation cows with severe mastitis may have contributed to lower correlation estimates for later lactations.

Mean correlation of LSCS and fat yield was about .02 (4, 7, 15, 22, 29, 41, 61, 62), which suggested little relationship of the two traits. Studies that looked at the genetic relationship of LSCS and protein yield (7, 22, 29, 41, 62) found estimates of correlations ranged from –.14 to .54 and had a mean of .17. The relationship was more positive than that of LSCS and milk yield in each study. The higher genetic correlation of LSCS and protein yield is especially noteworthy in view of recent trends to place greater selection emphasis on protein yield. However, the antagonistic rela-

tionship of SCS with yield traits means that genetic gain can be achieved in selection for lower SCS only by decreasing selection emphasis on milk and protein yields.

It is important to remember that the positive or antagonistic relationship of SCS and milk or protein yield is on a genetic basis. However, the phenotypic correlation of SCS and milk or protein is negative; i.e., increased SCS, which likely indicates subclinical or clinical mastitis, corresponds to reduced milk and protein yield. In recent studies (4, 7, 15, 22, 29, 41, 62), mean phenotypic correlations of SCS with milk or protein yields were both −.10. The positive genetic correlation and negative phenotypic correlation is a bit confusing. Apparently, cows with a genetic capacity for higher milk yield are genetically more predisposed to mastitis, possibly from the physical stress of increased yield, but incidence of subclinical or clinical mastitis reduces milk or protein yield. Thus, phenotypic milk yield is decreased, yet the genetic ability of the cow to produce milk is constant. Phenotypic correlations reflect both environmental and genetic causes of correlations.

Genetic correlations of LSCS with certain conformation traits may also be relevant to breeding programs because type is considered by most AI organizations. Rogers et al. (38) found genetic correlations of LSCS with udder depth, fore udder attachment, and front teat placement to be −.35, −.32, and −.22, respectively. Lower LSCS is associated with higher, more firmly attached udders with closer teat placement. Schutz et al. (42) found approximate genetic correlations of −.28, −.31, and −.21 for LSCS with the three type traits, respectively. Sire analysts from AI organizations usually screen prospective bull-dams for udder conformation traits and often eliminate cows with deep udders or wide front teats from consideration. Because of the favorable relationship between udder conformation and LSCS, screening on udder characteristics may have slowed the genetic increase in LSCS that could otherwise have accompanied selection for milk and protein yields.

Direct Value of Reduced SCS

Several economic incentives exist to decrease SCS in addition to reducing clinical and subclinical mastitis. Somatic cells have their own economic value. On July1, 1993, the National Conference on Interstate Milk Shipments reduced the legal limit for Grade A milk sold in the commercial market place from 1 million to 750,000 cells/ml. Although the genetic reduction of SCS will not occur quickly enough to meet this deadline, further reductions in legal limits likely will occur (5). Response to such demands through genetics may reduce the need for drug therapy, which improves milk product safety and consumer perception. However, most reduction of SCS to meet such limits must come through improved management practices.

Many dairy plants now are paying quality premiums for milk with low SCC. Milk with more somatic cells has decreased cheese yield and shorter shelf-life because of increased protease activity. Quality adjustments to milk pricing have been proposed in several Federal Milk Market Orders (30).

Genetic evaluations for somatic cells are calculated in several countries (6) and have been proposed for Canada and Germany. Inclusion of genetic evaluations for LSCS should dispel concerns of potential foreign buyers of US semen who were concerned that no indication of mastitis resistance existed in evaluations of US bulls.

GENETIC EVALUATION OF LSCS

Data

Records for LSCS have been contributed to USDA by seven of nine dairy records processing centers through the National Cooperative DHI Program. Some processing centers began contributing records for research purposes during 1987, and the total number of usable LSCS currently available is about 5 million. This number is fewer than the records that are available for milk yield evaluations for several reasons: 1) about 80% of DHIA cows are enrolled in SCC testing programs (64), which are optionally provided for an additional fee; 2) records are not currently contributed by all processing centers; and 3) historical data are available only since 1987, compared with 1960 for milk yield records. Much of the earlier information collected for somatic cells is not only sparse but is also of questionable value

because of inadequate calibration and standardization procedures for testing equipment.

The LSCS are recorded and reported by participating dairy records processing centers to USDA along with number of tests contributing to the mean and DIM on last sample day. For inclusion in evaluation procedures, LSCS must be from 0 to 9.99. Information for LSCS is included with the records of milk, fat, and protein used for yield evaluations. Upon receipt at USDA, records are subjected to the same edits required for inclusion in genetic evaluations for milk yield (33, 67). Some DHIA test plans, such as milk only or owner-sampler records, are not currently used for genetic evaluations. Edits are imposed to ensure the validity and consistency of information for animal identification, sire and dam identification, birth date, calving date, herd, and reported DIM. According to Norman et al. (33), lactation records are most often rejected for invalid or conflicting sire and dam identification and conflicting birth dates. Cow birth dates are compared with dam calving dates and parent birth dates for verification. Sire and dam identification is verified with pedigree information supplied by breed associations and DHIA.

Repeated record procedures, which allow records for more than one lactation per cow, will be used for genetic evaluation of LSCS. The literature is somewhat contradictory about the genetic correlation between LSCS from first versus later lactations but suggests that it is lower than that for milk yield and ranges from .55 to .81 (4, 11, 72). Coffey et al. (9) proposed that LSCS may be somewhat different genetically during first versus later lactations. There is a tradeoff between additional accuracy from including repeated records and the relatively lower repeatability between first and later parities, given that less information is available for LSCS than for milk yield and that PTA for LSCS will have lower REL than PTA for yield traits even for the same amount of information. In fact, the boost to REL from inclusion of additional lactations may be relatively greater for LSCS than for milk yield because of the lower heritability of LSCS. Also, if LSCS is genetically different for first versus later lactations and the overall goal is to enhance mastitis resistance throughout the productive lives of cows, LSCS records from later parities should be considered in evaluation procedures.

Only the first five lactations of cows are used in USDA-DHIA genetic evaluations because additional records contribute relatively little to accuracy. Only lactations of cows that have first lactations available are included in LSCS evaluations (65). Use of later records, if no first lactation record is available, biases results because such records would be only from cows that were not culled for mastitis during their first lactations.

For analysis, a further requirement is imposed: the number of SCC sample days must be representative for DIM; DIM at last score must be $\leq$60, 100, or 140 for 1, 2, or 3 tests, respectively (7). Records are then standardized for DIM at last SCS with separate adjustments for first or second and later lactation. Two sets of lactation adjustment factors are used: one for Guernseys and Jerseys and the other for Holsteins and other breeds. Adjustment for DIM accounts for dilution of SCC because of differing volumes of milk produced at various stages of lactation. Next, records are adjusted for age and season of calving. Influences of these effects on LSCS are well documented (19, 43). Solutions for LSCS increase with age; rates of increase are steeper after about 36 mo of age. Effects of month of calving are smaller than age at calving, but LSCS is highest for cows calving during midsummer. The seasonal fluctuation in LSCS is most pronounced in the Southeast, likely because of increased stress on cows and the presence of mastitis pathogens during hot and humid summer months (19). Thus, these effects are separate for four regions of the US (Northeast, Midwest, Southeast, and West) to reflect possible climatic influences on seasonal effects. Adjustment for systematic differences in stage of lactation and in calving age and season allows fairer comparison of a cow's record with records of her contemporaries that may be in different stages of lactation or have calved at a different age or during a different season.

Animal Model

National genetic evaluations for LSCS use an animal model similar to that used for milk, fat, and protein yields (65, 66). Essentially, an animal model is a simultaneous genetic evalua-

tion of males and females that uses the animal's own performance and the genetic merit of all relatives. The technique considers that a cow's performance is based on her genetic ability and her environment. The model is

$$y_{ijkl} = m_{ij} + a_{kl} + p_{kl} + c_{i'k} + e_{ijkl}$$

where y_{ijkl} = standardized LSCS record of cow kl (daughter l of sire k) in herd i and year-season, parity, and registration group j (i′ represents the herd of evaluation for first lactation). Terms in the model are fixed management group (m) and random animal breeding value (a), permanent environment (p), herd by sire interaction (c), and residual (e) (66).

Management Group. Records of cows calving in the same herd-year-season (2-mo seasons) and in the same lactation (first or later) are combined into management groups (m_{ij}). For Holsteins, cows must also have the same registration status (registered or grade). If fewer than five records occur in a management group, restrictions are relaxed until there are at least five records per group. For example, if a management group had only three records, the criteria would be expanded to a 4-mo season, then to a 6-mo season, and finally to combined registration status until at least five records were included in the management group. Further details are given by Wiggans et al. (65).

Animal. The animal effect (a_{kl}) is the genetic effect common to all records of a cow, and solutions are estimated breeding values. Through inclusion of known relationships among animals, animal models allow estimated breeding values to include information from related cows through breeding values of parents and records of progeny in addition to the animal's own yield. Thus, evaluations may contain contributions from sire, dam, offspring, sisters, cousins, and aunts. Contributions from offspring consider the breeding value of the other parent (merit of mates). Relationships among animals make use of extensive pedigree information from as long ago as 1950 (65). Unknown parents in pedigrees are grouped according to birth year of progeny, and the genetic merit of the group is estimated by the method of Westell et al. (63). For genetic evaluation, animal solutions are of primary interest. Inclusion of other effects in the model or preadjustment of records allows animal solutions to be predicted independently of systematic differences related to those effects.

Permanent Environment. The effect common to all records of a cow that does not arise through additive genetics and is not transmissible to offspring is her permanent environmental effect (p_{kl}). For example, a cow may suffer an injury to a teat early in first lactation, which would make her more susceptible to mastitis and elevated LSCS during all lactations. This effect also includes nonadditive genetic effects such as dominance (how alleles of a gene work in combination with other alleles) and epistasis (nonallelic gene interaction), which are common to all records of a cow.

Herd by Sire Interaction. The effect common to all daughters of a sire in a particular herd is the herd by sire interaction ($c_{i'j}$). This form of interaction between genotype and environment accounts for little variance of LSCS [.02 relative to a phenotypic variance of 1.00 (4, 43)]. Effect of interaction of herd and sire for a cow is based only on the herd of evaluation of the cow's first lactation (66). This effect is part of the phenotype of a cow but not part of her breeding value and, therefore, does not contribute to her sire's performance. Interaction between herd and sire is included in genetic evaluations for milk yield despite accounting for little variation of that trait (4). Inclusion of the effect in evaluation procedures at 5% of phenotypic variance limits the impact of sires with daughters in a single herd.

Residual. The part of the record unexplained by other effects in the model is the residual effect (e_{ijkl}). Thus, the residual is assumed to contain temporary environmental effects that change from lactation to lactation.

Evaluations use BLUP procedures in which the random effects of animal, permanent environment, herd by sire interaction, and residual are assumed to be normally distributed with variances of .10 (heritability), .20, .05, and .65, respectively, relative to a phenotypic variance of 1 (43). Solutions for individual levels of effects are regressed toward their mean value according to the amount of information contributing to prediction of the solution. For example, the solution for a sire with fewer daughters in fewer herds is regressed more toward the mean evaluation of its parents

because there is less confidence that mean performance of daughters accurately measures the sire's breeding value. Thus, more emphasis is given to the parent average and less to daughter performance.

Details about calculation of animal model evaluations have been reported in several studies (8, 65, 66). Essentially, the procedures used are iterative, and estimates for all effects are affected by estimates for all other effects during subsequent rounds of iteration. For example, genetic differences of animals in management groups are taken into account as management group effects are estimated. Similarly, records of a cow are adjusted for management group effects before the effect of her permanent environment is calculated. Processing begins by herd with estimation of management group effects, which are adjusted for solutions of other effects estimated during the previous round of iteration. Then, processing by sire within herd, permanent environmental effect and herd by sire effects are estimated using management group effects already estimated from the current round of iteration and animal effects from the previous round. Methods for including cows with records in more than one herd are reported by Wiggans and VanRaden (66). Finally, records adjusted for other effects in the model are accumulated, and breeding values are computed across herds. Iteration allows breeding values of animals to influence breeding values of all relatives from earlier or later generations after a number of rounds of iteration. Lactation length weights influence estimation of all effects in the model such that records based on few sample days (records in progress or those terminated before 305 DIM) receive less emphasis than records with at least 10 sample d.

Breeding values from animal model procedures are divided by 2 and are reported as PTA. Specific PTA are not directly applicable to a given herd, but rankings of PTA and differences among PTA are relevant for all herds if bulls are assumed to have been mated to cows of equal genetic merit. The difference between PTA for two animals predicts the expected mean differences between their progeny. As with yield traits, PTA of cows and bulls for SCS are adjusted so that mean PTA of cows born during 1985 is 0 (65).

TABLE 2. Mean of sample day $\log_2$ somatic cell scores (LSCS) for first lactation that were standardized for lactation length, calving age, and calving season for cows born during 1985.

Breed	LSCS
Ayrshire	3.08
Brown Swiss	3.04
Guernsey	3.40
Holstein	3.23
Jersey	3.48
Milking Shorthorn	3.83

RESULTS OF PRELIMINARY RESEARCH WITH SCS FOR EVALUATION

In conjunction with work to determine genetic parameters for LSCS for six breeds of cattle, Schutz et al. (43) computed preliminary genetic evaluations for LSCS of cows and bulls. Techniques and models used were similar to those already discussed, and details may be found in their report (43). Mean PTA of cows born in 1985 for the six breeds are in Table 2.

To determine an appropriate format for presenting PTA for LSCS to the dairy industry, USDA cooperated with the task force on genetic evaluations of the National Mastitis Council. Breeding values divided by 2 (PTA) should mostly range from −.5 to +.5 (4, 7, 43). Reporting values with this format might encourage overemphasis in breeding programs, because some breeders might avoid using any bull with a PTA >0 (higher LSCS), regardless of how good the bull may be for other traits, and effectively eliminate half of the available population of bulls. Although use of 0 as a natural selection threshold would allow progress in reducing somatic cells, the loss in overall merit would not be economically justifiable. This problem was circumvented by adding a constant to all evaluations so that all PTA for SCS are positive. The mean of standardized LSCS for first lactation cows born during the base year of 1985 was chosen as the constant. The adjusted values were termed PTA for SCS. First lactation LSCS was chosen because all cows are required to have first lactation records to be included in evaluations. Using standardized means more appropriately reflects the mean SCS for all lactations included in the evaluation procedure. Table 2 has the mean of standardized LSCS for first lacta-

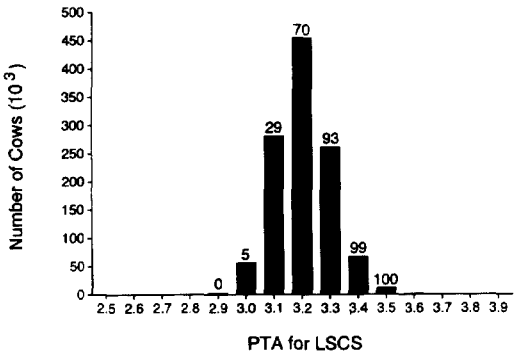

Figure 1. Distribution of PTA for lactation mean of sample day somatic cell scores (LSCS) rounded to the nearest tenth for Holstein cows with records (cumulative percentages reported above bars).

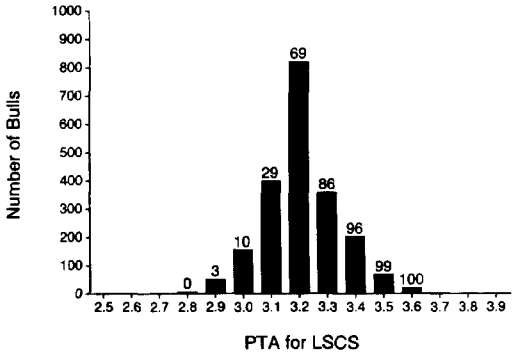

Figure 2. Distribution of PTA for lactation mean of sample day somatic cell scores (LSCS) rounded to the nearest tenth for Holstein bulls with ≥50 offspring (cumulative percentages reported above bars).

tion cows from the preliminary study (43). Means for Guernseys and Jerseys were higher than for other breeds. The mean for Milking Shorthorns likely is low because of the small sample size for that breed (43).

Distribution of PTA for SCS of 1,135,752 Holstein cows with records is in Figure 1 along with cumulative percentage of PTA for SCS rounded to the nearest tenth. The distribution was approximately normal. Nearly all PTA for SCS fell between 2.85 and 3.55, and 88% (5 to 93%) were between 3.05 and 3.35. The distribution of PTA for SCS of bulls with >50 offspring is in Figure 2. For bulls, PTA for SCS were in the same range as for cows, but the distribution was flatter. Most of the PTA for SCS were from 2.85 to 3.55, and only about 76% were from 3.05 to 3.35. Bulls with ≥50 progeny have relatively more genetic information than cows with their own records and few if any offspring. Thus, PTA for SCS of bulls would not be regressed as much as PTA for SCS of cows, which would allow a larger percentage of evaluations for bulls to lie toward the tails of the distribution.

An SCS of 3.2 is equivalent to an SCC of 114,850 cells/ml. Actual values for PTA for SCS do not directly apply to a given herd, but differences between PTA for SCS are important. For a herd exactly at breed mean, daughters of the bull with highest PTA for SCS (3.89) would have mean LSCS 1.14 higher than that of daughters of the bull with lowest PTA for SCS (2.75). More generally, the difference of 1.14 (3.89–2.75) in PTA for SCS means that daughters of the worst bull will have geometric means of SCC $2^{1.14}$ (2.2) times higher than daughters of the best bull. This relationship would be true at any herd level, because there is no appreciable interaction of genotype and environment for LSCS (4, 43). In general, the geometric mean SCC of daughters of the worse bull (higher PTA for SCS) is 2^D times the expected geometric mean SCC of progeny of the better bull (lower PTA for SCS), where D is the difference in PTA for SCS.

Widespread use of AI in the dairy industry allows some bulls to have a large influence on the population. Most genetic progress comes through intense selection of sires of sons by AI organizations (56). Table 3 has preliminary PTA for SCS of daughters of the 10 bulls with the most sons from USDA-DHIA genetic evaluations of January 1993 (L. W. Specht, 1993, unpublished data). The bulls are ranked within breed from best to worst according to PTA for SCS, but numbers are not comparable across breeds. Differences between the best and worst bulls were .43, .39, .38, .68, and .40 for Ayrshires, Brown Swiss, Guernseys, Holsteins, and Jerseys, respectively, which suggests reasonable variation among the bulls with a large impact on the breed. Eight of 10 of the Brown Swiss bulls were better than the breed mean of PTA for SCS. The largest difference in PTA for SCS was for Holsteins, which results from the greater number of bulls

TABLE 3. The PTA for lactation mean of sample day somatic cell scores (LSCS) for bulls with the most sons with USDA-DHIA genetic evaluations.[1]

Rank[2]	Ayrshire	Brown Swiss	Guernsey	Holstein	Jersey
1	2.88	2.70	3.17	3.01	3.28
2	2.97	2.81	3.17	3.03	3.34
3	2.98	2.83	3.19	3.04	3.37
4	3.01	2.84	3.24	3.18	3.38
5	3.03	2.86	3.28	3.23	3.39
6	3.06	2.86	3.30	3.33	3.40
7	3.16	2.94	3.35	3.39	3.50
8	3.25	3.01	3.35	3.44	3.55
9	3.28	3.05	3.39	3.54	3.56
10	3.31	3.09	3.55	3.89	3.68

[1]L. W. Specht (1993, unpublished data).

[2]Rank of bulls for PTA for LSCS within breed.

and the higher REL of evaluations. The worst Holstein bull with more than 20 sons was the fourth worst of all Holstein bulls with any offspring in the preliminary study (43). With positive genetic correlations of LSCS and milk or protein yield, higher PTA for SCS would be expected if all selection is on milk and protein yields. However, AI sire analysts select for other traits, such as udder conformation of daughters, and frequently eliminate bulls with dams that have serious mastitis from consideration as sires of sons. Hansen (17) reported evaluation results for mastitis resistance from other sources.

The task force on genetic evaluations of the National Mastitis Council strongly recommended that PTA for SCS also be reported as part of an economic index with SCS appropriately weighted relative to other economically important traits. Strandberg and Shook (54) found that selection for an index of total economic merit that included somatic cells slowed the rate of gain in yield traits by about 1 to 2% and decreased the rate of increase in clinical mastitis by 20 to 25%. Because yield traits are more highly heritable and are more important economically, optimal breeding programs did not reduce LSCS or clinical mastitis but merely slowed the rate of increase. Rogers (37) concluded that LSCS could be included in breeding programs along with yield, udder conformation, and feet and leg traits with an increase in net merit of 1 to 4%. He found that placing 5 to 8% as much emphasis on PTA for SCS as on PTA for yield traits optimized net merit and slowed the rate of increase in clinical mastitis by about 25%. Although breeders may choose to avoid or to use sparingly the bulls with extremely high PTA for SCS, the best option for including PTA for SCS in breeding programs is in an index with other traits to improve overall economic merit.

Evaluations for LSCS and productive life (longevity) were combined by USDA with those for milk, fat, and protein in an economic index for net merit. Four major economic impacts are associated with elevated SCS: increased subclinical milk loss, increased early culling, decreased value for low SCC milk, and increased costs of clinical mastitis (including discarded milk, labor, and treatment). Subclinical milk loss may reflect the largest cost but is already taken into account if PTA for milk yield is considered. Similarly, increased culling is accounted for by PTA for productive life. A mean value for SCS in terms of quality premiums is difficult to approximate because dairy plants use many different schedules for quality payment. Also, most payment schedules are based on SCC, not on log-transformed SCS. Nevertheless, the mean premium appears to be about $.0026/kg for a doubling or halving (1.00 on an SCS scale) of SCC at about breed mean. Heuven (20) reported the cost of clinical mastitis as 20.04 kg of milk per unit increase in SCS. By combining quality payments and clinical mastitis with the current economic index formula that includes PTA for milk, fat, and protein yields (**MFP$**) (H. D. Norman, March 19, 1993 memorandum to DHIA cooperators) but ignoring productive life, an example index might be

MFP$ – 28.2198(PTA for SCS). In terms of PTA standard deviations, the importance of LSCS relative to yield would be about 4% for such an index. Currently, MFP$ is not reduced for additional feed costs resulting from increased yield potential, but the relative weight of LSCS to yield increases if feed costs are deducted from the index.

REL FOR SCS EVALUATIONS

The REL of PTA for SCS is lower than REL of PTA for milk, fat, and protein yields with equal numbers of records, progeny, and relatives because of the lesser amount of genetic control (heritability). For USDA-DHIA procedures with the animal model, REL are estimated by summing daughter equivalents (DE) (55). One DE is equal to the amount of information contributed to a parent by a standard daughter, which is defined to have one record, an infinite number of management group mates, and the other parent with perfect REL. Total DE for an animal is

$$DE_{animal} = DE_{parents} + DE_{self} + DE_{progeny},$$

where DE_{animal} is the number of DE for the animal of interest, $DE_{parents}$ is the number of DE contributed by parents, DE_{self} is the contribution from cow's own yield, and $DE_{progeny}$ is the sum of the contributions from progeny. Contributions of various sources of information for either milk or LSCS in terms of DE are in Table 4. The conversion from DE to REL is explained by

$$REL_{animal} = DE_{animal}/(DE_{animal} + k_d),$$

where k_d is the ratio of error variance (with dam variance removed) to sire variance in terms of a sire model [$k_d = (4 - 2h^2)/h^2$] (55). For milk with heritability of .25, k_d is 14; for LSCS with heritability of .09, k_d is 42.4. The appearance of k_d in the denominator indicates that REL is lower for LSCS than for milk given the same DE. Note, however, from Table 4 that a proportional increase in information (e.g., from one to five lactations) is worth more DE for the trait with lower heritability.

Overall, REL of PTA for SCS is much smaller than REL of PTA for yield traits. For example, a bull graduating from a progeny-test program may have a sire with 99% REL, a dam with 60% REL, 20 daughters with two lactations, and 20 daughters with one lactation. Thus, DE = 9.2 + 0 + 20(1.4) + 20(1.0) = 57.2, and REL is .80 for milk yield. For LSCS, a likely situation for a similar bull is that the same sire would be at 90% REL and the dam at 30% REL. Because only 80% of DHIA cows are on SCS test, there may be only 16 daughters with two lactations and 16 daughters with one lactation. The DE would be 18.2 + 0 + 16(1.6) + 16(1.0) = 59.8, and the comparable REL would be .59.

Lower REL of PTA for SCS has implications for use in breeding programs. As with any trait, sires and dams should be selected on PTA. The PTA of animals with low REL already are regressed toward the mean of the PTA of their parents; PTA of animals with high REL may deviate more from mean parent PTA. For sire selection, REL should be considered after selection based on PTA. Among the selected bulls, those with higher REL might be used more frequently; PTA of higher REL should remain quite stable, but PTA of lower REL bulls may change as more daughter information becomes available. With low REL, the difference between true transmitting ability and PTA may be large.

Bulls recently graduated from progeny-test programs have a relatively small number of daughters (first crop daughters). Genetic evaluations based on these daughters (combined with other relatives) determine whether a bull has the genetic superiority to be used widely through AI in the population. Daughters of the best bulls that are conceived after this initial selection are second crop daughters. Usually, bulls do not attain high REL from first crop evaluations; however, REL are often adequate for yield traits if the bulls are well sampled. Reliabilities of PTA for SCS will be much lower and may only be high enough to warrant mating a bull to many females after the bull has a large number of second crop daughters that are lactating. Only the best bulls are chosen to be sires of sons, and often this selection is based on first crop information. Most bulls chosen to be sires of sons are selected before large numbers of second crop daughters have lactation records. By the time REL of PTA for SCS are high, most bulls are no longer in consideration for use as sires of sons and have

been replaced by younger bulls because of the rapid genetic progress for yield traits. Therefore, effectiveness of using PTA for SCS for selecting sires of sons may be limited.

The REL of cow PTA for SCS also is low. Perhaps only cows with several sons or with many daughters from embryo transfer will attain high REL. Because the sire pathway is most important for genetic progress, probably the most progress in selection for lower LSCS will come through screening sires of prospective bull-dams. Many of those sires will have attained reasonably high REL of PTA for SCS.

Lower REL of PTA for SCS provide further justification for incorporating PTA for SCS in breeding decisions through an economic index. The impact of low REL of PTA for SCS is less in an index because the REL of PTA for milk, fat, and protein yields and for productive life may be higher. Although REL of such an index is difficult to estimate, it would be much closer to the REL of PTA for traits given more emphasis and with higher REL.

DISCUSSION

Genetic evaluation for LSCS is possible, and genetic improvement for resistance to mastitis through selection for reduced somatic cells can be achieved. The genetic change in mastitis resistance is not proportional in size to the change in LSCS because the genetic correlation of the two traits is not perfect. As a trait, SCS is more easily measured and has higher heritability than clinical mastitis and, therefore, may serve as a substitute trait in breeding programs. There is also a small but direct value to reduced somatic cells from the additional value of milk with fewer somatic cells. The unfavorable correlations with milk and protein yields mean that selection emphasis on those traits must be sacrificed to make genetic progress in decreasing somatic cells.

Questions remain regarding whether or not SCC can become too low through long-term selection (12, 26). Schukken et al. (39) found that cows that were susceptible to mastitis following challenge with *Staph. aureus* had lower somatic cells before challenge than cows that were resistant to infection. The higher level of cells in cows resistant to the challenge was accompanied by higher infection with *Corynebacterium bovis*. By far the greatest cause of elevated SCC in cows is intramammary infection status (19); thus, the infection status of cows in the study by Schukken et al. (39) was probably the underlying factor for

TABLE 4. Example daughter equivalents (DE) from some sources of information contributing to reliability (REL) for milk yield and lactation mean somatic cell score (LSCS).

Relative	Information available	DE	
		Milk	LSCS
Parents[1]	Sire with 50% REL, dam with 20% REL	3.0	9.0
	Sire with 70% REL, dam with 20% REL	4.1	12.3
	Sire with 90% REL, dam with 30% REL	6.0	18.2
	Sire with 99% REL, dam with 60% REL	9.2	28.0
	Sire with 99% REL, dam with 99% REL	14.0	42.4
Self[2]	1 lactation record	4.7	4.2
	2 lactation records	6.7	6.7
	3 lactation records	7.8	8.4
	4 lactation records	8.5	9.5
	5 lactation records	9.0	10.4
Daughter[2,3]	1 lactation record	1.0	1.0
	2 lactation records	1.4	1.6
	3 lactation records	1.5	1.9
	4 lactation records	1.6	2.1
	5 lactation records	1.7	2.3

[1]Parent REL excludes information contributed by this offspring.

[2]Lactation records are assumed to have infinite management group mates.

[3]Other parent is assumed to have REL of 100%.

greater resistance. Goals of selection for lower LSCS are not to reduce the effectiveness or number of cells per se but to reduce clinical mastitis as indicated by high levels of cells. Indeed, work by Coffey et al. (10) showed a strong relationship between higher SCC during first lactation and greater mastitis incidence during later lactations. This relationship held true for all levels of SCC that were studied. Those researchers concluded that cows initially low for SCC are at no greater risk of subsequent infection. Further, McDaniel et al. (27) found that PTA for SCS of sires of cows with mastitis were higher than PTA for SCS of sires of cows with no recorded mastitis in three experimental herds. A unit increase in sire PTA for SCS (doubling of SCC) accounted for a 36% increase in mastitis incidence. As mentioned, previous work (37, 54) indicates that the current rate of genetic increase in LSCS would be slowed but not eliminated through selection in properly designed breeding programs, which helps to alleviate the controversy over whether SCC will become too low.

Concern has been expressed about whether or not selection to reduce LSCS will be effective in reducing mastitis caused by environmental pathogens. Environmental pathogens, including coliform bacteria and species of streptococci other than *Strep. agalactiae*, cause infections characterized by elevated SCC frequently returning to normal after a relatively short time (51). Exposure to environmental pathogens, which thrive in a cow's surroundings, often occurs between milkings and is not limited to the milking process as are the contagious pathogens. Some evidence exists that, as management practices have improved, the proportion of all mastitis caused by environmental pathogens has increased (52). However, because DHIA samples are taken only at 30-d intervals, a large proportion of environmental mastitis may not be detected. Smith et al. (51) reported mean duration of environmental infections was 9 to 17 d. On a lactational basis, LSCS frequently has as many as 10 sample d, and the influence of a single sample day would be greatly reduced. Impact of a small number of sample days (e.g., in early lactation) on genetic evaluation is small because short lactations receive less weight than complete records. Previous work (40) has shown little genetic difference between animals for environmental infections defined as markedly elevated SCC lasting for only 1 sample d. In contrast, chronic cases of elevated SCC were under greater genetic influence. Also, largest expenses from mastitis are likely to come from chronic mastitis infections, which are more likely to be detected with monthly sampling.

Obviously, selection to reduce clinical mastitis cannot replace proper management practices, which must be the first approach to mastitis prevention. Indeed, overemphasizing PTA for SCS in breeding programs would be economically counterproductive. Nevertheless, if properly used, genetic evaluations provide one more tool that producers may use to reduce the need for antibiotic therapy (and thereby the risk of dairy product contamination) to ensure milk quality and to enhance health of dairy cows.

REFERENCES

1 Afifi, Y. A. 1968. The influence of mastitis, milk production and ease of milking on leukocytes in the milk. Neth. Milk Dairy J. 22:83.

2 Ali, A.K.A., and G. E. Shook. 1980. An optimum transformation for somatic cell concentration in milk. J. Dairy Sci. 63:487.

3 Andrus, D. F., and L. D. McGilliard. 1975. Selection of dairy cattle for overall excellence. J. Dairy Sci. 58:1876.

4 Banos, G., and G. E. Shook. 1990. Genotype by environment interaction and genetic correlations among parities for somatic cell count and milk yield. J. Dairy Sci. 73:2563.

5 Bennett, R. 1992. Lead, follow, or get out of the way: the new PMO SCC policy. Page 52 *in* Proc. 31st Annu. Mtg. Natl. Mastitis Counc., Arlington, VA. Natl. Mastitis Counc., Arlington, VA.

6 Bierma, J. 1992. Mastitis scores from Scandinavia help to breed profitable Holsteins. Holstein World 89(16):120.

7 Boettcher, P. J., L. B. Hansen, P. M. VanRaden, and C. A. Ernst. 1992. Genetic evaluation of Holstein bulls for somatic cells in milk of daughters. J. Dairy Sci. 75:1127.

8 Cassell, B. G. 1988. What extension workers need to tell dairy farmers. J. Dairy Sci. 71(Suppl. 2):85.

9 Coffey, E. M., W. E. Vinson, and R. E. Pearson. 1985. Heritabilities for lactation average of somatic cell counts in first, second, and third or later parities. J. Dairy Sci. 68:3360.

10 Coffey, E. M., W. E. Vinson, and R. E. Pearson. 1986. Somatic cell counts and infection rates for cows of varying somatic cell count in initial test of first lactation. J. Dairy Sci. 69:552.

11 Da, Y., M. Grossman, I. Misztal, and G. R. Wiggans. 1992. Estimation of genetic parameters for somatic cell score in Holsteins. J. Dairy Sci. 75:2265.

12 Detilleux, J. C. 1993. Genetic study of immunological parameters in periparturient Holstein cows. Ph.D. Diss., Iowa State Univ., Ames.
13 Dobbins, C. N., Jr. 1977. Mastitis losses. J. Am. Vet. Med. Assoc. 170:1129.
14 Dunklee, J. S. 1991. Comparison of Holsteins selected for high and average milk production. M.S. Thesis, Iowa State Univ., Ames.
15 Emanuelson, U., B. Danell, and J. Philipsson. 1988. Genetic parameters for clinical mastitis, somatic cell counts, and milk production estimated by multiple-trait restricted maximum likelihood. J. Dairy Sci. 71: 467.
16 Falconer, D. S. 1989. Introduction to Quantitative Genetics. 3rd ed. Longman, Inc., New York, NY.
17 Hansen, L. B. 1993. The genetic framework of mastitis. Page 58 *in* Proc. 32nd Annu. Mtg. Natl. Mastitis Counc., Kansas City, MO. Natl. Mastitis Counc., Arlington, VA.
18 Hansen, L. B., C. W. Young, K. P. Miller, and R. W. Touchberry. 1979. Health care requirements of dairy cattle. I. Response to milk yield selection. J. Dairy Sci. 62:1922.
19 Harmon, R. J. 1994. Physiology of mastitis and factors affecting somatic cell counts in milk. J. Dairy Sci. 77:2104.
20 Heuven, H.C.M. 1987. Diagnostic and genetic analysis of mastitis field data. Ph.D. Diss., Univ. Wisconsin, Madison.
21 Jasper, D. E., J. S. McDonald, R. D. Mocherie, W. N. Philpot, R. J. Farnsworth, and S. B. Spencer. 1982. Bovine mastitis research: needs, funding, and sources of support. Page 184 *in* Proc. 21st Annu. Mtg. Natl. Mastitis Counc., Lexington, KY. Natl. Mastitis Counc., Arlington, VA.
22 Kennedy, B. W., M. S. Sethar, J. E. Moxley, and B. R. Downey. 1982. Heritability of somatic cell count and its relationship with milk yield and composition in Holsteins. J. Dairy Sci. 65:843.
23 Lawstuen, D. A., L. B. Hansen, G. R. Steuernagel, and L. P. Johnson. 1988. Management traits scored linearly by dairy producers. J. Dairy Sci. 71:788.
24 Lindstrom, U. B., and J. Syvajarvi. 1980. Breeding for mastitis resistance in cattle—practical possibilities. Page 130 *in* Proc. Conf. Resistance Factors and Genetic Aspects of Mastitis Control, Jablonna, Poland.
25 Lush, J. L. 1950. Inheritance of susceptibility to mastitis. J. Dairy Sci. 33:121.
26 Mallard, B. A., and D. A. Barnum. 1993. *S. aureus* mastitis: genetics and immunity. Page 27 *in* Proc. 32nd Annu. Mtg. Natl. Mastitis Counc., Kansas City, MO. Natl. Mastitis Counc., Arlington, VA.
27 McDaniel, B. T., R. W. Adkinson, and M. M. Schutz. 1993. Regression of incidence of clinical mastitis on sire evaluations for somatic cell score. J. Dairy Sci. 76(Suppl. 1):238.(Abstr.)
28 Miller, R. H. 1984. Traits for sire selection related to udder health and management. J. Dairy Sci. 67:459.
29 Monardes, H. G., and J. F. Hayes. 1985. Genetic and phenotypic relationships between lactation cell counts and milk yield and composition of Holstein cows. J. Dairy Sci. 68:1250.
30 National All-Jersey, Inc. 1992. Equity Newsletter XVII(5). Columbus, OH.
31 Nickerson, S. C., W. E. Owens, and R. L. Boddie. 1993. Effect of a *Staphylococcus aureus* bacterin on serum antibody, new infection, and mammary histology in nonlactating dairy cows. J. Dairy Sci. 76:1290.
32 Norman, H. D., and L. D. Van Vleck. 1972. Type appraisal: II. Variation in type traits due to sires, herds, and years. J. Dairy Sci. 55:1717.
33 Norman, H. D., L. G. Waite, G. R. Wiggans, and L. M. Walton. 1993. New editing system for national dairy genetics data base. J. Dairy Sci. 76(Suppl. 1): 151.(Abstr.)
34 O'Bleness, G. V., L. D. Van Vleck, and C. R. Henderson. 1960. Heritabilities of some type appraisal traits and their genetic and phenotypic correlations with production. J. Dairy Sci. 43:1490.
35 Powell, R. L. 1992. Breeding for profitable Holsteins in the U.S.A. Page 31 *in* Proc. 8th World Holstein Friesian Conf., Budapest, Hungary. MMI Nyomda, Budapest, Hungary.
36 Reneau, J. K. 1986. Effective use of Dairy Herd Improvement somatic cell counts in mastitis control. J. Dairy Sci. 69:1708.
37 Rogers, G. W. 1993. Index selection using milk yield, somatic cell score, udder depth, teat placement, and foot angle. J. Dairy Sci. 76:664.
38 Rogers, G. W., G. L. Hargrove, T. J. Lawlor, Jr., and J. L. Ebersole. 1991. Correlations among linear type traits and somatic cell counts. J. Dairy Sci. 74: 1087.
39 Schukken, Y. H., K. E. Lesslie, T.J.G.M. Lam, and J. Sol. 1993. *Staphylococcus aureus*: incidence, prevalence and risk factors for intramammary infection. Page 19 *in* Proc. 32nd Annu. Mtg. Natl. Mastitis Counc., Kansas City, MO. Natl. Mastitis Counc., Arlington, VA.
40 Schutz, M. M. 1988. Characterization of protein yield, quality, and composition in milk of dairy cattle. M.S. Thesis, Univ. Minnesota, St. Paul.
41 Schutz, M. M., L. B. Hansen, G. R. Steuernagel, J. K. Reneau, and A. L. Kuck. 1990. Genetic parameters for somatic cells, protein, and fat in milk of Holsteins. J. Dairy Sci. 73:494.
42 Schutz, M. M., P. M. VanRaden, P. J. Boettcher, and L. B. Hansen. 1993. Relationship of somatic cell score and linear type trait evaluations of Holstein sires. J. Dairy Sci. 76:658.
43 Schutz, M. M., P. M. VanRaden, and G. R. Wiggans. 1994. Genetic variation in lactation means of somatic cell scores for six breeds of dairy cattle. J. Dairy Sci. 77:284.
44 Sechrist, R. S. 1985. Summary of NCDHIP policies. Natl. Coop. DHI Progr. Handbook, Fact Sheet B-3, Washington, DC.
45 Seykora, A. J., and B. T. McDaniel. 1986. Genetic statistics and relationships of teat and udder traits, somatic cell counts, and milk production. J. Dairy Sci. 69:2395.
46 Shanks, R. D., P. J. Berger, A. E. Freeman, D. H. Kelly, and F. N. Dickinson. 1982. Projecting health cost from research herds. J. Dairy Sci. 65:644.
47 Shook, G. E. 1982. Approaches to summarizing somatic cell counts which improve interpretability. Page 150 *in* Proc. 21st Annu. Mtg. Natl. Mastitis Counc., Lexington, KY. Natl. Mastitis Counc., Arlington, VA.

48 Shook, G. E. 1989. Selection for disease resistance. J. Dairy Sci. 72:1349.
49 Shook, G. E., and M. M. Schutz. 1994. Selection on somatic cell score to improve resistance to mastitis in the United States. J. Dairy Sci. 77:648.
50 Simianer, H., H. Solbu, and L. R. Schaeffer. 1991. Estimated genetic correlations between disease and yield traits in dairy cattle. J. Dairy Sci. 74:4358.
51 Smith, K. L., D. A. Todhunter, and P. S. Schoenberger. 1985. Environmental mastitis: cause, prevalence, prevention. J. Dairy Sci. 68:1531.
52 Smith, K. L., D. A. Todhunter, and P. S. Schoenberger. 1985. Environmental pathogens and intramammary infection during the dry period. J. Dairy Sci. 68: 402.
53 Solbu, H., R. L. Spooner, and O. Lie. 1982. A possible influence of the bovine major histocompatibility complex (BoLA) on mastitis. Proc. 2nd World Congr. Genet. Appl. Livest. Prod., Madrid, Spain VIII:368.
54 Strandberg, E., and G. E. Shook. 1989. Genetic and economic responses to breeding programs that consider mastitis. J. Dairy Sci. 72:2136.
55 VanRaden, P. M., and G. R. Wiggans. 1991. Derivation, calculation, and use of national animal model information. J. Dairy Sci. 74:2737.
56 Van Tassell, C. P., and Van Vleck, L. D. 1991. Estimates of genetic selection differentials and generation intervals for four paths of selection. J. Dairy Sci. 74:1078.
57 Van Vleck, L. D., E. J. Pollak, and E.A.B. Oltenacu. 1987. Genetics for the Animal Sciences. W. H. Freeman and Co., New York, NY.
58 Ward, A. H. 1938. Preliminary report on inheritance of "susceptibility" to severe udder infection (mastitis). N.Z. J. Sci. Technol. 20:109A.
59 Watson, D. L. 1992. Vaccination against experimental staphylococcal mastitis in dairy heifers. Res. Vet. Sci. 53:346.
60 Weigel, K. A., A. E. Freeman, M. E. Kehrli, Jr., M. J. Stear, and D. H. Kelley. 1990. Association of class I bovine lymphocyte antigen complex alleles with health and production traits in dairy cattle. J. Dairy Sci. 73:2538.
61 Weller, J. I., A. Saran, and Y. Zeliger. 1992. Genetic and environmental relationships among somatic cell count, bacterial infection, and clinical mastitis. J. Dairy Sci. 75:2532.
62 Welper, R. D., and A. E. Freeman. 1992. Genetic parameters for yield traits of Holsteins, including lactose and somatic cell score. J. Dairy Sci. 75:1342.
63 Westell, R. A., R. L. Quaas, and L. D. Van Vleck. 1988. Genetic groups in an animal model. J. Dairy Sci. 71:1310.
64 Wiggans, G. R. 1992. NCDHIP participation as of January 1, 1992. Natl. Coop. DHI Progr. Handbook, Fact Sheet K-1, Washington, DC.
65 Wiggans, G. R., I. Misztal, and L. D. Van Vleck. 1988. Implementation of an animal model for genetic evaluation of dairy cattle in the United States. J. Dairy Sci. 71(Suppl. 2):54.
66 Wiggans, G. R., and P. M. VanRaden. 1990. Including information from records in later herds in animal model evaluations. J. Dairy Sci. 73:3336.
67 Wiggans, G. R., and L. G. Waite. 1985. Editing lactation records for USDA-DHIA genetic evaluations. Natl. Coop. DHI Progr. Handbook, Fact Sheet H-9, Washington, DC.
68 Wilcox, C. J. 1992. Genetics: basic concepts. Page 1 *in* Large Dairy Herd Management. H. H. Van Horn and C. J. Wilcox, ed. Am. Dairy Sci. Assoc., Champaign, IL.
69 Wilton, J. W., L. D. Van Vleck, R. W. Everett, R. S. Gutherie, and S. J. Roberts. 1972. Genetic and environmental aspects of udder infections. J. Dairy Sci. 55:183.
70 Young, C. W. 1992. Breeding dairy cattle for disease resistance. Page 42 *in* Large Dairy Herd Management. H. H. Van Horn and C. J. Wilcox, ed. Am. Dairy Sci. Assoc., Champaign, IL.
71 Young, C. W., J. E. Legates, and J. G. Lecce. 1960. Genetic and phenotypic relationships between clinical mastitis, laboratory criteria, and udder height. J. Dairy Sci. 43:54.
72 Zhang, W. C., J.C.M. Dekkers, G. Banos, and E. B. Burnside. 1994. Adjustment factors and genetic evaluation for somatic cell score and relationships with other traits of Canadian Holsteins. J. Dairy Sci. 77: 659.