BREEDING, SELECTION AND SOMATIC CELL COUNTS:

WHERE ARE WE TODAY?

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Genetics can help prevent mastitis and reduce somatic cell count. The purpose of this article is to inform producers, veterinarians, extension personnel, artificial breeding personnel, and genetics specialists about the methods and usefulness of genetics to improve milk quality. The USDA Animal Improvement Programs Laboratory [AIPL] has published genetic evaluations for Somatic Cell Score (SCS) since 1994 (Schutz et al., 1995; Shook and Schutz, 1994).

Despite the high economic cost of mastitis and the strong correlation between mastitis and SCS, genetic evaluations for SCS have attracted little attention among producers or within the AI industry. The tools of genetic improvement are not well understood by specialists in milk quality and mastitis. Likewise, the importance of mastitis and milk quality is not well known by specialists in dairy cattle genetics. Knowledge of the genetics of SCS and mastitis by specialists in milk quality and genetics, will add genetics to the array of tools for improving milk quality.

This article consists of the following sections:
  - Description of SCS data used in genetic evaluation
  - Predicted Transmitting Ability for SCS
  - Genetic base and changes in genetic base for PTA-SCS
  - Selection indexes and PTA-SCS
  - Recommendations for using PTA–SCS
  - Where to find PTA-SCS evaluations
  - Genetic relationship between SCS clinical mastitis
  - International genetic evaluations for SCS and clinical mastitis
  - Summary

Description of SCS Data Used in Genetic Evaluation

Genetic evaluations are computed from performance records of more than 1.7 million cows in Dairy Herd Improvement [DHI] herds throughout the US (Powell and Sanders, 2003). More than 90% of these cows are on somatic cell testing (AIPL, 2004b). The DHI laboratories perform monthly somatic cell counts [SCC] among other tests on milk samples of individual cows. Each SCC test is converted to SCS using a base 2 logarithmic function (Ali and Shook, 1980; Shook, 1993). Individual SCS values are averaged over all months of lactation and adjusted for effects of age, stage of lactation, and month of calving (Schutz et al., 1995). The adjusted average SCS for each lactation of each cow is used in the genetic analysis.
Table 1. Somatic cell counts [cells/ µl] associated with selected values of somatic cell score.

<table>
<thead>
<tr>
<th>SCS</th>
<th>1.0</th>
<th>1.5</th>
<th>2.0</th>
<th>2.5</th>
<th>3.0</th>
<th>3.5</th>
<th>4.0</th>
<th>4.5</th>
<th>5.0</th>
<th>5.5</th>
<th>6.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCC</td>
<td>25</td>
<td>35</td>
<td>50</td>
<td>71</td>
<td>100</td>
<td>141</td>
<td>200</td>
<td>283</td>
<td>400</td>
<td>566</td>
<td>800</td>
</tr>
</tbody>
</table>

Selected SCS and SCC values are shown in Table 1 to illustrate the nature of the conversion. A one point increase [or decrease] in SCS corresponds to a doubling [or halving] of SCC. The scores have a number of advantages over somatic cell counts (Shook, 1993):

1) Heritability, the portion of differences among cows that is due to genetics, is higher for SCS; the ability to distinguish genetic from environmental effects is improved;
2) The relationship between SCS and milk yield loss is linear for SCS;
3) SCS has a Normal frequency distribution – the familiar bell shaped curve; SCC has a strongly skewed frequency distribution in which the mean is higher than the median;
4) Mean SCS can be interpreted as a median; mean SCC is often much higher than the median and there is no consistent relationship between the mean and median;
5) The standard deviation of SCS is consistent among cows within herds or among daughters within sires; this makes it possible to use the same standard deviation within all herds or all sires, and the mean by itself characterizes the differences among herds or sires;
6) The power to distinguish between diseased and healthy cows is greater for SCS than SCC (Berning and Shook, 1992).

Predicted Transmitting Ability for SCS

Genetic evaluations for dairy cattle in the United States are called Predicted Transmitting Ability [PTA]. PTAs are published for cows and bulls for the yield traits – milk, fat, and protein – SCS, productive life, daughter pregnancy rate, calving difficulty, and several type traits. Although PTAs are computed for both cows and bulls, this discussion will focus mostly on bulls because more than 90% of genetic improvement is due to sire selection. New evaluations are published four times each year in February, May, August, and November. With each new evaluation, some AI bulls are removed from active service, new AI bulls are added, and additional daughters and lactations are included in the bulls’ PTAs. The PTAs are based on cows’ first five complete or partial lactations.

The difference between the PTA’s for two bulls is a prediction of the difference in average performance of the bulls’ future daughters. For example, PTA’s for two recently proven AI bulls are listed in Table 2; the bulls differ by 233 lb milk and nearly 0.60 points SCS. Future daughters of Oneida are expected to produce 233 lb more milk and average 0.59 points lower SCS than future daughters of Veto when the bulls’ daughters are in the same herd at the same time. PTA is a measure of the expected performance of a bull’s or a cow’s future daughters. PTA does not predict the average actual performance of bulls’ daughters, because daughter performance is influenced by herd management and other environmental factors. Instead, PTA should be used to forecast differences between bulls’ or cows’ future daughter averages in the same herd at the same time.
Table 2. PTA’s for two recently proven Holstein bulls and the differences between them.

<table>
<thead>
<tr>
<th>Bull</th>
<th>PTA–Milk</th>
<th>PTA–SCS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oneida</td>
<td>2,105</td>
<td>2.85</td>
</tr>
<tr>
<td>Veto</td>
<td>1,872</td>
<td>3.44</td>
</tr>
<tr>
<td>Difference</td>
<td>233</td>
<td>−0.59</td>
</tr>
</tbody>
</table>

Producers and milk quality specialists are more familiar with SCC than SCS. Using SCS in the genetic evaluations may be a barrier to use of PTA–SCS in sire selection. How can the differences among bulls in PTA–SCS be expressed in terms that are more familiar to producers and milk quality specialists? Two approaches are illustrated in Table 3.

The SCC ratio in Table 3 is the ratio of the geometric mean SCC for daughters of the high PTA–SCS bull to the geometric mean for daughters of the low PTA–SCS bull. Geometric means are discussed in detail elsewhere (Shook and Ruegg, 1999). Although the procedures discussed here are technically geometric means, disregarding this technicality is of little consequence. The mean SCC for daughters of the higher PTA bull is the mean SCC for daughters of the lower PTA bull times the SCC ratio in Table 3 that corresponds to the difference between PTA–SCS for the two bulls.

The clinical mastitis incidence rates in Table 3 show the clinical mastitis rates for daughters of the higher bull when the incidence of clinical mastitis for daughters of the lower bull is the value in the Base column. Results are shown for three base rates of clinical mastitis. These incidence rates are derived from the research of Nash, et al. (2000).

Table 3. Factors to express the PTA-SCS difference between two bulls in terms of the ratio of mean SCC and clinical mastitis incidence of their daughters.

<table>
<thead>
<tr>
<th>Base</th>
<th>0.1</th>
<th>0.2</th>
<th>0.3</th>
<th>0.4</th>
<th>0.5</th>
<th>0.6</th>
<th>0.7</th>
<th>0.8</th>
<th>0.9</th>
<th>1.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCC¹</td>
<td>1.07</td>
<td>1.15</td>
<td>1.23</td>
<td>1.32</td>
<td>1.41</td>
<td>1.52</td>
<td>1.62</td>
<td>1.74</td>
<td>1.87</td>
<td>2.00</td>
</tr>
<tr>
<td>CM²</td>
<td>10.0</td>
<td>10.5</td>
<td>11.1</td>
<td>11.6</td>
<td>12.2</td>
<td>12.8</td>
<td>13.5</td>
<td>14.1</td>
<td>14.8</td>
<td>15.5</td>
</tr>
<tr>
<td>CM</td>
<td>20.0</td>
<td>20.9</td>
<td>21.9</td>
<td>22.8</td>
<td>23.8</td>
<td>24.9</td>
<td>25.9</td>
<td>27.0</td>
<td>28.1</td>
<td>29.3</td>
</tr>
<tr>
<td>CM</td>
<td>30.0</td>
<td>31.2</td>
<td>32.4</td>
<td>33.6</td>
<td>34.9</td>
<td>36.2</td>
<td>37.5</td>
<td>38.8</td>
<td>40.2</td>
<td>41.5</td>
</tr>
</tbody>
</table>

¹Mean SCC for daughters of the bull with higher PTA-SCS is the mean SCC for daughters of the bull with lower PTA-SCS times these multipliers. From (Shook, 1993).

²Clinical mastitis (CM) incidence rate for daughters of the bull with higher PTA-SCS. Clinical mastitis incidence rate for daughters of the bull with lower PTA-SCS is in the Base column. Derived from (Nash et al., 2000).
Two examples will illustrate use of this table. One example uses the two bulls shown in Table 2. The second example uses Best Bull and Worst Bull based on the fact that the difference between the best and worst active AI bulls for PTA–SCS is usually about 1 point.

In Table 2 it is shown that the PTA-SCS for Veto is about 0.6 points higher than the PTA-SCS for Oneida. In Table 3, the SCC ratio that corresponds to a 0.6 point PTA-SCS difference between bulls is 1.52. Assume, for example, that in a particular herd the mean SCC for daughters of Oneida is 200 cells /µl; the mean SCC of daughters of Veto is expected to be 200 x 1.52 = 304 cells /µl. In a herd in which Oneida’s daughters average 500 cells /µl, Veto’s daughters would be expected to average 500 x 1.52 = 760 cells /µl. For Best Bull and Worst Bull, the PTA–SCS difference is 1 point and the SCC ratio in Table 3 is 2.0. In other words, the average SCC for daughters of the Worst Bull is expected to be twice the average SCC of daughters of Best Bull in any given herd. For example, if daughters of the Best Bull averaged 125 cells /µl, daughters of Worst Bull would be expected to average 250 cells. In a high SCS herd, if the daughters of the Best Bull averaged 350 cells /µl, daughters of Worst Bull would be expected to average 700 cells /µl.

The incidence of clinical mastitis among daughters of two bulls depends on the incidence among daughters of the lower PTA-SCS bull, given in the “Base” column in Table 3, and the PTA-SCS difference between the two bulls. For example, assume that in a given herd 20% of Oneida’s daughters have clinical mastitis. In that same herd, the clinical mastitis incidence would be 25.9% for the daughters of Veto. In a herd with higher mastitis incidence, if the Oneida daughters had 30% incidence, the Veto daughters are expected to have an incidence of 37.5%. In the extreme case, when the PTA–SCS difference is 1 point, the incidence of clinical mastitis among daughters of Worst Bull would be expected to be around 16% or 43%, respectively, in a herd where the mastitis incidence of Best Bull is 10% or 30%.

Nobody would argue that these differences among bulls in SCC and clinical mastitis are not important. Yet the narrow point scale used to measure SCS may give the illusion that differences among bulls in SCS are of little value.

Figure 1 shows the frequency distribution of PTA–SCS for active AI Holstein bulls from the May 2004 genetic evaluation run. The range of PTA–SCS was 2.64 to 3.65, a difference of 1 point. Notice in Figure 1 that 80 % of PTA’s lie in the middle four classes; a relatively low percentage of bulls are at the low and high extremes. This is due to the low heritability of SCS [10%] and comparatively low reliability of PTA–SCS. Increasing the numbers of cows on SCS testing and increasing the numbers of daughters per bull in young sire testing programs would contribute to increasing the percentage of bulls in the extreme PTA–SCS categories. The low percentage of extreme bulls limits the opportunity for genetic improvement of SCS.
Bulls with PTA–SCS 3.3 and higher should be avoided as herd sires unless they are truly superior for the economically important yield traits [see Figure 1]. This eliminates the worst 10% of bulls without affecting the opportunity for improvement in other traits. Upon inspection of a list of these high PTA–SCS bulls one will find several that have large numbers of daughters, many of which are not exceptional for any important traits. The economic cost of mastitis among daughters of these bulls is considerable and it can be avoided. Also, this group of high PTA–SCS bulls includes some of the top bulls for yield and other traits. Some of these may be worth using in spite of their high PTA–SCS. On the other hand, other bulls with similar genetic merit for the most important traits may be found that have lower PTA–SCS; these should be used in favor of the high PTA–SCS candidates.

Reliability of PTA is a measure of the accuracy of PTA. In statistical terms, reliability is the squared correlation between PTA and true transmitting ability. Reliability depends on heritability of the trait, the amount of performance information used in computing PTA, and the genetic relationship of the individual being evaluated with the animals involved in the performance measure. Reliability for bulls increases as additional daughters are included in successive genetic evaluation runs. In the May 2004 active AI Holstein sire summary, the reliability for PTA–Milk ranged from 54 to 99% and averaged 88%; for PTA–SCS reliability ranged from 35 to 99% and averaged 76% (NAAB, 2004). Reliability is higher for PTA–Milk.
because milk yield has a higher heritability than SCS [30% vs. 10%] and because more cows are recorded for milk than for SCS. When a bull’s reliability is low, say less than 65%, there is some chance that his PTA will increase or decrease as more daughters are included in future evaluations; when reliability is high, say above 90%, subsequent PTA’s are likely to change very little from their current values. *Producers should use high reliability bulls more heavily and low reliability bulls sparingly. Reliability should not be used as a basis for deciding which bulls to use.* Too often, producers will choose a bull with high reliability and low PTA in preference to a higher PTA bull with lower reliability; these decisions result in lower genetic value and poorer performance in the next generation of herd replacements.

**Genetic Base and Changes in Genetic Base for PTA-SCS**

New genetic bases are introduced for all traits every fifth year. The next base change will be done in 2005. Average PTA–SCS values may change when a new base is set. PTA’s are measured against a genetic base. An analogy to a genetic base is the zero point on the Celsius temperature scale. The freezing point of water is used as the base or zero point for Celsius temperature readings. *The genetic base is determined from the average of cows born in a specified year.* In August 2000, the base year was set by the average of cows born in 1995. In 2005, the base will be set by the average of cows born in 2000. Updating the base serves to keep the average PTA near current breed averages as genetic improvement raises the level in each breed. For most traits, the base value is set to zero; as an example, the average PTA for milk yield for cows born in 1995 is set to zero. The PTA for all cows and bulls for milk yield, then, is expressed as a difference from the average cow born in the base year which is set at zero. Each breed has its own genetic base, so PTA’s are not comparable between breeds.

**Table 4.** Breed averages for the previous [1990] and current [1995] genetic base years, genetic change in PTA–SCS from 1990 to 1995, and management change in PTA–SCS by breed [data from AIPL, 2004a].

<table>
<thead>
<tr>
<th>Breed</th>
<th>Breed Average</th>
<th>Genetic Change</th>
<th>Management Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ayrshire</td>
<td>3.15</td>
<td>0.02</td>
<td>-0.24</td>
</tr>
<tr>
<td>Brown Swiss</td>
<td>3.22</td>
<td>0.01</td>
<td>-0.15</td>
</tr>
<tr>
<td>Guernsey</td>
<td>3.35</td>
<td>-0.03</td>
<td>-0.10</td>
</tr>
<tr>
<td>Holstein</td>
<td>3.20</td>
<td>0.02</td>
<td>-0.09</td>
</tr>
<tr>
<td>Jersey</td>
<td>3.30</td>
<td>0.10</td>
<td></td>
</tr>
</tbody>
</table>

*1 Breeds average of first lactation SCS for cows born in the indicated base years

For SCS, the base is not set to 0; instead it is set equal to the average first lactation SCS of cows born in the base year. Table 4 shows breed averages for the 1990 and 1995 base years. Changes in breed average are due both to improvements in management and changes in genetics. Genetic change was calculated from the average PTA–SCS for cows born in 1995 minus the average for cows born in 1990; results are shown in Table 4. The desired genetic change for SCS is negative. Genetic change has been negligible in all breeds except Jersey; the Jersey breed continues to show a long term, slow, and unmistakable increase in genetic level of SCS (AIPL, 2004a). The changes in management from 1990 to 1995 shown in Table 4 indicate that
producers continued to improve their management of udder health as measured by SCS. This improvement in management was the major reason for the lower genetic base for PTA–SCS.

Selection Indexes and PTA-SCS

The two most widely used selection indexes used in dairy sire selection are Lifetime Net Merit [LNM] and the Holstein Association’s Type-Production Index™ [TPI] (VanRaden and Seykora, 2003; Holstein Association, 2004). The indexes are widely used for screening lists of dairy bulls. Indexes are an easy way to characterize and rank bulls while giving consideration to several traits simultaneously. An index considers a bull’s strengths and weaknesses for several traits to arrive at a single value that represents the bull’s overall merit. Ideally, the weights applied to the several traits in an index reflect the real economic values of the traits. Economic values for the yield traits are relatively well defined; for the non-yield traits, these values are more subjective. The LNM equation and its derivation are given elsewhere (VanRaden and Seykora, 2003). Index weights for the traits in LNM are given as dollars per unit of the trait. The formula for TPI is in Holstein Type-Production Sire Summaries (Holstein Association, 2004). Index weights for the traits in TPI are arbitrary TPI points per unit of the trait. The index weights are listed in Table 5. The weights on SCS are negative because high SCS are undesirable. Also shown in Table 5 are the standard deviations of transmitting ability [SDTA] for each trait.

Table 5. Standard deviation of transmitting ability [SDTA] and selection index weights for Lifetime Net Merit [LNM] and Type-Production Index (TPI).

<table>
<thead>
<tr>
<th>Trait, units</th>
<th>SDT A$^1$</th>
<th>LNM</th>
<th>TPI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Trait units/SD</td>
<td>$/ Trait unit</td>
<td>Points/ Trait unit</td>
</tr>
<tr>
<td>Milk, lbs/305 d</td>
<td>832</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Fat, lbs/305 d</td>
<td>32</td>
<td>2.54</td>
<td>2.64</td>
</tr>
<tr>
<td>Protein, lbs/305 d</td>
<td>25</td>
<td>4.81</td>
<td>6.25</td>
</tr>
<tr>
<td>Productive Life, mos</td>
<td>1.5</td>
<td>26.00</td>
<td>40.33</td>
</tr>
<tr>
<td>SCS, pts</td>
<td>.20</td>
<td>-166.00</td>
<td>-126.92</td>
</tr>
<tr>
<td>Dau. Preg. Rate, %</td>
<td>1.4</td>
<td>17.00</td>
<td>—</td>
</tr>
<tr>
<td>Serv. Sire Calv. Diff., %</td>
<td>1.7</td>
<td>-5.00</td>
<td>—</td>
</tr>
<tr>
<td>Dau. Calv. Diff., %</td>
<td>1.4</td>
<td>-5.00</td>
<td>—</td>
</tr>
<tr>
<td>Type, pts</td>
<td>.70</td>
<td>—</td>
<td>70.71</td>
</tr>
<tr>
<td>Size, pts</td>
<td>.94</td>
<td>-12.00</td>
<td>—</td>
</tr>
<tr>
<td>Udder, pts</td>
<td>.78</td>
<td>33.00</td>
<td>41.25</td>
</tr>
<tr>
<td>Feet and Legs, pts</td>
<td>.88</td>
<td>15.00</td>
<td>19.41</td>
</tr>
</tbody>
</table>

$^1$Standard Deviation of Transmitting Ability (from VanRaden and Seykora, 2003)

In one way, the selection index weights in Table 5 are easy to interpret because they show the values that are applied to the PTA for each trait in calculating a bull’s index value. A bull’s index value is obtained by multiplying the index weight by the PTA for each trait and summing the products over all traits. In another way, the index weights are potentially misleading and difficult to interpret because the genetic variability of the traits differs widely from one trait to
another. Genetic variability is shown in Table 5 as standard deviation of transmitting ability (SDTA). Comparing the weights for protein yield [\$4.81/ lb] and SCS [\$166/ point] in LNM, for example, it might appear that SCS is weighted very heavily in relation to protein. Multiplying the index weight for each trait by the SDTA for that trait shows that one standard deviation of change in protein has a value of \$120 while one standard deviation of change in SCS has a value of only \$33. Genetic change is proportional to SDTA. Therefore, it is appropriate to use the product of index weight times SDTA for each trait as a measure of the relative importance of each trait in the index. The Holstein Association uses standard deviations of PTA that are somewhat smaller than those shown here; the Holstein assumptions are shown in the Holstein Type-Production Sire Summaries (Holstein Association, 2004).

Table 6. Relative index weights for Net Merit $ (NMS), Lifetime Net Merit (LNM), and Type-Production Index (TPI).

<table>
<thead>
<tr>
<th>Trait</th>
<th>LNM</th>
<th>TPI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milk</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Fat</td>
<td>0.68</td>
<td>0.50</td>
</tr>
<tr>
<td>Protein</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Productive Life</td>
<td>0.32</td>
<td>0.31</td>
</tr>
<tr>
<td>SCS</td>
<td>-0.28</td>
<td>-0.14</td>
</tr>
<tr>
<td>Dau. Preg. Rate</td>
<td>0.20</td>
<td>—</td>
</tr>
<tr>
<td>Serv. Sire Calv. Diff.</td>
<td>-0.07</td>
<td>—</td>
</tr>
<tr>
<td>Dau. Calv. Diff.</td>
<td>-0.06</td>
<td>—</td>
</tr>
<tr>
<td>Type</td>
<td>—</td>
<td>0.42</td>
</tr>
<tr>
<td>Size</td>
<td>-0.09</td>
<td>—</td>
</tr>
<tr>
<td>Udder</td>
<td>0.21</td>
<td>0.28</td>
</tr>
<tr>
<td>Feet and Legs</td>
<td>0.11</td>
<td>0.14</td>
</tr>
</tbody>
</table>

The relative selection index weights in Table 6 show the importance of each trait in LNM and TPI relative to protein yield. These were calculated as the dollar value per standard deviation for each trait divided by the economic value per standard deviation for protein yield. In this way, it is quite easy to see the relative influence of traits on the index. The LNM index gives nearly 30% as much weight to SCS as it does to protein. In the TPI index, SCS has only 14% as much emphasis as protein yield. Because the yields of milk, fat and protein are highly correlated, the effect of the yield traits on the indexes is greater than the sum of their weights would imply. As a result, the non-yield traits actually have less influence on the indexes than these calculations suggest.

The assignment of index weights to SCS is controversial due to the lack of a comprehensive economic analysis of the association of SCS with mastitis prevention and treatment costs. Specifically, the need is for a documentation and analysis of the extent to which mastitis prevention and treatment costs change per unit of increase in SCS. Costs that should be included in such an analysis are: drugs used in treatment of clinical mastitis, veterinary treatment and diagnostic costs, milk discarded after antibiotic treatment, and herd labor involved in treatment and diagnosis. Among geneticists it is generally agreed that most of the yield losses from clinical and subclinical mastitis are reflected in measures of milk yield; for that reason, including yield loss in the index weight for SCS would be redundant. In addition to these real costs, there are
some intangibles that would justify even greater economic weight to SCS in the selection indexes. These intangibles include improved animal well being, reduced need for antibiotic treatments, and the nuisance of dealing with diagnosis and treatment of mastitis. In the absence of good information, the industry has taken a conservative approach to weighting SCS in the selection indexes. In my view, TPI considerably undervalues SCS. In the LNM index increasing the weight on SCS to a relative weight that is half the value of protein may be justifiable. This would not seriously reduce the genetic improvement for the yield traits, and it would create real improvement in genetic resistance to mastitis.

Recommendations for Using PTA–SCS

Although PTA–SCS was introduced more than a decade ago, the industry – producers, artificial breeding specialists, veterinarians, and milk quality specialists have been slow to use it as a tool for mastitis prevention. While mastitis is an important problem in dairy herds, it is only one of many concerns producers face. Also, the process of sire selection suffers from information overload; the large number of traits with genetic evaluations, measures of reliability, inbreeding, and other issues contribute to this problem. The use of a selection index consolidates much of this information into a single quantity. Producers are very aware that lower SCC increases milk quality premiums in many markets. The milk yield losses associated with high SCC and costs associated with clinical mastitis are frequently forgotten as benefits from improving milk quality.

From the perspective of the AI industry, PTA–SCS may do more to hinder than to enhance the marketing of bulls. A bull with desirable, low PTA–SCS and otherwise favorable genetic evaluations may not generate the additional revenue needed to offset the potential loss of sales from a bull with undesirable, high PTA–SCS that is outstanding in other respects. The AI industry does respond to market forces. Currently producers’ interest in type, even more than production, drives the semen market. Only a strong interest in PTA–SCS on the part of producers will raise the profile of PTA–SCS among decision-makers in the AI industry.

Veterinarians and milk quality specialists could be most helpful in raising awareness and use of PTA–SCS by producers. Genetics is an area in which these professionals do not normally work, so the comfort level with using genetic information is, understandably, quite low. Conversely, milk quality issues are almost never the concern of semen distributors. The following recommendations are provided to enable all segments of the industry to make appropriate use of PTA–SCS.

Recommendation 1: Include sire selection as a point on the check list when consulting with herds for mastitis and milk quality. Recognize that sire selection is a long term, not a short-term solution to improvement. For this reason, sire selection will not be a high priority for herds facing loss of their milk market, but it should be part of every herd’s long-term outlook. Because genetic improvement is a long-term process, even herds with excellent udder health management should avoid sires with high PTA–SCS.

Recommendation 2: Herds with low average SCC should give the same attention to PTA–SCS in sire selection as herds with high average SCC. Genetic differences among bulls’ daughters are expressed to the same extent in both high and low SCC herds (Banos and Shook, 1990). Consider for example two bulls whose PTA–SCS differ by .80 points. If the daughters of the low bull have average SCS of 2.0, daughters of the high bull will average 2.8; in a herd in which
the daughters of the low bull average 5.0, daughters of the high bull will average 5.8. Although few daughters of the high bull would be above the regulatory limit in either of these herds, that bull’s daughters will experience a higher incidence of mastitis in both herds.

Genetic improvement is a cumulative process. Using consecutive generations of sires with low PTA–SCS will produce future generations of cows with increasingly lower levels of SCS. The costs of diagnosing and treating mastitis can be reduced, even in well managed herds, when low SCS sires are used generation after generation.

**Recommendation 3**: Producers who can manage artificial insemination successfully should use AI bulls. A wealth of genetic information and true genetic superiority are available through AI that is not available for non-AI bulls. Producers who cannot manage AI, should purchase bulls from dams with favorable USDA-AIPL genetic evaluations and sired by well-qualified AI bulls. The same tools used for selecting AI bulls should be applied to selecting the sires and dams of non-AI bulls.

**Recommendation 4**: Use the LNM index as an initial screening for selecting AI bulls. This will provide a moderate amount of selection emphasis on PTA–SCS. Producers should create a ‘long list’ of candidate bulls using the LNM index. The TPI, which is favored by many producers, has a smaller amount of emphasis than LNM on PTA–SCS. If TPI is used in the initial screening, additional consideration should be given to PTA–SCS.

**Recommendation 5**: Include PTA–SCS among the criteria for selecting a ‘short list’ of service sires from the ‘long list’. Avoid the 5 to 10% of bulls with the highest PTA–SCS; for the current genetic base in Holsteins, these are the bulls with PTA–SCS 3.3 and higher. Other traits to consider in forming the short list are productive life, daughter pregnancy rate, calving ease, genetic defects, type, and semen cost.

**Where to Find PTA-SCS Evaluations**

Often the best source of information for genetic evaluations is the internet. Printed lists of bulls in the popular dairy magazines and AI company publications sometimes list PTA–SCS evaluations, but many do not. While each of the AI companies maintains a website with sire information, the genetic evaluation data aren’t always complete at these sites. Three sites that provide comprehensive genetic evaluation data are outlined here.

The National Association of Animal Breeders is the trade organization of the artificial breeding industry in the US. Their site, [http://www.naab-css.org/](http://www.naab-css.org/), provides the option to create a customized list of bulls that meet the user’s criteria. You may also download the complete list of active AI bulls as a comma delimited text file and place it into a spreadsheet or other personal computer application. Genetic evaluations for production, SCS, calving ease, fertility, and type traits are included in these files.

The USDA–AIPL computes genetic evaluations for yield traits, SCS, and productive life for all cows and their sires. The AIPL website, [http://aipl.arsusda.gov/](http://aipl.arsusda.gov/), provides extensive information about the genetic evaluation system and genetic improvement in the US. Results for both AI and non-AI bulls are available at the AIPL site. Facilities are provided to obtain complete information for individual cows and bulls and to obtain lists of bulls in various forms. Type
data are not available at the AIPL site. A useful feature of the AIPL site is its extensive list of links to the breed associations, domestic and international AI companies, Dairy Herd Improvement organizations, and others.

The “Dairy Bulls Dot Com” site, http://www.dairybulls.com/, provides genetic evaluations on AI bulls for SCS as well as all production and type traits. This site allows the user to look up information on individual bulls and to create ranked lists of bulls that meet criteria specified by the user.

Genetic Relationship Between SCS Clinical Mastitis

Including SCS among the criteria for sire selection is driven by the goal to improve genetic resistance to mastitis. Using SCS as a proxy for clinical mastitis in breeding programs is justified by the fact that the major factor affecting SCS is the presence of intramammary infections (Harmon, 1994). Several studies have shown a positive, unfavorable genetic correlation between milk yield and clinical mastitis (Emanuelson et al., 1988; Rogers et al., 1998, Shook, 1993); this implies that genetic improvement for milk yield is accompanied by increases in genetic susceptibility for mastitis. Therefore, it is important to place some selection emphasis on udder health traits to offset the undesirable genetic trend toward mastitis susceptibility that results from selection for increased milk yield.

Clinical mastitis treatments are routinely recorded in the Scandinavian countries, and these records are used in genetic evaluation programs (Emanuelson, 1988; Ruane et al., 1997). In the US, clinical mastitis records are not maintained as part of the DHI program, but SCC data are available for more than 90% of cows (AIPL, 2004b). How effective is SCS as a proxy for clinical mastitis in genetic improvement programs? The answer to this question hinges mostly on the genetic correlation between SCS and clinical mastitis. Earlier reviews have summarized the findings (Emanuelson et al., 1988; Mrode and Swanson, 1996, Nash et al., 2000; Shook, 1993); the focus here will be on two recent studies.

The relationship of cows’ clinical mastitis episodes in first and second lactations with their sires’ PTA–SCS was investigated for 1,860 cows in eight herds (Nash et al., 2000). Results consistently showed the lowest incidence of clinical mastitis and the fewest numbers of mastitis episodes occurred among daughters of bulls with lowest PTA–SCS, and the mastitis measures increased with increasing PTA–SCS. A one-point difference in the sire’s PTA–SCS was associated with around 10% increase in daughters’ mastitis prevalence in both first and second lactation (see Table 3).

Rogers et al. (1998) examined genetic correlations between daughters of Holstein bulls recorded in the US for SCS and daughters of the same bulls recorded in Denmark and Sweden for clinical mastitis. The study involved 80 bulls in the US–Denmark data and 85 bulls in the US–Sweden data. These bulls had >60% reliability for SCS in the US [average was >95%] and at least 50 daughter equivalents for clinical mastitis in Denmark or Sweden. The genetic correlations of SCS with clinical mastitis were .66 in Denmark and .49 in Sweden. Within the Swedish Holstein population, the correlation between SCS and clinical mastitis has been estimated at .70 (Philipsson et al., 1994). In the Danish portion of the study, the regression of clinical mastitis on PTA–SCS was .0972 ± .0163; a one point difference between PTA–SCS of two bulls is associated on average with nearly a 10% higher rate of clinical mastitis. In the Swedish data, the
regression of PTA–SCS on an index of clinical mastitis was $9.58 \pm 2.95$. This statistic is difficult to interpret in terms of the rate of clinical mastitis in bulls’ daughters because the trait is scaled to have a mean of 100 and standard deviation of around 6.

Genetic correlation estimates between clinical mastitis and SCS tend to have large standard errors, and the estimates vary rather widely due to their lack of precision. The several estimates that have been made are centered in the range of 65 to 70%. This is a strong association and indicates that selection of bulls for low SCS is effective as a means of producing new generations of cows that are genetically more resistant to mastitis. One should not expect these correlations to approach the upper limit of 1.0 because clinical mastitis is measured subjectively and only the more severe cases of mastitis are treated and recorded. The research is quite clear that daughters of bulls with the lowest PTA–SCS have the lowest rates of clinical mastitis.

**International Genetic Evaluations for SCS and Clinical Mastitis**

In 2001 Interbull, the International Bull Evaluation Service, introduced international genetic evaluations for somatic cell score and clinical mastitis (Interbull, 2004). International genetic evaluations for the yield traits have been computed since the early 1990’s. Such international evaluations of dairy bulls are possible because some bulls have daughters in more than one country and because many bulls have sons in several countries. These genetic ties across countries enable the use of performance records for genetic evaluation of bulls in all participating countries regardless of the bull’s country of origin. Each country conducts its own domestic genetic evaluation. Interbull uses these various domestic evaluations, the pedigree relationships among bulls, and genetic correlations among countries to compile an international evaluation for all bulls. Because the genetic correlations between countries are less than 1.0, each bull has a separate evaluation in each country. The Interbull results are expressed on the same scale and in relation to the same genetic base as each country’s domestic evaluation, in other words, each bull gets a different evaluation in each country. This makes it possible for producers and AI personnel to fairly evaluate the genetic merit of bulls regardless of the country of origin. In most countries, if the Interbull evaluation has a significantly higher reliability than the domestic evaluation due to a large number of daughters or sons proven in another country, the Interbull evaluation is published in place of the domestic one.

In May 2004, 20 countries participated in the international SCS evaluations for all five of the major US dairy breeds (Interbull, 2004). Three countries conduct Holstein evaluations for clinical mastitis: Denmark, Finland, and Sweden. Genetic correlations between countries for SCS are quite high. For example the genetic correlations between the US and other countries are: Canada, .92; Denmark, .86; Finland, .77; France, .92; Germany, .85; Great Britain, .90; Israel, .86; The Netherlands, .89; and Sweden, .91 (Mark et al., 2000; Mark et al., 2002). These high correlations may make it possible to obtain higher reliability evaluations for SCS for some US bulls by using data from other countries. Current methodology does not allow the use of clinical mastitis data in one country to contribute to the genetic evaluation for SCS in another country; such methods may be developed in the future.

**Summary**

Genetic differences among cows and bulls for clinical mastitis, SCS and SCC are substantial and economically important. Average SCC of daughters of the bulls with highest PTA–SCS is twice
the average SCC of daughters of bulls with the lowest PTA–SCS. In terms of clinical mastitis, daughters of the highest PTA–SCS bulls have clinical mastitis rates 10% higher than daughters of the lowest PTA–SCS bulls in the same herd environment.

While management is most effective in the short term for correcting milk quality and mastitis problems, genetic improvement must be a component of any effective long-term strategy for improving milk quality and udder health. Producers who have achieved excellent milk quality and mastitis management will benefit from selecting bulls with low PTA–SCS to the same extent as those with troublesome rates of mastitis or poor milk quality. Genetic change is a slow, long-term process, so attention to PTA–SCS and other measures of genetic merit must be applied consistently year after year to maintain continuous improvement.

Genetic evaluations of bulls for SCS have been underutilized as a tool for reducing incidence of mastitis. Ultimately, the market demands of knowledgeable producers will drive the change toward greater emphasis on PTA–SCS as a criterion for sire selection by the AI industry. Veterinarians, milk quality specialists, and geneticists all have roles to play in promoting appropriate use of PTA–SCS by producers. It is incumbent upon animal health and milk quality specialists to become familiar with PTA–SCS in order to create knowledgeable producers.

References


