

**IMPACT OF SIRE PTA_{SCS} ON MASTITIS RESISTANCE AND MEASURES OF
DAUGHTER PERFORMANCE**

by

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ABSTRACT

Research to determine the impact of PTA_{SCS} on incidence of mastitis, daughter response to infection, and other measures of daughter performance was conducted using data on 1st, 2nd, and 3rd lactation Holsteins obtained from the Virginia Tech herd and from VA DHI herds. Overall correlation of PTA_{SCS} to lactation average SCS ranged from 0.13 to 0.17 across all data sets. Correlations between PTA_{SCS} and 1st lactation SCS measures were higher than those between PTA_{SCS} and SCS in later parities, but higher correlations were found between 2nd and 3rd lactation SCS measures than between 1st and later parities. Correlation of lactation average SCS and incidence of clinical mastitis was 0.41. Regression of lactation average SCS and averages of test day SCS measures on PTA_{SCS} was significant in 1st, 2nd, and 3rd lactations. All significant relationships were linear and equal or close to 1.0. Relationships between PTA_{SCS} and number of cases of clinical mastitis (.79), number of treatments (2.0), number of days treated (7.0), changes in SCS from beginning to end of a lactation (-.26), and the slope of changes in test day SCS with DIM (5.9×10^{-4}) were significant only in 1st lactation. No significant relationships between PTA_{SCS} and measures of clinical mastitis or variation in test day SCS measures were found in 2nd or 3rd lactations. Heavy cull rates imposed on 1st lactation cows in the Virginia Tech herd explained lack of significance in the later parities in the herd study, but results in following analyses indicated that measures of SCS in 1st and later parities may be two different, but correlated, traits. The greatest impact of PTA_{SCS} on measures of daughter performance and profit was the negative relationship between PTA_{SCS} and herd life. Increased PTA_{SCS} resulted in the decreased ability to survive involuntary culling, and thus decreased opportunity for lifetime yield and profit. Selection on PTA_{SCS} should be an effective method of reducing incidence of clinical mastitis, lactation average SCS, and variation in SCS, or response to infection. The response, however, may be different in 1st lactation than in later parities.

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TABLE OF CONTENTS

ABSTRACT	ii
ACKNOWLEDGEMENTS	iii
LIST OF TABLES	v
LIST OF FIGURES	viii
INTRODUCTION	1
REVIEW OF LITERATURE	5
Introduction.....	5
Relationship of milk yield, measures of mastitis, and associated costs.....	9
Correlated responses in mastitis and health costs.....	10
Selection for Mastitis Resistance.....	13
Somatic cells in milk.....	13
Genetic Evaluation of SCS.....	15
Selection on PTA _{SCS} for improved resistance to mastitis.....	19
Impact of Selection on Defense Mechanisms.....	21
The Relationship of Economic Merit and Reduced Incidence of Mastitis...	22
 CHAPTER I:	
ABSTRACT	25
INTRODUCTION	26
MATERIALS AND METHODS	29
Data Set I.....	29
Data Set II.....	31
RESULTS AND DISCUSSION.....	35
Data Set I.....	35
Data Set II.....	47
CONCLUSIONS	60
 CHAPTER II:	
ABSTRACT	62
INTRODUCTION	63
MATERIALS AND METHODS	66
RESULTS AND DISCUSSION.....	69
CONCLUSIONS.....	80
CONCLUSION	87
REFERENCES	91
APPENDICES	96

LIST OF TABLES

Table 1:	Milk production loss per lactation associated with SCS.....	17
Table 2:	Responses in milk yield, mastitis, and total merit from selection indexes with different index traits.....	20
Table 3:	Values and costs for components in fluid and cheese markets.....	33
Table 1.1:	Means, ranges, and SD of PTAs of the 74 sires of daughters in the data set.....	35
Table 1.2:	Means, and SD for daughter variables analyzed for 1 st , 2 nd , and 3 rd lactations.....	36
Table 1.3:	Correlations of daughter variables and sire PTA _{SCS}	37
Table 1.4:	Regression coefficients and SE of 1 st lactation variables on PTA _{SCS}	38
Table 1.5:	Regression Coefficients and standard errors from 2 nd and 3 rd lactation analysis.....	40
Table 1.6:	Regression coefficients and SE of PTA _{SCS} on differences in variables related to mastitis resistance between lactations.....	41
Table 1.7:	Percentage of cows grouped by sire PTA _{SCS} culled after 1 st and 2 nd lactation.....	44
Table 1.8:	Cows grouped by number of lactations initiated.....	45
Table 1.9:	Means, SD, and correlation to sire PTA _{SCS} of measures of daughter performance and sire genetic evaluations.....	47
Table 1.10:	Linear regression coefficients and SE for measures of daughter performance on sire PTA _{SCS}	50
Table 1.11:	Linear and quadratic regression coefficients and SE for measures of daughter performance on sire PTA _{SCS}	52
Table 1.12:	Linear, quadratic, and cubic regressions and SE for measures of daughter performance on PTA _{SCS}	53
Table 1.13:	Comparison of R-Square values from linear, quadratic, and cubic regression of measures of daughter performance on PTA _{SCS}	54
Table 1.14:	Comparison of partial regressions of measures of daughter performance on PTA _{SCS} holding other sire PTA variables constant..	56
Table 2.1:	Means, SD, and correlations of test day SCS and sire PTA _{SCS} for all lactations	70
Table 2.2:	Means, SD, and correlations of test day SCS and sire PTA _{SCS} for 1 st , 2 nd , and 3 rd lactation records.....	72

Table 2.3:	Means, SD, and correlations of SCS test day segment averages for 1 st , 2 nd , and 3 rd lactations.....	73
Table 2.4:	Regression coefficients and SE of 1 st , 2 nd , and 3 rd lactation test day segment averages on PTA _{SCS}	74
Table 2.5:	Regression coefficients and SE of differences in SCS test day segment averages within a lactation on PTA _{SCS}	75
Table 2.6:	Means, SD, and correlation of PTA _{SCS} , lactation average SCS, and regression coefficients of test day SCS on test day DIM.....	76
Table 2.7:	Means, SD, and correlations of sire PTA _{SCS} and measures of variation in SCS, by lactation.....	77
Table 2.8:	Correlation of lactation average SCS measurements.....	78
Table 2.9:	Regression coefficients and SE of 1 st lactation measures of SCS on PTA _{SCS}	79
Table 2.10:	Linear and quadratic coefficients of 1 st lactation measures of SCS on PTA _{SCS}	79
Table 2.11:	Regression coefficients and SE of 2 nd lactation measures of SCS on PTA _{SCS}	80
Table 2.12:	Linear and quadratic coefficients of 2 nd lactation measures of SCS on PTA _{SCS}	81
Table 2.13:	Regression coefficients and SE of 3 rd lactation measures of SCS on PTA _{SCS}	81
Table 2.14:	Linear and quadratic coefficients of 3 rd lactation measures of SCS on PTA _{SCS}	82
Table 2.15:	Regression coefficients and SE of differences in measures of variation in SCS on PTA _{SCS} between lactations.....	83
Table 2.16:	Comparison of linear coefficients of measures of SCS on PTA _{SCS} in 1 st , 2 nd , and 3 rd , lactations.....	84
Table 2.17:	Means, SD, and ranges of test day segment data for all lactations	99
Table 2.18:	Means, ranges, and SD of test day segment data for 1 st lactation cows.	100
Table 2.19:	Means, ranges, and SD of test day segment data for 2 nd lactation cows.....	101
Table 2.20:	Means, ranges, and SD of test day segment data for 3 rd lactation cows.....	102
Table 2.21:	Correlation of test day SCS measurements between lactations 1 and 2.....	103
Table 2.22:	Correlation of test day SCS measurements between lactations 1 and 3.....	103

Table 2.23:	Correlation of test day SCS measurements between lactations 2 and 3.....	103
Table 2.24:	Linear and quadratic regression coefficients and standard errors of 1 st lactation test day segment averages on PTA _{SCS}	104
Table 2.25:	Linear, quadratic, and cubic regressions and standards errors of 1 st lactation SCS test day segment averages on PTA _{SCS}	104
Table 2.26:	Linear and quadratic regression coefficients and standard errors of 2 nd lactation test day segment averages on PTA _{SCS}	105
Table 2.27:	Linear, quadratic, and cubic regressions and standards errors of 2 nd lactation SCS test day segment averages on PTA _{SCS}	105
Table 2.28:	Linear and quadratic regression coefficients and standard errors of 3 rd lactation test day segment averages on PTA _{SCS}	106
Table 2.29:	Linear, quadratic, and cubic regressions and standards errors of 3 rd lactation SCS test day segment averages on PTA _{SCS}	106
Table 2.30:	Linear, quadratic, and cubic regressions and standards errors of 1 st lactation measures of SCS on PTA _{SCS}	107
Table 2.31:	Linear, quadratic, and cubic regressions and standards errors of 2 nd lactation measures of SCS on PTA _{SCS}	107
Table 2.32:	Linear, quadratic, and cubic regressions and standards errors of 3 rd lactation measures of SCS on PTA _{SCS}	107

LIST OF FIGURES

Figure 1: Cumulative Frequency of sire PTA_{SCS}	49
Figure 2: Linear, Quadratic, and Cubic effects of Number of lactations on PTA_{SCS}	96
Figure 3: Linear, Quadratic, and Cubic effects of DPL on PTA_{SCS}	96
Figure 4: Linear, Quadratic, and Cubic effects of TDIMM on PTA_{SCS}	96
Figure 5: Linear, Quadratic, and Cubic effects of Total Milk on PTA_{SCS}	97
Figure 6: Linear, Quadratic, and Cubic effects of Total Fat on PTA_{SCS}	97
Figure 7: Linear, Quadratic, and Cubic effects of Total Protein on PTA_{SCS}	97
Figure 8: Linear, Quadratic, and Cubic effects of Lactation Avg. SCS on PTA_{SCS}	98
Figure 9: Linear, Quadratic, and Cubic effects of RNIOCM on PTA_{SCS}	98
Figure 10: Linear, Quadratic, and Cubic effects of RNIOCF on PTA_{SCS}	98

INTRODUCTION

Sale of milk is the primary source of income for most dairy cattle producers. Thus, selection for high milk yield has been the primary focus of genetic improvement programs. As a result, the genetic potential for milk yield has escalated since the late 1960's (43). The intense selection and resulting increase in production has, however, raised questions as to the effects of higher yield on longevity and incidence of health disorders among dairy cattle. Of specific concern is the increase in incidence of mastitis that has accompanied increased milk yields.

Mastitis is defined as inflammatory reaction of udder tissue to bacterial, chemical, thermal, or mechanical injury. Mastitis disrupts production efficiency and causes multiple economic losses (35). Losses include: lower milk yield, milk discarded as a result of antibiotic treatment, loss of quality premiums, reduced longevity of animals, lower salvage value of animals, reduced genetic gain, and lost net income due to higher health care and treatment costs.

Generally, producers have selected for increase yields, and have ignored any correlated response in increased incidence of mastitis (43). The resulting elevation in incidence of mastitis has increased health costs. To justify increasing costs, net income from additional milk yield has to outweigh the added costs. Most studies have shown that income from increased milk yield more than exceeds the losses incurred as a result of mastitis. Research has seldom accounted for the total cost of disease, however, because the expense associated with replacing a diseased animal is rarely considered (44).

Approaches to mastitis prevention are prioritized based on feasibility, efficacy, and cost. Methods of mastitis prevention include: eradication, sanitation, genetic improvement, vaccination, isolation of infected animals, antibiotic therapy, and culling. To date, sanitation practices including: proper cleaning and drying of udders prior to milking, well maintained milking equipment, teat dipping after milking, and clean housing for cows prove to be the most effective means of mastitis prevention (41). Eradication would be the method of choice, but is not possible due to the numerous sources of mastitis infection, and the implausibility of eliminating all infectious pathogens (41, 44). Vaccination against some mastitis-causing pathogens shows promise as a preventative measure, but efficacy rates remain low. Treatment of mastitis with antimicrobial therapies is effective for some pathogens, but costly in terms of increased labor and treatment costs, and discarded milk due to withholding restrictions. Culling eliminates mastitis, but additional costs are incurred for replacements.

Selection for decreased incidence of mastitis is a plausible preventative measure. Effective selection for resistance to mastitis requires genetic evaluation of sires available to dairymen through artificial insemination. A sire's genetic evaluation would be based on incidence of mastitis measured in female offspring. No consistent method of recording incidence of mastitis exists in the U.S., however, and the cost of monthly bacterial testing of cows enrolled in DHI is prohibitive. Direct selection must be on an indicator trait which is strongly related to incidence of mastitis. Research suggests that monthly somatic cell counts (SCC) are a suitable indicator trait for selection to reduce incidence of mastitis (10,

41, 56). SCC are cost effective, easily measured monthly from milk samples collected for Dairy Herd Improvement (DHI) testing programs, and have a higher heritability (0.17) than incidence of mastitis (41, 45, 55). Furthermore, SCC are an acceptable indirect measure of mastitis in that elevation of SCC is indicative of both clinical and subclinical infection (34, 35).

The question remains in the minds of some as to whether selection for lower SCC will decrease incidence of mastitis or decrease a cow's ability to respond to invading pathogens. Somatic cells are comprised primarily of polymorphonuclear leukocytes (PMN), which are specific, and only migrate in response to infection. The critical question is therefore whether differences in somatic cells among cows is a reflection of differing abilities to respond to bacterial infection, or to preclude invading pathogens. Prior investigations (10, 30, 56) and reported correlations indicate the latter interpretation is correct. Correlations between early evaluations of sires for cell count and daughter infection rates were positive (10). Daughters of low PTA_{SCS} bulls should therefore phenotypically express lower average SCS, and decreased incidence of mastitis. The biological impact of selection on PTA_{SCS} to lower SCS and subsequent incidence of mastitis can be determined by evaluation of measures of daughter performance.

The objectives of this study were:

1. To determine the relationship between a bull's PTA_{SCS} and prevalence of cases of clinical mastitis, number of treatments for mastitis, number of days treated per lactation, and lactation average SCS among his daughters.
2. To determine the economic impact of a bull's PTA_{SCS} by examining the relationship between a bull's PTA_{SCS} and lifetime milk, fat, and protein production, number of lactations, days of productive life, lactation average SCS, total days in milk, and relative net income adjusted for opportunity cost calculated in fluid and manufacturing markets among his daughters.
3. To determine the relationship between PTA_{SCS} and incidence of mastitis among daughters as measured by SCS over test days and lactations.

LITERATURE REVIEW

Introduction

Heightened accuracy and intensity of selection for milk yield has resulted in rapid rates of genetic improvement. Research has suggested, however, that high milk yield was accompanied by greater a frequency in health problems and premature culling, and that single trait selection for greater milk yield increased susceptibility to mastitis, the most economically detrimental disease of dairy cattle (17, 21, 28, 33, 41, 43, 44).

Mastitis is the inflammatory reaction of the mammary gland to bacterial, chemical, thermal, or mechanical injury. The inflammatory response is characterized by an increase in blood proteins and somatic cells in the udder tissue and milk. Somatic cells found in milk are primarily leukocytes or white blood cells, whose principal function is to eradicate infections in the body, and repair damaged tissues (34, 51). A major percentage of the leukocytes are the PMN that enter the mammary gland from the blood via diapedesis. PMN phagocytize and destroy invading bacteria. Somatic cells are also comprised of macrophages, lymphocytes, and epithelial cells (34). T-lymphocytes orchestrate immune response via the secretion of lymphokines, or soluble immune factors, while B-lymphocytes produce antibodies that are essential in the binding and presentation of bacteria for phagocytosis (51). Epithelial cells are rarely found in udder secretions, and account for only a small percentage of the somatic cell population (34). Somatic cells are always present in the bloodstream, and infiltrate other areas of the body when damage or infection is detected (51).

Unlike other diseases common to dairy cattle, mastitis has several causative agents, the most common being bacteria or yeast. The chief pathogens involved in the development of mastitis are considered to be *Streptococcus agalactiae*, *Staphylococcus aureus*, *Streptococcus dysgalactiae*, *Streptococcus uberis*, and other environmental pathogens including *Escherichia coli* and *Klebsiella* species. *Corynebacterium bovis* is considered a minor pathogen. The causative organisms vary in mode of transmission, however, cow to cow is the most common path of infection (31). Poor sanitation, environmental or physiological stress, age of the cow, stage of lactation, and temperature are all factors that likewise increase the probability of infection. Bacteria can be transmitted to an uninfected udder via improper sanitation practices of farm personnel, poorly designed and/or maintained milking equipment, or multiple use of syringes for antibiotic treatments without proper sterilization (31).

Mastitis manifests in subclinical, clinical, and chronic forms. Subclinical mastitis is characterized by changes in the milk which occur in the absence of clinical symptoms such as udder edema and physiological abnormalities in the milk. Clinical mastitis is defined by gross abnormalities in the milk such as flakes, clots, or a watery appearance. Swelling of the mammary gland as well as fever, rapid pulse, and loss of appetite often accompanies clinical mastitis. Chronic mastitis implies a prolonged udder infection that occasionally flares to clinical status (31).

Several preventative measures have been considered in attempts to control mastitis. Eradication of pathogens that cause mastitis was examined, but dismissed as impractical due to the numerous pathogens associated with mastitis (41). A more practical approach was the control of invading pathogens through the application of sanitation practices. Milking equipment was more carefully maintained, and hygiene practices such as washing udders, drying udders with individual towels, and use of germicidal teat dips were instituted. Sanitation practices reduced the numbers of bacteria, and to date remain one of the most effective methods of controlling mastitis (41).

Routine culling and replacement of infected animals was an effective means of eliminating mastitis from a herd, but very expensive as a method of controlling mastitis due to costs associated with replacements. Dry cow and lactation therapy were also determined to be effective as methods of controlling mastitis. Antibiotics in a long-acting base administered intra-mammary at drying off proved effective in reducing incidence of mastitis at next calving (36). Lactation therapy, however, was not as successful and caused economic loss resulting from discarded milk.

In general, extensive use of antimicrobials in livestock was viewed as undesirable. Concerns have been reported regarding the rise in resistant strains of bacteria (12, 23). It has been speculated that prolonged use of antimicrobials in food animals has provided a selective pressure for resistant strains of bacteria. Since animals and humans were treated with the same antibiotics and shared the same environment, exchange of bacteria may have

occurred between animal and humans. For example, pathogenic *Salmonella* originating in the animal population has possibly been the cause of human disease (12, 23).

In conclusion, an obvious need exists for an effective method of mastitis prevention to augment current mastitis control practices. Selection to improve mastitis resistance warrants inclusion in a breeding program as it would decrease mastitis incidence and associated costs, and improve the quality of milk and related products. Direct selection to reduce incidence of mastitis, however, is not plausible. Selection must therefore be based on an indirect or associated measure. Selection to improve a cow's natural defense mechanisms has been proposed as the method to reduce incidence of mastitis. Of possible innate bovine defense mechanisms, phagocytosis, the mechanism associated with the recognition and digestion of foreign material, was of particular interest (38). Somatic cells that infiltrate the mammary gland in the presence of injury or infection possess phagocytic properties. Somatic cells are primarily PMN, which engulf and destroy invading pathogens. Somatic cell counts (SCC) are measured from monthly DHI milk samples and are converted to \log_2 base scores. Somatic cell scores (SCS) are therefore considered a general characterization of udder health on test day, and the average of monthly test day scores, the lactation average SCS, depicts the health of the cow throughout the lactation. Selection for lower somatic cell scores could therefore provide the opportunity to select for improved innate immune function.

Relationship of milk yield, measures of mastitis, and associated costs.

On an annual basis, mastitis caused greater monetary loss in commercial dairy production than any other disease (13, 17, 26, 27, 31, 40, 43, 44, 49). Losses associated with mastitis included: reduced milk yield, discarded milk as a result of antibiotic treatment, lower milk prices and loss of quality premiums, reduced longevity of animals, lower salvage value of animals, reduced genetic gain, decreased milk quality, and higher health care and treatment costs (13, 43). The largest contributing factor, reduced milk yield, accounted for 69 - 80% of the total cost (26).

On the average, cows with clinical mastitis exhibited a .5kg reduction in daily milk yield when compared to uninfected cows (27). Janzen reported milk loss per quarter per day in infected cows ranging from 0.35 to 2.7kgs (26). Estimates of the overall economic loss from mastitis ranged from \$100 to \$ 200 per cow per lactation, and were estimated to sum to more than \$2 billion annually in 1979 (5).

Several studies sought to quantify the genetic correlation between milk yield and mastitis. In a review published by Shook (44), the correlation between mastitis and milk yield ranged from -.07 to +.33 and averaged near .20. The positive correlation indicated that as the genetic merit for milk yield increased, so did genetic predisposition for mastitis. In an analogous study, genetic correlations between fat-corrected milk (FCM), and the number of clinical cases of mastitis were calculated from data collected on three populations of Swedish cattle. The estimated correlations ranged from -.10 to .32, and averaged .18 (17, 44). In a separate review, lactation milk yield and measures of mastitis infection were

reported to have a genetic correlation which averaged .04 (10). The correlation between milk per day of lactation and total mastitis cases per lactation was reported by Miller et al. (32) to be .33. Genetic correlations between mastitis and both milk and fat yield were estimated by O'Bleness et al. (37) to be .44 and -.12, respectively.

Correlated responses in mastitis and health costs: Studies examining the effect of selection for milk yield on costs for health care treatments in several cases revealed a positive correlation between yield and frequency of treatment for mastitis (4, 16, 19, 28, 42). Gilmore and McDaniel (19) reported a positive correlation between milk yield and number of treatments for mastitis. Total annual health costs were calculated (frequency x cost) and the resulting correlation between milk yield and mastitis cost was reported to be .18 (19).

Two separate long term selection projects, conducted in Iowa (21) and Minnesota (42), estimated health costs in response to selection for milk yield. In the Iowa study, Shanks et al. (42) compared cows with high milk yield pedigrees to cows with low milk yield pedigrees. The estimated breeding value (EBV) of the high group exceeded that of the low group by 760 kg of milk. When compared to the low pedigree group, the high pedigree cows produced an average of 435 kg more milk. Although the high pedigree cows had \$12.46 higher health cost, they netted \$45.80 more per lactation from increased milk yield (42). Shanks et al. (42) likewise reported that daughters of sires with high PTA for milk yield had \$9.69 higher health costs and \$77.64 higher net income per lactation when compared to daughters of average milk yield sires.

More recently, Dunklee et al. (16), and Bertrand et al. (4) analyzed the response to the Iowa selection experiment on two genetically diverse populations of cattle, a high line sired by superior PTA_{Milk} bulls, and a control line, sired by average PTA_{Milk} bulls. Sire selection was based on high and average milk yield, and the correlated response of fitness traits was measured. Bertrand et al. (4) noted a 21% increase in health costs for daughters of high PTA_{Milk} sires compared to daughters of sires which were breed average for milk yield. Similarly, Dunklee et al.(16) reported that per lactation, the high line averaged 3.8% higher total health care costs, and 11.2% higher mammary health costs than daughters of average sires.

In the Minnesota study, Hansen et al. (21) compared two populations of cattle, one maintained as a genetic constant, or control group, and the other in which sires were selected solely for milk yield. Both groups of cattle were subject to identical environment and management practices. Responses in milk yield and health disorders were measured on both populations of cattle over a period of 9 years, beginning in 1964. The performance of the high line exceeded the control line by 846 kg milk, and \$94 income over feed cost, while requiring an average of 34 more minutes of labor, and \$7.74 added health cost. Mammary disorders explained over 60% of the difference between the high and low lines for health expense, and approximately 30% of the differences in labor costs between lines (21, 44). In both the Iowa and Minnesota studies, the added health costs in the high pedigree milk yield groups accounted for 10-20% of the additional revenue from increased yields. Nonetheless, the added revenue from increased milk yield in both studies substantially surpassed extra feed and health care costs. The reported results are optimistic

however, because the costs associated with replacing diseased animals were not included (44).

In a later analysis of the Minnesota study, Jones et al. (28) investigated the correlated response of health care expense to selection for milk yield for both a control population, and cows bred for higher milk yield. Comparison of the two populations revealed that single trait selection for milk yield was correlated to an increase in health expenses. Differences in health costs between the genetic groups were \$23.11 for first lactation, and \$46.06 for the first 5 lactations. Furthermore, Jones et al. (28) reported that the majority of the discrepancy between groups for health costs was attributable to mastitis. Differences due to mastitis amounted for 82% of health costs for first lactation, and 56% across lactations.

Wilton et al. (58) reported cases of mastitis in second and later lactations would increase 1.3% per year per cow at the current rates of genetic improvement for yield. Similarly, Strandberg and Shook (49) estimated that a breeding program that included milk yield, but did not consider mastitis, would result in a .4% annual increase in incidence of mastitis (approximately .02 cases of mastitis per cow per year). Both of the latter simulation studies were based on an assumed genetic correlation between milk yield and incidence of mastitis of .30. Simultaneous genetic improvement for milk yield and mastitis was difficult due to positive genetic correlations between milk yield and incidence of mastitis. Nonetheless,

increased incidence of mastitis could be controlled by simultaneous selection for increased milk yield and decreased somatic cell score.

Selection for Mastitis Resistance

Methods of selection for mastitis resistance included bacteriological testing and direct measurement of mastitis. Bacteriological testing of quarters was considered the most accurate measure because it indicated both clinical and subclinical incidence of mastitis. Effective bacteriological testing required quarter milk samples from all cows in the herd, however, and monthly DHI samples are composite samples. Thus, monthly samples could not be used for bacteriological testing (44). Furthermore, high costs precluded bacteriological testing from inclusion in a breeding program.

Direct measurement of mastitis involved recording of each clinical case of mastitis. While out of pocket cost associated with direct measure was minimal, it failed to detect subclinical cases of mastitis which account for the vast majority of cases of mastitis. Likewise, no standard procedure for recording cases of mastitis existed, and producers were reluctant to record all cases of clinical mastitis. Selection for mastitis resistance therefore had to be based on an indicator trait correlated to mastitis. Since mastitis is an inflammatory response to invading pathogens, a trait related to the response to infection, or the innate defense mechanisms of a cow, was of particular interest (31). The most suitable parameter for selection to reduce incidence of mastitis proved to be milk somatic cells.

Somatic cells in milk: Somatic cells found in milk are white blood cells, or phagocytic cells, whose primary function is to eliminate pathogens and repair damaged tissues. Somatic cells circulate in the blood stream, and only infiltrate the mammary gland when infection or damage is detected. Somatic cells are comprised of PMN, macrophage, lymphocytes, and epithelial cells. Somatic cells are very specific, and are only elevated in the mammary gland when they are needed. Thus, factors that affect milk SCC should be considered. The first of these factors is the variation in SCC that is inherent from animal to animal. While several environmental factors contribute to variation in SCC, genetic variation is also a probable explanation (35). Two types of genetic variation should be examined when considering the heritability of SCC: the variation between cows during the presence of infection, and the variation in normal cell counts. Thus, the second factor that affects milk SCC is the presence or absence of infection. Determination of infection status is influenced by sampling time, number of quarters infected, and pathogen species.

Age of the cow likewise affected the variation in infection rates. Studies have shown that cell counts and the probability of infection increased with age of the cow (21, 32, 56, 60). Stage of lactation at sampling time is another factor which influenced milk SCC. In the first days of a lactation, a marked rise in SCC was frequent. The increase in cell counts early in lactation suggested that the frequency of new infections was higher at the beginning of a lactation (35). Elevated cell counts were also found at the end of a lactation (6). As a lactation progressed, inflammatory responses were more extreme and more pronounced by reduced milk yields.

The genetic correlation between milk yield and SCC was slightly lower than the correlation between milk yield and mastitis. Kennedy et al. (29) reported a correlation of 0.14 between milk yield and SCC while Emmanuelson et al. (17) found a higher correlation of .46. The phenotypic correlation between milk yield and mastitis, however, was negative and ranged from $-.10$ to $-.20$. It could be interpreted that cows with high SCC would produce less milk (45). Elevated counts as a result of mastitis infection thus reduced yields. Supporting studies found that, in general, milk yield decreased as SCC increased. Jones et al. (27) reported milk yield losses of 1.0kg for 1st lactation cows, and 3.0kg losses for 2nd and later lactation cows when SCC exceeded 200×10^3 . Jones et al. (27) likewise found that yield reductions and increases in SCC were more severe as lactation progressed. The conclusion was that lower milk yields and higher infection rates occur frequently with SCC of 200 to 400×10^3 (27).

Genetic Evaluation for Somatic Cell Score

Somatic cell testing programs were implemented within DHI programs in the U.S. in the late 1970's. Approximately 80% of all cows in the DHI program are currently on monthly somatic cell test, which accounts for close to 40% of all cows in the U.S. (44). Tests for somatic cells, conducted by DHI in widely varied intervals, are based on composite milk samples. Monthly sample scores are accumulated into a lactation average score, and are adjusted for age and season of calving (46).

Somatic cell counts (SCC) taken from monthly milk samples are a rough characterization of udder health throughout lactation. SCC are also an indication of the presence or absence

of infection, the number and duration of infections, the severity of infection, as well as the number of infected quarters (43). High SCC, in general, is an indication of poor udder health. Likewise, a low SCC is an indication of either a low level or absence of infection. Thus, low SCC is associated with low probability of clinical mastitis (43).

The distribution of SCC, however, possessed some statistically undesirable properties. Ali and Shook (1) determined that transformation of SCC to linear somatic cell scores (SCS) allowed for the application of traditional statistical methods of genetic evaluation. Individual SCC were converted by DHI Dairy Records Processing Center to SCS using a base 2 logarithm. A SCC of 100-cells/ μ l converted to a SCS of 3. A unit increase in SCS resulted in the doubling of the cell count. Conversely, counts were halved when scores decreased by 1 unit (43). The conversion of SCC to SCS was supported by Jones et al. (27) who reported that SCC data logarithmically transformed to SCS had a higher within lactation test repeatability, as well as a smaller within cow test day variance, and a smaller error variance.

As with SCC, a relationship existed between SCS and yield. The relationship between SCS and yield reduction due to mastitis is indicated in Table 1. With each increase in 1 unit in SCS, there was an estimated loss of 200 pounds of milk for first lactation animals, and a loss of approximately 400 pounds of milk for second or later lactation animals. From the data in Table 1, dollar loss due to subclinical mastitis was determined. As SCS increased by 1, the estimated annual loss due to mastitis increased by \$2,000 for a 50-cow herd, and \$4,000 for a 100-cow herd.

Table 1. Milk production loss per lactation associated with somatic cell scores (14).

Somatic Cell Score	Geometric Lact. avg. SCC	Decrease in milk yield (lbs.)	
		Lactation 1	Lactation 2 & up
0-2	----	0	0
3	100	200	400
4	200	400	800
5	400	600	1200
6	800	800	1600
7	1600	1000	2000

In an early study, Young et al. (59) reported correlation estimates between mastitis and leukocyte count of .80 and .98, derived by half-sib correlation, and daughter-dam regression, respectively. Data included 682 daughters of 342 sires. Emanuelson et al. (17) calculated estimates of the genetic correlation between SCC and mastitis for Swedish black and white cattle and Swedish red and white cattle. The resulting correlations were .46 and .78, respectively, with an average correlation of .62. Weller et al. (56) estimated the genetic correlation between SCS and clinical mastitis to be .30. The low estimate was explained by inaccurate recording of clinical mastitis. Shook and Shutz (43) reported the average genetic correlation of SCS and clinical mastitis to be somewhere between 60 and 80%, which was an indication of a strong genetic association between the two traits. Somatic cell score (SCS) proved to be a more reliable indicator trait than SCC, as SCS exhibited a linear relationship to milk loss, the correlation of mastitis and SCS (0.60) was higher than that between SCC and mastitis (43, 56), and SCS had a higher heritability (0.20) than SCC (10, 35, 41, 56).

Sire evaluations for somatic cell score were calculated and released to U.S. dairy producers for the first time in January of 1994 (46). Genetic evaluation of SCS was calculated on

dairy sires by USDA-AIPL using animal model procedures similar to those used for yield traits. A bull's evaluation was based on records from all known relatives weighted by their relationship to the bull (46). First lactation somatic cell information was required for a cow to be included in a genetic evaluation (41). Somatic cell counts of daughters were converted to base 2 logarithm scores (0 to 9 scale), then averaged across test days. The lactation average SCS for the first five lactations was adjusted for age and season of calving. Since the daughters of a bull were spread out over herds, records were deviated from the average performance of other animals in the same herd at the same time, to account for herd environment. A bull's evaluation was expressed relative to the average cow born in 1990, and was then added to the average first lactation SCS, standardized for age, month of calving, and length of lactation, of cows born in 1990 (24).

Evaluations were calculated using Best Linear Unbiased Prediction (BLUP) procedures in which the random effects of the animal, permanent environment, herd by sire interaction, and residual effects were all assumed to be normally distributed. Solutions for individual levels of effects were regressed toward their mean value depending on the amount of available information (41). Animal model procedures were iterative, thus estimates for all effects were influenced by estimates in subsequent rounds (41). Breeding values from animal model procedures were divided by 2 and reported as predicted transmitting ability (PTA). The difference in PTA_{SCS} between two bulls was therefore an estimate of the difference in lactation average SCS of future daughters in the same environment (46). The average PTA_{SCS} for Holsteins was 3.2 (24).

An assumption of no genotype-environment interaction was included in genetic evaluations for SCS. Geneticists expressed concern that bulls would be unable to demonstrate genetic differences in SCS in herds with low cell counts (8). Likewise, veterinarians were concerned that genetic differences in mastitis resistance could only be demonstrated when mastitis was prevalent in herds with high cell counts (8). Banos and Shook (3) confirmed that the difference between bull proofs based only on daughters in high-count herds and those based only on low cell count herds was insignificant. Thus, SCS from cows in both high cell and low cell count herds can contribute to a sire's genetic evaluation (8).

Due to low heritability ($h^2 = .10$ to $.15$), a bull's PTA_{SCS} for a given number of daughters was not as accurate as PTA calculated for production traits. A sire's PTA for SCS had a particularly low accuracy when only first lactation data was used in the estimation. Evidence indicated that incidence of mastitis increased with age of cows, thus SCS from later lactations were perhaps a more accurate indication of mastitis susceptibility than only first lactation scores (49). The rate of genetic gain for mastitis resistance as a result of selection for lower SCS will be slow, but economically feasible since the cost associated with genetic selection is small when compared to the cost of mastitis treatments .

Selection on PTA_{SCS} for improved resistance to mastitis.

Early experiments regarding the effects of bull selection on incidence of mastitis indicated that paternal half-sib groups with an elevated SCC in the initial lactation exhibited

increased susceptibility to mastitis in later lactations. Grootenhuis et al. (20) conducted a study in which comparisons were made between paternal half sibs grouped according to first lactation SCC. Results showed that high 1st lactation SCC daughters were more prone to mastitis in later lactations than the low SCC group. Vecht et al. (55) examined the effects of bull selection for SCC in 1st lactation on cell counts in later lactations, and concluded that daughter groups with low average SCC in 1st lactation continued the trend in later lactations, and had a lower percentage of quarters with mastitis pathogens than daughter groups with higher average cell counts.

In a simulation study, selection for mastitis resistance was accomplished by using sire genetic evaluations of SCS, an indicator trait. Strandberg and Shook (49) reported that inclusion of SCS in a breeding program slowed the rate of increase in cases of clinical mastitis by approximately 20%. Table 2 summarizes the response to selection indexes that include mastitis. The study (49) showed that selection for milk yield alone resulted in increased incidence of mastitis whereas including clinical mastitis slightly reduced milk yields, but slowed the rate of increase in mastitis. The same study showed that inclusion of SCS in a selection index was as effective as the index including mastitis. Clearly, inclusion of SCS in a breeding program will not eliminate mastitis, but would slow the increase in incidence of mastitis.

Table 2. Responses in milk yield, mastitis, and total merit from selection indexes with different index traits (49).

Mastitis related traits in the index	Response		
	Milk (kg)	Mastitis (cases/yr.)	Merit (\$)
None	53.5	.020	98.2
SCS	52.7	.016	98.6
Mastitis	52.4	.015	99.1

Impact of Selection on Defense Mechanisms.

Somatic cells are phagocytic cells which function to eradicate invading pathogens. The question has been raised as to whether selection for lower SCS would affect a cow's innate phagocytic defense mechanisms, or the cow's ability to avoid infections and mount immune responses (8, 11, 53). Miller and Schultze (35) partitioned phagocytic competence into two components: the competency of individual phagocytic cells, and the number of cells. Cell number was directly related to the speed with which the PMN responded to invading pathogens. Therefore, if selection emphasis were placed on lower number of SCC, would the ability of a cow to combat mastitis infection be compromised?

Coffey et al. (11) reported that cows with low SCC exhibited increased ability to avoid infection, or were able to respond quickly, eliminate the infection, and return to a low SCC. Timms (51) concluded that cell competency, the ability and speed of the cell to respond to mammary infection, was more important in infection prevention than strict cell number. Likewise, research (39) using intramammary devices (IMD) to artificially elevate SCC revealed lower infection rates in controlled challenge studies for IMD quarters, primarily due to the constant influx and higher speed at which somatic cells entered the gland. While

there is no question that a finite number of cells were needed once an infectious agent invaded the udder, no direct evidence implied that selection for reduced SCC would affect a cow's ability to mount an immune response.

The Relationship of Economic Merit and Reduced Incidence of Mastitis.

The overall goal of genetic improvement of dairy cattle was to maximize the total economic merit. Impacts that warrant selection for disease resistance therefore included the reduction of milk production costs, the improvement of the health of dairy animals and the improvement of the quality of milk and dairy products. Consideration likewise should be given to the impact of mastitis on the longevity of dairy cattle, and the costs associated with replacing mastitic animals. Andrus et al. (2) concluded that mastitis accounted for 14% of the variance of herd profit, and was the second most important trait when determining an animal's individual return per year of herd life.

Endeavors to measure and improve the overall economic merit of dairy cattle have led to the development of genetic evaluations upon which selection for healthier, longer-living cows can be practiced. While some natural selection for longevity exists in that animals that live longer usually have more progeny, direct selection for longevity increased profitability (54). Longer herd life contributed to overall profitability in two distinct ways. First, replacement costs were lowered when herd life was extended, and second, herds contained more cows producing at mature levels (25). Longer herd life also implied reduced culling levels, and lower health costs (25). Consequently, herd life is an important economic trait and is included in many breeding programs.

Herd life was largely defined by the culling decisions of individual producers. Most culling decisions pertained directly to economics, and were founded in the expectation that a replacement will yield a higher profit (15). The most common reasons cows left the herd included: low yield, mastitis, reproductive failure, sale for dairy purposes, or death (54). Many performance traits such as milk production, incidence of mastitis, age at first calving, days open, and days dry thus affected length of herd life and overall economic return (18).

Direct selection for longer herd life would be most efficient, but the heritability of herd life was low, and herd life was expressed at a later age than most traits in a selection index (53). Consequently, Van Raden and Klaaskate (54) proposed direct selection for an alternative trait, productive life (PL). PL was defined as total months in milk through 84 months of age. A method to include predicted PL as a data source was developed (54) to allow for calculation of PL earlier in an animals' life. Genetic evaluations of PL measured differences in months of milk for cows in the same herd at the same time, and likewise reflected all culling reasons (53). Because a cow expresses PL only once, permanent environmental effects were not considered in the evaluation (53).

In addition to selection for longevity, selection for healthier cows with low incidence of mastitis has been proposed using SCS as a correlated trait (43). Established positive correlations between SCS and incidence of mastitis rendered SCS a valuable indicator trait and thus made it reasonable to assume selection for lower SCS would manifest in lower incidence of mastitis (43, 44). Since mastitis was superseded as a reason for culling only

by low production and reproductive failure, it was accurate to assume a relationship existed between mastitis resistance and increased longevity (43).

The lifetime income of an individual dairy animal can be factored into income per unit of time and length of productive life (54). Profit generated by individual animals is not measured directly, but rather estimated with functions of Dairy Herd Improvement Association (DHIA) variables (18). Relative Net Income (RNI) is one such profitability function. RNI estimates profit using the total net values of the amounts of both milk and fat produced as well as number of calves born, and the total fixed and variable costs associated with rearing, feed, and labor collected over the lifetime of the animal (57). Van Arendonk (52) conjectured that RNI would be inflated unless adjusted for the revenue forfeited when a cow was kept for another lactation instead of being replaced by a fresh cow. The forfeited revenue associated with keeping a cow in the herd is referred to as opportunity cost (OC). RNI adjusted for opportunity cost is therefore referred to as RNIOC. The heritabilities associated with RNI and RNIOC were found to be .12 and .17, respectively (57).

Relative net income variables are calculated based on lifetime yields and are thus directly affected by length of productive life. Should a negative relationship exist between length of productive life and SCS, selection for lower SCS could likewise have a positive effect on the overall profit realized over the lifetime of individual cows. Selection for lower SCS may not only reduce incidence of clinical and subclinical mastitis, but likewise produce more profitable cows that remain longer in the herd

CHAPTER I

THE RELATIONSHIP BETWEEN SIRE PTA_{SCS} AND MEASURES OF DAUGHTER PERFORMANCE

ABSTRACT

The relationship between sire PTA_{SCS} and incidence of mastitis in daughters was analyzed for 304 Holsteins first freshening since 1991 in the Va. Tech dairy herd. No direct sire selection for PTA_{SCS} had been practiced. In first lactation, linear regressions of lactation average SCS (1.34), number of cases of mastitis (.794), days treated (6.99), and number of treatments (2.01) on PTA_{SCS} were significant. Quadratic and cubic relationships were not significant. Linear, quadratic, and cubic relationships between PTA_{SCS} and the variables of interest for second and third lactation were not significant. Heavy culling imposed on first lactation cows resulted in a group of highly selected second and third lactation cows.

The relationship of sire PTA_{SCS} and number of lactations, DPL, TDIMM, lifetime milk, fat, and protein production, 1st lactation average SCS, and RNIOC calculated for fluid and manufacturing markets were also determined for 2,494,195 cows freshening between 1983 and 1990. Linear relationships between PTA_{SCS} and number of lactations (-.31), DPL (-85.6), total milk (-1567.6), fat (-79.0), and protein (-46.8) production, TDIMM (-71.3), 1st lactation average SCS, and RNIOC values for both the manufacturing and fluid markets (-6.7 and -15.8, respectively) were significant. Quadratic and cubic relationships were significant. No quadratic or cubic relationship existed between SCS and PTA_{SCS}. When PTA_{Milk}, and PTA_{Protein} were held constant, the effect of PTA_{SCS} on yield and profit variables became more severe. Effects were reduced when PTA_{Fat} was held constant due to the lack of correlation between PTA_{Fat} and PTA_{SCS}. When PTA_{PL} was held constant, the relationship between PTA_{SCS} and DPL, TDIMM, and RNIOC variables became positive. Results indicated that loss previously explained by increase in PTA_{SCS} was also explained by variation in PTA_{PL}.

Key Words (PTA_{SCS}, mastitis, SCS, sire selection)

Abbreviation key: SCS = **somatic cell score**. DPL = **Days of productive life**. TDIMM = **Total days in milk**. RNIOC = **Relative net income adjusted for opportunity cost**.

INTRODUCTION

Genetic selection for increased milk potential has had detrimental effects on the longevity, reproductive efficiency, and incidence of mastitis in dairy cattle (4, 16, 21, 28, 33, 43, 44, 45, 47). The increase in incidence of mastitis associated with the stress of increased yield is of particular economic concern given the high losses associated with mastitis. The multiple losses suffered as a result of discarded milk, lost production, costs of antibiotics, veterinary expenses, and the cost of replacements summed to an estimated \$2 billion in 1979 (5).

Current methods of preventing mastitis include eradication, vaccination, hygienic practices, prophylactics, and selection for mastitis resistance. Eradication and vaccination exhibited marginal efficacy due to the numerous mastitis causing pathogens. Hygiene practices have been effective in reducing bacteria counts, but have had no permanent effects on the reduction in incidence of mastitis. Antimicrobials have been used to treat established infections. Research has suggested, however, that extensive use of antibiotics in livestock populations has introduced a selective pressure favoring resistant strains of bacteria (12, 23). Public concern regarding drug use in agricultural production systems is thus pressuring producers to limit use of antibiotics and seek alternate means of mastitis control.

The selection for mastitis resistance is currently under consideration. No means of direct selection for mastitis resistance is available. Selection on a correlated indicator trait is, however, possible. The genetic correlation between mastitis and somatic cell count is between .60 and .80 (43). Somatic cells are present in the mammary gland and milk

primarily in response to bacterial toxins and are therefore a rough indication of udder health. Likewise, low cell counts are associated with low incidence of mastitis, thus selection for lower cell counts could reduce number of cases of mastitis. Monthly SCC converted to \log_2 scores (SCS) are used to estimate transmitting abilities. Somatic cell scores are more accurate measures upon which selection can be practiced. Genetic evaluations for SCS (PTA_{SCS}), which first became available in January of 1994, provide the opportunity for effective selection for lower SCS in a breeding program (46).

Selection for increased longevity can be practiced using the genetic evaluation for productive life (54). Possible relationships between PTA_{SCS} and measures of daughter performance including days of PL (longevity), total days in milk, and relative net income adjusted for opportunity cost could likewise prove to be factors that support the inclusion of PTA_{SCS} in selection indexes. While selection on PTA_{SCS} may have negative effects on selection to improve milk yield, the negative effects could be outweighed by the improvement in overall health, longevity, and economic value of dairy animals associated with lower SCS. Inclusion of PTA_{SCS} in a selection index with production would seem to offer the greatest opportunity for improving net merit in dairy cows.

The objectives of this study were:

1. To determine the relationship between a bull's PTA_{SCS} and the incidence of mastitis, number of treatments for mastitis, number of days treated per lactation, and lactation average SCS among his daughters.

2. To determine the economic impact of a bull's PTA_{SCS} by examining the relationship between a bull's PTA_{SCS} and lifetime milk, fat, and protein production, number of lactations, days of productive life, lactation average SCS, total days in milk, and relative net income adjusted for opportunity cost calculated in fluid and manufacturing markets among his daughters.

MATERIAL AND METHODS

Data Set I

Data were obtained from two sources. Data set I contained production records (305 day M.E.), lactation average SCS, number of cases of mastitis, number of days treated, and number and type of treatments per lactation for 1st through 3rd lactation cows in the Virginia Tech dairy herd. First freshening dates were between January 1991 and December 1997. All data were obtained from PCDART (DHI) records. Total number of cases of clinical mastitis, total number of days treated, and total number of treatments were calculated per lactation. An incidence of mastitis was defined as a new case when it was over 14 days (10) since the last treatment in the initial quarter, or when occurrence was recorded for a different quarter. Total days treated were calculated from the date of first treatment until the day of last treatment and summed for all cases. Number of treatments were summed for all cases per lactation.

Sire PTA_{SCS}, milk, fat, and protein were obtained from the USDA May 1998 Sire Summary. Range of sire PTA_{SCS} of cows in the data set was from 2.76 to 3.84. Mean sire PTA_{SCS} of cows in the data set was 3.3. Corresponding standard deviation was .24. Sire PTA_{SCS} was merged with the cow data.

Prior to analysis, data were edited to remove records of cows by sires without PTA_{SCS}. Cows culled prior to first test day were likewise removed from the data set. A total of 576 records were analyzed using the General Linear Models procedure in SAS (Carey, NC). The 576 records included 304 first lactation observations, 172 second lactation, and 100 third lactation observations.

PTA_{SCS} < 3.24, group 4 were 3.24 < PTA_{SCS} < 3.39, group 5 3.39 < PTA_{SCS} < 3.50, and group 6 sires were those with PTA_{SCS} > 3.51. Percentage of daughters in each sire group were analyzed to compare cull rates from 1st to 2nd and 2nd to 3rd lactations.

Further analyses were completed to investigate the impact of PTA_{SCS} on the longevity of cows in the data set. Cows were grouped according to number of lactations initiated. Group 1 cows were those cows that had initiated more than one lactation prior to being culled, and Group 2 cows were cows culled during or after initiating only one lactation. Means and standard deviations for number of cases of clinical mastitis, days treated, total number of treatments, milk, fat, and protein production as well as lactation average SCS were calculated based on 1st lactation data for each group of cows and compared.

Data Set II

Data set II included all cows scored between 1983 and 1990 for descriptive linear traits prior to 42 months by the Holstein Association. Data included lifetime milk, fat, and protein production, cow identification including sire, dam, and herd number, total days in milk (TDIMM), days of productive life (DPL), first lactation average SCS, number of lactations, and Relative Net Income adjusted for opportunity cost (RNIOC) values calculated for fluid and manufacturing markets (48). The file, after edits, contained records on 2,494,195 cows from 18,729 herds (48). Only 152,588 of the 2,494,195 cows had first lactation SCS.

The RNIOC values were based on 84 month opportunity and used the animal's birth date, and the last freshening date for the herd included in the data. All production records started prior to the end of the 84 month opportunity length were included. RNI values were calculated as follows:

$$\text{RNI} = \sum_{i=1}^n \left[\sum_{j=1}^3 \text{Component}_{ij} (\text{Value}_j - \text{Cost}_j) \right]$$

+ (number of lactations)(net value of calf)

+ net salvage value

- rearing cost
- (total days in milk)(maintenance, fixed, and labor cost for 2x or 3x per day in milk)
- (total days dry)(maintenance, fixed, and labor costs per day dry)

where j is the total milk, fat, or protein component in the ith lactation initiated before the end of the 84 month opportunity length (48). A base price of \$30.86/100kg was used in the fluid market, in which no value was given to protein. A base price of \$26.46/100kg was used in the manufacturing market. Values for the components in both markets were the same as used by both Smith et al. and Weigel et al. (48, 57). Values and costs for components in both markets are shown in Table 3.

Table 3. Values and costs for components in fluid and cheese markets (48).

Trait	Cost	Value	
		Fluid Market	Cheese Market
Fluid, per kg	.020	.254	.1159
Fat, per kg	.567	1.280	1.280
Protein, per kg	1.020	N/A	3.00

Sire PTA_{SCS} , PTA_{Milk} , PTA_{Fat} , $PTA_{Protein}$, and PTA_{PL} (productive life) were obtained from the August 1998 Sire Summary. Sire PTA data were merged with daughter measures of herd life and overall economic merit.

Effects of herd-year were absorbed. Herd-year refer to the herd and year of first freshening. Some cows changed herds, but belonged to only one herd-year group (48).

Data were analyzed using the General Linear Models procedure in SAS (Carey, NC) and the following model [Model 1]:

$$\text{Model: } Y_{ijk} = \alpha + H_i + \beta_1 X_k + \beta_2 X_k^2 + \beta_3 X_k^3 + \epsilon_{ijk}$$

Where: Y_{ijk} =

- Number of lactations
- Days of Productive Life
- Total days in milk
- Total milk production (lifetime)
- Total fat (lifetime)
- Total protein (lifetime)
- 1st Lactation average SCS
- RNIOCM
- RNIOCF

For the j^{th} daughter of the k^{th} sire freshening in the i^{th} herd year.

H_i = the i^{th} effect of herd - year of first freshening (absorbed).

$\beta_1 X_k$, $\beta_2 X_k^2$, $\beta_3 X_k^3$ = regression linear, quadratic, and cubic on PTA_{SCS} of the k^{th} sire.

ϵ_{ijk} = residual variance of the j^{th} daughter of the k^{th} sire freshening in the i^{th} herd year.

Two reduced models were used to examine linear [Model 2] and quadratic [Model 3] relationships.

Additional models also included sire PTA for milk [Model 4], fat [Model 5], protein [Model 6], MF\$ and PL [Model 7], and MFP\$ and PL [Model 8] as continuous variables. All models were analyzed using the General Linear Models procedure in SAS (Carey, NC). The effects of herd-year were absorbed.

RESULTS AND DISCUSSION

Data Set I

Means, and standard deviations for sire PTA_{SCS} , PTA_{Milk} , PTA_{Fat} , and $PTA_{Protein}$ are in Table 1.1. The average PTA_{SCS} of the 74 sires of cows in the data set was 3.25, slightly higher than 3.22, the current breed average (24). The data set average suggested that sire selection on SCS in the VA Tech herd was virtually nonexistent. The range of sire PTA_{SCS} was 2.76 to 3.84. The 1.08 unit difference between the lowest and highest bulls for PTA_{SCS} likewise supported the assumption of no selection. Mean sire PTA_{Milk} , PTA_{Fat} , and $PTA_{Protein}$ were 580.7kg, 13.5kg, and 12.9kg, respectively.

Table 1.1. Means, ranges, and SD of PTAs of the 74 sires of daughters in the data set.

Variable	Range		Mean	SD
	Minimum	Maximum		
PTA_{SCS}	2.76	3.84	3.25	.24
PTA_{Milk} (kg)	-145.6	1490.9	580.7	314.9
PTA_{Fat} (kg)	-11.8	47.6	13.5	11.8
$PTA_{Protein}$ (kg)	-7.7	46.7	12.9	9.9

Means and standard deviations for number of cases of clinical mastitis, number of days treated, total treatments, measures of production, and lactation average SCS of cows for each lactation are in Table 1.2. Average number of cases of clinical mastitis, days treated, and number of treatments increased with lactation number. Average number of cases of mastitis exhibited by first lactation cows was .43, with average number of days treated and total treatments of 2.8 and 1.1, respectively. Higher averages were calculated for second lactation cows, with number of cases averaging .68, days treated 7.0, and total treatments 1.4. Third lactation cows averaged .71, 8.5, and 1.5 for cases of clinical mastitis, days treated, and total treatments, respectively. The increase in average number of cases of

mastitis with lactation number coincided with literature reports of higher frequency of clinical mastitis in later lactations (22, 34). Average number of treatments in each lactation was slightly lower than the value of 1.91 reported by Coffey et al. (10).

Table 1.2. Means and SD for daughter variables analyzed for 1st, 2nd, and 3rd lactations.

Variable	Lact. 1 304 cows		Lact. 2 172 cows		Lact. 3 100 cows	
	Mean	SD	Mean	SD	Mean	SD
Cases mastitis (#)	.43	.96	.68	1.2	.71	1.3
Total days treated (days)	2.8	12.1	7.0	24.8	8.5	32.8
Number of treatments (#)	1.1	2.7	1.4	2.3	1.5	3.2
Lac. average SCS	2.5	1.5	2.6	1.7	3.1	1.8
305-d M.E Milk (kg) ¹	10575.7	1481.5	10908.9	1524.0	10678.8	1894.3
305-d M.E Fat (kg)	371.7	55.7	386.4	64.5	376.4	75.8
305-d M.E Protein (kg)	318.8	43.0	326.6	44.1	323.4	57.1

¹M.E. = Mature Equivalent.

Lactation average SCS increased with lactation number. The mean first lactation average SCS was 2.5, while second lactation cows averaged 2.6, and third lactation cows averaged a SCS of 3.1. Virginia state average for SCS was 2.8 for first lactation cows, and 3.9 for cows in second or later lactations, indicating that cows in the Tech herd were below state average for SCS in all lactations reported.

The averages for M.E. milk, fat, and protein production were 10575.7kg, 371.7kg, and 318.8kg, 10908.9kg, 386.4kg, and 326.6kg, and 10678.8kg, 376.4kg, and 323.4kg for first, second, and third lactation cows, respectively. The small decrease evident in third lactation yield corresponded to the reported negative phenotypic correlation between SCS and milk yield (3, 7, 17, 27), and the subsequent increase in lactation average SCS from 2.6 in second lactation to 3.1 in third lactation.

Correlations calculated between sire PTA_{SCS} , cases of clinical mastitis, lactation average SCS, days treated, and number of treatments for records on all lactations combined (n=576) are in Table 1.3. All correlations were positive. The correlations of sire PTA_{SCS} to cases of clinical mastitis, lactation average SCS, days treated, and number of treatments were .10, .13, .04, and .13, respectively. The correlation between PTA_{SCS} and number of clinical cases (.10) was lower than correlations reported (.20 to .38) in a previous field study (9). The correlation of PTA_{SCS} and days treated (.04) fell within the reported (.02 to .13) range (9).

Table 1.3. Correlations of daughter variables and sire PTA_{SCS} .

Variable	PTA_{SCS}	Case	SCS	Days	Treatments
PTA_{SCS}	1.0	.10	.13	.04	.13
Cases of mastitis	----	1.0	.41	.66	.77
Lact. Avg. SCS	----	----	1.0	.34	.28
Days treated	----	----	----	1.0	.26
# Treatments	----	----	----	----	1.0

Correlations between number of clinical cases and lactation average SCS, days treated, and number of treatments were .41, .66, and .77, respectively. The moderate phenotypic correlation (.41) between cases of clinical mastitis and lactation average SCS fell within the reported range of .36 to .67 (10), but was lower than the reported average of .60 (43). Weller et al. (56) reported an even lower correlation of .30, and attributed the lower value to inaccuracy of recording field data. While the correlation between clinical mastitis and SCS obtained in this study falls within the reported range, cases of mastitis that occurred between test days were not reflected in test day cell counts, and are an explanation of the low phenotypic correlation.

The results of 1st lactation regression of mastitis and yield variables on PTA_{SCS} are in Table 1.4. In first lactation, number of cases of mastitis, number of days treated, number of treatments, and lactation average SCS exhibited significant ($P < 0.05$) linear relationships with sire PTA_{SCS}. Number of cases of clinical mastitis increased by .80 cases per each unit increase in PTA_{SCS}. The corresponding standard error was .24. The positive relationship between PTA_{SCS} and cases of clinical mastitis reflected the positive correlation between PTA_{SCS} and clinical mastitis determined in this and previous studies (10, 17).

Table 1.4. Regression coefficients and SE of 1st lactation variables on PTA_{SCS}.

Dependent Variable	β	SE
Cases of mastitis ¹ (#)	.80	.24
Total days treated ¹ (days)	7.0	3.0
Number of treatments ¹ (#)	2.0	.65
Lact. Avg. SCS ²	1.3	.40
305-d M.E Milk ^{3,4} (kg)	2817.3	1737.5
305-d M.E Fat ^{2,4} (kg)	-26.9	64.0
305-d M.E Protein ^{2,4} (kg)	-2.8	49.6

¹Number of cows – 304. All variables significant ($P < 0.05$).

²Number of cows – 302. Variable significant ($P < 0.05$).

³Number of cows – 288. Variable not significant ($P > 0.05$).

⁴M.E = Mature equivalent.

A unit increase in PTA_{SCS} was associated with a 7 day increase in number of days treated and a 2 treatment increase in total number of treatments. Corresponding standard errors were 3.0 and .65, respectively. Lactation average SCS likewise showed a significant ($P < 0.05$) linear relationship with PTA_{SCS}. Each unit increase in PTA_{SCS} corresponded to a 1.3 increase in 1st lactation average SCS. There was no significant relationship between sire PTA_{SCS} and 305-day milk, fat, and protein production. Regression coefficients were negative for fat and protein, but positive for milk yield. Quadratic and cubic relationships

were likewise investigated, but no quadratic or cubic effects were significant. The effects of year-season were not significant.

The results for 2nd and 3rd lactation regression analysis are in Table 1.5. Data from 172 second lactation and 100 third lactation records were analyzed to determine linear effects. Harmon (22) reported higher infection rates for third and later lactation groups. Despite the increase in average number of cases seen in 3rd lactation which supported previous reports, regression analyses identified no significant relationships between sire PTA_{SCS} and the variables of interest for 2nd or 3rd lactations. Regression coefficients for total cases of clinical mastitis, days treated, and number of treatments remained positive across lactations, but did not exhibit a significant relationship with sire PTA_{SCS} ($P > 0.05$). The resulting regression coefficients for number of cases of mastitis for 2nd and 3rd lactations were .46 and .25, respectively. Days treated yielded coefficients of 9.3 and .84, and coefficients for total treatments were 1.4 and 1.0 for 2nd and 3rd lactations, respectively. The linear relationship between 2nd lactation average SCS and PTA_{SCS} approached significance ($P \approx .07$) with a regression coefficient of 1.0. Second and third lactation data were analyzed for quadratic and cubic relationships, but no significant effects were detected. The effects of year-season were not significant.

Table 1.5. Regression coefficients and SE for 2nd and 3rd lactation variables on PTA_{SCS}.

Lactation (#cows)	2 (172)		3 (100)	
Variable	β	SE	β	SE
Cases of mastitis ¹ (#)	.46	.40	.25	.60
Total days treated ¹ (days)	9.3	8.1	.84	16.1
Number of treatments ¹ (#)	1.4	.80	1.0	1.4
Lact. Avg. SCS ³	1.0	.55	-.88	.86
305-d M. E Milk ^{2,4} (kg)	544.2	2507.1	4197.9	3965.2
305-d M. E Fat ^{2,4} (kg)	-188.5	102.8	103.4	156.6
305-d M. E Protein ^{2,4} (kg)	- 62.9	81.2	43.3	129.6

¹ All variables not significant (P > 0.05).

² All variables not significant (P > 0.05).

³ All variables not significant (P > 0.05).

⁴ M.E. = Mature equivalent.

Regression coefficients of measures of mastitis on PTA_{SCS} declined with each lactation, going from .80 in 1st lactation analysis to .46 in 2nd lactation, and .25 in 3rd lactation analysis. Number of cows represented decreased from 304 to 172, to 100 with lactation number. The average percent of cows culled for mastitis per lactation in a previous study (13) was about 13% of the total disposals. Percent of cows culled for mastitis in this study was not known, but overall cull rates were very high. The heavy cull rate imposed on 1st lactation cows produced a population of highly selected 2nd and 3rd lactation daughters, and may be the major reason for the decrease in magnitude of the regression coefficients with increasing lactation number.

In first lactation, year season and PTA_{SCS} accounted for 11.5% of the variability in number of cases of mastitis, 8.6% of the variability associated with number of days treated, 12.1% of the variability associated with number of treatments, and 14.3% of the variability inherent in lactation average SCS. The R-square values were within expected ranges given the amount of genetic variation associated with mastitis was reported to be about 10% (41). Despite the lower regression coefficients calculated for 2nd and 3rd lactation (.46 and .25),

R-square values increased. The increase in R-square values was attributed to the decrease in overall variability resulting from the removal (culling) of the 1st lactation cows with a high incidence of clinical mastitis.

Differences between first and second, first and third, and second and third lactations in each daughter variable were also analyzed. Results of the analyses are in Table 1.6. None of the linear, quadratic, or cubic relationships between sire PTA_{SCS} and the differences in the variables between lactations were significant. Regression coefficients for total number of cases remained positive for analyses of 1st and 2nd lactation differences, 1st and 3rd lactation differences, and 2nd and 3rd lactation differences, but did not exhibit a significant relationship with sire PTA_{SCS} (P>0.05). Analysis of the difference between 1st and 2nd lactation number of cases of mastitis on PTA_{SCS} yielded a regression coefficient of .13. The difference in number of cases between 1st and 3rd lactation was higher at .24, but negligible between 2nd and 3rd lactations.

Table 1.6. Regression coefficients and SE of differences in variables related to mastitis resistance between lactations on PTA_{SCS}.

Variable	<u>Lact 2-1^a</u>		<u>Lact 3-1^a</u>		<u>Lact 3-2^a</u>	
	β	SE	β	SE	β	SE
Cases of mastitis (#)	.13	.05	.24	.63	.01	.67
Total days treated (days)	.48	9.1	-.89	16.6	.74	16.7
Number of treatments (#)	-.27	1.1	1.0	1.6	-.13	1.4
Lact. Avg. SCS	-.50	.55	1.14	.89	1.6	.86

^a All variables not significant P> 0.05

The resulting regression coefficients for the difference in days treated between 1st and 2nd lactations and 2nd and 3rd lactations on PTA_{SCS} were positive (.48 and .74, respectively), but

not significant ($P>0.05$). The coefficient resulting from the regression of the differences in number of days treated between 1st and 3rd lactations was negative (-.89) and not significant ($P>0.05$). Differences in total treatments per lactation maintained a negative linear relationship with sire PTA_{SCS} for differences between 1st and 2nd lactations and 2nd and 3rd lactations (-.27 and -.13, respectively), but exhibited a positive (1.0), though not significant ($P>0.05$), relationship to PTA_{SCS} for the difference between 1st and 3rd lactations. The regression of the difference between lactation 1 and 2 lactation average SCS on PTA_{SCS} was negative (-.50), but not significant. Differences between 1st and 3rd lactations and 2nd and 3rd lactations (1.14 and 1.6, respectively) were both positive but not significant ($P>0.05$).

Results of 1st lactation analyses confirmed the premise that a relationship existed between sire PTA_{SCS} and the incidence of clinical mastitis in his daughters. The positive correlations between PTA_{SCS} and mastitis variables in this study, as well as the correlations between the variables, were consistent with previous research (10, 17) and indicated a relationship between SCS and incidence of clinical mastitis. The expected relationship was seen in all lactations, however, it was not significant in second and third lactation. Lack of significance in 2nd and 3rd lactations may be attributable to intense culling of 1st lactation cows. Furthermore, the results of the study, while an indication of the relationship existing between a sire's PTA_{SCS} and the incidence of mastitis in his daughters, must be reviewed with consideration for potential biases. The small number of records analyzed, the fact that all the cows shared the same environment, and the heavy cull rate imposed on first lactation cows introduced additional sources of variation. Likewise, the heavy cull rate imposed on

first lactation cows was unrealistic for practical production situations, and resulted in a group of highly selected 2nd and 3rd lactation cows upon which the analyses did not prove meaningful.

To determine the effects of PTA_{SCS} on the longevity of female offspring, sires were grouped by PTA_{SCS} to determine cull rate of their daughters from 1st to 2nd lactation, and from 2nd to 3rd lactation. Results are in Table 1.7. Group 1 consisted of daughters of sires with a $PTA_{SCS} \leq 2.99$. These cows represented 19.4% of the 304 total cows in the data set. Thirty-nine percent (39%) of the group 1 daughters were culled after 1st lactation, and 36% of group 1 daughters were culled after 2nd lactation. Group 1 cull rates fell within the expected state average range of 35 – 38% and were thus not viewed as heavy or impractical. Furthermore, Group 1 cows had the highest rate of survival to 3rd lactation, and were surpassed in survival to 2nd lactation only by the group 2 cows (daughters of bulls with a $2.99 \leq PTA_{SCS} \leq 3.12$). Since it was determined that cows with high incidence of mastitis were culled in 1st lactation, it was inferred that group 1 cows had lower infection rates in 1st lactation and thus remained in the herd for later lactations. Group 2 cows represented 22% of the total cows and likewise had acceptable cull rates after 1st and 2nd lactation (37% and 38%, respectively). Likewise, both Group 1 and Group 2 were comprised of cows sired by bulls below breed average for PTA_{SCS} (3.2). These findings were supported by the work of Grootenhuis, who found that sire progeny groups with low average SCS had lower infection rates in first and later parities (20).

Table 1.7 Percentage of cows (number culled by sire PTA_{SCS}) culled during 1st and 2nd lactation.

Group	PTA _{SCS}	# Cows	% Culled (cows)	
			Lact 1-2	Lact 2-3
1	$x \leq 2.99$	59	39% (23)	36% (13)
2	$2.99 \leq x \leq 3.12$	67	37% (25)	38% (16)
3	$3.12 \leq x \leq 3.24$	55	44% (24)	48% (15)
4	$3.24 \leq x \leq 3.39$	45	51% (23)	32% (7)
5	$3.39 \leq x \leq 3.50$	44	45% (20)	63% (15)
6	$x \geq 3.51$	34	50% (17)	35% (6)
Total		304		

Group 6 was comprised of daughters of sires with the highest PTA_{SCS} (PTA_{SCS} ≥ 3.51) and represented 11.2 % of the total cows in the data set. Of group 6 cows, 50% were culled after 1st lactation, and 35% were culled after 2nd lactation. The cull rate of group 6 cows in 1st lactation was well above state average, and considered heavy. Comparing cull rate of daughters of low PTA_{SCS} bulls (≤ 2.99) versus cull rate of daughters of high PTA_{SCS} bulls (≥ 3.51) further supported the theory that high PTA_{SCS} bulls sire cows with increased incidence of mastitis and therefore a decreased opportunity for longer herd life.

The impact of the mastitis traits on longevity of cows in the data set was also examined by grouping cows by number of lactations initiated. Group 1 consisted of cows that survived culling after 1st lactation that had the opportunity for more than one lactation. Group 2 represented cows culled before 2nd lactation. Means and standard deviations of the variables analyzed for the 3 groups are in Table 1.8.

Table 1.8. Cows grouped by number of lactations initiated.

Group	1 n=172		2 n=90	
Variable	Mean	SD	Mean	SD
Cases of mastitis (#)	.40	.89	.63	1.2
Total days treated (days)	2.9	14.9	3.1	7.6
Number of treatments(#)	.98	2.4	1.8	3.5
305-d M.E Milk ¹ (kg)	10673.9	1255.8	10091.7	1806.8
305-d M.E Fat ¹ (kg)	374.6	51.4	351.2	59.7
305-d M.E Protein ¹ (kg)	320.2	38.4	303.9	49.0
Lact. Avg. SCS	2.2	1.3	3.1	1.8

¹ M.E. = Mature equivalent.

Group 1 consisted of 57% of the total cows in the data set, group 2, 29%, and group 3, 13% of the total cows. One hundred seventy two cows, of the 262 with opportunity for more than one lactation survived, while 90 cows were culled after 1st lactation. Comparison of Group 1 and Group 2 cows revealed that Group 2 cows exhibited .23 more cases of mastitis, .20 more days of treatment, and nearly double the number of treatments seen in Group 1 cows. Yields, however, were lower in Group 2 than in Group 1, indicating that the heavy culling imposed on 1st lactation cows focused both on production and incidence of mastitis.

Conclusions

Significant relationships were found between PTA_{SCS} and number of cases of clinical mastitis, days treated, number of treatments, and lactation average SCS in 1st lactation. Correlation between clinical mastitis and lactation average SCS were both positive and within reported range (10). Frequency of clinical mastitis, number of days treated, and number of treatments increased with lactation average SCS. The linear relationship between PTA_{SCS} and lactation average SCS was slightly greater than 1 ($\beta = 1.34$) in 1st

lactation, but not significant for 2nd ($\beta = 1.0$) or 3rd ($\beta = -.88$) lactations. Lack of significance in the later lactations could be explained by the intense cull rate imposed on 1st lactation animals which produced a population of selected animals, or by the theory that perhaps SCS early and late in life may be genetically different traits (17). The negative relationship in lactation 3 was attributed to the high level of prior culling.

Daughters of sires below breed average for PTA_{SCS} (3.22) had the highest survival rate to 3rd lactation, and thus longer productive life values than daughters of sires above breed average for PTA_{SCS} . Daughters of sires above breed average for PTA_{SCS} exhibited higher average number of cases of mastitis, days treated, and number of treatments, and exhibited decreased rates of survival.

While the number of records analyzed were not great enough to draw definitive conclusions, and the intense culling of 1st lactation cows was both practically unrealistic and yielded a selected population of 2nd and 3rd lactation cows, the study provided an optimistic indication of the relationship between sire PTA_{SCS} and incidence of mastitis in female offspring. Daughters of low PTA_{SCS} sires had lower incidence of clinical mastitis, and remained in the herd longer than daughters of high PTA_{SCS} bulls. Results indicate that selection for lower PTA_{SCS} would result in lower incidence of clinical mastitis in female offspring.

Data Set II

Means and standard deviations of lifetime economic variables of 2,494,195 cows, means and standard deviations of PTA_{SCS}, PTA_{Milk}, PTA_{Fat}, and PTA_{Pro} for sires of the cows, and the resulting correlations of each variable to sire PTA_{SCS} are in Table 1.9. Means for each sire PTA were weighted by number of daughters in the data set. Correlations of PTA_{Milk}, PTA_{Fat}, PTA_{Pro} and PTA_{SCS} were .19, .04, and .21, respectively. Though positive, PTA_{SCS} and PTA_{Fat} were virtually uncorrelated. PTA_{PL} exhibited a negative (biologically positive) relationship with PTA_{SCS}, with a correlation of only $-.03$. Mean PTA_{Milk}, PTA_{Fat}, PTA_{Protein} and PTA_{PL} were -72.7kg , -3.5kg , -3.2kg , and $-.02$ days, respectively. Negative values were not surprising considering the cows in the data set were born between 1979 and 1987 and the PTAs are on a 1990 genetic base. Average PTA_{SCS} was 3.2, the current breed average for U.S. Holsteins (24).

Table 1.9 Means, SD, and correlation to sire PTA_{SCS} of measures of daughter performance and sire genetic evaluations.

Variable	Mean	SD	Corr. to PTA _{SCS}
PTA Milk (kg)	-72.7	317.7	.19
PTA Fat (kg)	-3.5	11.8	.03
PTA Protein (kg)	-3.2	9.4	.21
PTA PL ¹	-.02	1.4	-.03
PTA SCS ²	3.2	.18	1.0
No. Lactations	2.9	1.7	-.04
Days PL ¹	911.4	586.4	-.03
Total Days in milk	736.1	445.5	-.03
SCS ²	2.6	1.4	.13
Lifetime Milk (kg)	20586.0	14207.0	-.02
Lifetime Fat (kg)	745.8	516.3	-.02
Lifetime Protein (kg)	651.1	445.9	-.02
RNIOCM ⁴ (\$)	26.8	737.1	.002
RNIOCF ³ (\$)	83.1	858.5	.001

¹ PL= Productive life.

² SCS= Somatic cell score.

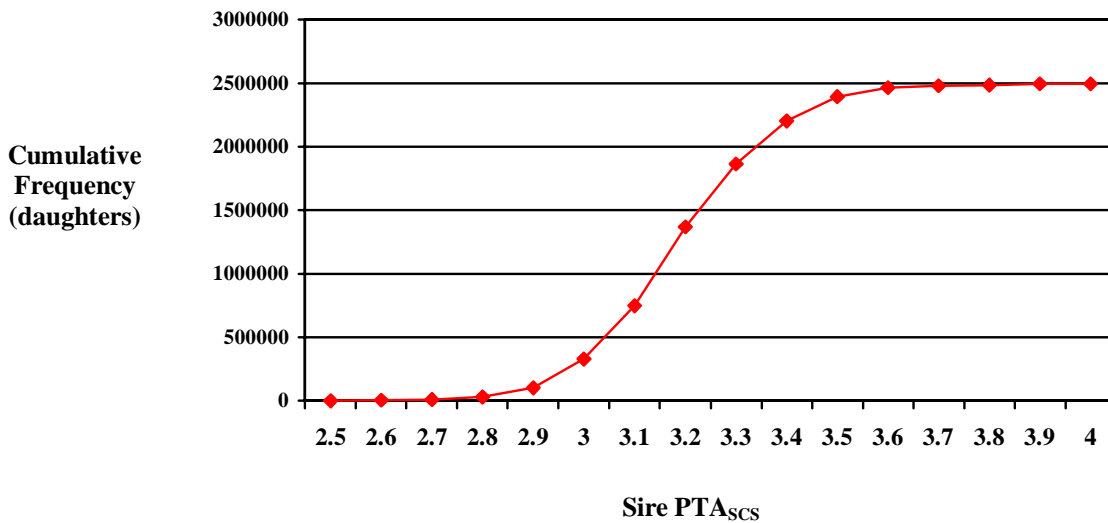
³ RNIOCF = Relative net income adjusted for opportunity cost in a fluid market.

⁴ RNIOCM = Relative net income adjusted for opportunity cost in a manufacturing market.

Of the daughter traits, number of lactations, DPL, TDIMM, and lifetime yield variables were all negatively correlated to PTA_{SCS} (range -0.02 to -0.04). Though negative relationships, the respective correlations of measures of daughter performance and sire PTA_{SCS} were nearly zero. The correlation of PTA_{SCS} and 1st lactation average SCS was $.13$. The RNIOC variables exhibited positive correlations to PTA_{SCS} , with coefficients of $.001$ and $.002$ respectively, but were also virtually zero. Cows in the data set averaged 2.9 lactations, 911 DPL, and 736 TDIMM. Average milk, fat, and protein yield were, 20586kg, 745kg, and 651kg, respectively. Relative net income adjusted for opportunity cost averaged \$83.1 in the fluid market, and \$26.8 in the cheese market. Average 1st lactation SCS for cows with SCS data ($n=152,588$) was 2.6.

The cumulative frequency of daughters of sires in the data set, by sire PTA_{SCS} , is in Figure 1. Examination of the cumulative frequency of daughters revealed that 98% of the cows in the data set had sires with PTA_{SCS} between the range of 2.81 and 3.62. Cows sired by bulls outside the PTA_{SCS} range of 2.81 and 3.62 were few in number and low in frequency. Sire PTA_{SCS} in the data set ranged from 2.55 to 3.97, a 1.4 unit range. The effective range in sire PTA_{SCS} , however, was $.81$.

Figure 1. Cumulative frequency of daughters of sires by sire PTA_{SCS} .



Results of the regression of measures of daughter performance on sire PTA_{SCS} [Model 2] are in Table 1.10. The linear regressions for all variables were significant ($P < 0.05$). The linear regression coefficient for number of lactations on PTA_{SCS} was -0.31 , which was equivalent to a predicted loss of $.31$ lactations per lifetime per unit increase in PTA_{SCS} . While the predicted loss in lifetime number of lactations per unit increase in sire PTA_{SCS} was statistically significant, the loss actually accounted for less than one-fifth of a standard deviation in number of lactations.

Table 1.10. Linear regression coefficients and standard errors for measures of daughter performance on sire PTA_{SCS}.

Variable	β	SE	β/σ_p^2
Number of Lactations ¹	-.31	.01	-.18
Days productive life ¹	-85.8	2.2	-.15
TDIMM ^{1,4}	-71.3	1.7	-.16
Total milk ¹ (kg)	-1567.5	53.4	-.11
Total fat ¹ (kg)	-79.0	1.9	-.15
Total protein ¹ (kg)	-46.8	1.7	-.10
1 st Lact. Avg. SCS ²	.99	.02	.70
RNIOCM ^{1,5} (\$)	-6.7	2.9	-.01
RNIOCF ^{3,6} (\$)	-15.8	3.3	-.02

¹ Number of cows = 2,494,195. All variables significant (P<0.05).

² Number of cows = 152,588. Significant (P<0.05).

³ Number of cows = 2,457 033. Significant (P<0.05).

⁴ TDIMM= Total days in milk.

⁵ RNIOCM= Relative net income adjusted for opportunity cost in a manufacturing market.

⁶ RNIOCF= relative net income adjusted for opportunity cost in a fluid market.

The regression of DPL on PTA_{SCS} yielded a coefficient of -85.8. The relationship was significant (P< 0.05), and implied a predicted loss of approximately 86 days of productive life per unit increase in PTA_{SCS}. Likewise, the relationship between PTA_{SCS} and TDIMM was significant (P<0.05). A unit increase in PTA_{SCS} would, on average, result in loss of 71.3 lifetime days in milk. The correlation between DPL and TDIMM of cows in the data set was .98, which explained the harmony of the respective regression coefficients. Again, while statistically significant, the predicted loss per unit increase in PTA_{SCS} accounted for less than two-tenths of one standard deviation in both DPL and TDIMM (-.15 and -.16, respectively). The low correlation between daughter DPL, TDIMM, and sire PTA_{SCS} (-.03) was an explanation for the small impact of the relationships.

The regression coefficients of lifetime yield variables on sire PTA_{SCS} were -1567.6, -79.0, and -46.8, for total milk, fat, and protein, respectively. All relationships were significant (P< 0.05). Thus, on the average, two bulls differing in one unit for PTA_{SCS} would be expected to have daughters that differ by approximately 1500kg in lifetime milk yield

Lifetime fat and protein were found to decrease by 79kg and 47kg per unit increase in PTA_{SCS} , respectively. Reduced production was due primarily to shorter herd life as was evident in the relationship between PTA_{SCS} and DPL and TDIMM. The relationship between PTA_{SCS} and 1st lactation average SCS was significant ($P < 0.05$). The regression of lactation average SCS on PTA_{SCS} yielded the expected coefficient of .99, which implied a one unit increase in lactation average SCS per each unit increase in PTA_{SCS} .

Analysis of the relationship between adjusted relative net income variables and PTA_{SCS} revealed a significant ($P < 0.05$) relationship. Per unit increase in PTA_{SCS} , relative net income adjusted for opportunity cost was found to decrease by -\$6.71 in the cheese market, and by -\$15.81 in the fluid market. The relationship seen between daughter RNIOC measures and increases in PTA_{SCS} supported earlier research concerning the effects of selection for increased yield on the overall economic merit of dairy cattle (50). Selection for maximum production as a means to increase profit seldom accounts for the increased health, labor, and feed costs which accompany higher yields. The RNIOC calculations analyzed did not include several of the important expenses associated with mastitis including: cost of treatments, discarded milk, and lower salvage value. Had the RNIOC values included costs associated with mastitis, the observed impact of increased PTA_{SCS} may have been greater.

Results of the investigation of possible quadratic relationships [Model 3] between measures of daughter performance and sire PTA_{SCS} are in Table 1.11. The linear (for all traits) and quadratic (for all traits but SCS) effects of the regression of daughter traits on PTA_{SCS} were

significant ($P < 0.05$). All linear regression coefficients, excepting that which resulted from the regression of 1st lactation average SCS on PTA_{SCS} , were negative. All the quadratic regression coefficients, with the exception of 1st lactation average SCS, were positive.

Table 1.11. Linear and quadratic regression coefficients and standard errors for measures of daughter performance on sire PTA_{SCS} .

Variable	Linear		Quadratic		P > F ^a
	β	SE	β	SE	
Number of Lactations ¹	-0.02	.001	2.1×10^{-5}	2.2×10^{-6}	**
Days productive life ¹	-3.2	.50	4.0×10^{-3}	8.0×10^{-4}	**
TDIMM ^{1,4}	-2.4	.37	3.0×10^{-3}	6.0×10^{-4}	**
Total milk ¹ (kg)	-101.6	11.8	.13	.018	**
Total fat ¹ (kg)	-3.4	.43	4.1×10^{-3}	8.0×10^{-4}	**
Total protein ¹ (kg)	-2.3	.37	3.0×10^{-3}	6.0×10^{-4}	**
1 st Lact. Avg. SCS ²	-5.6	.63	9.0×10^{-3}	.001	*
RNIOCM ^{1,5} (\$)	.012	.01	-4.0×10^{-6}	1.0×10^{-5}	**
RNIOCF ^{3,6} (\$)	-8.3	.74	.013	1.1×10^{-3}	**

¹Number of cows = 2,494,195.

²Number of cows = 152,588.

³Number of cows = 2,457,033.

⁴TDIMM= Total days in milk.

⁵RNIOCM= Relative net income adjusted for opportunity cost in a manufacturing market.

⁶RNIOCF= relative net income adjusted for opportunity cost in a fluid market.

^aStatistical significance is indicated with asterisk marks as follows: *, Linear effects, ($P < 0.05$), quadratic effects, ($P > 0.05$); **, Linear and quadratic effects ($P < 0.05$).

Further analysis was conducted to determine the presence of possible cubic relationships. Results of the analysis of linear, quadratic, and cubic effects [Model 1] of measures of daughter performance on sire PTA_{SCS} are in Table 1.12. The pattern of significance for measure of daughter performance on PTA_{SCS} was the same as for the quadratic for all traits other than SCS.

Table 1.12. Linear, quadratic, and cubic regressions and standards errors for measures of daughter performance on PTA_{SCS}.

Variable	Linear		Quadratic		Cubic		P > F ^a
	β	SE	β	SE	β	SE	
Number of Lactations ¹	-0.27	0.02	0.001	1.0x10 ⁻⁴	-1.0x10 ⁻⁶	6.0x10 ⁻⁸	***
Days productive life ¹	-92.8	6.0	0.28	0.02	-3.0x10 ⁻⁴	2.0x10 ⁻⁵	***
TDIMM ^{1,4}	-67.1	4.5	0.20	0.01	-2.0x10 ⁻⁴	1.4x10 ⁻⁵	***
Total milk ¹ (kg)	-2510.1	144.4	7.6	0.45	-0.008	5.0x10 ⁻⁴	***
Total fat ¹ (kg)	-98.5	5.2	0.30	0.02	-3.0x10 ⁻⁴	2.0x10 ⁻⁵	***
Total protein ¹ (kg)	-77.9	4.5	0.24	0.01	-2.0x10 ⁻⁴	1.4x10 ⁻⁵	***
1 st Lact. Avg. SCS ²	-149.6	7.7	0.50	0.02	-5.0x10 ⁻⁴	3.0x10 ⁻⁵	NS
RNIOCM ^{1,5} (\$)	-0.08	0.06	3.0 x 10 ⁻⁴	2.0x10 ⁻⁴	-3.0x10 ⁻⁷	2.0x10 ⁻⁷	***
RNIOCF ^{3,6} (\$)	-157.6	9.0	0.50	0.03	-5.0x10 ⁻⁴	3.0x10 ⁻⁵	***

¹ Number of cows = 2, 494, 195.

² Number of cows = 152, 588.

³ Number of cows = 2, 457, 033.

⁴ TDIMM= Total days in milk.

⁵ RNIOCM= Relative net income adjusted for opportunity cost in a manufacturing market.

⁶ RNIOCF= relative net income adjusted for opportunity cost in a fluid market.

^a Statistical significance is indicated with asterisk marks as follows: *** Linear, quadratic, cubic (P<0.05). NS=not significant.

The impact of these coefficients is best seen in Figures 2 – 10 (Appendix A) which compare the results of the linear, quadratic, and cubic models. For most traits the relationship is almost linear over the range that includes sires of 98% of the cows (Figure 1). Comparison of R-square values from linear, quadratic and cubic regressions in Table 1.13 reveal no additional variation explained by quadratic or cubic relationships, and support the conclusion of a linear relationship. Quadratic and cubic relationships were more likely bull effects caused by outlier bulls than actual significant quadratic and cubic effects.

Table 1.13. Comparison of R-Square values for linear, quadratic, and cubic regressions of measures of daughter performance on PTA_{SCS}.

Variable	Linear	Quadratic	Cubic
	R ²	R ²	R ²
Number of Lactations ¹	.1168	.1168	.1169
Days productive life ¹	.1116	.1116	.1117
TDIMM ^{1, 4}	.1139	.1139	.1139
Total milk ¹ (kg)	.1157	.1157	.1158
Total fat ¹ (kg)	.1186	.1186	.1187
Total protein ¹ (kg)	.1162	.1162	.1163
RNIOCM ^{1, 5} (\$)	.0606	.0606	.0608
RNIOCF ^{3, 6} (\$)	.0690	.0691	.0692

¹Number of cows = 2, 494, 195.

²Number of cows = 152, 588.

³Number of cows = 2, 457, 033.

⁴TDIMM= Total days in milk.

⁵RNIOCM=Relative net income adjusted for opportunity cost in a manufacturing market.

⁶RNIOCF= Relative net income adjusted for opportunity cost in a fluid market.

Comparison of the results of the partial regression analyses of measures of daughter performance on PTA_{SCS} and PTA_{Milk}, PTA_{Fat}, PTA_{Protein}, and PTA_{PL} are in Table 1.14. All regressions were significant, except the relationship between PTA_{SCS} and RNIOCM holding PTA_{Fat} constant, and the relationship between PTA_{SCS} and RNIOCF, holding PTA_{PL} and MFPS constant. Simple regression of number of lactations on PTA_{SCS} was $-.31$ which translated to a .31 loss in lifetime number of lactations per unit increase in PTA_{SCS}. Results of the partial regression analysis of number of lactations on PTA_{SCS} and PTA_{Milk} [Model 4] was $-.41$ which corresponded to a loss in approximately 0.41 lactations per unit increase in PTA_{SCS} when variation in PTA_{Milk} was held constant. Similar partial regression analyses including PTA_{Fat} [Model 5] and PTA_{Protein} [Model 6] yielded coefficients of $-.30$ and $-.42$, respectively. Results indicated that PTA_{SCS} accounted for a higher loss in number of lifetime lactations when variation in PTA_{Milk} and PTA_{Protein} were held constant. Significant genetic correlations existed between PTA_{SCS} and PTA_{Milk} and PTA_{Protein} (.19 and .21, respectively). Increased milk and protein yields offset the loss in

number of lactations explained by increase in PTA_{SCS} when neither PTA_{Milk} or $PTA_{Protein}$ were considered; thus, holding PTA_{Milk} and $PTA_{Protein}$ constant revealed greater loss in number of lactations explained by increases in PTA_{SCS} . Holding variation in PTA_{Fat} constant yielded results similar to the simple linear regression of number of lactations on PTA_{SCS} . The similarity of the relationships was a result of the lack of correlation between PTA_{SCS} and PTA_{Fat} (0.03). Thus, holding PTA_{Fat} constant had virtually no effect on the relationship between number of lactations and PTA_{SCS} . When PTA_{PL} and $MFPS$ [Model 8] were included in the analysis, the relationship between PTA_{SCS} and number of lactations became positive. Per unit increase in PTA_{SCS} , predicted number of lifetime lactations increased by .028 lactations when the variation in PTA_{PL} and $MFPS$ were held constant. A similar relationship was found between PTA_{SCS} and PTA_{PL} and MF [Model7]. With the variation in PTA_{PL} and MF held constant, number of lifetime lactations was predicted to increase by .031 lactations per unit increase in PTA_{SCS} . The positive relationship between PTA_{SCS} and number of lifetime lactations when PTA_{PL} was considered resulted from the fact that loss in number of lifetime lactations explained by increases in PTA_{SCS} was also explained by increases in PTA_{PL} . Results indicate that the most negative impact of increased PTA_{SCS} on measures of daughter performance was through the effect of PTA_{SCS} on length of productive life. Increases in PTA_{SCS} resulted in a reduction in lifetime number of lactations (longevity), and thus reduced the potential for increased lifetime yields.

Table 1.14. Comparison of partial regressions of measures of daughter performance on PTA_{SCS} holding other sire PTA variables constant.

Variable	$\beta_{y, SCS}^6$	$\beta_{y, SCS, M}^7$	$\beta_{y, SCS, F}^8$	$\beta_{y, SCS, P}^9$	$\beta_{y, SCS, MFP\$, PL}^{10}$	$\beta_{y, SCS, MF\$, PL}^{11}$
Number of Lactations ¹	-.31	-.41	-.30	-.42	.028	.031
Days of productive life ¹	-85.8	-124.6	-83.2	-----	7.0	10.1
TDIMM ^{1,3}	-71.3	-98.8	-69.5	-102.3	8.0	9.9
Total milk ¹ (kg)	-1567.6	-2911.0	-----	-----	-----	-----
Total fat ¹ (kg)	-79.0	-----	-75.3	-----	-----	-----
Total protein ¹ (kg)	-46.8	-----	-----	-94.4	-----	-----
RNIOCM ^{1,4} (\$)	-6.7	-100.6	-0.73 ^a	-115.3	7.0	25.5
RNIOCF ^{2,5} (\$)	-15.8	-125.5	-10.1	-122.7	3.4 ^a	12.9

¹ Number of cows = 2, 494, 195.

² Number of cows = 2, 457, 033.

³ TDIMM= Total days in milk.

⁴ RNIOCM= Relative net income adjusted for opportunity cost in a manufacturing market.

⁵ RNIOCF= relative net income adjusted for opportunity cost in a fluid market.

⁶ Linear regression of y variables on PTA_{SCS}.

⁷ Partial regression of y variables on PTA_{SCS} holding PTA_{Milk} constant.

⁸ Partial regression of y variables on PTA_{SCS} holding PTA_{Fat} constant.

⁹ Partial regression of y variables on PTA_{SCS} holding PTA_{Protein} constant.

¹⁰ Partial regression of y variables on PTA_{SCS} holding PTA_{PL} and dollar value for protein constant.

¹¹ Partial regression of y variables on PTA_{SCS} holding PTA_{PL} and dollar value for milk and fat constant.

^a Variable not significant (P>0.05).

Partial regression of days of productive life on PTA_{SCS}, holding the variation in PTA_{Milk} constant [Model 4] revealed a loss in predicted DPL of approximately 125 days. A similar partial regression analysis including PTA_{Fat} [Model 5] yielded the coefficient of -83.2. Results indicated that PTA_{SCS} accounted for a higher loss in DPL when PTA_{Milk} was held constant. Partial regression of DPL on PTA_{SCS} while including PTA_{PL} and MFP\$ [Model 8] was 7.0, which indicated a 7 day increase in predicted DPL per unit increase in PTA_{SCS} when variation in PTA_{PL} and MFP\$ were held constant. When MF\$ and PTA_{PL} were held constant, [Model 7] the partial regression analysis yielded a coefficient of 10.1 which implied a 10 day increase in predicted DPL per unit increase in PTA_{SCS}. The change from a negative to positive relationship after the inclusion of PTA_{PL} as a continuous variable reflected the relationship between PTA_{SCS} and number of lactations, and implied that loss

in DPL previously explained by increase in PTA_{SCS} was also explained by variation in PTA_{PL} .

The partial regression of TDIMM on PTA_{SCS} holding PTA_{Milk} constant [Model 4] implied a loss of 99 lifetime days in milk per unit increase in PTA_{SCS} . Predicted decrease in TDIMM per unit increase in PTA_{SCS} was 70 days when PTA_{Fat} was held constant [Model 5]. The regression of TDIMM on PTA_{SCS} and $PTA_{Protein}$ was -102.3 [Model 6]. Partial regression of TDIMM on PTA_{SCS} , PTA_{PL} , and MFPS [Model 8] estimated an 8 day increase in TDIMM per unit increase in PTA_{SCS} . A positive relationship between TDIMM and PTA_{SCS} was likewise evidenced when variation in PTA_{PL} and MF\$ [Model 7] were held constant. Partial regression of TDIMM on PTA_{SCS} , PTA_{PL} , and MF\$ estimated a 10 day increase in TDIMM per unit increase in PTA_{SCS} . When variation in PTA_{PL} was held constant, it was evident that the decrease in TDIMM which was previously attributed to increase in PTA_{SCS} was also accounted for by PTA_{PL} . The similarity in results between number of lactations, DPL and TDIMM was explained by the high phenotypic correlations between all three traits.

Partial regression of total milk on PTA_{SCS} and PTA_{Milk} [Model 4] was -2911.0, which implied a loss of 2911kg in predicted lifetime milk production per unit increase in PTA_{SCS} when PTA_{Milk} was held constant. Because of a positive correlation between PTA_{SCS} and PTA_{Milk} , selection for higher PTA_{Milk} caused selection for higher PTA_{SCS} . Increased milk yield per lactation offset part of the actual loss in lifetime milk production associated with the increase in PTA_{SCS} . Loss in predicted lifetime milk was thus greater when variation in PTA_{Milk} was held constant.

Partial regression analysis of total fat production on PTA_{SCS} and PTA_{Fat} [Model 5] was -75.3. When PTA_{Fat} was held constant, decrease in predicted lifetime fat production was 75kg. The two estimates were very similar due to the virtually nonexistent correlation ($r = 0.03$) between PTA_{SCS} and PTA_{Fat} . Thus, holding PTA_{Fat} had little effect on the loss in lifetime fat production associated with increased PTA_{SCS} .

Regression of lifetime protein on PTA_{SCS} including $PTA_{Protein}$, [Model 6] however, suggested a 94 kg loss in predicted protein production per unit increase in PTA_{SCS} . Not accounting for increase in $PTA_{Protein}$ in the simple regression caused the loss explained by increase in PTA_{SCS} to be underestimated. The relationship evident between 1st lactation average SCS and sire PTA_{SCS} was linear and significant ($P < 0.05$). The resulting partial regression coefficients did not differ significantly from the linear regression coefficient.

A \$6.70 loss in RNIOCM was associated with a unit increase in PTA_{SCS} . When PTA_{Milk} was held constant, the loss in RNIOCM per unit increase in PTA_{SCS} was \$100.60. While the increase seemed drastic, it accounted for approximately one-eighth of a standard deviation of the difference in RNIOCM values. When PTA_{Fat} was held constant, the loss in RNIOCM value explained by a unit increase in PTA_{SCS} was reduced to \$ 0.73. When $PTA_{Protein}$ was held constant, the loss in RNIOCM associated with PTA_{SCS} increased to \$115.30.

When MFP\$ and PTA_{PL} were held constant, the relationship between RNIOCM and PTA_{SCS} became positive. A unit increase in PTA_{SCS} was associated with a \$7.00 increase in RNIOCM in a manufacturing market, and a \$25.50 increase in RNIOCM in a fluid market. As was seen in the partial regressions of number of lactations, DPL, and TDIMM on PTA_{SCS} when PTA_{PL} was held constant, losses attributed to increase in PTA_{SCS} were also explained by variation in PTA_{PL}.

Comparison of the simple and partial regression analyses of RNIOCF on PTA_{SCS} revealed relationships similar to those seen with RNIOCM. Loss in RNIOCF increased to -\$125.50 and -\$122.70 when variation in PTA_{Milk} [Model 4] and PTA_{Protein} [Model 6] were held constant, respectively. The loss associated with a unit increase in PTA_{SCS} when variation in PTA_{Fat} [Model 5] was held constant was -\$10.10. As seen in other analyses, the relationship between RNIOCF and PTA_{SCS} when PTA_{PL} and MFP\$ [Model 8] were held constant became positive. Per unit increase in PTA_{SCS}, RNIOCF values increased \$3.40 when PTA_{PL} and MFP\$ were held constant. Likewise, when PTA_{PL} and MF\$ [Model 7] were held constant, a unit increase in PTA_{SCS} was associated with a \$12.90 increase in RNIOCF values.

Past research has shown that highest profits are gained from high yielding cows that remain in the herd for several lactations (18, 54). Dairy producers are concerned, however, that high yielding cows may not survive very long (54). Cows are culled for a variety of reasons, the most common being low production, reproductive disorders, and mastitis. Most culling decisions are for economic reasons. Characteristics that affect culling

decisions can be partitioned into either production or other traits (15). Other traits include mastitis, reproductive performance, other health disorders, and management traits (15). Reduced milk production accounts for 69 – 80% of the total loss attributable to mastitis (6, 19). Mastitis could therefore be both a direct and indirect reason for culling. Given the established relationship between elevated SCS and incidence of mastitis (10, 16, 43, 56), an indirect relationship existed between SCS and herd life. The indirect relationship between SCS and longevity was evident in the study as losses in number of lactations, DPL, TDIMM, RNIOCM, and RNIOCF explained by increased PTA_{SCS} in simple regression analysis became positive relationships when variation in PTA_{PL} was held constant. The negative impact of sire PTA_{SCS} on measures of daughter performance therefore proved to be the negative relationship which existed between increased SCS and length of herd life.

Conclusions

Significant relationships were found between measures of daughter performance and sire PTA_{SCS} . Generally, lifetime yields, measures of herd life, and measures of profitability decreased as sire PTA_{SCS} increased, indicating that increased PTA_{SCS} decreased herd life and thus reduced potential lifetime yields and profit. The negative effect of PTA_{SCS} on herd life was evident in the relationship discovered between PTA_{SCS} and PTA_{PL} . Partial regression analyses including PTA_{PL} revealed positive relationships between PTA_{SCS} and measures of herd life and daughter profitability, whereas linear relationships were negative. The implication of the reversal of the relationship from negative to positive was that losses explained by increase in PTA_{SCS} were also explained by PTA_{PL} .

It is evident that the most profitable cows are those with high yields that remain in the herd for many lactations and produce multiple offspring. Thus, for dairy cattle, the ability to survive involuntary or premature culling is a trait of economic importance. If a cow is culled as a result of high SCS or incidence of mastitis, her opportunity for multiple lactations and increased days of productive life (longer herd life) are cut short. Increased herd life, on the other hand, decreases costs associated with raising or purchasing replacements. Likewise, longer herd life increases average production in the herd because if incidence of mastitis is low, culling can be based strictly on production, and there would be a higher percentage of mature cows producing at higher levels. The most negative impact of increased PTA_{SCS} on measures of daughter performance is thus the relationship which exists between increased incidence of mastitis and decreased herd life. Selection on PTA_{SCS} would result in lower incidence of mastitis and increased profitability due to increased longevity.

CHAPTER II

THE RELATIONSHIP BETWEEN SIRE PTA_{SCS} AND VARIATION IN TEST DAY SCS MEASURES ACROSS MULTIPLE LACTATIONS

ABSTRACT

The relationship between sire PTA_{SCS} and variation in daughter test day SCS measurements was analyzed for 25,333 cows freshening prior to 1994. A total of 59,426 1st, 2nd, and 3rd lactations records were analyzed. Average sire PTA_{SCS} of daughters in the data set was 3.24. No prior sire selection on PTA_{SCS} had been practiced. Correlation of PTA_{SCS} and lactation average SCS ranged from 0.17 to 0.14 for 1st through 3rd lactation cows, respectively. Correlations of lactation average SCS between lactations was higher between 2nd and 3rd lactation (.50) than between 1st and later lactations. Significant linear relationships were found between averages of test day SCS records and PTA_{SCS} in 1st, 2nd, and 3rd lactations. All linear regressions of averages of test day scores on PTA_{SCS} in 1st, 2nd, and 3rd lactations were equal or close to 1.0. Correlations between 1st lactation SCS measurements and 2nd and 3rd lactation measurements were positive and greater between 2nd and 3rd lactation than between 1st and later lactations. In 1st lactation, significant relationships were found between PTA_{SCS} and the difference in SCS test day measures from the beginning to the end of lactation (-.26). No significant impact of PTA_{SCS} on the changes in test day SCS measures from beginning to end of lactation were found in 2nd or 3rd lactation. The regression of test day SCS on test day DIM was calculated for 1st, 2nd, and 3rd lactations. In 1st lactation, the regression of changes in test day SCS with test day DIM on PTA_{SCS} was significant (5.9×10^{-4}). No significant relationship between PTA_{SCS} and changes in test day SCS with test day DIM was seen in 2nd or 3rd lactation. In all lactations, the regression of lactation average SCS on PTA_{SCS} was significant. All regressions of lactation average SCS on PTA_{SCS} were equal or close to 1.0. Results indicate that the relationship between PTA_{SCS} and test day SCS measurements in 1st lactation may be different than the relationship in later parities. Lack of significant relationships in 2nd and 3rd lactations and higher correlations between 2nd and 3rd lactation SCS measures indicate that SCS in young and mature cows may be two different but correlated traits. Selection on PTA_{SCS} to lower SCS should be effective in reducing the variation in SCS, but selection in 1st lactation may not be effective in reducing SCS in later lactations.

Key Words (PTA_{SCS}, SCS, selection)

INTRODUCTION

Genetic improvement of dairy cattle is aimed at increasing total economic merit, thus the inclusion of mastitis in a breeding program should be examined. Consideration of mastitis could reduce milk production costs and involuntary cull rates as well as improve milk quality and the health of dairy cattle. Direct selection for reduced incidence of mastitis could be based on bacteriological testing or direct measurement, but neither are routinely practiced in the United States because of high associated costs and the lack of a standardized means of recording cases of mastitis. Thus, selection for increased resistance to mastitis has been directed towards a cow's innate defense mechanisms (35). Selection for lower SCS is indirect selection for reduced incidence of mastitis. Currently, PTA_{SCS} is the primary selection criteria for reducing incidence of mastitis.

Somatic cells in milk are primarily white blood cells that migrate from the blood to the mammary gland in response to damage or infection. Somatic cells are comprised of PMN, which possess phagocytic qualities, and thus represent a primary defense mechanism of the cow to eliminate infections and repair damaged tissue (38, 51). When somatic cells are high in number, a mammary infection is indicated, as somatic cells are specific and only elevated in the presence of infection.

Transformation of SCC to linear somatic cell scores (SCS) allowed for the application of traditional statistical methods of genetic evaluation (1). Individual SCC were converted by DHI Dairy Records Processing Center to SCS using a base 2 logarithm. SCS proved to be a

more reliable indicator trait than SCC, as SCS exhibited a linear relationship to milk loss, the correlation of mastitis and SCS (0.60) was higher than that between SCC and mastitis (43, 56), and SCS had a higher heritability (0.20) than SCC (10, 35, 41, 56). Therefore, selection for decreased somatic cell scores (SCS) should be a means of decreasing mastitis infections (7, 21).

Dairy Herd Improvement (DHI) tests for somatic cells are taken at monthly intervals, and are based on bucket milk samples. Therefore, monthly SCS are an estimate of the health status of all four quarters at the time of the sample. An average of the monthly tests, referred to as the lactation average SCS, is a rough characterization of the udder health of a cow throughout a lactation. Typically, a low SCS is an indication of the absence or low level of infection (43).

Genetic evaluations for SCS are calculated for cows and bulls by animal model procedures using data collected through DHI Associations (41, 46). Animal breeding values are estimated simultaneously with management groups, herd-sire interaction effects, and permanent environmental effects (43). Differences in sire predicted transmitting abilities for SCS are therefore estimates of the difference in lactation average SCS of future daughters in the same environment (46).

Selection for lower SCS, however, raises the question as to whether differences in SCS reflect differences among cows in their ability to respond to or preclude invading pathogens in the udder (30, 35). Selection on low SCS favors cows with low incidence of

infection as well as cows with a low somatic cell response to the presence of infection. Is the low response to infection a reflection of the cow's ability to respond quickly and return to normal cell levels, or of the decreased ability of the cow to mount a response to infection? To attempt to answer these questions, the effects of PTA_{SCS} on incidence of mastitis among daughters as measured by SCS over test days and lactations should be examined.

Several factors have previously been identified as sources of variation in SCS among cows including: presence or absence of infection, causative agent, age, and stage of lactation. If PTA_{SCS} is found to impact changes in SCS, a relationship could be drawn between PTA_{SCS} and a cow's response to infection. Furthermore, a significant relationship between PTA_{SCS} and test day SCS, changes in test day SCS from test day to test day, changes in test day SCS from the beginning to the end of a lactation, or lactation average SCS would indicate that selection for lower SCS via the inclusion of PTA_{SCS} in a breeding program could reduce incidence of mastitis in female offspring.

The objective of this study was:

1. To determine the relationship between PTA_{SCS} and incidence of mastitis among daughters as measured by SCS over test days and lactations.

MATERIALS AND METHODS

Test day data on 82,866 registered and grade Holsteins from 362 Virginia dairy herds were obtained from the Animal Improvement Programs Laboratory (AIPL) in Beltsville, Maryland. Lactations were comprised of test day segments, with each segment including days in milk at test day, test day pounds of milk, test day fat and protein percent, test day somatic cell score (SCS), and test day frequency of milking. Initially, the number of lactations represented was 180,885. Preliminary edits required a first freshening date of 1994 or earlier. Only records on 1st, 2nd, and 3rd lactations were included. Following initial edits, data from 75,222 lactations remained.

Further edits removed records with 1st test day DIM greater than 60 days. Likewise, records with greater than 60 days between 1st and 2nd test days were removed. After these additional edits, data from 59,426 lactations remained. Sire PTA_{SCS} was obtained from the November, 1998 Sire Summary and merged with the cow data. The range of sire PTA_{SCS} in the data set was 2.7 to 4.0, with an average and standard deviation of 3.24 and 0.21, respectively.

Means were calculated for all test day segments (n=59,426) including lactation number, total days in milk for each lactation, number of test day segments, test day days in milk, milk yield, fat and protein percents, test day SCS, and frequency of milking on test day. Means were likewise calculated for all test day measurements by lactation. Means were calculated for averages of test day segments 1 and 2, 3 and 4, 5 and 6, 7 and 8, and 9 and

10. To investigate the relationship between PTA_{SCS} and test day measures of SCS, segment averages in each lactation were regressed on PTA_{SCS} using the GLM procedure in SAS (Carey, NC). To investigate possible genetic factors influencing changes in SCS, differences between segment averages within each lactation were regressed on PTA_{SCS} . Correlations of test day SCS measurements, averages of test day SCS measurements, and PTA_{SCS} were calculated using the CORR procedure in SAS (Carey, NC).

Somatic cell scores were regressed on test day DIM for each lactation record containing at least 6 test day segments (n=50,433). Lactation average SCS, averages of test day SCS records, differences between averages of test day SCS records, the regression coefficients of test day SCS on test day DIM, and differences in regression coefficients between lactations were regressed on PTA_{SCS} using the GLM procedure in SAS (Carey, NC). The later two analyses were done to determine the relationship of PTA_{SCS} and changes in SCS over time. In each analysis, the effects of herd, year, and season of calving were absorbed. The following model was used for each analysis:

$$\text{Model: } Y_{ijk} = \alpha + hys_i + \beta_1 x_k + \beta_2 x_k^2 + \beta_3 x_k^3 + \epsilon_{ijk}$$

Where: Y_{ijk} = Average of test day SCS records
Differences between averages of test day SCS records
Regression coefficients of test day SCS on test day DIM
Lactation average SCS

For the j^{th} daughter of the k^{th} sire freshening in the i^{th} herd-year-season.

hys_i = the i^{th} effect of herd, year, and season of freshening.

$\beta_1 x_k$, $\beta_2 x_k^2$, $\beta_3 x_k^3$ = linear, quadratic, and cubic regression on PTA_{SCS} of the kth sire.

ε_{ijk} = residual variance of the ijkth observation.

Two reduced models were used to examine linear, and quadratic relationships.

RESULTS AND DISCUSSION

There were 59,426 lactation records in this data set with 25,269 first lactation records, 20,043 second lactation records, and 14,114 third lactation records. Means and standard deviations of all test day lactation records ($n = 59,426$) and sire genetic evaluations are in Appendix B. Cows in the data set averaged 1.8 lactations, with all cows required to have a first lactation, and only records up to and including 3rd lactation considered. Across all lactations, the average number of test day segments was 8.0. Average number of days in milk at last test day was 248.5d. Average DIM at first test day was 24.2d, and average DIM at the 10th test day was 291.6d. First test day milk yield average was 30.6kg, and last test day milk yield average was 21.4kg. Average PTA_{SCS} of sires of daughters in the data set was 3.24, and average PTA_{Milk} was 239.1kg.

Means, standard deviations, and correlations of PTA_{SCS} with all test day measures of SCS are in Table 2.1. The average PTA_{SCS} of daughters in the data set was 3.24, close to breed average for Holsteins (24). All cows had a first test day, but not all had a first test day SCS measurement. Across all lactations, cows with a first test day SCS ($n=55,547$) averaged an SCS of 3.1. Tenth test day SCS averaged 3.4. The lowest average test day SCS measurement was 2nd test day, and the highest test day SCS occurred on the 9th test day. A gradual increase in test day SCS was seen after the 3rd test day. There was a slight decline in test day SCS from the 9th to 10th test day (3.5 to 3.4, respectively). The decrease could indicate that some high SCS cows were culled or dried off between 9th and 10th test day.

Table 2.1. Means, SD, and correlations of test day SCS and sire PTA_{SCS} for all lactations .

Variable	N	Mean	SD	Corr. to PTA _{SCS}
PTA _{SCS}	59419	3.24	.21	1.0
SCS 1	55547	3.2	2.0	.10
SCS 2	52806	2.9	2.0	.11
SCS 3	51905	3.0	2.0	.12
SCS 4	50938	3.1	2.0	.11
SCS 5	49858	3.2	1.9	.11
SCS 6	48407	3.3	1.9	.12
SCS 7	46560	3.3	1.9	.11
SCS 8	42925	3.4	1.8	.12
SCS 9	35318	3.5	1.8	.11
SCS 10	12148	3.4	1.8	.11

Correlation of test day SCS measurements and PTA_{SCS} ranged from .10 to .12 across all lactations. Correlations were positive, which coincided with earlier reports (10) of positive correlations between daughter cell counts and sire genetic evaluations. Correlations were lower than those seen between genetic evaluations for yield and yield traits, due partly to the failure of the monthly sampling scheme to detect all cases of mastitis caused by environmental pathogens. Infections caused by environmental pathogens such as *Escherichia coli* are becoming more prevalent, and are characterized by elevations in SCS which last only a short duration (43). Since SCS are only measured once a month, chances of detecting all cases of mastitis caused by environmental pathogens are reduced. Test day SCS typically reflect only 10 – 20% of the cases of mastitis caused by environmental pathogens (43), thus lowering both the correlation between SCS and mastitis, and the correlation between SCS and PTA_{SCS}. An additional reason for the correlation between SCS and PTA_{SCS} to be lower than correlations seen for yield traits, however, was decreased genetic influence and more environmental effects contributing to the control of SCS.

Means and standard deviations of test day measurements and sire genetic evaluations, by lactation, are in Appendix C. First lactation cows averaged 8.2 test day segments, and 251.6 total DIM. Second lactation animals averaged 8.0 test day segments and 249.3 DIM, while third lactation averages were 7.7d and 241.8d, respectively. Average DIM at first test day was 24.2d for 1st lactation cows, 23.9d for 2nd lactation cows, and 24.5d for 3rd lactation cows. Corresponding average DIM on the 10th test day were 292.3d, 291.5d, and 290.1d for 1st, 2nd, and 3rd lactation cows, respectively. First lactation cows averaged 25.2kg of milk on first test day, while 2nd and 3rd lactation cows averaged 33.8kg, and 35.7kg, respectively. Final test day yields were 21.7kg, 21.0kg, and 21.3kg for 1st, 2nd, and 3rd lactation animals. Mean sire PTA_{SCS} of the 1st lactation cows in the data set was 3.24, while sire PTA_{Milk} averaged 239.9kg. For 2nd and 3rd lactation cows, sire PTA_{SCS} and PTA_{Milk} averaged 3.24 and 241.3kg, and 3.23 and 234.3kg, respectively.

Means, standard deviations, and correlations of PTA_{SCS} with test day measures of SCS, by lactation, are in Table 2.2. Initial test day SCS averages were 3.3, 3.0, and 3.3, for 1st, 2nd, and 3rd lactation cows, respectively. Final test day SCS averages were 3.1, 3.6, and 4.0, respectively. A decrease in test day SCS on the second test day was seen in 1st, 2nd, and 3rd lactation cows (2.8, 2.8 and 3.2, respectively). In first lactation, the initial test day SCS was higher than all later test day SCS during the lactation. The higher first test day SCS measurement in 1st lactation was similar to the findings of Jones et al. (27) who reported higher first test day SCC than second and later test day SCC for cows in first lactation. In general, test day SCS exhibited a gradual increase after the third test day across all parities. Likewise, test day somatic cell scores increased with stage of lactation and lactation

number. Higher test day SCS were evident in the later lactations, which was consistent with the findings of Jones et al. (27) who reported higher SCC during second and later lactations.

Table 2.2. Means, SD, and correlations of test day SCS and sire PTA_{SCS} for 1st, 2nd, and 3rd lactation records.

Variable	Lact. 1 ^a			Lact. 2 ^b			Lact. 3 ^c		
	Mean	SD	r ^d	Mean	SD	r ^d	Mean	SD	r ^d
PTA _{SCS}	3.24	.21	1.0	3.23	.21	1.0	3.23	.21	1.0
SCS 1	3.3	1.9	.11	3.0	1.9	.10	3.3	2.1	.10
SCS 2	2.8	1.9	.12	2.8	2.0	.11	3.2	2.2	.10
SCS 3	2.8	1.9	.13	2.9	2.0	.11	3.3	2.2	.11
SCS 4	2.9	1.9	.13	3.1	2.0	.10	3.4	2.1	.11
SCS 5	2.9	1.9	.12	3.2	1.9	.12	3.5	2.0	.11
SCS 6	3.0	1.8	.14	3.3	1.8	.11	3.7	2.0	.10
SCS 7	3.0	1.8	.14	3.4	1.8	.11	3.8	1.9	.11
SCS 8	3.1	1.8	.13	3.5	1.8	.12	3.9	1.8	.11
SCS 9	3.1	1.8	.13	3.6	1.7	.10	4.0	1.7	.10
SCS 10	3.1	1.8	.13	3.6	1.7	.11	4.0	1.7	.10

^a Number of records = 25269.

^b Number of records = 20043.

^c Number of records = 14114.

^d Correlation to PTA_{SCS}.

Correlations of test day SCS measurements and PTA_{SCS} ranged from .10 to .14. The highest correlations were seen between PTA_{SCS} and 1st lactation SCS test day measures. Highest correlations were evident in mid-lactation (test days 4 – 8). In general, the magnitude of the correlation decreased with lactation number. Correlation of individual test day SCS measures between lactations 1 and 2, 1 and 3, and 2 and 3 are in Appendix D. Correlations of test day SCS measures between lactations ranged from .10 to .35. In general, correlations of test day SCS measurements between lactations increased in magnitude with stage of lactation. Correlations were slightly higher between 1st and 2nd lactation than between 1st and 3rd lactations. The highest correlations between test day SCS measures, however, were evident between 2nd and 3rd lactations. Lack of unity in the

correlation of test day SCS between 1st and later parities support earlier findings (3, 17), as well as the indication that SCS in young and mature cows may be two different but correlated traits. Different genetic factors may be influencing SCS in 1st and later lactations (3).

Means, SD, and correlations of the averages of SCS test day measurements and sire PTA_{SCS}, by lactation, are in Table 2.3. As seen with the individual test day segments, little variation was seen in segment averages, but a gradual increase in average scores was evident with stage of lactation. The lowest segment averages were the averages of 1st and 2nd test days, and 3rd and 4th test days. The highest average scores were those averaged between test days 7 and 8, and test days 9 and 10. All correlations between segment averages and PTA_{SCS} were positive, and nearly identical to the correlations measured between PTA_{SCS} and individual test day scores. Highest correlations were evident between sire PTA_{SCS} and daughter SCS averages in 1st lactation. Generally, magnitude of the correlations decreased with lactation number.

Table 2.3 Means, SD, and correlations of SCS test day segment averages for 1st, 2nd, and 3rd lactations.

Variable	Lact 1			Lact 2			Lact 3		
	Mean	SD	r ^a	Mean	SD	r ^a	Mean	SD	r ^a
SCS Avg1 ¹	3.1	1.6	.13	3.0	1.8	.11	3.3	1.9	.11
SCS Avg2 ²	2.9	1.7	.15	3.1	1.8	.11	3.4	1.9	.11
SCS Avg3 ³	3.0	1.7	.14	3.3	1.7	.13	3.6	1.8	.10
SCS Avg4 ⁴	3.1	1.7	.15	3.5	1.6	.12	3.8	1.7	.11
SCS Avg5 ⁵	3.1	1.6	.14	3.6	1.5	.11	3.9	1.6	.11
Sire PTA _{SCS} ⁶	3.24	.21	1.0	3.23	.21	1.0	3.23	.21	1.0

¹ Avg. SCS of test days 1 & 2. Number of records in 1st lactation = 21898, 2nd lactation = 12709, 3rd lactation = 8312.

² Avg. SCS of test days 3 & 4. Number of records in 1st lactation = 20762, 2nd lactation = 12524, 3rd lactation = 7996.

³ Avg. SCS of test days 5 & 6. Number of records in 1st lactation = 19951, 2nd lactation = 12392, 3rd lactation = 7727.

⁴ Avg. SCS of test days 7 & 8. Number of records in 1st lactation = 18205, 2nd lactation = 11243, 3rd lactation = 6731.

⁵ Avg. SCS of test days 9 & 10. Number of records in 1st lactation = 6307, 2nd lactation = 3205, 3rd lactation = 1371.

^a Correlation to PTA_{SCS}.

Results of the regression of test day segment averages on PTA_{SCS} in 1st, 2nd, and 3rd lactations are in Table 2.4. Across all lactations, linear relationships between the average of test day SCS measurements and sire PTA_{SCS} were significant ($P<0.05$). Likewise, all regression coefficients were close to or equal to 1.0, which indicated a one unit increase in the predicted average of test day SCS measurements per unit increase in PTA_{SCS} . Results confirm the expected linear relationship between daughter SCS measurements, and sire PTA_{SCS} .

Table 2.4 Regression coefficients and SE of 1st, 2nd, and 3rd lactation test day segment averages on PTA_{SCS} .

Variable	Lact 1 ^a		Lact 2 ^a		Lact 3 ^a	
	β	SE	β	SE	β	SE
SCS Avg1 ¹	1.0	.06	.95	.08	1.0	.11
SCS Avg2 ²	1.2	.06	.92	.08	.98	.11
SCS Avg3 ³	1.1	.06	1.1	.08	.89	.11
SCS Avg4 ⁴	1.1	.06	.99	.08	.96	.11
SCS Avg5 ⁵	1.1	.11	.92	.15	.94	.25

¹ Avg. SCS of test days 1 & 2. Number of records in 1st lactation = 21898, 2nd lactation = 12709, 3rd lactation =8312.

² Avg. SCS of test days 3 & 4. Number of records in 1st lactation = 20762, 2nd lactation = 12524, 3rd lactation =7996.

³ Avg. SCS of test days 5 & 6. Number of records in 1st lactation = 19951, 2nd lactation = 12392, 3rd lactation =7727.

⁴ Avg. SCS of test days 7 & 8. Number of records in 1st lactation = 18205, 2nd lactation = 11243, 3rd lactation =6731.

⁵ Avg. SCS of test days 9 & 10. Number of records in 1st lactation = 6307, 2nd lactation = 3205, 3rd lactation=1371.

^a All coefficients significant ($P<0.05$).

Results of the investigation of possible quadratic and cubic effects of the average of test day SCS measurements on PTA_{SCS} in 1st, 2nd, and 3rd lactations (Appendix E) revealed significant quadratic relationships in lactation 2 between the average of test days 7 and 8 and PTA_{SCS} as well as between the average of test days 9 and 10 and PTA_{SCS} . In third lactation, significant quadratic relationships existed between the average of test day segments 5 and 6, the average of test days 7 and 8, and PTA_{SCS} . No significant quadratic

relationships existed in 1st lactation. No significant cubic relationships existed between the averages of test day SCS measurements and PTA_{SCS} in 1st, 2nd, or 3rd lactation.

Results of the regression of differences in SCS test day segment averages within a lactation on PTA_{SCS} are in Table 2.5. Significant differences were found between SCS test day segment averages for segments 1 and 2 and segments 9 and 10 in 1st lactation. Significance was also found in 1st lactation between the averages of segments 1 and 2 and segments 7 and 8. No significant differences were found between SCS test day segment averages in 2nd or 3rd lactations. Quadratic and cubic relationships were investigated for all lactations, but no significant effects were discovered.

Table 2.5. Regression coefficients and SE of differences in SCS test day segment averages within a lactation on PTA_{SCS}.

Comparison	Lact. 1			Lact. 2			Lact. 3		
	β	SE	P>F ^a	β	SE	P>F ^a	β	SE	P>F ^a
SCS Avg5 – SCS Avg1 ¹	.26	.13	*	.03	.20	NS	.50	.34	NS
SCS Avg4 – SCS Avg1 ²	.22	.07	*	.08	.10	NS	-.01	.13	NS
SCS Avg3 – SCS Avg1 ³	.10	.06	NS	.12	.09	NS	-.05	.12	NS

¹ Number of records 1st lact = 5663, 2nd lact = 2695, 3rd lact = 1179.

² Number of records 1st lact = 16578, 2nd lact = 9638, 3rd lact = 5925.

³ Number of records 1st lact = 18177, 2nd lact = 10748, 3rd lact = 6864.

^a Statistical significance is indicated with asterisk marks as follows: * (P < 0.05), NS = Not significant.

First lactation results indicate that sire PTA_{SCS} impacts the increase in SCS evident with increased stage of lactation. Elevated SCS at the beginning of a lactation has previously been explained by the higher incidence of new mastitis infections in the early phase of lactation (35). Cell counts tend to increase throughout lactation. Higher SCS towards the end of a lactation result because inflammatory responses become more severe and exaggerated in later stages of lactation due to prolonged exposure and reduced milk yield

(6). Other factors that cause increases in SCS are season, stress, and environmental temperatures (35). Results obtained in this analysis indicate sire PTA_{SCS} likewise impacts increases in SCS that occur with stage of lactation. Lack of significance in later parities could be a result of different genetic factors influencing SCS in 2nd and 3rd lactations or the build up of environmental effects over time .

Means, standard deviations, and correlation's of PTA_{SCS}, lactation average SCS, and regression coefficients of test day SCS on test day DIM (SCS lactation slope) are in Table 2.6. The coefficient average SCS lactation slope was 2.8×10^{-3} . The correlation of PTA_{SCS} and the SCS lactation slope was negative, but virtually zero, which implied that an increase in PTA_{SCS} had essentially no effect on the estimate of the relationship between test day SCS and test day DIM.

Table 2.6. Means , SD, range, and correlation of PTA_{SCS}, lactation average SCS, and regression coefficients of test day SCS on test day DIM.

Variable	Mean	SD	Min	Max	r ^a
β_{SCSDIM}	.0028	.009	-.13	.15	-.004
Lactation Avg. SCS	3.1	1.4	.10	9.0	.15
PTA _{SCS}	3.24	.21	2.7	4.0	1.0

¹ Regression of test day SCS on test day DIM. Number of observations = 50176.

² Lactation average SCS. Number of observations = 50201.

^a Correlation to PTA_{SCS}.

Across lactations, the mean lactation average SCS was 3.1. The correlation between PTA_{SCS} and lactation average SCS was .15, a slightly higher value than the correlation obtained when test day segments were considered individually or when test day segment measurements were averaged. Average PTA_{SCS} of sires of cows with greater than 6 test day segments per lactation was 3.24.

Means, standard deviations, and correlations of PTA_{SCS}, lactation average SCS, and the SCS lactation slope, by lactation, are in table 2.7. In general, estimates of the relationship between test day SCS and test day DIM increased in magnitude with lactation number. This increase in magnitude was expected given the established increase in SCS with stage of lactation and lactation number (22). The average SCS lactation slopes were 7.9×10^{-4} , 4.1×10^{-3} , 4.5×10^{-3} for 1st, 2nd, and 3rd lactation, respectively. The correlation of PTA_{SCS} and the SCS lactation slope was positive, but weak in 1st and 2nd lactations (.013 and , 2.6×10^{-4} , respectively), and negative in 3rd lactation (-.02).

Table 2.7. Means, SD, and correlations of sire PTA_{SCS} and measures of variation in SCS, by lactation.

Variable	Lact 1			Lact 2			Lact 3		
	Mean	SD	r ^a	Mean	SD	r ^a	Mean	SD	r ^a
β_{SCSDIM}	7.9×10^{-4}	.008	.013	4.1×10^{-3}	.008	2.6×10^{-4}	4.5×10^{-3}	.009	-.02
Lact. Avg. SCS	2.9	1.4	0.17	3.1	1.4	0.15	3.5	1.5	0.14
PTA _{SCS}	3.24	0.21	1.0	3.24	0.21	1.0	3.23	0.21	1.0

¹ Regression of test day SCS on test day DIM. Number of records 1st lact = 21804, 2nd lact = 16866, 3rd lact = 11506.

² Lactation average SCS. Number of records 1st lact = 21814, 2nd lact = 16878, 3rd lact = 11509.

^a Correlation to PTA_{SCS}.

Lactation average SCS also exhibited a gradual increase in magnitude with lactation number. First lactation average SCS was 2.9, while second lactation cows averaged an SCS of 3.1, and third lactation cows had a mean lactation average SCS of 3.5. Correlations between PTA_{SCS} and lactation average SCS decreased in magnitude from .17 in 1st lactation to .15 and .14 in 2nd and 3rd lactations, respectively. Average sire PTA_{SCS} fluctuated only slightly across lactations, going from 3.24 in 1st and 2nd lactations to 3.23 in 3rd lactation.

Correlations of lactation average SCS, by lactation, are in Table 2.8. Phenotypic correlations of lactation average SCS were strong and positive across lactations. Correlations were higher between 2nd and 3rd lactations than between 1st and later parities. The phenotypic correlation of 1st lactation average SCS and 2nd lactation average SCS was .40, which was lower than the genetic correlation, .70 to .80, reported by Banos and Shook (3). The correlation of 1st and 3rd lactation average SCS was .30. The correlation of 2nd lactation average SCS to 3rd lactation, however, was higher at .50. The higher correlation found between the later parities further supports the indication that SCS in 1st and later lactations are impacted by some different genes.

Table 2.8. Correlation of lactation average SCS measurements.

Variable	1 st LASCS	2 nd LASCS	3 rd LASCS
1 st LASCS ¹	1.0	0.40	0.30
2 nd LASCS ²	----	1.0	0.50
3 rd LASCS ³	----	----	1.0

¹ 1st lactation average SCS. Number of records = 21814.

² 1st lactation average SCS. Number of records = 16878.

³ 1st lactation average SCS. Number of records = 11509.

Results of the regression of lactation average SCS and the SCS lactation slope (regression of test day SCS on test day DIM) on PTA_{SCS} in 1st lactation are in Table 2.9. Linear relationships between PTA_{SCS} and 1st lactation average SCS and the SCS lactation slope were significant (P<0.05). The regression coefficient of lactation average SCS on sire PTA_{SCS} (1.0) reflected the expected linear relationship, and implied a one unit increase in lactation average SCS per unit increase in PTA_{SCS}. Likewise, the regression coefficient of the SCS lactation slope on sire PTA_{SCS} (5.9×10^{-4}) implied a small, but positive increase in the SCS lactation slope per unit increase in PTA_{SCS}. The relationship discovered between PTA_{SCS} and the SCS lactation slope implied that PTA_{SCS} not only impacts the change in

SCS from the beginning of a lactation to the end of a lactation, but also the changes in SCS which occur from test day to test day.

Table 2.9. Regression coefficients and SE of 1st lactation measures of SCS on PTA_{SCS}.

Variable	β	SE	R ²	P > F ^a
β_{SCSDIM}^1	5.9×10^{-4}	2.7×10^{-4}	.14982	*
Lact. Avg. SCS ²	1.0	.04	.21767	*

¹Regression of test day SCS on test day DIM. Number of records = 21801.

²Lactation average SCS. Number of records = 21811.

^aStatistical significance is indicated with asterisk marks as follows: * (P<0.05). NS=not significant.

Results of the analysis of possible quadratic relationships between PTA_{SCS} and 1st lactation average SCS and the SCS lactation slope are in Table 2.10. No significant quadratic relationships were found between PTA_{SCS} and the SCS lactation slope, implying only a significant linear relationships existed between the two traits. Significant quadratic relationships were found, however, between PTA_{SCS} and 1st lactation average SCS. Cubic relationships were likewise investigated, but no significant effects were found (Appendix F). Comparison of R-square values resulting from the investigation of linear effects between PTA_{SCS} and 1st lactation average SCS and the SCS lactation slope (.21767 and .14982, respectively) and the R-square values resulting from the investigation of possible linear and quadratic relationships (.21771 and .14984, respectively) revealed no additional variation was explained by the quadratic relationship.

Table 2.10. Linear and quadratic coefficients of 1st lactation measures of SCS on PTA_{SCS}.

Variable	Linear		Quadratic		R ²	P > F ^a
	β	SE	β	SE		
β_{SCSDIM}^1	4.4×10^{-3}	6.0×10^{-3}	-5.8×10^{-4}	9.1×10^{-4}	.14984	NS
Lact. Avg. SCS ²	2.0	.97	-.15	.15	.21771	*

¹Regression of test day SCS on test day DIM. Number of records = 21801.

²Lactation average SCS. Number of records = 21811.

^aStatistical significance is indicated with asterisk marks as follows: *, Linear effects (P < 0.05), quadratic effects P>0.05, **, Linear and quadratic effects P<0.05, NS, not significant.

Results of 2nd lactation analysis of the relationship between PTA_{SCS} and lactation average SCS and the SCS lactation slope are in Table 2.11. No significant relationship was found between the SCS lactation slope in 2nd lactation and PTA_{SCS}. A linear relationship did exist, however, between 2nd lactation average SCS and PTA_{SCS}. Per unit increase in PTA_{SCS}, 2nd lactation average SCS was predicted to increase by .93 units, a near perfect linear relationship. Lack of significance between PTA_{SCS} and the SCS lactation slope could be explained by the smaller number of 2nd lactation records, and the decreased correlation between PTA_{SCS} and test day SCS measures evident in 2nd lactation.

Table 2.11. Regression coefficients and SE of 2nd lactation measures of SCS on PTA_{SCS}.

Variable	β	SE	R ²	P > F ^a
β_{SCSDIM}^1	-1.8x10 ⁻⁴	3.4x10 ⁻⁴	.17693	NS
Lact. Avg. SCS ²	.93	.05	.27955	*

¹ Regression of test day SCS on test day DIM. Number of records = 16864.

² Lactation average SCS. Number of records = 16876.

^a Statistical significance is indicated with asterisk marks as follows: * (P<0.05). NS=not significant.

Results of the investigation of possible quadratic effects between 2nd lactation average SCS, SCS lactation slope, and PTA_{SCS} are in Table 2.12. A significant quadratic relationship was found between 2nd lactation average SCS and PTA_{SCS}. No quadratic relationship existed between PTA_{SCS} and the SCS lactation slope. Investigation of potential cubic relationships (Appendix F) revealed no significance for either trait on PTA_{SCS}. Comparison of R-square values from linear and linear and quadratic analyses revealed no additional variation explained by quadratic relationships.

Table 2.12. Linear and quadratic coefficients of 2nd lactation measures of SCS on PTA_{SCS}.

Variable	Linear		Quadratic		R ²	P > F ^a
	β	SE	β	SE		
β _{SCSDIM} ¹	.007	.008	-.001	.001	.17699	NS
Lact. Avg.	2.8	1.1	-.28	.20	.27968	*

¹Regression of test day SCS on test day DIM. Number of records = 16864.

²Lactation average SCS. Number of records = 16876.

^aStatistical significance is indicated with asterisk marks as follows: *, Linear effects (P < 0.05), quadratic effects P > 0.05, **, Linear and quadratic effects P < 0.05, NS, not significant.

Results of 3rd lactation analysis of the relationship between PTA_{SCS} and lactation average SCS and the SCS lactation slope are in Table 2.13. Results in 3rd lactation were similar to those evident in 2nd lactation. No significant relationship was found between the SCS lactation slope in 3rd lactation and PTA_{SCS}, but a linear relationship existed between 3rd lactation average SCS and PTA_{SCS}. Per unit increase in PTA_{SCS}, 3rd lactation average SCS was predicted to increase by .99 units. As in 2nd lactation, lack of significance between PTA_{SCS} and the SCS lactation slope could be explained by the even smaller number of 3rd lactation records, and the decreased correlation between PTA_{SCS} and test day SCS measures evident in 3rd lactation.

Table 2.13. Regression coefficients and SE of 3rd lactation measures of SCS on PTA_{SCS}.

Variable	β	SE	R ²	P > F ^a
β _{SCSDIM} ¹	-2.7x10 ⁻⁴	4.5x10 ⁻⁴	.21238	NS
Lact. Avg. SCS ²	.99	.07	.31588	*

¹Regression of test day SCS on test day DIM. Number of records = 11506.

²Lactation average SCS. Number of records = 11509.

^aStatistical significance is indicated with asterisk marks as follows: * (P < 0.05). NS=not significant.

Results of the investigation of possible quadratic effects between 3rd lactation average SCS, SCS lactation slope, and PTA_{SCS} are in Table 2.14. A significant quadratic relationship was found between PTA_{SCS} and the SCS lactation slope in 3rd lactation, but no quadratic

relationship existed between lactation average SCS and PTA_{SCS}. Investigation of potential cubic relationships (Appendix F) revealed no significance for either trait on PTA_{SCS} in 3rd lactation. Comparison of R-square values from linear and linear and quadratic analyses revealed no additional variation was explained by quadratic relationships, thus indicating any existing relationships were linear.

Table 2.14. Linear and quadratic coefficients of 3rd lactation measures of SCS on PTA_{SCS}.

Variable	Linear		Quadratic		R ²	P > F ^a
	β	SE	β	SE		
β _{SCSDIM} ¹	.025	.01	-.004	.002	.21292	**
Lact. Avg. SCS ²	3.8	1.6	-.42	.24	.31611	NS

¹Regression of test day SCS on test day DIM. Number of records = 11506.

²Lactation average SCS. Number of records = 11509.

^aStatistical significance is indicated with asterisk marks as follows: *, Linear effects (P < 0.05), quadratic effects P > 0.05, **, Linear and quadratic effects P < 0.05, NS, not significant.

Results of the analysis of the difference in lactation average SCS and the difference in the SCS lactation slopes between lactations are in Table 2.15. No significant linear relationships were found between the difference in the two traits and PTA_{SCS} between 1st and 2nd lactations, 1st and 3rd, or 2nd and 3rd lactations. Results indicate that PTA_{SCS} has a significant impact on changes in SCS within a lactation, but the relationship holds only within the lactation, and not between lactations. Lack of significance found between the SCS lactation slopes was most likely a result of the lack of significance found between the SCS lactation slopes and PTA_{SCS} in 2nd and 3rd lactations. Lack of significance between lactation average SCS measurements could be a result of the lower than expected correlations found between the lactation average SCS measurements. Potential quadratic

and cubic effects were investigated as well, but no significant relationships were discovered.

Table 2.15. Regression coefficients and SE of differences in measures of variation in SCS on PTA_{SCS} between lactations.

Variable	Lact 2-1		Lact 3-1		Lact 3-2		P>
	β	SE	β	SE	β	SE	
β_{SCSDIM}^1	8.6×10^{-4}	5.0×10^{-4}	1.1×10^{-3}	6.7×10^{-4}	1.8×10^{-5}	6.9×10^{-4}	NS
Lact. Avg. SCS ²	-.09	.07	-.03	.10	.04	.09	NS

¹Regression of test day SCS on test day DIM. Lact 2-1 No.records=13276. Lact 3-1 No.records=8130. Lact 3-2 No.records=7569.

²Lactation average SCS. Lact 2-1 No.records=13281. Lact 3-1 No.records=8131. Lact 3-2 No.records=7572.

Comparison of the coefficients resulting from the regression of various measures of SCS on PTA_{SCS} for 1st, 2nd, and 3rd lactation is in Table 2.16. Coefficients resulting from the regression of test day SCS record averages on PTA_{SCS} were positive, and close to or slightly greater than 1.0 for all parities. All coefficients supported the expected one unit increase in SCS per unit increase in PTA_{SCS}. Regression of the differences in averages of test day scores on PTA_{SCS} resulted in a mixture of positive and negative relationships. In 1st lactation, all relationships between differences in the averages of test day SCS and PTA_{SCS} were negative. In 2nd lactation, relationships were positive, excepting the relationship between PTA_{SCS} and the difference in the average of SCS from test days 7 and 8, and SCS from test days 1 and 2, which was negative. The reverse was seen in 3rd lactation, where once again all relationships between PTA_{SCS} and the differences in averages of test day SCS were negative, excepting the relationship between PTA_{SCS} and the difference in the average of SCS from test days 7 and 8, and SCS from test days 1 and 2, which was positive. All relationships were not significant (P>0.05), however, excepting the relationship evident between PTA_{SCS} and the differences between the average of 1st and

2nd test day SCS and both the averages of 7th and 8th and 9th and 10th test day SCS. Results indicate that PTA_{SCS} has a significant impact of changes in SCS from the beginning to the end of a lactation in 1st lactation, but that the relationship does not hold for later parities.

Table 2.16. Comparison of linear coefficients of measures of SCS on PTA_{SCS} in 1st (L1), 2nd (L2), and 3rd (L3) lactations.

Variable	$\beta_{L1.SCS}$	$\beta_{L2.SCS}$	$\beta_{L3.SCS}$
SCS Avg1 ¹	1.0	.95	1.0
SCS Avg2 ²	1.2	.92	.98
SCS Avg3 ³	1.1	1.1	.89
SCS Avg4 ⁴	1.1	.99	.96
SCS Avg5 ⁵	1.1	.92	.94
SCS Avg5 – SCS Avg1 ⁶	-.26	.09 ^a	-.51 ^a
SCS Avg4 – SCS Avg1 ⁷	-.23	-.01 ^a	.05 ^a
SCS Avg3 – SCS Avg1 ⁸	-.10 ^a	.014 ^a	-.003 ^a
β_{SCSDIM} ⁹	5.9x10 ⁻⁴	-1.8x10 ^{-4a}	-2.7x10 ^{-4a}
Lact. Avg. SCS ¹⁰	1.0	.93	.99

¹ Avg. SCS of test days 1 & 2. Number of records in 1st lactation = 21898, 2nd lactation = 12709, 3rd lactation = 8312.

² Avg. SCS of test days 3 & 4. Number of records in 1st lactation = 20762, 2nd lactation = 12524, 3rd lactation = 7996.

³ Avg. SCS of test days 5 & 6. Number of records in 1st lactation = 19951, 2nd lactation = 12392, 3rd lactation = 7727.

⁴ Avg. SCS of test days 7 & 8. Number of records in 1st lactation = 18205, 2nd lactation = 11243, 3rd lactation = 6731.

⁵ Avg. SCS of test days 9 & 10. Number of records in 1st lactation = 6307, 2nd lactation = 3205, 3rd lactation = 1371.

⁶ Number of records 1st lact = 5663, 2nd lact = 3084, 3rd lact = 1465.

⁷ Number of records 1st lact = 16992, 2nd lact = 12407, 3rd lact = 8641.

⁸ Number of records 1st lact = 19180, 2nd lact = 14565, 3rd lact = 10592.

⁹ The regression of test day SCS on test day DIM. Number of records 1st lact = 21801, 2nd lact = 16864, 3rd lact = 11506.

¹⁰ Lactation average SCS. Number of records 1st lact = 21811, 2nd lact = 16876, 3rd lact = 11509

^a Variable not significant (P>0.05).

The coefficients resulting from the investigation of possible relationships between PTA_{SCS} and the SCS lactation slope (regression of test day SCS on test day DIM) revealed a significant relationship in 1st lactation only. Relationships between SCS lactation slope and PTA_{SCS} in 2nd and 3rd lactations were negative and not significant (P>0.05). Results indicate that PTA_{SCS} has a significant impact on changes in test day SCS with DIM in 1st lactation, but that the relationship did not hold in 2nd or 3rd lactations. The regression of lactation average SCS on PTA_{SCS} was positive, significant, and close to or equal to 1.0 for all parities. Thus, the expected one unit increase in lactation average SCS per unit increase in PTA_{SCS} was found to hold for all lactations.

Conclusions

Positive correlations were found between PTA_{SCS} and individual SCS test day measures, averages of test day SCS records, and lactation average SCS in 1st, 2nd, and 3rd lactations. Correlations between test day SCS measures and lactation average SCS were greater between 2nd and 3rd lactation than between 1st lactation and later parities. Significant relationships were found in 1st, 2nd, and 3rd lactations between PTA_{SCS} and averages of test day SCS measures and lactation average SCS. Likewise, in 1st lactation, significant relationships were found between PTA_{SCS} and changes in SCS measures from the beginning of a lactation to the end of a lactation, and the changes in test day SCS with test day DIM (SCS lactation slope). The relationships between PTA_{SCS} and variation in SCS measurements within a lactation were not evident in later lactations.

Cows differ innately in their normal SCS measurements. Several factors have been identified as potential sources of variation in SCS from cow to cow. The first of these factors is the positive genetic correlation found between milk yield and SCC, which characterizes daughters of high yield sires as also having higher SCS (11, 18, 43). Another factor is the presence or absence of mastitis infection, pathogen species, number of quarters infected, and sampling time (7, 33). Age and stage of lactation have likewise been identified as sources of variation in SCS measurements (54). Results of this study likewise indicate that PTA_{SCS} affects the variation in SCS seen from beginning to end of a lactation, and from test day to test day. The relationship between variation in test day SCS measures and PTA_{SCS} indicates a genetic influence over variation in SCS. Thus it is logical to conclude that selection on PTA_{SCS} could decrease variation in SCS. Since variation (or

elevation) in SCS is most often the response to infection, selection for lower SCS should result in reduced incidence of mastitis.

The relationship between changes in SCS and PTA_{SCS} was evident only in 1st lactation. Likewise, higher correlations between SCS measurements and PTA_{SCS} were found in 1st lactation. In contrast, higher correlations between individual test day SCS records and lactation average SCS were found between 2nd and 3rd lactations. The results indicate that SCS in 1st and later lactations are two different, but correlated, traits. The lower correlations found between PTA_{SCS} and lactation average SCS in 2nd and 3rd lactations could indicate decreased genetic influence and increased environmental effects on incidence of mastitis in the later parities. As a result, inclusion of PTA_{SCS} in a breeding scheme should produce daughters with reduced incidence of mastitis and lower SCS, but, selection in 1st lactation may not be effective in reducing SCS in later lactations. Nonetheless, records from later lactations may be better indicators of mastitis resistance, because despite lower correlations between 2nd and 3rd lactation SCS measurements and PTA_{SCS} , higher correlations were found between 2nd and 3rd lactation SCS. The higher correlations were most likely due to higher frequency of mastitis infection.

CONCLUSION

Due to the continuing genetic increase in incidence of mastitis, much effort has been focused on developing and implementing new methods of improving resistance to mastitis. Lack of a direct means of selecting for lower incidence of mastitis, and theories of enhancing an animal's innate defense mechanisms led researchers to investigate the relationship between SCS and mastitis. Measurement of SCS was easy and cost effective, and correlations between measures of mastitis and SCS were both high and positive, indicating that selection for lower SCS could be an effective means of reducing incidence of mastitis. Selection for lower SCS was made possible through the development of sire genetic evaluations for SCS which were released in 1994. Most of the research regarding the relationship between SCS and mastitis, however, was done prior to the calculation of PTA_{SCS} . The role of this study was to determine the impact of PTA_{SCS} on measures of mastitis and SCS, and to support or negate the inclusion of PTA_{SCS} in a breeding program as a means to reduce incidence of mastitis and increase overall profitability.

Results from data sets I, II, and III indicate that a relationship exists between PTA_{SCS} and measures of mastitis, variation in SCS, and measures of herd life. Positive correlations existed between PTA_{SCS} , measures of mastitis, test day SCS, and lactation average SCS. The correlation of PTA_{SCS} and mastitis (.41) was higher than the correlation of PTA_{SCS} and test day SCS (average correlation approximately .11). Significant linear relationships were found between PTA_{SCS} and number of cases of mastitis, number of days treated, number of treatments for mastitis, and lactation average SCS. PTA_{SCS} was also found to impact the

changes in test day SCS from the beginning to the end of a lactation, as well as changes in test day SCS with DIM (data set III). Increases in PTA_{SCS} also resulted in higher rates of involuntary culling, and decreased herd life. Losses in number of lactations, DPL, TDIMM, lifetime milk, fat, and protein, and relative net income variables that were attributable to increases in PTA_{SCS} were also explained by variation in PTA_{PL} . The relationship between PTA_{SCS} and decreased herd life was consistent across multiple lactations.

The relationships evident between PTA_{SCS} and measures of mastitis and SCS, excepting lactation average SCS, however, were significant only in 1st lactation. Results imply a genetic influence over variation in SCS as well as incidence of mastitis. Selection on PTA_{SCS} should be an effective means of reducing the variation in SCS, lowering lactation average SCS, and reducing incidence of mastitis. The impact of selection, however, may differ in 1st and later parities.

Several factors logically contributed to the lack of significance between PTA_{SCS} and incidence of mastitis, and variation in test day SCS records evident in 2nd and 3rd lactation analyses. Inadequate number of records in the Va. Tech study likely affected overall implications of the study, and diminished the significance of the relationship between PTA_{SCS} and measures of clinical mastitis in the later parities. Likewise, the apparent heavy cull rate imposed on 1st lactation cows was higher than state average, and resulted in a population of highly selected animals. Nonetheless, the effects of PTA_{SCS} on incidence of mastitis as measured by test day SCS measures in data set III resulted in the same lack of

significance in 2nd and 3rd lactations. In data set III, number of records was not a factor, and acceptable cull rates existed from 1st to later lactations. In addition, correlations of test day SCS between first and later parities was less than unity. Correlations between 2nd and 3rd lactation SCS records were higher than correlations between SCS in 1st lactation and the later lactations. Likewise, the correlation between lactation average SCS was higher between 2nd and 3rd lactation than between 1st and later parities. Interesting to note, however, were the higher correlations evident between PTA_{SCS} and measures of SCS in 1st lactation. The disparity between 1st and later parity SCS measures support earlier findings (3, 17), and suggest that perhaps SCS in young and mature cows are two different traits. Selection on PTA_{SCS} should be effective in reducing incidence of mastitis, decreasing changes in SCS, and reducing lactation average SCS. The response in 1st lactation cows, however, may be different than the response in later parities.

Absence of PTA_{SCS} in a breeding program, however, will result in the continued rise in incidence of clinical mastitis and in the rate of premature culling. Selection to reduce SCS is thus economically justified. The most negative impact of increased PTA_{SCS} on profitability of dairy cattle was determined to be the relationship between PTA_{SCS} and decreased herd life. Losses in measures of lifetime performance and profitability explained by PTA_{SCS} were likewise explained by PTA_{PL}. The implication was that PTA_{SCS} had a more profound than expected impact on the ability to survive involuntary culling, and thus increases in PTA_{SCS} often resulted in shorter herd life, and reduced opportunity for yield.

While the results of this research are positive, and support the inclusion of PTA_{SCS} in a breeding program, the research was limited to a classical quantitative genetics approach to the selection for reduction in incidence of mastitis. Molecular genetic techniques are emerging as a possible means of increasing disease resistance. Bovine lymphocyte antigen (BoLa) types may provide the opportunity to identify animals with superior genotypes for mastitis resistance (45). Likewise, a more advanced understanding of the role of the MHC and MHC polymorphism in resistance and susceptibility to disease may lead to the production of transgenic animals with resistance to specific diseases such as mastitis, or the production of more effective vaccines against mastitis. Disease resistance is a complex mechanism involving a large number of genes. Identification of major genes affecting disease resistance through the application of molecular techniques should both complement and enhance selection on SCS to reduce incidence of mastitis in the future.

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APPENDIX A. Graphic illustration of linear, quadratic, and cubic effects of measure of daughter performance on PTA_{SCS}

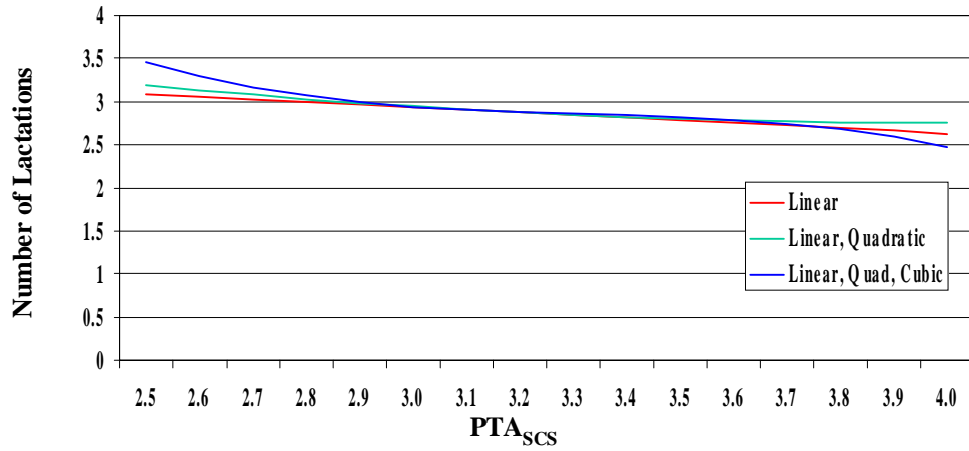


Figure 2. Linear, Quadratic, and Cubic Effects of Number of Lactations on PTA_{SCS} .

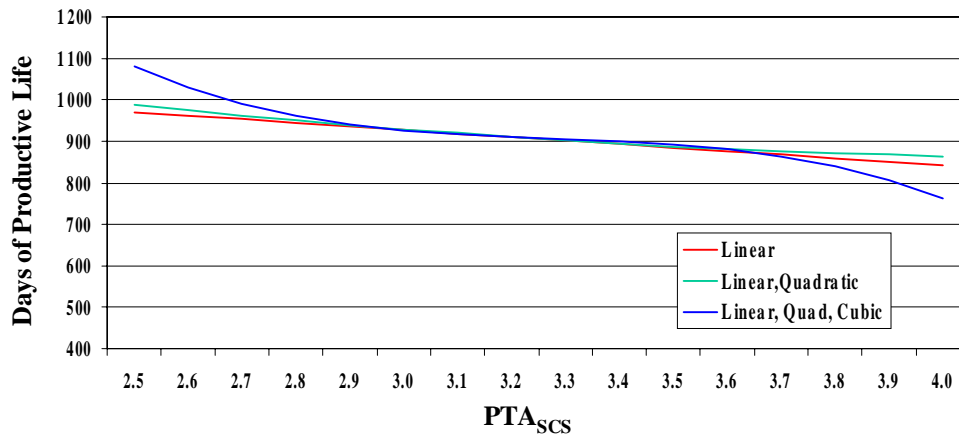


Figure 3. Linear, Quadratic, and Cubic Effects of DPL on PTA_{SCS}

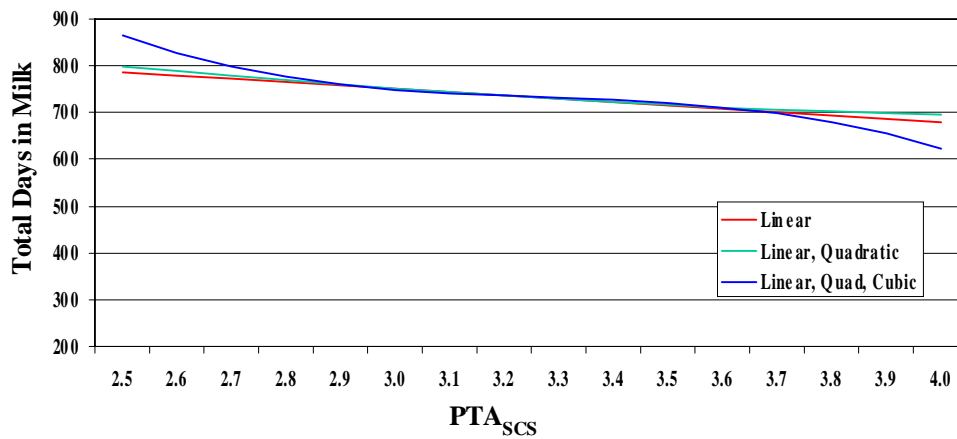


Figure 4. Linear, Quadratic, and Cubic Effects of TDIMM on PTA_{SCS}

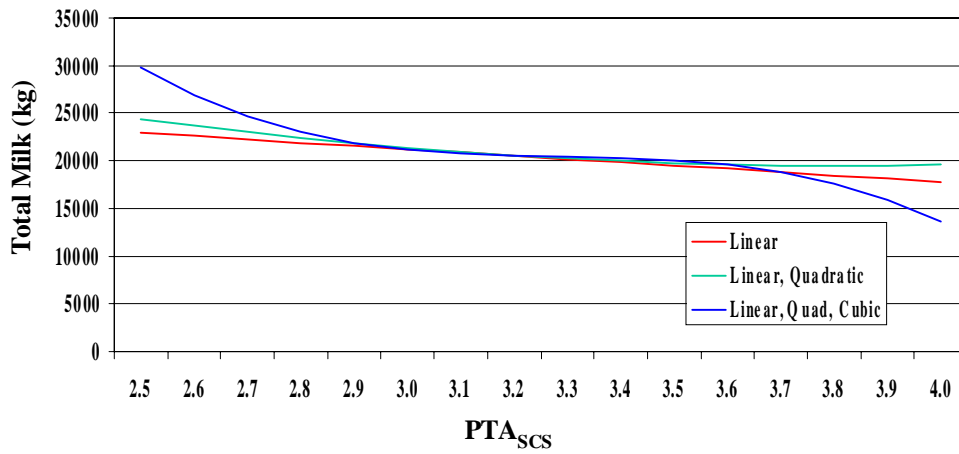


Figure 5. Linear, Quadratic, and Cubic Effects of Total Milk on PTA_{SCS}

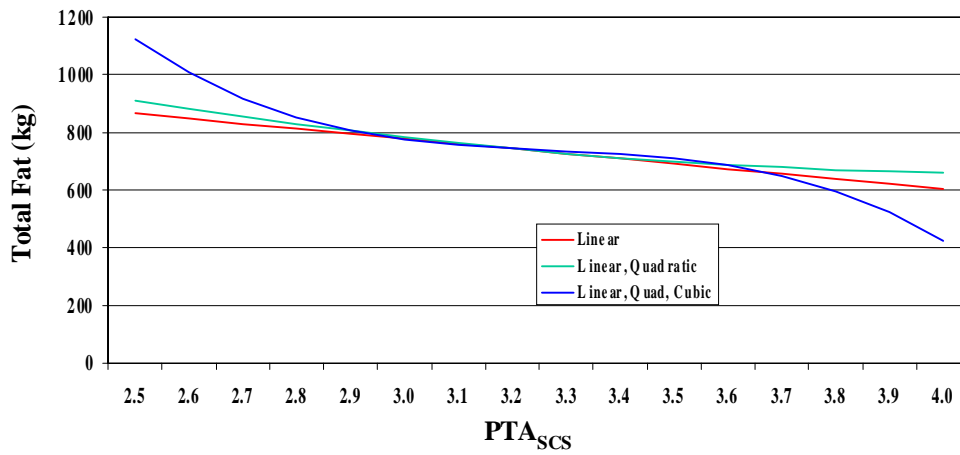


Figure 6. Linear, Quadratic, and Cubic Effects of Total Fat on PTA_{SCS}

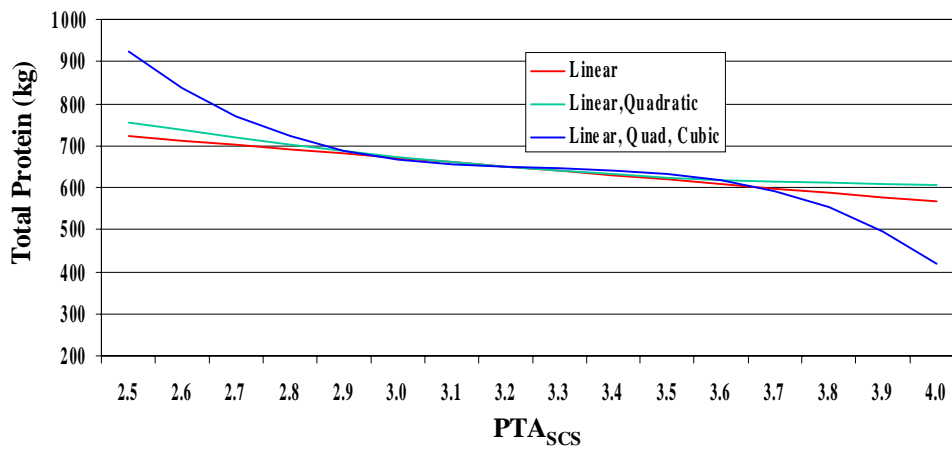


Figure 7. Linear, Quadratic, and Cubic Effects of Total Protein on PTA_{SCS}

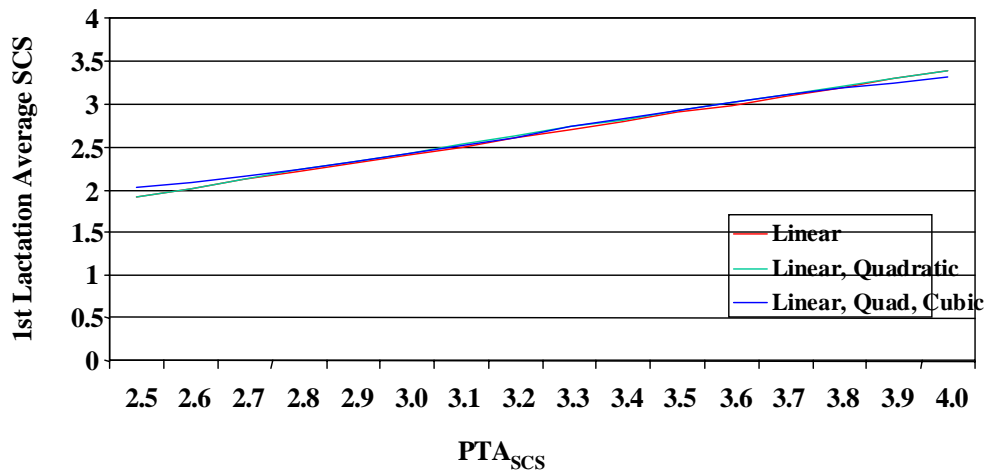


Figure 8. Linear, Quadratic, and Cubic Effects of 1st Lactation Average SCS on PTA_{SCS}

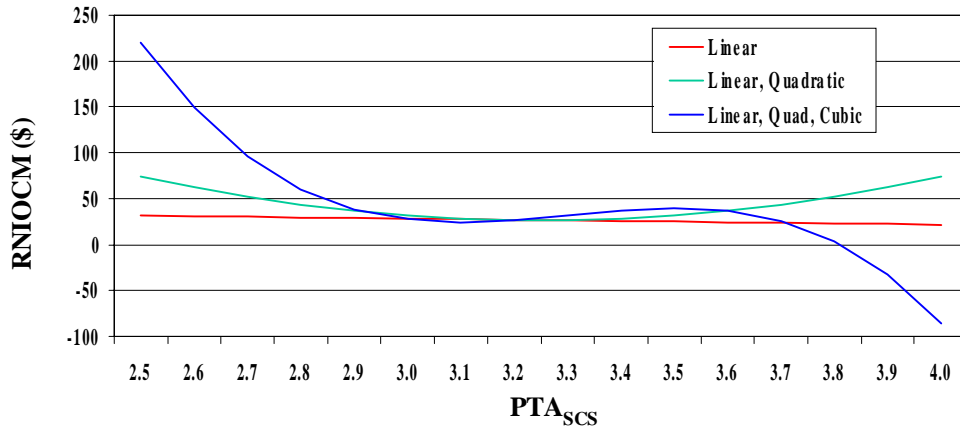


Figure 9. Linear, Quadratic, and Cubic Effects of RNIOCM on PTA_{SCS}

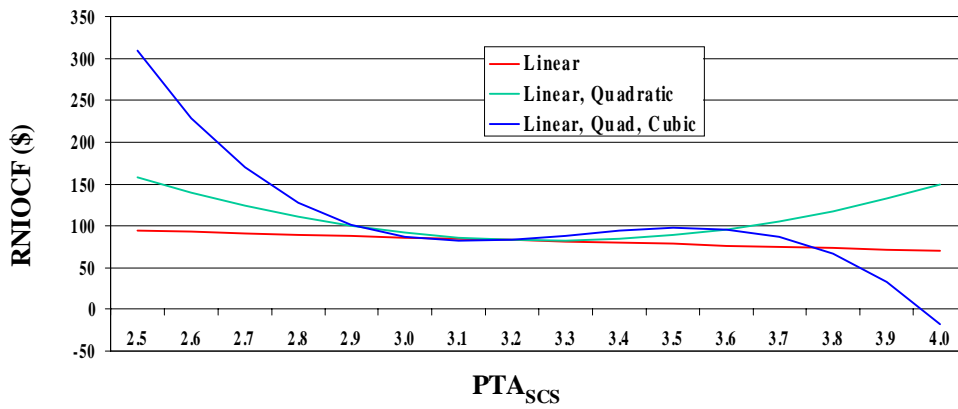


Figure 10. Linear, Quadratic, and Cubic Effects of RNIOCF on PTA_{SCS}

APPENDIX B. Means and standard deviations of test day segment averages for all lactations.

Table 2.17. Means, SD , and ranges of test day segment data for all lactations.

Variable	N	Mean	SD	Min	Max
No. Lacs (#)	59426	1.8	0.80	1.0	3.0
No. Segment (#)	59426	8.0	2.5	0	20.0
DIM (days)	59426	248.5	74.8	7.0	305.0
Dimtd1 (days)	59426	24.2	11.2	5.0	60.0
Dimtd2 (days)	57403	55.6	12.6	8.0	120.0
Dimtd3 (days)	55603	88.1	15.6	37.0	299.0
Dimtd4 (days)	53836	120.2	17.9	38.0	304.0
Dimtd5 (days)	52167	152.4	20.2	65.0	305.0
Dimtd6 (days)	50427	184.5	22.0	66.0	305.0
Dimtd7 (days)	48217	215.6	22.1	97.0	305.0
Dimtd8 (days)	44338	244.3	19.2	98.0	305.0
Dimtd9 (days)	36353	270.0	13.9	127.0	305.0
Dimtd10 (days)	19390	291.6	14.1	2.0	305.0
SCS1	55547	3.2	2.0	.10	9.6
SCS2	52806	2.9	2.0	.10	9.6
SCS3	51905	3.0	2.0	.10	9.8
SCS4	50938	3.1	2.0	.10	9.7
SCS5	49858	3.2	1.9	.10	9.6
SCS6	48407	3.3	1.9	.10	9.8
SCS7	46560	3.3	1.9	.10	9.6
SCS8	42925	3.4	1.8	.10	9.6
SCS9	35318	3.5	1.8	.10	9.6
SCS10	12148	3.4	1.8	0	9.6
Milk1 (kg)	59426	30.6	8.8	2.3	82.9
Milk2 (kg)	57403	33.1	8.5	1.8	71.7
Milk3 (kg)	55603	32.1	7.9	.91	71.7
Milk4 (kg)	53836	30.8	7.5	.91	75.4
Milk5 (kg)	52167	29.3	7.0	.70	67.6
Milk6 (kg)	50427	27.7	6.7	.60	69.1
Milk7 (kg)	48217	26.2	6.5	.95	86.2
Milk8 (kg)	44338	24.6	6.4	.91	58.2
Milk9 (kg)	36353	23.3	6.2	.91	56.5
Milk10 (kg)	19190	21.4	7.0	.27	50.8
PTA _{SCS}	59419	3.24	.21	2.7	4.0
PTA _{Milk} (kg)	59419	239.1	281.0	-1334.4	1465.1

APPENDIX C. Means and standard deviations of test day segment averages by lactation.

Table 2.18. Means, ranges, and SD of test day segment data for 1st lactation cows.

Variable	N	Mean	SD	Min	Max
No. Lacs (#)	25269	1.0	0	1.0	1.0
No. Segment (#)	25269	8.2	2.4	1.0	18.0
DIM (days)	25269	251.6	73.9	7.0	305.0
Dimtd1 (days)	25269	24.2	11.0	5.0	60.0
Dimtd2 (days)	24423	55.4	12.1	8.0	118.0
Dimtd3 (days)	23661	87.2	14.2	37.0	294.0
Dimtd4 (days)	22995	119.0	16.0	38.0	286.0
Dimtd5 (days)	22393	150.9	18.0	65.0	305.0
Dimtd6 (days)	21820	182.7	19.7	66.0	304.0
Dimtd7 (days)	21133	213.8	20.3	97.0	304.0
Dimtd8 (days)	19849	243.2	18.1	98.0	305.0
Dimtd9 (days)	16797	269.6	13.4	127.0	305.0
Dimtd10 (days)	9256	292.3	8.8	30.0	305.0
SCS1	23788	3.3	1.9	.10	9.6
SCS2	22814	2.8	1.9	.10	9.6
SCS3	22194	2.8	1.9	.10	9.8
SCS4	21737	2.9	1.9	.10	9.7
SCS5	21226	2.9	1.9	.10	9.6
SCS6	20700	3.0	1.8	.10	9.8
SCS7	20078	3.0	1.8	.10	9.6
SCS8	18884	3.1	1.8	.10	9.6
SCS9	16074	3.1	1.8	.10	9.6
SCS10	6573	3.0	1.8	0	9.6
Milk1 (kg)	25269	25.2	6.0	2.7	53.1
Milk2 (kg)	24426	27.9	5.9	2.0	54.6
Milk3 (kg)	23661	27.9	5.8	.91	70.8
Milk4 (kg)	22995	27.4	5.7	4.1	62.6
Milk5 (kg)	22393	26.6	5.6	.70	53.1
Milk6 (kg)	21820	25.8	5.6	.60	58.1
Milk7 (kg)	21133	25.0	5.6	2.0	49.4
Milk8 (kg)	19849	24.0	5.5	.91	51.8
Milk9 (kg)	16797	23.1	5.4	2.3	48.5
Milk10 (kg)	9252	21.7	6.3	.45	50.6
PTA _{SCS}	25266	3.24	.21	2.7	4.0
PTA _{Milk} (kg)	25266	239.9	282.6	-1334.4	1465.1

Table 2.19. Means, ranges, and SD of test day segment data for 2nd lactation cows.

Variable	N	Mean	SD	Min	Max
No. Lacs (#)	20043	2.0	0	2.0	2.0
No. Segment (#)	20043	8.0	2.4	1.0	18.0
DIM (days)	20043	249.3	72.6	7.0	305.0
Dimtd1 (days)	20043	23.9	11.1	7.0	60.0
Dimtd2 (days)	19463	55.5	12.6	24.0	120.0
Dimtd3 (days)	18918	88.1	15.8	54.0	290.0
Dimtd4 (days)	18323	120.5	18.5	69.0	302.0
Dimtd5 (days)	17722	152.8	21.1	91.0	303.0
Dimtd6 (days)	17081	185.0	22.9	98.0	304.0
Dimtd7 (days)	16205	215.9	22.6	120.0	305.0
Dimtd8 (days)	14769	244.4	19.4	132.0	305.0
Dimtd9 (days)	12023	270.0	14.2	147.0	305.0
Dimtd10 (days)	6384	291.5	13.8	2.0	305.0
SCS1	18494	3.0	2.0	.10	9.6
SCS2	17552	2.8	2.0	.10	9.6
SCS3	17450	2.9	2.0	.10	9.6
SCS4	17216	3.1	2.0	.10	9.6
SCS5	16913	3.2	1.9	.10	9.6
SCS6	16440	3.3	1.8	.10	9.6
SCS7	15774	3.4	1.8	.10	9.6
SCS8	14434	3.5	1.8	.10	9.6
SCS9	11782	3.6	1.7	.10	9.6
SCS10	3823	3.6	1.7	0	9.6
Milk1 (kg)	20043	33.8	7.9	3.6	82.9
Milk2 (kg)	19463	35.9	7.7	1.8	70.6
Milk3 (kg)	18918	34.3	7.4	1.6	66.2
Milk4 (kg)	18323	32.4	7.2	.91	75.4
Milk5 (kg)	17722	30.4	6.9	.91	61.3
Milk6 (kg)	17081	28.6	6.8	2.0	69.1
Milk7 (kg)	16205	26.7	6.8	.95	62.5
Milk8 (kg)	14769	24.9	6.7	.91	57.6
Milk9 (kg)	12023	23.2	6.6	.91	56.4
Milk10 (kg)	6313	21.0	7.4	.45	48.5
PTA _{SCS}	20041	3.24	.21	2.7	4.0
PTA _{Milk} (kg)	20041	241.3	280.1	-1276.3	1465.1

Table 2.20. Means, ranges, and SD of test day segment data for 3rd lactation cows.

Variable	N	Mean	SD	Min	Max
No. Lacs (#)	14114	3.0	0	3.0	3.0
No. Segment (#)	14114	7.7	2.6	1.0	20.0
DIM (days)	14114	241.9	79.2	7.0	305.0
Dimtd1 (days)	14114	24.5	11.5	6.0	60.0
Dimtd2 (days)	13517	56.4	13.3	15.0	120.0
Dimtd3 (days)	13024	89.6	17.7	43.0	299.0
Dimtd4 (days)	12518	122.1	20.0	44.0	304.0
Dimtd5 (days)	12052	154.7	22.6	72.0	302.0
Dimtd6 (days)	11526	187.3	24.5	73.0	305.0
Dimtd7 (days)	10879	218.5	24.2	106.0	305.0
Dimtd8 (days)	9720	246.5	20.6	107.0	305.0
Dimtd9 (days)	7533	270.9	14.6	135.0	305.0
Dimtd10 (days)	3750	290.1	22.6	2.0	305.0
SCS1	13265	3.3	2.1	.10	9.6
SCS2	12440	3.2	2.2	.10	9.6
SCS3	12261	3.3	2.2	.10	9.6
SCS4	11985	3.4	2.1	.10	9.6
SCS5	11719	3.5	2.0	.10	9.6
SCS6	11267	3.7	2.0	.10	9.6
SCS7	10708	3.8	1.9	.10	9.6
SCS8	9607	3.9	1.8	.10	9.6
SCS9	7462	4.0	1.7	.10	9.6
SCS10	1752	4.0	1.7	0	9.6
Milk1 (kg)	14114	35.7	8.8	2.3	74.8
Milk2 (kg)	13517	38.3	8.5	1.8	71.7
Milk3 (kg)	13024	36.7	8.2	2.3	71.7
Milk4 (kg)	12518	34.5	7.9	2.0	71.0
Milk5 (kg)	12052	32.4	7.8	.70	67.6
Milk6 (kg)	11526	30.0	7.6	2.1	64.7
Milk7 (kg)	10879	27.8	7.4	2.5	86.2
Milk8 (kg)	9720	25.6	7.3	1.8	58.2
Milk9 (kg)	7533	23.8	7.2	.91	56.5
Milk10 (kg)	3625	21.3	8.1	.30	50.8
PTA _{SCS}	14112	3.23	.21	2.7	4.0
PTA _{Milk} (kg)	14112	234.3	279.3	-1276.3	1465.1

APPENDIX D. Correlations of test day SCS between lactations.

Table 2.21. Correlation of test day SCS measurements between lactations 1 and 2.

	SCS1 ²	SCS2 ²	SCS3 ²	SCS4 ²	SCS5 ²	SCS6 ²	SCS7 ²	SCS8 ²	SCS9 ²	SCS10 ²
SCS1 ¹	.15	.14	.13	.13	.14	.14	.13	.13	.12	.13
SCS2 ¹	.17	.18	.17	.17	.16	.16	.15	.16	.14	.10
SCS3 ¹	.18	.19	.20	.19	.18	.18	.17	.17	.16	.14
SCS4 ¹	.19	.19	.20	.21	.20	.20	.19	.18	.17	.16
SCS5 ¹	.19	.21	.21	.22	.22	.21	.20	.19	.19	.16
SCS6 ¹	.20	.21	.23	.23	.23	.23	.22	.21	.20	.18
SCS7 ¹	.20	.21	.23	.23	.23	.24	.22	.21	.21	.18
SCS8 ¹	.22	.23	.24	.23	.24	.25	.23	.23	.22	.21
SCS9 ¹	.21	.24	.24	.24	.24	.26	.23	.24	.23	.21
SCS10 ¹	.20	.22	.23	.21	.21	.24	.24	.22	.21	.16

¹ Lactation 1 test day SCS measurements.

² Lactation 2 test day SCS measurements.

Table 2.22. Correlation of test day SCS measurements between lactations 1 and 3.

	SCS1 ²	SCS2 ²	SCS3 ²	SCS4 ²	SCS5 ²	SCS6 ²	SCS7 ²	SCS8 ²	SCS9 ²	SCS10 ²
SCS1 ¹	.12	.12	.12	.11	.12	.11	.11	.11	.10	.13
SCS2 ¹	.14	.16	.16	.15	.14	.15	.14	.14	.14	.11
SCS3 ¹	.14	.16	.16	.15	.15	.15	.14	.14	.14	.12
SCS4 ¹	.14	.17	.16	.16	.16	.15	.15	.15	.15	.16
SCS5 ¹	.15	.17	.17	.17	.16	.16	.16	.16	.17	.17
SCS6 ¹	.15	.17	.17	.18	.17	.17	.16	.17	.16	.17
SCS7 ¹	.16	.19	.18	.18	.18	.18	.17	.17	.18	.18
SCS8 ¹	.16	.19	.19	.19	.19	.19	.19	.19	.20	.22
SCS9 ¹	.17	.19	.20	.20	.19	.19	.19	.16	.19	.19
SCS10 ¹	.17	.17	.18	.21	.19	.19	.20	.19	.19	.21

¹ Lactation 1 test day SCS measurements.

² Lactation 3 test day SCS measurements.

Table 2.23. Correlation of test day SCS measurements between lactations 2 and 3.

	SCS1 ²	SCS2 ²	SCS3 ²	SCS4 ²	SCS5 ²	SCS6 ²	SCS7 ²	SCS8 ²	SCS9 ²	SCS10 ²
SCS1 ¹	.23	.23	.23	.20	.21	.20	.18	.19	.19	.19
SCS2 ¹	.26	.24	.27	.25	.26	.24	.23	.22	.21	.23
SCS3 ¹	.26	.27	.29	.28	.29	.26	.25	.25	.25	.28
SCS4 ¹	.24	.26	.27	.29	.29	.27	.26	.26	.26	.26
SCS5 ¹	.24	.27	.28	.30	.32	.29	.28	.28	.28	.28
SCS6 ¹	.24	.26	.29	.30	.32	.31	.31	.31	.31	.29
SCS7 ¹	.24	.26	.29	.30	.32	.31	.31	.32	.31	.29
SCS8 ¹	.23	.26	.28	.30	.32	.33	.33	.34	.34	.33
SCS9 ¹	.23	.25	.26	.28	.30	.30	.31	.33	.32	.32
SCS10 ¹	.26	.25	.26	.28	.29	.30	.32	.31	.34	.35

¹ Lactation 2 test day SCS measurements.

² Lactation 3 test day SCS measurements.

APPENDIX E. Linear, quadratic, and cubic regressions of 1st, 2nd, and 3rd lactation test day SCS segment averages on PTA_{SCS}.

Table 2.24. Linear and quadratic regression coefficients and standard errors of 1st lactation test day segment averages on PTA_{SCS}.

Variable	Linear		Quadratic		P > F ^a
	β	SE	β	SE	
SCS Avg1 ¹	0.98	1.0	-0.11	0.16	NS
SCS Avg2 ²	1.25	1.2	-0.02	0.18	NS
SCS Avg3 ³	2.8	1.2	-0.30	0.18	*
SCS Avg4 ⁴	3.0	1.3	-0.30	0.19	*
SCS Avg5 ⁵	-1.1	2.4	0.33	0.36	NS

¹ Avg. SCS of test days 1 & 2. Number of records = 29434

² Avg. SCS of test days 3 & 4. Number of records = 22628

³ Avg. SCS of test days 5 & 6. Number of records = 21051

⁴ Avg. SCS of test days 7 & 8. Number of records = 18661

⁵ Avg. SCS of test days 9 & 10. Number of records = 6307

^a Statistical significance is indicated with asterisk marks as follows: *, Linear effects (P < 0.05), quadratic effects P > 0.05, **, Linear and quadratic effects P < 0.05, NS, not significant.

Table 2.25. Linear, quadratic, and cubic regressions and standards errors of 1st lactation SCS test day segment averages on PTA_{SCS}.

Variable	Linear		Quadratic		Cubic		P > F ^a
	β	SE	β	SE	β	SE	
SCS Avg1 ¹	-2.2	16.2	0.93	4.8	-0.10	0.50	NS
SCS Avg2 ²	-3.0	18.2	1.3	5.5	-0.13	0.55	NS
SCS Avg3 ³	-5.0	18.9	2.1	5.6	-0.23	0.56	NS
SCS Avg4 ⁴	-10.9	19.9	3.9	6.0	-0.41	0.60	NS
SCS Avg5 ⁵	22.8	39.2	-6.8	11.7	0.71	1.2	NS

¹ Avg. SCS of test days 1 & 2. Number of records = 29434

² Avg. SCS of test days 3 & 4. Number of records = 22628

³ Avg. SCS of test days 5 & 6. Number of records = 21051

⁴ Avg. SCS of test days 7 & 8. Number of records = 18661

⁵ Avg. SCS of test days 9 & 10. Number of records = 6307

^a Statistical significance is indicated with asterisk marks as follows: *, Linear effects (P < 0.05), quadratic effects P > 0.05, **, Linear and quadratic effects P < 0.05, NS, not significant.

Table 2.26. Linear and quadratic regression coefficients and standard errors of 2nd lactation test day segment averages on PTA_{SCS}.

Variable	Linear		Quadratic		P > F ^a
	β	SE	β	SE	
SCS Avg1 ¹	2.4	1.4	-0.21	0.21	NS
SCS Avg2 ²	2.4	1.5	-0.22	0.22	NS
SCS Avg3 ³	1.7	1.4	-0.11	0.22	NS
SCS Avg4 ⁴	4.3	1.5	-0.51	0.22	**
SCS Avg5 ⁵	7.3	3.1	-0.98	0.46	**

¹ Avg. SCS of test days 1 & 2. Number of records = 20303

² Avg. SCS of test days 3 & 4. Number of records = 17811

³ Avg. SCS of test days 5 & 6. Number of records = 16874

⁴ Avg. SCS of test days 7 & 8. Number of records = 14535

⁵ Avg. SCS of test days 9 & 10. Number of records = 3719

^a Statistical significance is indicated with asterisk marks as follows: *, Linear effects (P < 0.05), quadratic effects P > 0.05, **, Linear and quadratic effects P < 0.05, NS, not significant.

Table 2.27. Linear, quadratic, and cubic regressions and standards errors of 2nd lactation SCS test day segment averages on PTA_{SCS}.

Variable	Linear		Quadratic		Cubic		P > F ^a
	β	SE	β	SE	β	SE	
SCS Avg1 ¹	-25.3	21.5	8.1	6.4	-0.82	0.64	NS
SCS Avg2 ²	-15.9	22.9	5.2	6.9	-0.54	0.68	NS
SCS Avg3 ³	-6.3	22.3	2.3	6.7	-0.24	0.66	NS
SCS Avg4 ⁴	-39.4	22.9	12.6	6.9	-1.3	0.68	NS
SCS Avg5 ⁵	-51.5	51.8	16.6	15.5	-1.7	1.5	NS

¹ Avg. SCS of test days 1 & 2. Number of records = 20303

² Avg. SCS of test days 3 & 4. Number of records = 17811

³ Avg. SCS of test days 5 & 6. Number of records = 16874

⁴ Avg. SCS of test days 7 & 8. Number of records = 14535

⁵ Avg. SCS of test days 9 & 10. Number of records = 3719

^a Statistical significance is indicated with asterisk marks as follows: *, Linear effects (P < 0.05), quadratic effects P > 0.05, **, Linear and quadratic effects P < 0.05, NS, not significant.

Table 2.28. Linear and quadratic regression coefficients and standard errors of 3rd lactation test day segment averages on PTA_{SCS}.

Variable	Linear		Quadratic		P > F ^a
	β	SE	β	SE	
SCS Avg1 ¹	0.60	1.9	0.10	0.30	NS
SCS Avg2 ²	4.7	2.0	-0.55	0.30	*
SCS Avg3 ³	5.1	1.9	-0.63	0.30	**
SCS Avg4 ⁴	5.0	1.9	-0.61	0.30	**
SCS Avg5 ⁵	2.6	5.2	-0.26	0.80	NS

¹ Avg. SCS of test days 1 & 2. Number of records = 13619

² Avg. SCS of test days 3 & 4. Number of records = 12740

³ Avg. SCS of test days 5 & 6. Number of records = 11946

⁴ Avg. SCS of test days 7 & 8. Number of records = 9839

⁵ Avg. SCS of test days 9 & 10. Number of records = 1733

^a Statistical significance is indicated with asterisk marks as follows: *, Linear effects (P < 0.05), quadratic effects P > 0.05, **, Linear and quadratic effects P < 0.05, NS, not significant.

Table 2.29. Linear, quadratic, and cubic regressions and standards errors of 2nd lactation SCS test day segment averages on PTA_{SCS}.

Variable	Linear		Quadratic		Cubic		P > F ^a
	β	SE	β	SE	β	SE	
SCS Avg1 ¹	-44.2	29.3	13.5	8.8	-1.3	0.87	NS
SCS Avg2 ²	-14.2	30.7	5.1	9.2	-0.56	0.91	NS
SCS Avg3 ³	22.7	29.3	-5.9	8.8	0.52	0.87	NS
SCS Avg4 ⁴	-32.2	29.9	10.5	8.9	-1.1	0.89	NS
SCS Avg5 ⁵	-40.3	90.9	12.6	27.2	-1.3	2.7	NS

¹ Avg. SCS of test days 1 & 2. Number of records = 13619

² Avg. SCS of test days 3 & 4. Number of records = 12740

³ Avg. SCS of test days 5 & 6. Number of records = 11946

⁴ Avg. SCS of test days 7 & 8. Number of records = 9839

⁵ Avg. SCS of test days 9 & 10. Number of records = 1733

^a Statistical significance is indicated with asterisk marks as follows: *, Linear effects (P < 0.05), quadratic effects P > 0.05, **, Linear and quadratic effects P < 0.05, NS, not significant.

APPENDIX F. Linear, quadratic, and cubic regressions of 1st, 2nd, and 3rd lactation measures of SCS on PTA_{SCS}.

Table 2.30. Linear, quadratic, and cubic regressions and standards errors of 1st lactation measures of SCS on PTA_{SCS}.

Variable	Linear		Quadratic		Cubic		P > F ^a
	β	SE	β	SE	β	SE	
β _{SCSDIM} ¹	-0.02	.09	.006	.03	-6.4x10 ⁻⁴	.003	NS
Lact. Avg. SCS ²	-2.1	15.1	1.1	4.5	-.12	.45	NS

¹ Number of records = 21801

² Number of records = 21811

^a Statistical significance is indicated with asterisk marks as follows: *, Linear effects (P < 0.05), quadratic effects P > 0.05, **, Linear and quadratic effects P < 0.05, NS, not significant.

Table 2.31. Linear, quadratic, and cubic regressions and standards errors of 2nd lactation measures of SCS on PTA_{SCS}.

Variable	Linear		Quadratic		Cubic		P > F ^a
	β	SE	β	SE	β	SE	
β _{SCSDIM} ¹	.014	.12	-.003	.04	2.1x10 ⁻⁴	.003	NS
Lact. Avg. SCS ²	-24.9	17.9	8.0	5.4	-.82	.53	NS

¹ Number of records = 16864

² Number of records = 16876

^a Statistical significance is indicated with asterisk marks as follows: *, Linear effects (P < 0.05), quadratic effects P > 0.05, **, Linear and quadratic effects P < 0.05, NS, not significant.

Table 2.32. Linear, quadratic, and cubic regressions and standards errors of 3rd lactation measures of SCS on PTA_{SCS}.

Variable	Linear		Quad		Cubic		P >
	β	SE	β	SE	β	SE	
β _{SCSDIM} ¹	.15	.15	-.04	.05	.004	.005	NS
Lact. Avg. SCS ²	-17.8	24.4	6.0	7.3	-.64	.73	NS

¹ Number of records = 11506

² Number of records = 11509

^a Statistical significance is indicated with asterisk marks as follows: *, Linear effects (P < 0.05), quadratic effects P > 0.05, **, Linear and quadratic effects P < 0.05, NS, not significant.

VITAE

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