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## **Body Condition Score and Dairy Form as Indicators of Dairy Cattle Disease and Reproductive Performance.**

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To the Graduate Council:

I am submitting herewith a dissertation written by Chad D. Dechow entitled "Body Condition Score and Dairy Form as Indicators of Dairy Cattle Disease and Reproductive Performance.." I have examined the final electronic copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Animal Science.

Gary W. Rogers, Major Professor

We have read this dissertation and recommend its acceptance:

Dr. Kenneth Stalder, Dr. Arnold Saxton, Dr. F. David Kirkpatrick, Dr. Fred Hopkins

Accepted for the Council:

Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

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Dr. Fred Hopkins

Acceptance for the Council:

Anne Mayhew  
Vice Provost and Dean of  
Graduate Studies

(Original signatures are on file with official student records.)

**BODY CONDITION SCORE AND DAIRY FORM AS INDICATORS OF DAIRY  
CATTLE DISEASE AND REPRODUCTIVE PERFORMANCE**

A Dissertation  
Presented for the  
Doctor of Philosophy  
Degree  
The University of Tennessee, Knoxville

Chad Dechow  
August 2003

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## Abstract

The objectives of this research were to estimate heritabilities and correlations between body condition score (BCS) from various sources, determine the genetic relationship among BCS, dairy form, cow health and reproductive performance and investigate various models to analyze BCS and dairy form. BCS was obtained from herds using PCDART dairy management software and from linear type appraisals by Holstein classifiers. Cow health data was obtained from several herds recording disease treatments. Genetic evaluations for cow health in Denmark were also obtained. Reproductive data and yield data were provided by DRMS and AIPL-USDA. Heritabilities and correlations among traits were estimated with REML using animal and sire models. Random regression and repeatability sire models were compared. Fixed effects for all models included contemporary group effects, age, and days in milk (DIM) when available. Random effects were sire or animal and error. The heritability estimate of BCS from linear type appraisal was 0.22. The genetic correlation estimate between BCS from PCDART records and linear type appraisals was 0.87, between BCS and dairy form was  $-0.72$  and between BCS and strength was 0.69. The genetic correlation estimates from random regression models between DIM 0 in lactation 1 and DIM 305 in lactation 3 were estimated to be 0.77 for BCS and 0.60 for dairy form. Higher BCS and lower dairy form were significantly correlated with lower milk yield, less metabolic disease and fewer days open. The relationship among BCS, dairy form, cow health and reproductive disease remained significant after adjustment for milk yield. The relationship between BCS and cow health and reproductive performance tended to be non-significant after adjustment for dairy form. Supplementing direct genetic evaluations for days open with evaluations for dairy form increased reliability of days open by an average of 0.06 for 19 recently proven bulls. Selection for lower dairy form or higher BCS will slow the deterioration of cow health and reproductive performance that accompanies selection for increased yield.

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# Chapter 1

## Introduction

Dairy cattle selection programs have been successful in improving yields of milk, fat and protein (AIPL-USDA, 2003). Unfortunately, cow health and reproductive performance have declined in response to selection for yield. Production is unfavorably correlated with incidences of metritis, ketosis, milk fever, cystic ovaries, lameness, mastitis and other diseases (Kadarmideen et al., 2000; Pösö et al., 1996; Shanks et al., 1978; Tveit et al., 1992; Van Dorp et al., 1998). Abdallah and McDaniel (2000) estimated that days open increased at a rate of 1.1 days from 1980 to 1993 as a correlated response to selection for increased yield in five North Carolina experimental herds. A trend toward less voluntary culling of low producing cows has been documented (Weigel et al., 2003), which is likely the result of poorer fertility and cow health.

Concerns over declining cow health and reproductive performance have led to efforts to select cows that are healthier and more reproductively fit. Genetic variation for many fertility measures is substantial, indicating potential to select for improved fertility. Weigel and Rekaya (2000) reported ranges in sire breeding values for 60 day non-return rates of 16% for several California herds to 30% in several Minnesota herds. The genetic standard deviation of first service conception rate was near 0.05% in two studies (Berry et al., 2003, Veerkamp et al., 2001). The genetic standard deviation of calving interval was reported to be 7 days (Pryce et al., 2002) and 9 days (Veerkamp et al., 2001).

The presence of a genetic component for disease resistance has been documented for several diseases of the dairy cow. Dystocia, retained placenta, metritis, ovarian cysts, milk fever, mastitis, lameness, displaced abomasum and ketosis all have heritable genetic components (Lin et al., 1989; Tveit et al., 1992; Van Dorp et al., 1998).

Heritability estimates of reproductive and health traits from large data sets are generally low. Disease treatments are recorded on a large scale in some countries, including Denmark. The heritabilities for disease traits in Denmark's national genetic evaluations are all 0.05 (for clinical mastitis) or lower (Danish Cattle Federation, 2002). Many countries (including the US) do not have a centralized recording scheme to

facilitate large scale recording of health data necessary to generate genetic evaluations for cow health.

National genetic evaluations in the US for daughter pregnancy rate (DPR) are now available (VanRaden et al., 2002). The heritability of DPR (which is derived from days open data) was estimated to be 0.04.

Selection for health and reproductive performance in the US has largely been ignored until recently because of the low heritabilities associated with health and reproductive performance. Genetic improvement for such traits will take time and unfavorable correlations with other economically important traits (like yield) make selection for improved health or reproductive performance difficult. However, it was estimated that ignoring these traits in selection programs decreases the overall potential for improved economic efficiency by 15 to 25% (Philipsson et al., 1994) and anecdotal evidence suggests that many dairy producers are frustrated with the ability of their cows to conceive and resist disease.

An alternative to direct selection for lower disease incidence or improved reproductive performance is to select using traits that are genetically correlated to improved cow fitness levels. Dairy producers in the US have selected for improved udder morphology for some time. Cows genetically inclined to have shallower udders and stronger fore udder attachments are less prone to clinical mastitis (Nash et al., 2000, Rogers et al., 1998). Hansen (2000) speculated that selection for improved udder composite might explain a relatively constant cost for mastitis in a line of cows selected for higher yield compared to an increase in costs related to metabolic and reproductive diseases.

Productive life has a strong and favorable genetic relationship to cow health and has been used as an indicator to select for improved cow fitness in the US. Productive life evaluations are associated with decreased incidence of clinical mastitis in the US and Scandinavia and diseases other than mastitis in Scandinavia (Nash et al., 2000, Rogers et al., 1998; Rogers et al., 1999). While productive life evaluations for a sire do reflect the general reproductive fitness and health of a bull's daughters, it has a heritability of less than 0.10 and is recorded late in a cow's life, limiting the effectiveness of productive life

evaluations. Productive life itself must be supplemented with genetic evaluations for other traits to improve reliability for recently proven bulls (Weigel et al., 1998).

Somatic cell score evaluations are also used to select indirectly for lower clinical mastitis incidence. Bulls that sire daughters with high somatic cell score also have daughters with increased clinical mastitis incidence (Nash et al., 2000; Rogers et al., 1998). The heritability of somatic cell count (0.10) is also low, but a large number of daughters with somatic cell count data can be obtained fairly early during a bull's active service period.

While effective indicator traits do exist for mastitis resistance, productive life is the only indicator for reproductive performance and other disease traits and is limited in effectiveness. Clearly, more effective indicator traits are needed to aid selection for improved reproductive performance and resistance to most diseases. Body condition scores (BCS) and dairy form may be effective indicators for both reproductive performance and cow health.

Body condition scores are a subjective measure of body tissue reserves and are commonly used to monitor energy balance during the lactation (Wildman et al, 1982). Negative energy balance in early lactation requires cows to mobilize body tissue in support of lactation. Negative energy balance and excessive body tissue mobilization are associated with increased incidence of metabolic disorders and poor fertility (Baird, 1982; Butler et al, 1981; de Vries and Veerkamp, 2000; Loeffler et al., 1999).

Dairy form in the US is a measure of openness of rib (the spacing between a cow's ribs) and is related to BCS. Genetic correlation estimates between angularity (a similar trait to US dairy form) and BCS range from  $-0.47$  to  $-0.77$  (Veerkamp and Brotherstone, 1997). It is likely that many US classifiers consider the overall angularity of a cow when assigning dairy form scores and do not solely analyze openness of rib.

Body condition scores are genetically correlated with improved reproductive performance after adjustment for milk yield (Dechow et al., 2001, Pryce et al., 2000, Veerkamp et al., 2001). Higher dairy form is genetically correlated with an increase in disease incidence (Hansen et al., 2002, Rogers et al., 1999). Selection for higher BCS or

lower dairy form may help to increase stores of energy and decrease early lactation negative energy balance, which could improve cow health.

Changes in BCS and dairy form across the lactation reflect changes in energy balance. Cows lose BCS during negative energy balance in early lactation and regain BCS as the lactation progresses and daily milk yield declines. Those changes in BCS (or dairy form) might be heritable and could be related to cow fitness. Random regression models allow estimation of genetic merit for change in a trait (Jamrozik et al., 1997). Random regression models have been used in Europe to analyze BCS in first lactation cows (Jones et al., 1999; Veerkamp et al., 2001). Random regression models have also been used to analyze changes in genetic parameters for selected linear type traits and final score with age and to investigate changes in genetic parameters over time in the US (Tsuruta et al., 2002a; Tsuruta et al., 2002b; Uribe et al., 2000). Genetic evaluations for change in BCS or dairy form might be more effective indicators of cow health or reproductive performance than the level of BCS or dairy form.

The objectives of this study were to:

- 1) Estimate heritability and correlations among BCS, early lactation BCS loss, milk yield and reproductive performance in commercial dairy herds using producer recorded BCS.
- 2) Estimate the heritability of BCS recorded in a national linear type appraisal system that could be used to generate national genetic evaluations for BCS.
- 3) Estimate correlations among BCS, dairy form and other commonly recorded linear type traits.
- 4) Estimate correlations among BCS from various sources and recording schemes.
- 5) Determine the effectiveness of random regression models and repeatability models to generate evaluations for BCS and dairy form.
- 6) Examine the genetic and phenotypic relationship among BCS, dairy form and measures of cow health.



- 7) Determine the effectiveness of supplementing national fertility evaluations with genetic evaluations for BCS or dairy form.

## Chapter 2

### **Heritability and Correlations Among Body Condition Score Loss, Body Condition Score, Production and Reproductive Performance**

This chapter is a slightly modified version the following paper by the same name published in the Journal of Dairy Science in 2002 by C.D. Dechow, G.W. Rogers and J.S. Clay:

Dechow, C.D., G.W. Rogers, and J.S. Clay. 2002. Heritability and correlations among body condition score loss, body condition score, production and reproductive performance. *J. Dairy Sci.* 85: 3062-3070.

My contributions to this paper include: All data analysis, gathering and interpretation of literature and all writing of the manuscript except for editing of other authors and reviewers.

#### **ABSTRACT**

The objectives of this study were to estimate the heritability of body condition score loss (BCSL) in early lactation and estimate genetic and phenotypic correlations among BCSL, body condition score (BCS), production and reproductive performance. Body condition scores at calving and postpartum, mature equivalents for milk fat and protein yield, days to first service and services per conception were obtained from Dairy Records Management Systems in Raleigh, NC. Body condition score loss was defined as BCS at calving minus postpartum BCS. Heritabilities and correlations were estimated with a series of bi-variate animal models with average-information REML. Herd-year-season effects and age at calving were included in all models. The length of the prior calving interval was included for all second lactation traits and all non-production traits were analyzed with and without ME milk as a covariable. Initial correlations between BCS and BCSL were obtained using BCSL and BCS observations from the same cows. Additional genetic correlation estimates were generated through relationships between a group of cows with BCSL observations and a separate group of cows with BCS observations. Heritability estimates for BCSL ranged from 0.01 to 0.07. Genetic correlation estimates between BCSL and BCS at calving ranged from -0.15 to -0.26 in

first lactation and from  $-0.11$  to  $-0.48$  in second lactation. Genetic correlation estimates between BCSL and postpartum BCS ranged from  $-0.70$  to  $-0.99$  in first lactation and from  $-0.56$  to  $-0.91$  in second lactation. Phenotypic correlation estimates between BCSL and BCS at calving were near  $0.54$ , whereas phenotypic correlation estimates between BCSL and postpartum BCS were near  $-0.65$ . Genetic correlations between BCSL and yield traits ranged from  $0.17$  to  $0.50$ . Genetic correlations between BCSL and days to first service ranged from  $0.29$  to  $0.68$ . Selection for yield appears to increase BCSL by lowering postpartum BCS. More loss in BCS was associated with an increase in days to first service.

**(Key Words:** body condition score loss, heritability, production, reproduction)

**Abbreviation Key:** BCS = body condition score, BCSL = body condition score loss  
DFS = days to first service, ME = Mature Equivalent, SPC = services per conception.

## INTRODUCTION

Negative energy balance in early lactation requires cows to mobilize body tissue in support of lactation. Negative energy balance and excessive body tissue mobilization are associated with increased incidence of metabolic disorders and poor fertility (Baird, 1982; Butler et al, 1981; de Vries and Veerkamp, 2000; Loeffler et al., 1999). Body condition scores (BCS) are a subjective measure of body tissue reserves and are commonly used to monitor energy balance during the lactation (Wildman et al, 1982).

Genetic parameters for BCS have been reported by several authors (Dechow et al., 2001; Jones et al., 1999; Koenen et al., 2001, Veerkamp, 1998). Cows genetically inclined to have higher BCS during the lactation are reported to have fewer days to first service (DFS), fewer services per conception (SPC) and a shorter calving interval than cows that are genetically thin (Dechow et al., 2001; Pryce et al., 2000; Pryce et al, 2001). The genetic correlation between energy balance and first luteal activity was reported to be moderately negative after adjustment for yield (Veerkamp et al., 2000). Additionally, bulls that sire daughters with high dairy form scores (and likely more angular and thin) have daughters with higher incidences of metabolic, reproductive and foot and leg diseases (Hansen et al., 2002; Rogers et al., 1999).

Direct estimates of the heritability of body condition score loss (BCSL) and the genetic relationship among BCSL, production and reproductive performance are limited. The heritability of BCS change from week 1 to week 10 of lactation was reported to be 0.09 in an experimental herd (Pryce et al., 2001). Additionally, genetic correlation estimates between BCS measured at various points during the lactation has been reported to be high, indicating that genetic variation for BCSL may be limited (Dechow et al., 2001; Jones et al., 1999; Koenen et al., 2001). Body condition score loss from week 1 to week 10 of lactation was reported to be genetically correlated with higher yield, and extended DFS, days to first heat and calving interval in an experimental herd (Pryce et al., 2001).

The genetic relationship between BCSL and BCS has not been defined, but may be important to understand to the impact of selection for yield on energy balance and BCS. The objectives of this study were to estimate the heritability of BCSL and estimate genetic and phenotypic correlations among BCSL, BCS, production and reproductive performance in commercial dairy herds.

## **MATERIALS AND METHODS**

### **Data**

Body condition scores were obtained from the Dairy Records Management System in Raleigh, NC. Dairy producers or herd-consultants using PCDART dairy management software recorded BCS on a scale of 1 (thin) to 5 (fat) at one or more of the following scoring periods: calving, postpartum, first service, pregnancy check, before dry-off and dry-off. In the current study, only BCS at calving and postpartum were considered. The days in milk when a BCS was evaluated was not reported. However, the order given above is presumed to correspond to the order BCS was recorded during lactation. Therefore, postpartum BCS would be recorded after calving and before first service. The genetic correlation between postpartum BCS and BCS at first service was previously reported to be 1.0, and mean postpartum BCS and BCS at first service were similar (Dechow et al, 2001). Therefore, the average days in milk when postpartum BCS was recorded is likely to be slightly less than the average days to first service. The

average days to first service is 86.6 in first lactation and 88.4 in second lactation, so postpartum BCS were, on average, likely to be recorded in the second or third month of lactation.

Mature equivalents (ME) for milk, fat and protein production, DFS and two sources of SPC were available. The first source of SPC was used to investigate the genetic and phenotypic relationship between BCSL and SPC, whereas the second source of SPC was used to investigate the phenotypic relationship between SPC and DFS. SPC did not include services not resulting in pregnancy.

Services per conception for genetic analyses were recovered from cows that had conceived and subsequently calved. Records from second and higher lactations reported the number of times a cow had been inseminated in the previous lactation. Thus, SPC in first lactation was obtained from a cow's second lactation record. Likewise, SPC in second lactation was recovered from a cow's third lactation record. Cows that had conceived, but not subsequently calved, would not have a SPC record from this source.

Lactation records reported a cow's pregnancy status, if known, and the number of times that cow had been inseminated. Approximately 10% of cows were confirmed pregnant and had both SPC and DFS recorded. These records were used to investigate the phenotypic relationship between DFS and SPC.

The initial data set included 310,071 lactation records. Not all lactation records had BCS data available. Records were edited to include those cows with a valid identification, a registered Holstein sire and a Holstein dam. Valid birth dates and calving dates were required and lactations initiated by an abortion were eliminated. First lactation cows that had calved prior to 20 months of age or later than 36 months of age were eliminated, whereas second lactation cows were required to have calved no earlier than 10 months and no later than 24 months after first calving. Records were required to have a ME milk of at least 4,536 kg. Services per conception records were edited to include only those cows that required fewer than 10 inseminations to conceive, whereas DFS records were edited to include those cows that were first served between 25 and 200 days after calving.

In total, records for at least one trait were available for 51,195 cows after edits. The number of observations and mean of ME Milk, DFS and SPC are reported in Table 1. Not all cows that had production or reproductive performance data available had BCS data available.

A total of 27,817 cows from 236 herds in first lactation and 20,936 cows from 235 herds in second lactation had BCS available at one or more scoring periods. A breakdown of the above number of cows that had BCS observations in each scoring period, mean BCS in each scoring period, average age at calving, average calving interval, and the number of sires and herds represented in each scoring period are reported in Table 2. The number of cows with BCSL, mean BCSL, average age at calving, average calving interval, and the number of sires and herds represented for cows with BCSL are also reported in Table 2.

In first lactation, 9,656 cows had BCS available in one scoring period only, 6,524 in two scoring periods, 6,430 in 3 scoring periods and 5,167 in four or more scoring periods. Body condition scores were available for 6,496, 5,300, 5,287 and 3,853 for one, two, three, and four or more scoring periods, respectively, in second lactation.

Heritabilities, genetic and phenotypic correlation estimates among BCS at all six scoring periods, production and reproductive performance were previously reported by Dechow et al. (2001) using the same data. The focus of the current study was to investigate the relationship among BCSL in early lactation, BCS, production and reproductive performance.

It was determined that BCS at calving and postpartum BCS were the most suitable scoring periods to investigate BCSL in early lactation for two reasons. First, postpartum BCS had more observations available than BCS at first service. Secondly, the mean BCS at pregnancy check was higher than that of postpartum BCS or BCS at first service, indicating that early lactation BCSL had ceased and that external body fat was beginning to be deposited by pregnancy check. The average postpartum BCS for all cows that had postpartum BCS available was 2.91 in first lactation and 2.82 in second lactation (Table 2). The average BCS at pregnancy check was 2.96 and 2.92 in first and second lactation, respectively. Of those cows that had BCS available at both postpartum and

Table 1. Number of observations and mean of ME milk, days to first service (DFS) and services per conception (SPC).

	Observations (n)	Mean
<b>1<sup>st</sup> Lactation</b>		
ME Milk	48,332	10,409 kg
DFS	11,319	86.6
SPC <sup>1</sup>	34,681	2.40
SPC <sup>2</sup>	4,596	2.36
<b>2<sup>nd</sup> Lactation</b>		
ME Milk	32,796	10,688 kg
DFS	10,192	88.4
SPC <sup>1</sup>	20,179	2.40
SPC <sup>2</sup>	3,622	2.26

<sup>1</sup>Services per conception used in genetic analyses. <sup>2</sup>Services per conception used to investigate the phenotypic relationship between SPC and DFS.

Table 2. Observation numbers for body condition scores (BCS), sires, herds, average age at calving (in months), average calving intervals (C.I.) and mean BCS at each specific scoring period in first and second lactations.

Scoring Period	Observations (n)	BCS	Calving Age (Months)	C.I. (Days)	Sires	Herds
1 <sup>st</sup> Lactation						
Calving	17,316	3.18	25.29	-	2,325	188
Postpartum	10,728	2.91	25.30	-	1,806	178
First service	5,828	2.87	26.08	-	1,384	177
Pregnancy check	12,405	2.96	25.87	-	2,003	205
Before dry off	9,639	3.12	26.26	-	1,781	204
Dry off	9,214	3.31	25.26	-	1,620	155
BCS Loss <sup>1</sup>	7,424	0.30	24.97		1,381	115
2 <sup>nd</sup> Lactation						
Calving	13,937	3.07	39.03	421.51	2,097	203
Postpartum	8,308	2.82	39.14	425.17	1,654	177
First service	4,562	2.81	39.83	422.12	1,193	177
Pregnancy check	8,865	2.92	39.36	420.90	1,600	210
Before dry off	6,328	3.19	39.96	425.78	1,373	196
Dry off	8,105	3.45	38.78	415.41	1,617	171
BCS loss <sup>1</sup>	6,092	0.29	38.81	423.79	1,317	125

<sup>1</sup>Body condition score loss is defined as BCS at calving minus postpartum BCS.



pregnancy check, the mean postpartum BCS was 2.89 in first lactation and 2.8 in second lactation, while BCS at pregnancy check averaged 2.96 and 2.93 in first and second lactation, respectively. Body condition scores were available at both calving and postpartum for 7,424 cows in first lactation and 6,092 cows in second lactation.

### **Analyses**

The traits included in the genetic analyses included BCS at calving and postpartum, BCSL in early lactation, three production traits (ME milk, ME fat and ME protein) and two reproductive traits (DFS and SPC). Body condition score loss was defined as BCS at calving minus postpartum BCS. Higher values for BCSL represent more loss of BCS in early lactation.

Heritabilities, genetic and phenotypic correlations among BCSL and production, reproductive performance and BCS were estimated using a series of bi-variate analyses. Analyses were performed with the average-information algorithm of the derivative-free REML program (Meyer, 1998). Standard errors for genetic correlations were calculated according to Falconer and Mackay (1996).

The basic statistical model used in the analyses was:

$$\mathbf{y} = \mathbf{b} * \text{age} + \mathbf{hys} + \mathbf{animal} + \mathbf{e}$$

where  $\mathbf{y}$  = a vector of BCSL and one of the following: BCS at calving or postpartum, ME milk, ME fat, ME protein, DFS, or SPC,

$\text{age}$  = age at calving in months,

$\mathbf{b}$  = vector of regression coefficients on age at calving in months,

$\mathbf{hys}$  = vector of fixed effects for herd-year-season of calving,

$\mathbf{animal}$  = a vector of random animal effects and

$\mathbf{e}$  = a vector of normally distributed random residuals.

Sire identification for all cows and dam identification for most cows were available and included in the pedigree for any cow with a record for one or more traits. Maternal grand-sire identification was also available and included in the pedigree as sire

of the dam. Sire and maternal grandsire pedigrees were traced for five generations and all ancestors were included in the pedigree. The final pedigree included all 51,195 cows from 5,390 sires and 43,488 dams. A total of 100,718 animals were included in the pedigree when ancestors were included.

All models for second lactation traits also included the length of the prior calving interval as a covariable. Non-production traits (BCSL, BCS, DFS, SPC) were analyzed with and without ME milk as a covariable.

The season of calving effects were defined as January through April, May through August and September through December. Since the number of days in milk when postpartum BCS was recorded was not known, days in milk was not included in the model. However, days in milk when postpartum BCS was recorded should be consistent within a herd and would be recorded before first service.

Because BCSL was derived from BCS at calving and postpartum, genetic correlation estimates between BCSL and BCS at calving or postpartum could be biased by part-whole influences when one trait is a function of another. Therefore, genetic correlations were estimated using two approaches. First, correlations between BCSL and BCS were estimated by allowing cows to contribute observations for both BCSL and BCS at either calving or postpartum, depending on which BCS trait was being analyzed. All cows that had an observation for BCSL, by the definition of BCSL, would also have observations for BCS at calving and postpartum.

Second, genetic correlations between BCSL and BCS at calving or postpartum were estimated only through pedigree linkages. Cows with BCS available at both calving and postpartum contributed BCSL observations, as in the first method. However, cows with BCSL observations were not allowed to contribute an observation for BCS at calving or postpartum. Only cows that had an observation available for BCS at calving and no postpartum BCS observation available (thus BCSL could not be calculated) contributed records for BCS at calving. Likewise, only cows that did not have a record for BCS at calving contributed records for postpartum BCS. Genetic correlation estimates between BCSL and BCS would then be through relationships in the pedigree described above between a group of cows with BCSL and a separate group of cows with BCS

observations. There was no residual covariance between BCSL and BCS with this approach because no cows had observations for both traits.

The second approach allows for estimation of genetic parameters free of any part-whole influences that might otherwise impact parameter estimates when one trait is a function of another trait. Using both approaches should give a reasonable estimate of the genetic relationship between BCSL and BCS and not simply reflect the definition of BCSL used in this study.

The number of days in milk when a cow is inseminated is likely to impact the success of that insemination. Adjusting SPC for DFS may result in more accurate correlations between SPC and other traits, particularly if those traits are correlated with DFS. Unfortunately, the number of cows with both SPC and DFS data in the same lactation was too small to facilitate accurate genetic analyses. Attempts to perform genetic analyses with the second set of SPC observations and DFS either did not converge or resulted in solutions at the boundary of the parameter space. Sufficient observations were available to determine the phenotypic relationship between SPC and DFS however.

Multiple regression was performed with ASREML (Gilmour, 2000) to determine the phenotypic relationship between DFS and SPC. The model used to investigate the relationship between SPC and DFS was:

$$y_i = b_1 * \text{age} + \text{hys}_i + \sum_{j=2}^5 b_j * \text{DFS}^{j-1} + e_i$$

where  $y = \text{SPC}$ ,

$\text{age} = \text{age at calving in months}$ ,

$b_1 = \text{regression coefficient on age at calving in months}$ ,

$b_j = \text{regression coefficients of order 1 to 4 for DFS}$ ,

$\text{hys}_i = \text{ith fixed effect for herd-year-season of calving}$ ,

$e_i = \text{random residuals}$ .

A minimum of five cows per HYS group was required. Fourth-order polynomials of DFS were significant ( $p < .05$ ) in first and second lactation, whereas fifth-order polynomials were not ( $p > .24$ ).

## RESULTS

Heritabilities and correlations among BCS at calving and postpartum, ME milk, fat and protein, DFS and SPC are reported in Dechow et al., 2001. Heritability estimates for BCS at calving were reported to be 0.10 in first lactation and 0.13 in second lactation. Heritability estimates for postpartum BCS were reported to be 0.15 in first lactation and 0.14 in second lactation.

Mean BCS are reported in Table 2. Mean BCS at calving was 3.18 in first lactation and 3.07 in second lactation. Mean postpartum BCS were 2.91 and 2.82 in first and second lactation, respectively. Of those cows that had both BCS at calving and postpartum BCS, an average of 0.30 and 0.29 BCS was lost in early first and second lactation, respectively. Heritability estimates for BCSL ranged from 0.05 to 0.07 in first lactation and from 0.01 to 0.03 in second lactation.

Genetic correlation estimates between BCS and BCSL are reported in Table 3. When cows were allowed to contribute both BCS at calving and BCSL observations, genetic correlation estimates ranged from  $-0.11$  to  $-0.29$ . Genetic correlations were stronger (negative) when estimated through pedigree linkages only, ranging from  $-0.24$  to  $-0.48$ .

Genetic correlation estimates between BCSL and postpartum BCS ranged from  $-0.70$  to  $-0.91$  when cows were allowed to contribute BCS and BCSL observations. Genetic correlation estimates ranged from  $-0.56$  to  $-0.99$  when estimates were through pedigree linkages only. Including ME milk as a covariable in the model did not change the genetic correlation estimates between BCSL and BCS beyond one standard error.

Phenotypic correlations between BCSL and BCS at calving ranged from 0.53 to 0.55. Phenotypic correlations between BCSL and postpartum BCS ranged from  $-0.62$  to  $-0.69$ .

Correlations between BCSL and production traits are reported in Table 4. Increased BCSL was correlated with increased ME milk, fat and protein yield both genetically and phenotypically. Genetic correlation estimates ranged from 0.17 to 0.50, whereas phenotypic correlations ranged from 0.06 to 0.10.

Correlations between BCSL and reproductive traits are reported in Table 5.

Table 3. Genetic and phenotypic correlations between body condition score loss and body condition score (BCS) at calving and postpartum BCS in first and second lactation.<sup>1</sup>

	Genetic Correlations		Phenotypic Correlations	
	First Lactation	Second Lactation	First Lactation	Second Lactation
Calving <sup>2</sup>	-0.15	-0.29	0.55	0.53
Calving <sup>2,4</sup>	-0.17	-0.11	0.55	0.54
Calving <sup>3</sup>	-0.26	-0.48	...	...
Calving <sup>3,4</sup>	-0.24	-0.44	...	...
Postpartum <sup>2</sup>	-0.72	-0.89	-0.69	-0.64
Postpartum <sup>2,4</sup>	-0.70	-0.91	-0.68	-0.62
Postpartum <sup>3</sup>	-0.81	-0.56	...	...
Postpartum <sup>3,4</sup>	-0.99	-0.64	...	...

<sup>1</sup> Approximate standard errors of genetic correlations average 0.12 and are less than 0.19 in first lactation. Approximate standard errors of genetic correlations average 0.22 and are less than 0.41 in second lactation.

<sup>2</sup>Correlations derived using all available BCS and BCS loss observations. Cows with BCS loss observations also had BCS observations.

<sup>3</sup>Correlations derived through pedigree linkages only. Cows with BCS observations were not the same cows as those with BCS loss observations.

<sup>4</sup>ME Milk included as a covariable.

Table 4. Genetic and phenotypic correlations between body condition score loss and ME milk, fat and protein in first and second lactation.<sup>1</sup>

	Genetic Correlations		Phenotypic Correlations	
	First Lactation	Second Lactation	First Lactation	Second Lactation
ME Milk	0.50	0.17	0.10	0.09
ME Fat	0.40	0.46	0.09	0.06
ME Protein	0.41	0.30	0.09	0.06

<sup>1</sup> Approximate standard errors of genetic correlations average 0.08 and are less than 0.09 in first lactation. Approximate standard errors of genetic correlations average 0.18 and are less than 0.19 in second lactation.

Table 5. Genetic and phenotypic correlations between body condition score loss and days to first service (DFS) and services per conception (SPC) in first and second lactation.<sup>1</sup>

	Genetic Correlations		Phenotypic Correlations	
	First Lactation	Second Lactation	First Lactation	Second Lactation
DFS	0.68	0.44	0.09	0.06
DFS <sup>2</sup>	0.52	0.29	0.08	0.05
SPC	0.20	-0.21	0.02	-0.01
SPC <sup>2</sup>	0.16	-0.46	0	-0.03

<sup>1</sup> Approximate standard errors of genetic correlations average 0.18 and are less than 0.23 in first lactation. Approximate standard errors of genetic correlations average 0.40 and are less than 0.44 in second lactation.

<sup>2</sup>ME milk included as covariate in model.

Genetic correlation estimates between BCSL and DFS were positive in first and second lactation before and after adjustment for ME milk, ranging from 0.29 to 0.68. Phenotypic correlation estimates were not as strong, but were still positive, ranging from 0.05 to 0.09.

The genetic correlation estimates between BCSL and SPC were positive in first lactation, but negative in second lactation before and after adjustment for ME milk. Phenotypic correlation estimates ranged from -0.03 to 0.02.

A plot of the regression of SPC on DFS is shown in Figure 1. In general, SPC decline as DFS increase until around 175 days in milk. The average SPC within a HYS group for cows that were first served at 175 days was approximately half the SPC required at 25 days.

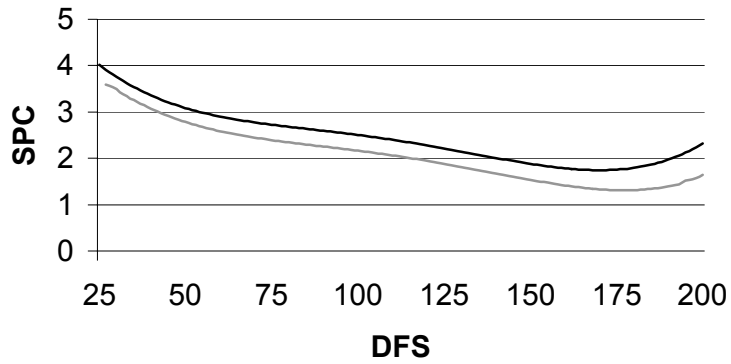
## **DISCUSSION**

The heritability of BCSL was lower than that of BCS at calving or postpartum in this study. The genetic correlations between BCS at calving and postpartum BCS were reported to be 0.74 in first lactation and 0.87 in second lactation using the same data (Dechow et al., 2001). However, the phenotypic correlations between BCS at calving and postpartum BCS were reported to be only 0.26 in first lactation and 0.35 in second lactation. While the amount of BCSL in early lactation varied between cows, the genetic component contributing to that variation was relatively small.

The heritability of BCS change from week one to week ten was reported to be 0.09, while the heritability of BCS was 0.28 at week one and 0.27 at week ten in a research herd (Pryce et al., 2001). Over a range of studies, genetic correlation estimates between BCS measured at different points during the lactation are strong. Genetic correlation estimates between BCS at the beginning of lactation and BCS at the end of lactation were reported to be 0.69 by Jones et al. (1999), 0.99 and 0.87 by Koenen et al. (2001), and 0.84 and 0.93 by Dechow et al. (2001). Genetic correlation estimates in all three studies tend to be highest for BCS measured in consecutive months or stages of lactation.



Figure 1. Regression of services per conception (SPC) on days to first service (DFS) in first (—) and second (—) lactation after adjustment for herd-year-season of calving and age at calving.



A second factor likely contributing to the low heritability for BCSL in this study was the inability to account for the days in milk when postpartum BCS was assigned. This was assumed to be the major factor contributing to lower heritability estimates for BCS compared to other estimates in Dechow et al. (2001). Presumably, postpartum BCS would have been assigned after calving and before first service, but that may not be the practice in all herds, especially those that do not record BCS at first service. While the genetic component contributing to BCSL does not appear to be high, small genetic differences between cows may exist in the amount of BCS lost during early lactation. Jones et al. (1999) reported differences in the shape of the average daughter BCS curve for six sires with >1500 daughters using random regression models.

Phenotypically, a higher BCS at calving was associated with more BCSL in early lactation in this and other studies (Treacher, 1986; Garnsworthy and Jones, 1987). Genetically, an increase in BCS at calving was correlated with less BCSL during early lactation. Management and environmental conditions that increased BCS at calving resulted in more BCSL in early lactation. However, cows that were genetically inclined to have higher BCS at calving appeared to maintain more BCS in early lactation than genetically thin cows.

The genetic and phenotypic relationship between postpartum BCS and BCSL was strong and negative. Management and environmental conditions that limited loss of BCS in early lactation resulted in higher postpartum BCS. Likewise, cows that were genetically inclined to have relatively high postpartum BCS tended to lose less BCS in early lactation.

There were differences in the magnitude of genetic correlation estimates between the two approaches used to estimate genetic correlations between BCS and BCSL. The definition of BCSL used in this study forced phenotypic correlations between BCSL and BCS at calving to be positive, while phenotypic correlations between BCSL and postpartum BCS must be negative. This would likely cause bias due to part-whole influence to result in genetic correlation estimates between BCS at calving and BCSL that are stronger (positive) than the true genetic correlation. Likewise, bias due to part-

whole relationships might result in genetic correlation estimates between postpartum BCS and BCSL that are stronger (negative) than the true genetic correlation.

Some part-whole influence on genetic correlation estimates might have occurred when BCS and BCSL observation were from the same cows, particularly for genetic correlation estimates between BCS at calving and BCSL. The correlation estimates between BCSL and BCS at calving were stronger (negative) in both first and second lactation when estimates were obtained through pedigree linkages only (Table 3). This would seem to indicate that the genetic correlation estimates were positively biased when obtained using observations of BCS at calving and BCSL from the same cows. Genetic correlation estimates between BCSL and postpartum BCS were stronger (negative) in first lactation, but stronger (positive) in second lactation when estimates were obtained through pedigree linkages only.

Despite some potential part-whole bias, there was one pattern that was consistent across all analyses; genetic correlation estimates between postpartum BCS and BCSL were stronger (negative) than estimates between BCS at calving and BCSL. This indicates that selection programs that increase BCSL are likely to do so by lowering postpartum BCS levels more than BCS at calving.

Cows in an experimental herd that have been selected for increased yield have higher dairy form scores (and are thus more angular and thin) than control herd-mates that are bred to maintain a 1964 genetic level for production (Boettcher et al., 1993). However, the selected line has higher incidences of metabolic diseases normally associated with cows that are over-conditioned at calving than the control line (Jones et al., 1994). As cows become genetically thinner, the amount of BCS lost during early lactation is likely to increase at a given level of BCS at calving. Continued selection, whether directly or indirectly, for thinner cows is likely to continue to increase negative energy balance and BCSL in early lactation. Additionally, the target levels for BCS at calving that are recommended to dairy producers may need to reflect genetic trends for BCS.

The genetic and phenotypic correlations between BCSL and production were low to moderately positive. Cows that are genetically inclined to lose more BCS in early

lactation tend to have higher yields of milk, fat and protein. Genetic correlations were similar in magnitude to those previously reported (range -0.06 to -0.31) for production and postpartum BCS (Dechow et al., 2001). Waltner et al. (1993), reported that BCSL in the range of 0.5 to 1.5 BCS was associated with higher production. However, Garnsworthy and Jones (1987) reported that thinner cows had higher dry matter intakes, produced a larger proportion of milk directly from food, and produced milk more efficiently than fatter cows that mobilized more body condition. Selection programs that increase yield without increasing levels of BCSL may result in more efficient dairy production than those that do not account for BCSL.

The genetic relationship between BCSL and DFS was unfavorable before and after adjustment for ME milk. The magnitude of the genetic correlations between BCSL and DFS were similar to those reported by Dechow et al. (2001) for postpartum BCS and DFS (range -0.57 to -0.76). Cows genetically inclined to maintain BCS in early lactation and have higher postpartum BCS are inseminated earlier in the lactation. Cows in negative energy balance are reported to have delayed luteal activity and estrus (Butler et al., 1981; de Vries et al., 1999; Harrison et al., 1990). Cows that are genetically inclined to lose more BCS and have low levels of postpartum BCS are subject to more negative energy balance in early lactation, which appears to delay onset of luteal activity and first estrus.

Genetic correlations between BCSL and SPC were positive in first lactation, but negative in second lactation (Table 5). The standard errors for the genetic correlations between BCSL and SPC were high however, ranging from 0.22 to 0.44. Several authors have reported that fertility decreases as BCSL increases (Domecq et al., 1997; Gillund et al., 2001; Loeffler et al., 1999). The effects of DFS on SPC were not accounted for in the genetic analyses. The effects of BCSL on SPC may not be observed when DFS is not considered and may have resulted in inconsistent genetic correlation estimates between BCSL and SPC.

Cows were losing BCS and in negative energy balance until near pregnancy check in this study. Services per conception decreased from nearly four when DFS was 25 to less than two when DFS was 175. Some of the relationship observed between SPC and

DFS may be exaggerated by the nature of the data set. If successful inseminations were more likely to be reported by producers than unsuccessful inseminations, SPC are likely to be under-reported and SPC would be expected to decline as DFS increases. However, the trend was strong and likely reflects more than recording inaccuracies. The reduction in SPC as DFS increases likely reflects the effect of negative energy balance on fertility.

The effect of DFS on SPC could have implications for reproductive management. Inseminating a large proportion of cows in the first two months of lactation is likely to lower herd conception rates and increase semen expenditures. However, waiting to inseminate cows when they are likely to be most fertile will increase calving intervals. Moreover, average BCS at the following calving could be higher because of an extended lactation, which is likely to result in greater negative energy balance the following lactation. An alternative may be to use less expensive or young sire semen in early lactation and more expensive semen later in lactation. Additionally, tracking BCS change could help determine which cows are in more severe negative energy balance and therefore candidates for delayed DFS or less expensive semen.

## **CONCLUSIONS**

Selection for higher yield increases BCSL in early lactation. Genetic correlations were low to moderate between BCSL and milk yield. However, higher levels of yield are attainable while limiting the amount BCSL in early lactation. Increased BCSL as a correlated response to selection occurs by lowering postpartum BCS more than BCS at calving. Increases in BCSL and lower postpartum BCS are associated with an increase in DFS.

Body condition score loss has a strong negative correlation with postpartum BCS both genetically and phenotypically, but has a lower heritability than postpartum BCS. Moreover, genetic correlation estimates between BCSL and both production and reproductive performance are similar in magnitude to the genetic correlation estimates between postpartum BCS and performance. Selection for higher postpartum BCS would likely be more efficient in maintaining or improving reproductive performance than selection for reduced BCSL.

## Chapter 3

### **Heritabilities and Correlations Among Body Condition Score, Dairy Form and Selected Linear Type Traits**

This chapter is a slightly modified version of a paper by the same name published in the Journal of Dairy Science in 2003 by C.D. Dechow, G.W. Rogers, L. Klei and T.J. Lawlor:

Dechow, C.D., G.W. Rogers, L. Klei, and T.J. Lawlor. 2003. Heritabilities and correlations among body condition score, dairy form and selected linear type traits. J. Dairy Sci. 86:2236-2242.

My contributions to this paper include: All data analysis, gathering and interpretation of literature and all writing of the manuscript except for editing of other authors and reviewers.

#### **ABSTRACT**

The objectives of this study were to estimate the heritability of body condition score (BCS) with data that could be used to generate genetic evaluations for BCS in the US, and to estimate the relationship among BCS, dairy form and selected type traits. Body condition score and linear type trait records were obtained from Holstein Association USA Inc. Because BCS was a new trait for classifiers, scoring distribution and accuracy was not normal. Records from 11 of 29 classifiers were eliminated to generate a data set that should represent BCS data recorded in the future. Edited data included 128,478 records for analysis of first lactation cows and 207,149 records for analysis of all cows. Heritabilities and correlations were estimated with ASREML using sire models. Models included age at calving nested within lactation, 5<sup>th</sup> order polynomials of days in milk, fixed herd-classification visit effects and random sire and error. Genetic correlation estimates were generated between first lactation data that had records from 11 classifiers removed and data with no classifiers removed. Genetic correlation estimates were 0.995 and above between data with and without classifiers removed for scoring distributions, but heritability estimates were higher with the classifiers edited from the data. Heritability estimates for type traits and final score were similar to previously reported estimates. The heritability estimate for BCS was 0.19 for first lactation cows and

0.22 for all cows. The genetic correlation estimate for first lactation cows between BCS and dairy form was  $-0.73$ , whereas the genetic correlation estimate between BCS and strength was  $0.72$ . Genetic correlation estimates were nearly identical when cows from all lactations were included in the analyses. Body condition score had a genetic correlation with final score closer to zero ( $0.08$ ) than correlations of final score with dairy form, stature or strength.

(Key Words: **body condition score, heritability, genetic correlation**) Abbreviation Key: **BCS** = body condition score, **HD\_CL** = herd-classification visit.

## INRODUCTION

Body condition score (BCS) evaluations may be useful as an indicator trait in selection for improved reproductive performance and cow health. Body condition scores are genetically correlated with improved reproductive performance after adjustment for milk yield (Dechow et al., 2001, Pryce et al., 2000, Veerkamp et al., 2001). Moreover, higher dairy form is genetically correlated with an increase in disease incidence (Hansen et al., 2002, Rogers et al., 1999). Genetic correlation estimates between angularity (a similar trait to US dairy form) and BCS range from  $-0.47$  to  $-0.77$  (Veerkamp and Brotherstone, 1997).

Heritability estimates for BCS when recorded during routine on farm linear type appraisal range from  $0.25$  to  $0.38$  in Europe (Koenen et al., 2001; Pryce et al., 2000; Veerkamp et al., 2001). Heritability estimates for BCS in the US have been generated with field data and are lower ( $0.07$  to  $0.20$ ) than heritability estimates from other studies (Dechow et al., 2001). While studies with field data have helped estimate the genetic relationship among BCS, production and reproductive performance in the US, national genetic evaluations for BCS are not likely to be generated with such data. The Holstein Association USA Inc. began to record BCS during routine linear type evaluations in the fall of 1997 is the probable source of any national genetic evaluations for BCS in the US.

The objectives of this study were to estimate the heritability of BCS with data that could be used to generate genetic evaluations for BCS in the US, and to estimate the relationship among BCS, dairy form and selected type traits.

## MATERIALS AND METHODS

### Data and Editing

Records for BCS and type traits were obtained from Holstein Association USA Inc., which began recording BCS in the fall of 1997. Therefore, records from October of 1997 through June of 2000 were available. The initial data set included 728,597 records from 613,338 cows. Body condition scores are recorded on a scale of 1 (thin) to 50 (fat) to be consistent with the scale used for linear type traits.

Body condition score distributions were not normal for some classifiers. Scoring procedures for BCS will improve in the future as classifiers become accustomed to evaluating BCS. Therefore, two edits were applied to generate a BCS data set that would represent data used to generate genetic evaluations for BCS in the future.

The standard deviation of BCS in first lactation was 6.37. Therefore, the random number generator in Microsoft® Excel 2000 was used to generate 10,000 observations for a trait with a mean of 25 and standard deviation of six. The most frequent number generated occurred 6.7% of the time. However, it is possible to use a limited number of scores when evaluating body condition and still determine differences in body condition among cows accurately. For example, body condition in some European type classification systems is scored on an integer scale of 1 to 9 (Koenen et al., 2001; Pryce et al., 2000; Veerkamp et al., 2001). Therefore, randomly generated scores were grouped in intervals of five, resulting in 10 interval groups. Randomly generated scores fell in the most frequent interval 30.1% of the time. Data from 10 classifiers that assigned a single BCS more than 30.1% of the time for first lactation cows was eliminated.

A second set of classifier edits was applied using the approach of Veerkamp et al. (2002) to identify classifiers that were scoring traits inconsistently when compared with other classifiers. Genetic correlations between BCS recorded by a single classifier and BCS recorded by all other classifiers were generated. Low genetic correlations would indicate that classifier is scoring body condition inconsistent with other classifiers. Veerkamp et al. (2002) reported genetic correlations for BCS between a single classifier



and BCS for all other classifiers to be greater than 0.92 for classifiers that had scored at least 1,000 cows.

Of the remaining 19 classifiers, 18 had evaluated at least 1000 first lactation cows. Genetic correlations between BCS from a single classifier and BCS from all other classifiers were generated for those 18 classifiers. The same edits and procedures described below for other analyses were applied. Seventeen of the classifiers had genetic correlations for BCS with all other classifiers of greater than 0.90. One classifier had a genetic correlation for BCS with all other classifiers of 0.75 and data from that classifier was removed.

One classifier had only 137 first lactation records and a genetic correlation between that classifier and all other classifiers could not be estimated, but data from that classifier was not removed. In total, records from 11 of 29 classifiers were eliminated.

Other data edits included a requirement of 20 daughters per sire and 10 cows for each herd-classification visit. Records from cows that had calved before 20 months of age and after 60 months of age were eliminated. Records from cows that have calved after 60 months of age are not used by Holstein Association USA Inc. to generate genetic evaluations for type traits because final score is not allowed to decline after 60 months of age. Records that were recorded after 305 days in milk were also eliminated.

The data edits used here differ from those that would be used for national genetic evaluations. All cows from contemporary groups of two or more are included in national genetic evaluations and no limit is placed on the number of daughters per sire (10 or more daughters are required for an official proof).

An initial data set was formed to determine the effect of editing for classifier on genetic parameter estimates. Unedited traits for BCS, dairy form, stature, strength and final score were formed that included first lactation records from all classifiers if the record was associated with an odd numbered herd-classification date. A second set of edited traits was formed that included first lactation records only from classifiers that were not eliminated by classifier edits and that were associated with even numbered herd-classification date. This data set included 106,257 records that were not edited for classifier and 46,292 records that were edited for classifier.

A data set consisting of 128,478 first lactation cows and a data set consisting of 207,149 cows between the ages of 20 to 60 months were formed only from records not removed by classifier edits. The cows were sired by 1,645 bulls and records were from 11,998 herd-classification visits. Three generations of ancestors were traced for each sire and the final pedigree file included 3,156 animals.

## **Analyses**

All analyses were performed in ASREML (Gilmour et al., 2002) using single to three-trait sire models. Single trait models were used to estimate heritability and repeatability for BCS, dairy form, stature, strength and final score. A series of two-trait sire models were used to estimate correlations among most traits. Three-trait sire models were used if the relationship among three traits was of interest. For example, correlation estimates among BCS, foot angle and rear legs side view were generated with a three-trait model, as were correlations among BCS, dairy form and strength in first lactation.

Sire models were chosen because of reduced computational demands, especially for three-trait models, and because data were from a less than three year window which would minimize the number of daughter-dam pairs, especially in the first lactation data. Nearly all pedigree ties in the data are among paternal half-siblings with related sires, and heritability estimates would be expected to be nearly identical to those obtained with an animal model.

Initial analyses were between first lactation records edited for classifier and records not edited for classifier for the following traits: BCS, dairy form, stature, strength and final score.

All other analyses were performed with data that had been edited for classifier. Analyses for first lactation cows only were between BCS and dairy form, and between BCS or dairy form and the following selected type traits: stature, strength, body depth, thurl width, rear legs side view, foot angle, udder composite, frame, feet and legs composite, body size composite, dairy composite and final score.

The final set of analyses were performed with records from all cows calving between 20 and 60 months of age for the following traits: BCS, dairy form, stature,

strength and final score. The set of cows that would be used by the Holstein Association USA Inc. for national genetic evaluations for BCS are most closely represented by this set of analyses.

The statistical model used in the analyses is shown below:

$$\mathbf{y} = \mathbf{b}_1 * \text{age}(\text{lact}) + \sum_{m=2}^6 \mathbf{b}_x * \text{DIM}^{m-1} + \text{Sire} + \text{HD\_CL} + \boldsymbol{\varepsilon}$$

where  $\mathbf{y}$  = BCS or a selected type trait for univariate models, a vector of length two for two-trait models, or a vector of length three for three-trait models,

$\mathbf{b}_1$  = a vector of regression coefficients on age at calving nested within lactation, age=age at calving in months,

lact= fixed lactation number,

$\mathbf{b}_x$  = a vector of regression coefficients of order 1 to 5 on DIM,

DIM=days in milk,

Sire=a vector of random effects for sire,

HD\_CL=a vector fixed effects for herd-classification visit, and

$\boldsymbol{\varepsilon}$  = random error.

To reduce the number of effects required in the model, cows were not allowed to contribute more than one record in any analyses with one exception; uni-variate models were used to estimate repeatability for BCS, dairy form, stature, strength and final score using records from all cows that had calved from 20 to 60 months of age. A random, permanent environment effect was included for those analyses. A total of 20,973 cows had more than one record available for this analysis (total number of records=228,122). For all other analyses, the earliest record was chosen for cows with multiple records within lactation. Likewise, for cows with records from multiple lactations, the earliest lactation with a record was chosen.

The Holstein Association USA Inc. accounts for age and stage of lactation effects by including fixed group effects for age and stage of lactation. This was not done in this study because BCS changes over the lactation period more than other traits, particularly in early lactation. Secondly, average BCS at a given age is affected by the average stage of lactation. Average values for traits like stature would not be expected to increase and decrease with age depending on stage of lactation as would BCS.

## RESULTS AND DISCUSSION

### Data Edits

The polynomial regression solutions for BCS on DIM from the two-trait model of BCS before edits for classifier and BCS after edits for classifier are shown in Figure 2. Regression solutions for BCS decreases from calving until 69 DIM for both traits. There was more change in BCS after edits for classifier. The change from calving to day 69 DIM was 2.68 for the unedited data set and 3.42 for the edited data set. The change in BCS from 69 DIM to 305 DIM was 2.97 for the unedited data set and 3.07 for the edited data set. The fixed classes used in the national genetic evaluations to model stage of lactation effects would account for much of the change shown in Figure 2, but solutions would not be as smooth as the curves shown here.

Heritabilities and genetic correlations among records edited and not edited for classifier for BCS, dairy form, stature, strength and final score are shown in Table 6. Genetic correlations between edited and unedited data for all traits were 0.995 or higher. Heritability estimates were highest in the edited data set for all traits. The largest change in heritability estimate was for BCS. The heritability estimate of BCS increased from 0.14 in the unedited data set to 0.19 for the edited data set.

The edits made for classifier appear to have had their intended effect. The high genetic correlation between traits edited and unedited for classifier indicates that classifier edits did not select records from cows that were genetically different for BCS. Thus, the genetic correlation estimates between the BCS records edited for classifier and selected type traits will be accurate. However, the edited data likely had more accurate BCS records, resulting in a higher heritability estimate.

The heritability estimate for BCS reported here is lower than heritability estimates for BCS in first lactation from Europe (Koenen et al., 2001; Pryce et al., 2000; Veerkamp et al., 2001). A sire model was used here, while other reported estimates have been obtained with an animal model or a sire-maternal grandsire model. However, that is not likely the major cause of the lower heritability estimate. The Holstein Association USA Inc. has estimated the heritability of BCS using current national evaluation procedures

Figure 2. Regression solutions for BCS on DIM in first lactation before and after edits for classifier.

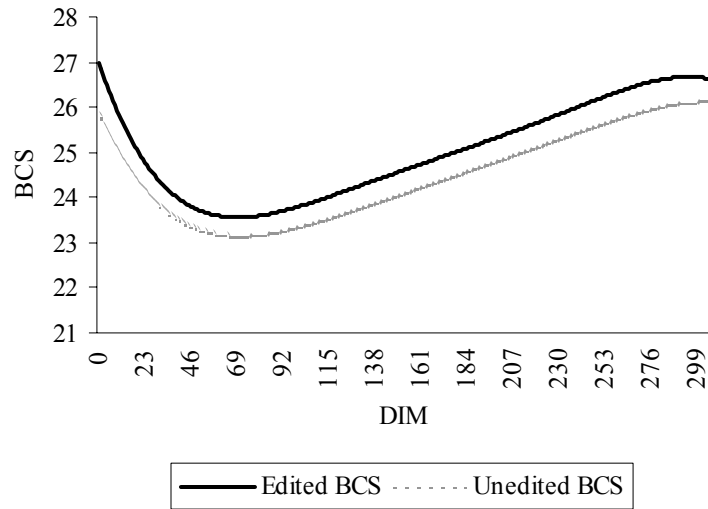


Table 6. Heritabilities ( $h^2$ ) and genetic correlations ( $r_a$ ) between data edited for classifier and data unedited for classifier for body condition score (BCS), dairy form, stature, strength and final score.<sup>1</sup>

	$h^2$ edited data	$h^2$ unedited data	$r_a$
BCS	0.19	0.14	0.995
Dairy Form	0.25	0.23	0.995
Stature	0.33	0.32	1.00
Strength	0.23	0.22	1.00
Final Score	0.24	0.21	1.00

<sup>1</sup>Standard errors for heritability estimates ranged from 0.01 to 0.02. Approximate standard errors for the genetic correlations ranged from 0 to 0.01.

(which includes an animal model) to be 0.15 using Method  $\mathfrak{R}$  (L. Klei, 2002, personal communication). Classifiers that gave a large proportion of cows the same BCS were eliminated, but edits were not as stringent as those used in this study.

Other heritability estimates for BCS in the US are lower than heritability estimates for BCS in Europe (Dechow et al., 2001). Body condition scores used in genetic studies in Europe may have been recorded more consistently than those used in genetic studies in the US. The heritability of BCS in the US may increase as classifiers become more accustomed to scoring body condition. It is not clear that management conditions in Europe would result in less environmental variance for BCS, which would also result in higher heritability estimates.

It is possible that the genetic variance for BCS is lower in the US than in Europe. Average BCS was reported to decrease from 5.4 to 4.4 for first lactation Dutch-Friesian cows as the percentage of North American Holstein genes increased from 50% to 100% (Koenen et al., 2001). A lower average BCS for US dairy cows could be associated with less genetic variance for BCS. Higher yield is genetically correlated with lower BCS (Dechow et al., 2001; Veerkamp et al., 2001). More intense selection for milk yield in the US may have resulted in a lower average BCS and a reduced genetic variance for BCS.

The heritability estimates reported in Table 6 for stature, strength, dairy form and final score are also lower than other estimates published by the Holstein Association USA Inc. (Holstein Association USA Inc., 2002). The estimates reported in Table 6 are for first lactation cows only, however, and estimates that include all lactations are similar to published estimates.

### **First Lactation Cows**

Correlation estimates between BCS, dairy form and selected type traits in first lactation are reported in Table 7. The genetic correlation estimate between BCS and dairy form was  $-0.73$  while the phenotypic correlation was  $-0.44$ . Genetic correlations between BCS at calving and angularity (similar to US dairy form) in a research herd were reported to range from  $-0.47$  to  $-0.77$  (Veerkamp and Brotherstone, 1997). Cows with high dairy form scores tend to be angular and thin. Body condition score and dairy form are not

Table 7. Genetic ( $r_a$ ) and phenotypic ( $r_p$ ) correlation estimates among body condition score (BCS), dairy form, selected linear type traits and final score for first lactation cows.<sup>1</sup>

	BCS		Dairy Form	
	( $r_a$ )	( $r_p$ )	( $r_a$ )	( $r_p$ )
Dairy Form	-0.73	-0.44	1.00	1.00
Strength	0.72	0.50	-0.16	-0.02
Stature	0.20	0.19	0.23	0.19
Body Depth	0.40	0.34	0.20	0.23
Thurl Width	0.27	0.22	0.16	0.12
Body Size Composite	0.43	0.36	0.18	0.19
Frame	0.25	0.21	0.24	0.26
Rear Legs Side View	-0.38	-0.19	0.35	0.11
Foot Angle	0.38	0.18	-0.21	0.01
Feet and Legs Composite	0.19	0.14	-0.03	0.10
Dairy Composite	-0.75	-0.46	0.93	0.84
Udder Composite	0.10	0.03	0.10	0.18
Final Score	0.05	0.05	0.34	0.41

<sup>1</sup>Standard errors for genetic correlation estimates ranged from 0.007 to 0.055, while standard errors for phenotypic correlation estimates ranged from 0.001 to 0.004.



entirely the same trait, however, and dairy form evaluates more than level of body condition.

There was a strong genetic correlation (0.72) and phenotypic correlation (0.50) between BCS and strength. Others have reported moderate to strong genetic correlation estimates between BCS and measures similar to strength. Genetic correlation estimates between BCS and chest width ranged from 0.32 to 0.73, whereas the genetic correlation between BCS and heart girth circumference was reported to be 0.34 (Gallo et al., 2001; Veerkamp and Brotherstone, 1997). Additionally, dairy character had a negative genetic correlation (-0.47) with muscularity (Koenen and Groen, 1998). Genetic correlations between BCS and muscularity would thus be expected to be positive.

Body condition score was also positively correlated with body size composite and other body dimension traits including stature, body depth, frame and thurl width both genetically (range 0.20 to 0.43) and phenotypically (range 0.19 to 0.36). Genetic correlation estimates between BCS and stature were reported to range from -0.09 to 0.32 (Veerkamp and Brotherstone, 1997). Negative genetic correlation estimates were reported for first lactation cows only and were positive for cows of all ages. Genetic correlation estimates between BCS and body depth ranged from -0.24 to 0.26 in the same study, with negative correlations occurring in first lactation cows only. Veerkamp and Brotherstone (1997) reported the genetic correlation between BCS and live weight to be 0.67, while Enevoldsen et al. (1997) reported the phenotypic correlation between BCS and body weight to be 0.53. Cows with more body condition have more body fat and muscle, and thus appear to be stronger, have somewhat larger body dimensions and weigh more.

The genetic correlation estimate between dairy form and strength was -0.16, while the phenotypic correlation was -0.02. Genetic and phenotypic correlation estimates between dairy form and body size composite and other body dimension traits were positive, ranging from 0.12 to 0.26.

Genetic correlation estimates between dairy character and measures of body size, including heart girth, hip height, body depth, size and rump width, were reported to range from 0.14 to 0.70, whereas phenotypic correlations ranged from 0.08 to 0.61 (Koenen and

Groen, 1998). Dairy character was reported to be positively correlated with body weight both genetically (0.15) and phenotypically (0.11) in the same study. Veerkamp and Brotherstone (1997) reported negative genetic correlations (range  $-0.07$  to  $-0.56$ ) between angularity and live weight however.

Cows with high dairy form scores likely have larger body dimensions, but the relationship between dairy form and body weight is less clear because cows with higher dairy form are also thinner. Cows with higher dairy form also appear to have slightly lower strength scores and may have less muscularity.

Genetic correlation estimates between BCS and feet and legs composite, foot angle and rear legs side view were 0.19, 0.38 and  $-0.38$ , respectively. Phenotypic correlation estimates were 0.14, 0.18 and  $-0.19$  between BCS and feet and legs composite, foot angle and rear legs side view, respectively. The genetic and phenotypic correlation estimates between dairy form and feet and legs composite were  $-0.03$  and 0.10, respectively, between dairy form and foot angle were  $-0.21$  and 0.01, respectively and between dairy form and rear legs side view were 0.35 and 0.11, respectively.

Cows with higher BCS would be expected to be heavier and heavier cows have more foot and leg trouble than smaller cows. Cows that have been selected for higher body size weighed 51kg more after calving than herd mates selected for smaller body size in an experimental herd in Minnesota (Hansen et al., 1999). The cows selected for larger body size were culled more often for leg and foot problems than cows that were selected for small body size. The authors speculated that the higher body weight of the larger cows resulted in greater stress on the cow's feet and legs and the larger cows may have been more prone to foot and leg injuries. However, cows with lower BCS (and are thus likely to weigh less) had slightly lower feet and legs composite, a lower foot angle and more set to the hock in this study.

Cows that were genetically inclined to have higher dairy form scores also had more set to the hock and had slightly lower foot angles. Moreover, Rogers et al. (1999) reported that sires with daughters that had high dairy form also had daughters with poorer foot and leg health. Cows that are thin appear to have more set to the hock, a lower foot angle and poorer foot and leg health despite likely having a lower body weight. Perhaps

cows that are inclined to be thin and angular are susceptible to more stress on their feet and legs. Poor locomotion could also reduce BCS because of reduced feed intake. The relationship between body weight and foot and leg conformation or foot and leg health might be even more apparent if BCS or dairy form is considered in the model.

The genetic and phenotypic correlation estimates between BCS and final score were 0.05. The genetic and phenotypic correlation estimates between dairy form and final score were 0.34 and 0.41, respectively. Despite the strong genetic correlation between BCS and dairy form, BCS is not included in calculation of final score and is therefore more independent of final score than is dairy form.

### **All Cows**

Heritabilities, repeatabilities and variances for BCS, dairy form, stature, strength and final score for all cows are in Table 8. Heritability estimates were lowest for BCS (0.22) and highest for stature (0.37). The heritability estimates for all traits, except dairy form (0.24), were higher for all cows than for first lactation cows only. Heritability estimates for stature and dairy form reported previously using a sire-maternal grandsire model were identical to those reported here, whereas the heritability estimate for strength was 0.29 (Short et al., 1991). The heritability estimates are slightly lower than those obtained using an animal model. Heritability estimates using an animal model have been reported to range from 0.41 to 0.42 for stature, 0.29 to 0.30 for strength, and were reported to be 0.28 for dairy form and 0.29 for final score (Misztal et al., 1992; Misztal et al., 1995).

Repeatability estimates ranged from 0.33 for BCS to 0.85 for final score. Repeatability estimates obtained using an animal model are nearly identical to those for stature and strength, but lower (0.46) for dairy form (Misztal et al., 1995).

Genetic and phenotypic correlations among BCS, dairy form, stature, strength and final score for all cows are reported in Table 9. Genetic and phenotypic correlation estimates between BCS and dairy form and stature, strength and final score were similar to those reported for first lactation cows only. The genetic correlation estimate between final score and BCS (0.08) was lower than genetic correlation estimates between final score and dairy form, stature and strength (range 0.34 to 0.56). Phenotypic correlations

Table 8. Genetic variance ( $V_a$ ), permanent environmental variance ( $V_{pe}$ ), residual variance ( $V_e$ ), heritability ( $h^2$ ) and repeatability (rpt) for body condition score (BCS), dairy form, selected linear type traits and final score for all cows ages 20 to 60 months.<sup>1</sup>

	( $V_a$ )	( $V_{pe}$ )	( $V_e$ )	$h^2$	rpt
BCS	7.16	3.77	22.18	0.22	0.33
Dairy Form	10.16	15.6	16.47	0.24	0.61
Stature	18.84	13.91	18.30	0.37	0.64
Strength	10.8	8.98	20.80	0.27	0.49
Final Score	5.12	12.65	2.95	0.25	0.85

<sup>1</sup>Standard errors for heritability and repeatability estimates ranged from 0.01 to 0.02.

Table 9. Genetic (above diagonal) and phenotypic (below diagonal) correlation estimates among body condition score (BCS), dairy form, selected linear type traits and final score for all cows ages 20 to 60 months.<sup>1</sup>

	BCS	Dairy Form	Stature	Strength	Final Score
BCS		-0.72	0.27	0.69	0.08
Dairy Form	-0.44		0.21	-0.11	0.34
Stature	0.20	0.19		0.73	0.56
Strength	0.49	0.00	0.57		0.42
Final Score	0.06	0.41	0.36	0.32	

<sup>1</sup>Standard errors for the genetic correlation estimates ranged from 0.02 to 0.04, while standard errors for the phenotypic correlation estimates ranged from 0.002 to 0.003.

between final score and BCS (0.06) were also lower than phenotypic correlation estimates between final score and dairy form, stature and strength (range 0.36 to 0.41).

Genetic and phenotypic correlation estimates previously reported among dairy form, stature and strength are similar to those reported in this study (Misztal et al., 1992). Previously reported genetic correlations between final score and stature (0.75) and strength (0.62) were somewhat higher than reported in this study (Misztal et al., 1992).

### **CONCLUSIONS**

Body condition scores routinely recorded by Holstein Association USA Inc. provide an extensive and consistent source of BCS observations. The editing and evaluation procedures used in this study differ than those used currently for national evaluations and the parameter estimates used in a national genetic evaluation might vary slightly from those reported here. The heritability estimate of BCS is expected to increase as classifiers become more accustomed to evaluating cows for BCS, however. Body condition score is highly correlated with dairy form and strength. Body condition score is not highly correlated with final score and BCS evaluations may be influenced less by final score than many type traits. Previously reported relationships between BCS and reproductive performance coupled with heritable variation for BCS may warrant generation of national BCS evaluations. Body condition score evaluations would likely be useful as an early indicator of reproductive fitness.

## Chapter 4

### **Heritability and Correlations for Body Condition Score and Dairy Form Within and Across Lactation and Age**

This chapter is a slightly modified version of a paper by the same name accepted for publication in the Journal of Dairy Science by C.D. Dechow, G.W. Rogers, L. Klei and T.J. Lawlor.

My contributions to this paper include: All data analysis, gathering and interpretation of literature and all writing of the manuscript except for editing of other authors and peer reviewers.

#### **ABSTRACT**

The objectives of the current study were to investigate the relationship between body condition score (BCS) and dairy form and changes in genetic parameters for BCS and dairy form within and across lactations and age. Body condition score and dairy form were obtained from the Holstein Association USA Inc. Records were edited to include those cows classified between 24 and 60 months of age and between 0 and 335 days in milk. A minimum of 20 daughters per sire and 15 cows per herd-classification visit were required. The data set consisted of 135,178 records from 119,215 cows. Repeatability, multiple trait and random regression models were used to analyze the data. All models included fixed effects for herd-classification visit, age within lactation 1, 2 and 3 or higher, and 5<sup>th</sup> order polynomials for DIM. Random effects included sire and permanent environment for all models. Random regression models included age at classification nested within sire or DIM and lactation number nested within sire. Genetic variance for both BCS and dairy form was lowest in early lactation and highest in mid lactation. Genetic correlations within and across lactations were high. The genetic correlation between DIM 0 in lactation 1 and DIM 305 in lactation 3 was estimated to be 0.77 for BCS and 0.60 for dairy form. The genetic correlation estimate between 30 months of age at classification and 50 months of age at classification was 0.94 for both dairy form and

BCS. The repeatability models appeared to generate accurate evaluations for BCS or dairy form at all ages and stages of lactation.

**(Key Words:** body condition score, dairy form, random regression)

**Abbreviation Key:** **BCS** = body condition score, **BCS<sub>x</sub>** = BCS on DIM X, **BCH<sub>X-Y</sub>** = BCS on DIM X minus BCS on DIM Y, **BCH<sub>DP</sub>** = BCS on DIM 0 in lactation 2 – DIM 305 in lactation 1, **DF<sub>x</sub>** = dairy form on DIM X, **DCH<sub>X-Y</sub>** = dairy form on DIM X minus dairy form on DIM Y, **HEV** = heterogeneous residual variance, **HOV** = homogeneous residual variance, **L** = lactation number, **LG** = lactation group, **LG1** = lactation group 1, **LG2** = lactation group 2, **LG3** = lactation group 3, **LP** = Legendre polynomial, **LP0** = intercept, **LP1** = linear Legendre polynomial, **LP2** = quadratic Legendre polynomial, **MDRR** = multidimensional random regression, **MT** = multiple trait, **PE** = permanent environment, **PTA** = predicted transmitting ability, **RPT** = repeatability, **RRA** = random regression on age.

## INTRODUCTION

Body condition score (BCS) and dairy form are genetically similar traits that are related to production, cow health and reproductive performance. The genetic correlation between BCS and dairy form score in the US has been estimated to be  $-0.72$  (Dechow et al., 2003).

Body condition score is favorably correlated genetically with days to first heat, days to first service, conception rates and calving intervals (Dechow et al., 2001; Pryce et al., 2001; Veerkamp et al., 2001). Higher levels of BCS are also genetically correlated with lower milk yield in the above studies, but the genetic relationship between BCS and reproductive performance exists after adjustment for yield. Higher BCS loss during early lactation is also related to higher production and poorer reproductive performance (Dechow et al., 2002; Pryce et al., 2001).

Dairy form has been genetically correlated with increased disease incidence after adjustment for milk yield (Hansen et al., 2002; Rogers et al., 1999). Despite the antagonistic relationship between dairy form and measures of cow health, selection has been practiced for higher dairy form in the US because of a favorable relationship with



production. The genetic correlation between dairy form and milk yield was reported to be 0.52 (Short and Lawlor, 1992).

Random regression models have been used to analyze BCS in first lactation in Europe (Jones et al., 1999; Veerkamp et al., 2001). Random regression models have also been used to analyze changes in genetic parameters for selected linear type traits and final score with age and to investigate changes in genetic parameters over time in the US (Tsuruta et al., 2002a; Tsuruta et al., 2002b; Uribe et al., 2000).

Random regression models have not been used to analyze changes in BCS or dairy form within lactation in the US. Moreover, changes in BCS or dairy form with age or lactation number have not been investigated. Multidimensional random regression models allow investigation of changes both within lactation and across lactation number or age simultaneously (Jensen, 2001).

The objectives of the current study were to: 1) investigate changes in genetic parameters for BCS and dairy form within and across lactations using multidimensional random regression models, 2) investigate changes in genetic parameters with age for BCS and dairy form using random regression models, and 3) further investigate the relationship between BCS and dairy form.

## **MATERIALS AND METHODS**

### **Data**

Body condition and dairy form scores were obtained from the Holstein Association USA Inc. The initial data set included 728,597 records on 613,338 cows that were recorded from October of 1997 through June of 2000. Body condition score is recorded on a scale of 1 (thin) to 50 (fat). Body condition score was a new trait for classifiers and BCS were not distributed normally for many classifiers. Therefore, records from classifiers that assigned BCS abnormally were eliminated with the same procedures used in Dechow et al. (accepted).

Cows that were classified before 24 months of age or later than 60 months of age were eliminated to be consistent with the data editing procedures used for the national genetic evaluations. Classification scores do not decline for cows greater than 60 months

of age in the US. Cows that were more than 335 days in milk were eliminated. Additional data edits included a requirement of 20 daughters per sire and 15 cows for each herd-classification visit. Edits for a minimum number of daughters per sire and cows per herd-classification visit were necessary to make parameter estimation computationally feasible. However, there is no minimum number of daughters required for the national genetic evaluations and all cows from contemporary groups of two or more are retained.

The final data set included 135,178 records from 119,215 cows. There were 80,967 first lactation records, 40,468 second lactation records and 13,743 records from third to fifth lactations. Within a given lactation, 4,768 cows had two records and 26 cows had 3 records. Across lactations, 10,301 cows had records in two lactations while 421 cows had records in three lactations.

The cows were sired by 827 bulls and were evaluated in 4,726 herd-classification visits. Three generations of sires and dams were traced for each sire resulting in a pedigree file that included 1,654 animals.

## Analyses

Sire models for BCS or dairy form were performed with ASREML (Gilmour et al., 2002). Several models described below were used to analyze the data. Likelihood ratio tests were used to test the significance of random effects in random regression models (Gilmour et al., 2002).

**Repeatability models (RPT).** Body condition and dairy form scores on the same cow at different DIM and in different lactations were considered repeated observations of the same trait. The statistical model is described below:

$$y_{ijklm} = hd\_cl_i + b_1 * age(LG_j) + \sum_{k=2}^6 b_{kj} * DIM^{k-1}(LG_j) + Sire_l + PE_m + \varepsilon_{ijklm},$$

[1] where  $y_{ijklm}$  = BCS or dairy form. Fixed effects were:  $hd\_cl_i$  = herd-classification visit  $i$ ,  $b_1$  = regression coefficient on age at calving nested within lactation group  $j$ ,  $b_{kj}$  = regression coefficients on DIM of order 1 to 5 nested within lactation group  $j$ , and  $LG_j$  were lactation groups consisting of first lactation cows (LG1), second lactation cows (LG2) and third through fifth lactation cows (LG3). Random effects

included:  $Sire_l$  = effect of sire l,  $PE_m$  = permanent environmental effect for cow m, and  $\varepsilon_{ijklm}$  = random error.

**Random regression on age at classification models (RRA).** Changes in random genetic and permanent environment effects were considered a function of age at classification with the model described below:

$$y_{ijklmnp} = hd\_cl_i + b_1 * age(LG_j) + \sum_{k=2}^6 b_{kj} * DIM^{k-1}(LG_j) + \sum_{n=0}^1 Sire_l * b_{ln} * age_p^n + \sum_{n=0}^1 PE_m * b_{mn} * age_p^n + \varepsilon_{ijklmnp}, \quad [2]$$

where  $y_{ijklmnp}$  = BCS or dairy form, and the fixed effects are the same as described for model 1 except age at calving is replaced with age at classification,  $b_{ln}$  = random regression coefficients of order 0 to 1 on age at classification p for sire l,  $b_{mn}$  = random regression coefficients of order 0 to 1 on age at classification p for the permanent environmental effects of cow m, and  $\varepsilon_{ijklmnp}$  = random error.

Convergence was not obtained for BCS or dairy form models that included random regression coefficients for  $age_p^2$ . Random error variance was allowed to vary for the following age at classification groups: 24 to 30 months, 31 to 35 months, 36 to 40 months, 41 to 45 months, 46 to 50 months, 51 to 55 months, and 56 to 60 months.

This model allows generation of sire transmitting abilities for any age at classification. Additionally, sire transmitting abilities for change in daughter BCS or dairy form as they mature can be calculated.

**Multidimensional random regression on DIM and lactation number models (MDRR).** Changes in random genetic and permanent environment effects were considered a function of DIM and lactation number and are described by the model

below:

$$y_{ijkmnpq} = \text{fixed effects}_i + \sum_{p=0}^2 Sire_{jp} \phi_{pk} + Sire_{jm} L_m + Sire_{j1m} \phi_1 L_m + \sum_{q=0}^x PE_{nq} \phi_{qk} + PE_{nm} L_m + \varepsilon_{ijkmnpq}, \quad [3]$$

where  $y_{ijkmnpq}$  = BCS or dairy form, fixed effects<sub>i</sub> are the ith fixed effects and are identical to those described for model 1,  $Sire_{jp}$  = random regression coefficient for sire j on a Legendre polynomial (LP) for DIM of order p,  $\phi_{pk}$  = LP of order p (LP0 = intercept, LP1 = linear and LP2 = quadratic) for DIM k,  $Sire_{jm}$  = random regression coefficient for sire j on lactation number m ( $L_m$ ),  $Sire_{j1m}$  = random regression coefficient for sire j on LP1 x

$L_m$ ,  $PE_{nq}$  = random regression coefficient for permanent environmental effect of cow  $n$  on LP for DIM of order  $q$ ,  $\phi_{qk}$  = LP of order  $q$  for DIM  $k$ ,  $x = 1$  for BCS and 0 for dairy form, and  $\varepsilon_{ijkmnpq}$  = random error.

Legendre polynomials are orthogonal and can be used with random regression models to model smooth curves and growth trajectories (Kirkpatrick et al., 1990). Legendre polynomials are standardized to range from -1 (day 0) to 1 (day 335). LP0 is a constant and for these analyses was set to 1. Cubic LP for sire effects would not converge for BCS, and did not improve the log-likelihood ( $p = 0.52$ ) for dairy form.

Initial analyses attempted to fit sire and PE effects to identical order of LP. However, analyses of BCS including PE\*LP2 would not converge so only PE\*LP1 was fit. For analyses of dairy form, models including PE\*LP1 or PE\*LP2 would not converge.

A lack of cows with multiple records within lactation may have limited successful modeling of higher order LP for PE. Only 4,768 cows had two observations within a single lactation and very few (26) cows had three observations within a single lactation.

Including  $Sire_{j1m} \phi_1 L_m$  improved the log likelihood significantly for dairy form ( $p < .001$ ) and was included in the analysis of dairy form, but  $Sire_{j1m} \phi_1 L_m$  was not included in analysis of BCS ( $p = 0.75$ ).

Two types of residual error structures were compared. Residual variance was assumed to remain constant across DIM and lactation for the first analyses. This first model assumed homogeneous residual variance (HOV). Residual variance was allowed to vary by month within LG for the second set of analyses. Consecutive months with similar residual variance were then grouped, resulting in the following five residual variance groups for each LG: month 1, month 2, months 3 through 8, months 9 and 10, and month 11. In LG3, only 266 records were available for month 11, so month 11 was grouped with months 9 and 10. This second model assumed heterogeneous residual variance (HEV).

The above models will allow the generation of sire transmitting abilities for any DIM between 0 and 335 in any lactation 1 through 3 (there were only 163 observations in lactations 4 and 5). Moreover, sire transmitting abilities can be generated for change in

BCS or dairy form between any two DIM in any lactation. Sire transmitting abilities for daughter change in BCS or dairy form as lactations progress could be generated.

**Random regression model assumptions.** Models 2 and 3 can be written in matrix notation as

$$\mathbf{y} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}_1\mathbf{a} + \mathbf{Z}_2\mathbf{p} + \mathbf{e},$$

where  $\mathbf{y}$  = a vector of BCS or dairy form,  $\mathbf{X}$  is an incidence matrix for fixed effects,  $\boldsymbol{\beta}$  is a vector of fixed effects,  $\mathbf{Z}_1$  is an incidence matrix for sire effects,  $\mathbf{a}$  is a vector of random regression coefficients for sire effects,  $\mathbf{Z}_2$  is an incidence matrix for permanent environmental effects,  $\mathbf{p}$  is a vector of random regression coefficients for permanent environmental effects,  $\mathbf{e}$  is a vector of residual effects. It was assumed that

$$\begin{pmatrix} \mathbf{a} \\ \mathbf{p} \\ \mathbf{e} \end{pmatrix} \sim \mathbf{N}(\mathbf{0}, \mathbf{V})$$

and

$$\mathbf{V} = \begin{pmatrix} \mathbf{G} \otimes \mathbf{A} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{P} \otimes \mathbf{I}_1 & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{R} \end{pmatrix}$$

where

$\mathbf{G}$  and  $\mathbf{P}$  are the covariance matrices of random regression coefficients for sire and permanent environmental effects, respectively, and are assumed to be the same for all sires,  $\mathbf{A}$  is the additive genetic relationship among sires,  $\otimes$  is the direct product,  $\mathbf{I}$  is an identity matrix with order equal to the number of cows, and  $\mathbf{R}$  is a diagonal matrix of residual variances for models that assume HEV, or residual variance for models that assume HOV.

For analysis of dairy form using model 3,

$$\mathbf{G} = \mathbf{V} (a_{\phi 0}, a_{\phi 1}, a_{\phi 2}, a_L, a_{L*\phi 1})'.$$

**Multiple trait models (MT).** Body condition score and dairy form were analyzed with two types of MT models. First, MT models with either BCS or dairy form in LG1,

LG2 and LG3 treated as different traits were compared. A three-trait model was used for analysis of BCS. However, a three-trait model would not converge with dairy form because of genetic correlation estimates near the boundary of the parameter space. Therefore, three bi-variate analyses were performed for dairy form to generate correlations among LG1, LG2 and LG3.

A second set of MT models were used to estimate correlations between BCS and dairy form at different lactation stages. The traits analyzed were BCS or dairy form in the following five lactation periods: months 1 through 2, 3 through 4, 5 through 6, 7 through 8, and 9 through 11.

The statistical model is as follows:

$$\mathbf{y} = \mathbf{b}_1 * \text{age} + \mathbf{hd\_cl} + \sum_{m=2}^6 \mathbf{b}_x * \text{DIM}^{m-1} + \mathbf{Sire} + \mathbf{PE} + \boldsymbol{\varepsilon}, \quad [4]$$

where  $\mathbf{y}$  = a vector of length three for BCS with LG1, LG2 and LG3 treated as separate traits, a vector of length two with dairy form from two LG, or a vector of length two with BCS and dairy form from the same lactation period,  $\mathbf{b}_1$  = a vector regression coefficients on age at calving,  $\mathbf{hd\_cl}$  = vector of fixed effects for herd-classification visit,  $\mathbf{b}_x$  = a vector of regression coefficients on DIM polynomials of order 1 through 5,  $\mathbf{Sire}$  = a vector of random effects for sire,  $\mathbf{PE}$  = a vector of random permanent environmental effects for cow, and  $\boldsymbol{\varepsilon}$  = random error.

Heritabilities, correlations and PTAs generated with the MT models were used to help assess the accuracy and fit of RPT and random regression models for a given lactation. The MT models for lactation period will also help assess the relationship between BCS and dairy form at different stages of lactation.

## Variance Derivation

The matrix of random regression coefficients for sire with MDRR (model 3) is as follows for analysis of dairy form:

$$C_S = \begin{bmatrix} \sigma^2_{\phi_0} & & & & \\ \sigma_{\phi_0, \phi_1} & \sigma^2_{\phi_1} & & & \\ \sigma_{\phi_0, \phi_2} & \sigma_{\phi_1, \phi_2} & \sigma^2_{\phi_2} & & \\ \sigma_{\phi_0, L} & \sigma_{\phi_1, L} & \sigma_{\phi_2, L} & \sigma^2_L & \\ \sigma_{\phi_0, \phi_1 * L} & \sigma_{\phi_1, \phi_1 * L} & \sigma_{\phi_2, \phi_1 * L} & \sigma_{L, \phi_1 * L} & \sigma^2_{L * \phi_1} \end{bmatrix}$$

The last row and column would not be included in  $C_S$  for BCS. The coefficient matrix for PE\* effects ( $C_{PE^*}$ ) will have the same general form as the coefficient matrix for sire effects. Because a sire model was used for this study,  $\frac{3}{4}$  of the genetic variance is associated with the PE variance estimate. Therefore, PE\* variance will refer to PE variance +  $\frac{3}{4}$  of the genetic variance, and PE variance to actual PE variance. The  $C_{PE^*}$  for BCS will not contain the third and last rows and columns, while  $C_{PE^*}$  for dairy form would only contain the first and fourth rows and columns.

The design matrix for random sire effects for DIM w and x in lactations y and z for dairy form is:

$$D_{wx,yz} = \begin{bmatrix} 1 & \phi_{1w} & \phi_{2w} & L_y & \phi_{1w} * L_y \\ 1 & \phi_{1x} & \phi_{2x} & L_z & \phi_{1x} * L_z \end{bmatrix}$$

The design matrix for BCS sire effects will not have the last column, while the design matrix for BCS PE effects will not have the third and last columns and the design matrix for dairy form PE effects will have only the first and fourth columns.

The sire variance/covariance matrix for DIM w and x in lactations y and z would then be:  $\sigma^2_{Sire,wx,yz} = D_{wx,yz} C_S D_{wx,yz}'$ . Likewise, the PE\* variance/covariance matrix for DIM w and x in lactations y and z would be:  $\sigma^2_{PE^*,wx,yz} = D_{wx,yz} C_{PE^*} D_{wx,yz}'$ .

To determine sire or PE variance for change in BCS or dairy form from day x to y, the design matrix coefficients for day y are subtracted from the design matrix coefficients for x. For example, to determine the sire variance/covariance among BCS on

DIM 0 and 70, and change in BCS from DIM 0 to 70 in first lactation, the following design matrix would be used.

$$D_{0,70} = \begin{bmatrix} 1 & -1.225 & 1.580 & 1 \\ 1 & -0.713 & 0.013 & 1 \\ 0 & -0.512 & 1.567 & 0 \end{bmatrix}$$

The variance/covariance matrix among BCS on day 0 and 70, and change in BCS from day 0 to 70 in first lactation is then derived as:  $D_{0,70} C_S D_{0,70}'$ .

Genetic variance on day x in lactation y ( $\sigma_{A,xy}^2$ ) was calculated as  $4 * \sigma_{Sire,xy}^2$ . The PE variance on day x in lactation y can be calculated as  $\sigma_{PE,xy}^2 = \sigma_{PE^*,xy}^2 - 3 * \sigma_{Sire,xy}^2$ .

The phenotypic variance was calculated as  $\sigma_{P,xy}^2 = \sigma_{A,xy}^2 + \sigma_{PE,xy}^2 + \sigma_{\epsilon,z}^2$ , where day x in lactation y was included in residual group z. Heritability ( $h_{xy}^2$ ) was  $\sigma_{A,xy}^2 / \sigma_{P,xy}^2$ . The phenotypic covariance between day w and x in lactation y and z was calculated as  $\sigma_{P,wx,yz} = \sigma_{A,wx,yz} + \sigma_{PE,wx,yz}$ . Phenotypic variance for the change in BCS or dairy form from day w to x in lactations y and z could then be calculated as:  $\sigma_{P,wx,yz}^2 = \sigma_{P,wy}^2 + \sigma_{P,xz}^2 - 2 * \sigma_{P,wx,yz}$ .

Generating variances with model 2 (RRA) is similar to model 3 (MDRR). Days in milk is not included in the model so the size of the coefficient and design matrices are smaller. Lactation number is replaced with age at classification and the procedures used to calculate variances and variance ratios for MDRR are used.

### **Predicted Transmitting Abilities (PTAs)**

Sire predicted transmitting abilities for BCS and dairy form from model 4 (MT) and model 1 (RPT) were obtained from ASREML output. Average PTAs for lactations 1, 2 and 3 were generated from model 3 (MDRR). Additionally, PTAs for 30 and 50 months of age at classification were generated from model 2 (RRA).

Correlations were generated among all of the PTAs. Low correlation estimates between PTAs from the RPT models and PTAs from random regression models could indicate ill-fitted random regression models or that RPT models are inadequate for certain DIM or ages. Correlations among specific lactation PTAs from MT and MDRR models



that were higher than correlations between MT and RPT PTAs would indicate that MDRR models are more accurate for specific lactations than RPT models. These correlations can also help determine at what age or lactation number national genetic evaluations are most accurate.

## **RESULTS AND DISCUSSION**

### **RPT Models**

Generalized least square solutions from RPT models for BCS and dairy form across LG1, LG2 and LG3 are shown in Figure 3. The solutions represent average BCS or dairy form on an average herd-classification visit for a cow that calved at an average age for each LG (26.7 in LG1, 40.5 for LG2 and 51.2 for LG3). Nadir BCS was attained at DIM 80, 62 and 68 for LG1, LG2 and LG3, respectively. Dairy form increased in early lactation, and maximum dairy form was attained at DIM 113 in LG1, DIM 70 in LG2 and DIM 73 in LG3.

In general, dairy form is increasing as BCS decreases. Dairy form peaks 33 days after minimum BCS in first lactation and the shape of the dairy form curve is different in first lactation than second or third lactation. Dairy form increases with lactation number more than average BCS declines. A different phenotypic relationship between dairy form and stage of lactation in first lactation compared to later lactations may have resulted in a significant interaction between DIM and lactation number with model 3 (MDRR). The heritability of BCS was estimated to be 0.20, whereas the repeatability estimate was 0.32. The heritability and repeatability estimates for dairy form were 0.26 and 0.61, respectively.

### **RRA Models**

Heritabilities and correlations were derived from model 2 (RRA) for BCS at 30 months of age ( $BCS_{30}$ ), 50 months of age ( $BCS_{50}$ ) and change in BCS from 50 - 30 months of age ( $BCH_{50-30}$ ). The heritability estimate changed minimally from 30 months (0.21) to 50 months (0.20). The genetic correlation between  $BCS_{30}$  and  $BCS_{50}$  was high (0.94), but the phenotypic correlation estimate was only 0.27.

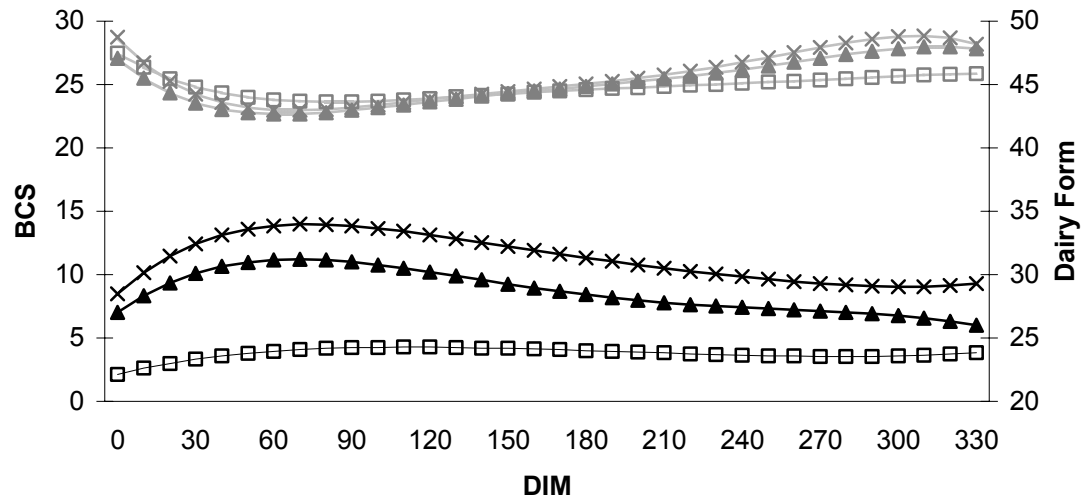


Figure 3. Generalized least squares solutions for body condition score (—) and dairy form (—) in lactation 1 (□), lactation 2 (▲) and lactations 3 and higher (×).

The heritability estimate for  $BCH_{50-30}$  was 0.03. When the genetic correlation between BCS at two DIM is high, then genetic variation for change between those two DIM must be low. The phenotypic correlation was not high, indicating that there is appreciable phenotypic variation for change in BCS.

Heritabilities and correlations were derived from the RRA model among dairy form at 30 ( $DF_{30}$ ), 50 ( $DF_{50}$ ) and 50 - 30 months of age ( $DCH_{50-30}$ ). The heritability estimate for  $DF_{30}$  (0.28) was similar to that of  $DF_{50}$  (0.27). As with BCS, the genetic correlation between  $DF_{30}$  and  $DF_{50}$  was 0.94, but the phenotypic correlation estimate was higher (0.56) for dairy form. The heritability estimate for  $DCH_{50-30}$  was 0.05.

Random regression models have been used to describe genetic changes in final score with age at classification (Tsuruta et al., 2002a). Tsuruta et al. (2002a) assumed a constant residual variance across ages and fitted quadratic polynomials for random effects. Random quadratic effects for age at calving would not converge for BCS or dairy form in this study. Average-Information REML was used here, while Tsuruta et al. (2002a) used REMLF90, which uses an EM-REML algorithm and can be more stable but takes longer to converge (Misztal et al., 2000). Convergence was not attained because of limited variation for a quadratic effect of sire on age and would change our results minimally.

Results from random regression on age at classification for stature, rump angle, thurl width, rear leg set, rear udder width, rear udder height, udder depth and fore udder attachment were reported by Uribe et al. (2000). In general, traits related to body structure (stature, rump angle and thurl width) were genetically similar traits across ages, while estimated breeding values for rear leg set and udder traits tended to change with age. Dairy form is related to body structure and appears to change minimally with age genetically, as does BCS.

### **MDRR Models**

Multidimensional random regression models (model 3) were successfully fit for both BCS and dairy form. The advantage of such a model over a multiple trait random regression model with each LG treated as a separate trait is that the number of parameters

to be estimated is greatly reduced (Jensen, 2001). A three-trait model with sire interacted with LP0, LP1 and LP2 and nested within LG would require estimation of 45 (co)variance parameters for the sire effect alone. The current model for BCS required estimation of 10 (co)variance parameters for sire, while the model for dairy form required 15 (co)variance parameters be estimated. Separate breeding values for each lactation can still be generated however.

**BCS.** The estimated heritability curves for BCS in lactations 1, 2 and 3 are shown in Figure 4. Heritability estimates for lactations 1 are from both HEV and HOV models. Heritability estimates for lactation 2 and 3 are from HEV models only.

Heritability estimates peak in mid lactation for all lactations. Heritability estimates are highest across first lactation and increase from 0.15 at DIM 0 to 0.24 at DIM 200. The curve of heritability estimates obtained from the HOV model is nearly identical to the curve obtained from the HEV model.

Variance component estimates for lactation 1 from HOV and HEV models are shown in Figure 5. The estimate of genetic variance is nearly identical for both models. Residual variance was estimated to be lower in first lactation and during early lactation for the HEV model. This appears to have resulted in a lower PE variance estimate across lactation 1, especially in early lactation, for the HOV model. Likewise, residual variance is estimated to be somewhat higher in late lactation with the HEV model, corresponding to a higher estimate of PE variance with the HOV model. The end result is a heritability estimate that is nearly identical for both models.

Cubic LP for DIM were used by both Jones et al. (1999) and Veerkamp et al. (2001) to model genetic variation for BCS across the lactation. Attempts to fit cubic LP did not converge in this study. The sire variance reported in this study displays a similar trend to the genetic variance estimated from a quadratic LP by Veerkamp et al. (2001). Genetic variance was reported to be highest in mid lactation and lower at the beginning and end of lactation. The estimate of genetic variance increased near the end of lactation when cubic LP were fit, but the cubic term explained only 0.016% of the genetic variation (Veerkamp et al., 2001).

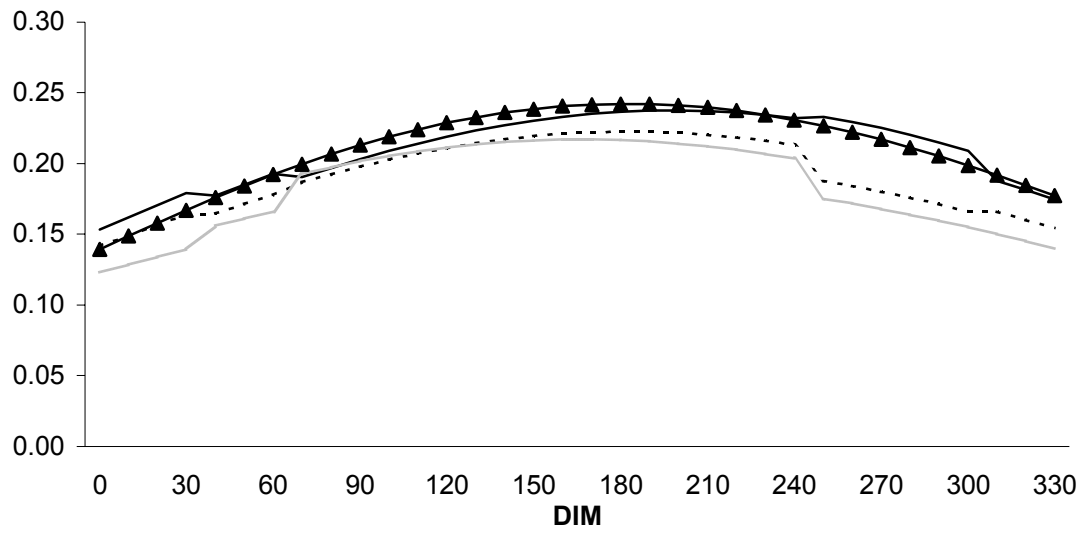


Figure 4. Heritability of body condition score in lactation 1 (—), lactation 2 (---), lactation 3 (—) assuming heterogeneous residual variance. Heritability of body condition score in lactation 1 (▲) assuming homogeneous residual variance.

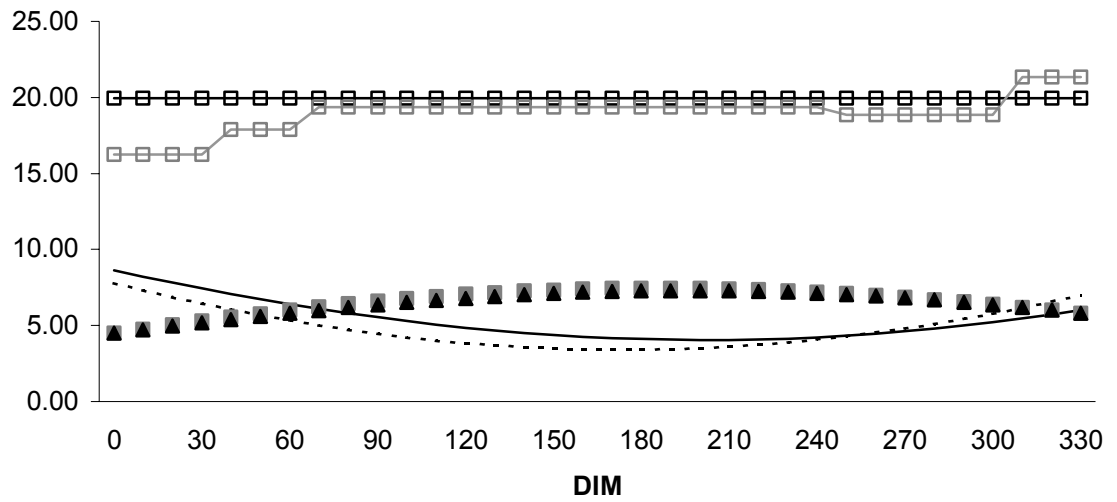


Figure 5. Genetic variation estimate (▲), permanent environmental variation estimate (—), and residual variation estimate (□) for body condition score in lactation 1 assuming heterogeneous residual variance. Genetic variation estimate (■), permanent environmental variation estimate (---), and residual variation estimate (□) for body condition score in lactation 1 assuming homogeneous residual variance.

Heritabilities, genetic and phenotypic correlation estimates among BCS at DIM 0, 70 and 305 in first, second, and third lactation are given in Table 10. Additionally, heritability and correlation estimates among BCS and change in BCS from DIM 0 - 70 ( $BCH_{0-70}$ ), 305 - 70 ( $BCH_{305-70}$ ) and change from DIM 0 in lactation 2 - DIM 305 in lactation 1 ( $BCH_{DP}$ ) are also given in Table 10. Correlations among the above traits and DIM 305 - 178 ( $BCH_{305-178}$ ) and DIM 178 - 70 ( $BCH_{178-70}$ ) were calculated but not shown.

These points were chosen to represent BCS at calving (DIM 0), nadir BCS (DIM 70) and BCS at the end of lactation (DIM 305). A high PTA for  $BCH_{0-70}$  indicates that daughters of that sire lose more body condition than average from DIM 0 to DIM 70, while a high PTA for  $BCH_{305-70}$  indicates that daughters of that sire gain more body condition than average from DIM 70 to DIM 305. A high value for  $BCH_{DP}$  indicates that daughters of that sire gain more body condition during the dry period than average. Genetic correlation estimates among BCS at DIM 0, 70 and 305 in lactation 1 through 3 range from 0.77 (between DIM 0 in lactation 1 and DIM 305 in lactation 3) to 0.99. Genetic correlation estimates within a lactation are 0.90 and above, whereas genetic correlation estimates at the same DIM in different lactations are 0.88 and above. Several authors have found high genetic correlations between BCS at different DIM and in different lactations using both random regression models and multiple trait models where BCS from different stages of lactation were considered different traits (Dechow et al., 2001; Gallo et al., 2001; Jones et al., 1999; Koenen et al., 2001).

Phenotypic correlation estimates are lower than the genetic correlation estimates, ranging from 0.09 to 0.41. It may be possible that PE covariances, and thus phenotypic correlations, were underestimated. Few cows would have BCS observations at or near two particular DIM and only LP1 could be fit for PE effects. Thus, the PE estimate for a particular cow on a given DIM is based on a straight line with only one to three observations available to estimate that line. If PE covariance is underestimated, the estimated residual variance for change in BCS would be overestimated, and heritability underestimated. The heritability of  $BCH_{0-70}$  and  $BCH_{305-70}$  were estimated to be 0.01, whereas the heritability estimate of  $BCH_{DP}$  was 0.03. Other estimates of the heritability

Table 10. Heritabilities (diagonal), genetic (below diagonal) and phenotypic (above diagonal) correlations estimates among body condition score (BCS) at days in milk 0, 70 and 305 in lactations 1, 2 and 3, loss in BCS from day 0 to 70 (0-70), gain in BCS from day 70 to 305 (305-70) and change during the first dry period (Dry, DIM 0 in lactation 2 – DIM 305 in lactation 1).

Lactation	DIM	1			2			3			BCS Change		
		0	70	305	0	70	305	0	70	305	0-70	305-70	Dry
1	0	0.15	0.41	0.31	0.34	0.32	0.20	0.21	0.21	0.09	0.02	-0.08	0.01
	70	0.99	0.19	0.32	0.33	0.31	0.23	0.22	0.23	0.14	0.01	-0.05	-0.01
	305	0.90	0.96	0.19	0.29	0.31	0.32	0.26	0.30	0.29	-0.02	0.04	-0.07
2	0	0.97	0.96	0.90	0.14	0.31	0.26	0.27	0.29	0.22	0.00	-0.02	0.00
	70	0.96	0.98	0.96	0.99	0.19	0.29	0.29	0.32	0.27	-0.01	0.00	-0.02
	305	0.85	0.92	0.98	0.90	0.96	0.17	0.30	0.36	0.39	-0.04	0.09	-0.07
3	0	0.88	0.88	0.84	0.97	0.95	0.89	0.12	0.35	0.32	-0.02	0.03	0.00
	70	0.89	0.91	0.91	0.96	0.98	0.96	0.98	0.19	0.40	-0.03	0.06	-0.03
	305	0.77	0.83	0.92	0.86	0.92	0.98	0.90	0.96	0.15	-0.05	0.13	-0.07
BCS Change	0-70	-0.60	-0.72	-0.88	-0.60	-0.73	-0.87	-0.57	-0.71	-0.84	0.01	-0.02	0.02
	305-70	-0.21	-0.06	0.22	-0.14	0.01	0.30	-0.06	0.08	0.36	-0.62	0.01	-0.05
	Dry	-0.29	-0.43	-0.63	-0.23	-0.39	-0.59	-0.16	-0.32	-0.52	0.89	-0.75	0.03



of BCS loss in the first 2 to 3 months of lactation range from 0.01 to 0.09 (Berry et al., 2002; Dechow et al, 2002; Pryce et al., 2001). While the heritability of BCS change could be underestimated here, it seems clear that the genetic correlation among BCS at different stages of lactation and across lactations are high, and that the heritability of BCS change is much less than the heritability of the level of BCS.

The heritability estimates of BCS change are low and genetic correlations among levels of BCS and BCS change should be interpreted with caution. Correlations among levels of BCS and BCS change do appear to be consistent with other reports, however. Genetic correlation estimates between  $BCH_{0-70}$  and the level of BCS range from  $-0.60$  to  $-0.87$  (Table 10). Genetic correlations are stronger (negative) between  $BCH_{0-70}$  and the level of BCS at DIM 70 and 305 than at DIM 0. Dechow et al. (2002) reported that BCS loss during the first third of lactation was genetically correlated more strongly with postpartum BCS (range  $-0.56$  to  $-0.99$ ) than with BCS at calving, (range  $-0.11$  to  $-0.48$ ). It appears that cows genetically inclined to have higher BCS at calving lose less body condition during the first months of lactation and have higher BCS later in lactation.

Early lactation cows are in negative energy balance and must mobilize body condition to support early lactation production (Bauman and Currie, 1980). There is variation on the severity and duration of negative energy balance. Cows genetically inclined to have higher levels of BCS appear to lose less BCS, and thus have less severe negative energy balance, in early lactation. Additionally, selection that increases negative energy balance in early lactation would do so by lowering BCS during the lactation more than BCS at calving.

The genetic correlation between  $BCH_{0-70}$  and  $BCH_{305-70}$  was  $-0.62$ . Cows genetically inclined to lose more body condition than average from DIM 0 to 70 appear to gain less BCS from DIM 70 to 305. However, the genetic correlation between  $BCH_{0-70}$  and BCS change from  $BCH_{305-178}$  is 0 and the genetic correlation estimate between  $BCH_{0-70}$  and  $BCH_{178-70}$  is  $-0.95$  (not shown). Body condition score gain from any DIM after 178 to 305 is positively correlated with  $BCH_{0-70}$ . Genetic correlation estimates between  $BCH_{DP}$  and BCS at DIM 0, 70 and 305 were negative, ranging from  $-0.16$  to  $-0.63$ , while the genetic correlation estimate between  $BCH_{DP}$  and  $BCH_{0-70}$  was 0.89.

This appears to agree with observations of Berry et al. (2002). They reported a genetic correlation between BCS at DIM 5 - 60 and BCS at DIM 180-120 of  $-0.26$  (the authors used DIM 60 – 5 so the signs have been reversed here). The genetic correlation between BCS at DIM 5 – 60 and DIM 240 – 180 was reported to be  $0.37$ .

Cows genetically inclined to have a high level of BCS at calving (DIM 0) appear inclined to lose less body condition from DIM 0 to DIM 70, and gain less BCS during the dry period. Cows genetically inclined to lose more BCS from DIM 0 to 70 appear to gain less body condition through mid lactation and then gain more body condition than average in late lactation and the dry period.

The relationship between the level of BCS and changes in BCS may be due, in part, to their relationship with milk production. While slightly positive genetic correlation estimates between BCS at calving and production have been reported, genetic correlation estimates for BCS during the lactation and milk, fat or protein production are moderately negative (Berry et al., 2002; Dechow et al., 2001; Gallo et al., 2001; Veerkamp et al., 2001). Genetic correlation estimates between BCS loss in early lactation and total lactation milk production range from  $0.09$  to  $0.50$  (Dechow et al., 2002; Pryce et al., 2001).

Genetic correlation estimates between BCS and production are not constant across the lactation, however. When BCS at different stages of lactation are treated as separate traits, genetic correlation estimates between production and BCS were reported range from  $0.22$  to  $-0.27$  at or near calving,  $-0.06$  to  $-0.43$  in mid lactation, and from  $0.07$  to  $-0.31$  in late lactation (Dechow et al., 2001). Veerkamp et al. (2001) reported that genetic correlations between BCS and production became stronger (negative) as the lactation progressed using random regression models.

Cows genetically inclined to produce higher levels of milk tend to have lower levels of BCS, lose more BCS in early lactation and have more severe negative energy balance in early lactation. Higher producing cows likely partition more nutrients toward production and less toward replenishing body condition during mid to late lactation, and then recover body condition at a more rapid rate in late lactation and the dry period.

**Dairy form.** The estimated heritability curve of dairy form across lactations one, two and three is given in Figure 6. From the HEV model, heritability estimates for lactation 1 are highest at the beginning of lactation (0.31), but early lactation heritability estimates are lowest for lactations 2 and 3. The heritability estimate for first lactation is lowest at the beginning of the lactation if homogeneous residual variance is assumed. The heritability estimate for second lactation peaks at DIM 240 (0.30). In lactation 3, heritability increased across the lactation and was highest (0.36) at DIM 240 and DIM 335.

The estimated variance components for lactation 1 from HEV and HOV models are given in Figure 7. Genetic variance estimates from both models are nearly identical. As with BCS, residual variance in first lactation was lower than average residual variance in second and third lactations, resulting in a lower PE variance when residual variance was held constant. Because models that included PE interacted with LP did not converge, PE variance estimates did not compensate for lower early lactation residual variance, resulting in lower heritability estimates for dairy form with the HOV model at DIM 0 for lactations 1 (7% lower), 2 (3% lower) and 3 (4% lower).

Residual variance was particularly low in the first month of lactation 1, resulting in a high heritability estimate in the first month of lactation 1 with the HEV model. This is likely an effect of type appraisal procedures for early first lactation cows. Classifiers have the option of not classifying early lactation cows if they feel that a cow has not had time to recover from the stress of calving and is not in proper condition. Cows that are scored in that first month are therefore a select group. Early first lactation cows that are in condition to be classified and that producers would like to have scored are likely to be above average for type, which could bias variance estimates in early lactation. The average final score of first lactation cows evaluated during the first month of lactation was 79.4 in this dataset, whereas the average final score of cows evaluated after the first month of first lactation was 76.5.

Many random regression models have displayed a rapid increase in heritability estimates at the ends of the measured time scale, which is usually DIM (Misztal et al., 2000). This increase is likely an artifact of the random regression model. Stages of

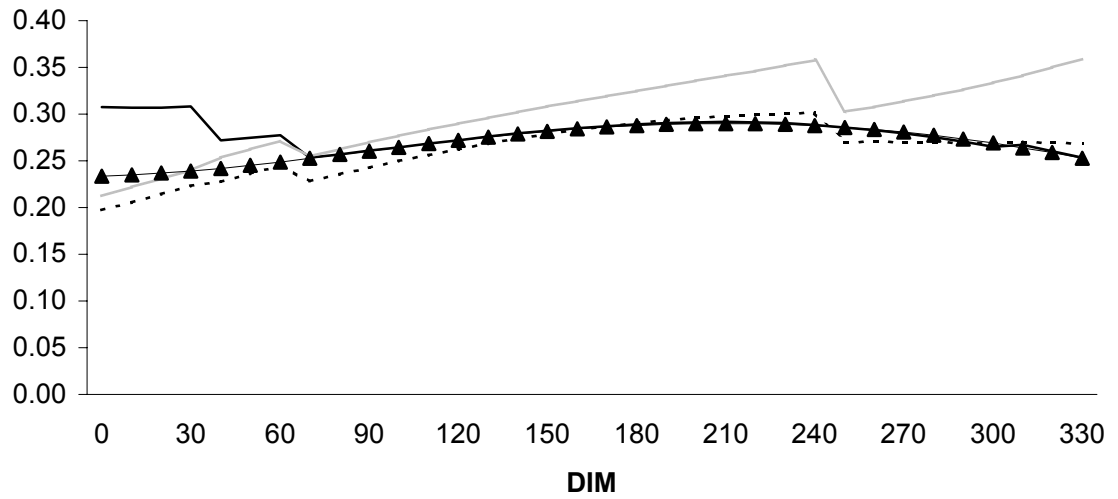


Figure 6. Heritability of dairy form in lactation 1 (—), lactation 2 (---), lactation 3 (—) assuming heterogeneous residual variance. Heritability of dairy form in lactation 1 (—▲) assuming homogeneous residual variance.

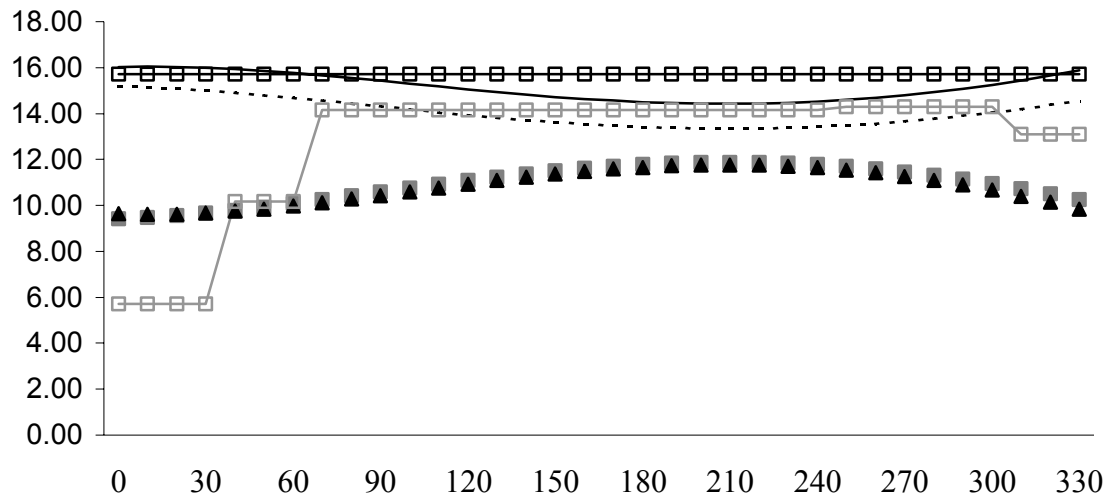


Figure 7. Genetic variation estimate (▲), permanent environmental variation estimate (—), and residual variation estimate (◻) for dairy form in lactation 1 assuming heterogeneous residual variance. Genetic variation estimate (■), permanent environmental variation estimate (---), and residual variation estimate (◻) for body condition score in lactation 1 assuming homogeneous residual variance.

lactation where observations are abundant may be modeled well, while the beginning and end of lactations, which typically contain fewer observations, are modeled poorly (Misztal et al., 2000). The increasing heritability estimate across third lactation is likely an artifact of the model used here. Lactation 3 in late lactation is near the end of the observation for two time scales (lactation number and DIM), and there were only 266 observations in month 11 for lactations 3 and higher.

Genetic correlation estimates among dairy form scores at DIM 0, 70 and 305 and in lactations 1 through 3 (not shown) ranged from 0.60 to 0.98. The heritability estimates of change in dairy form scores in lactation 1 from DIM 0-70 ( $DCH_{0-70}$ ) was 0.05, as was the heritability estimate for change in dairy form from DIM 305-70.

Because an interaction between DIM and lactation effects was fitted, change in dairy form in one lactation was not necessarily the same in other lactations. The genetic correlation estimate for  $DCH_{0-70}$  among lactations 1, 2 and 3 ranged from 0.43 (between lactations 1 and 3) to 0.88 (between lactations 2 and 3).

The genetic correlation estimates between the level of dairy form and  $DCH_{0-70}$  in the corresponding lactation was 0.08 at DIM 0 in lactation 1 and negative elsewhere (range  $-0.23$  to  $-0.90$ ). A negative value for  $DCH_{0-70}$  represents an increase in dairy form from DIM 0 to 70. It appears that cows with a high level of dairy form tended to increase in dairy form in early lactation, especially later lactations. Genetic correlations estimates tended to be strongest between  $DCH_{0-70}$  and DIM 305 (range  $-0.55$  to  $-0.90$ ).

### **MT and MDRR Models**

Genetic parameter estimates obtained from MDRR (model 3) for lactations 1, 2 and 3 were similar to those obtained from MT (model 4). Genetic correlation estimates obtained from MT models among BCS in LG 1, LG 2 and LG 3 ranged from 0.94 to 0.98, whereas genetic correlation estimates obtained from MDRR models among first, second and third lactations ranged from 0.92 to 0.98. Standard errors of the genetic correlations for the MT model ranged from 0.02 to 0.03. The heritability estimates from the MT model ranged from 0.20 (lactations 2 and 3) to 0.22 (lactation 1), while

heritability estimates for the MDRR model ranged from 0.18 (lactation 3) to 0.21 (lactation 1).

Genetic correlation estimates ranged from 0.96 to 1.01 among LG 1, LG 2 and LG3 for dairy form for the MT model. Standard errors for the genetic correlations ranged from 0.01 to 0.02. The two trait model with dairy form in LG2 and LG3 failed to remain positive definite. If the genetic correlation between two traits is near 1, a genetic correlation greater than 1 may be within the sampling error. Rather than bending covariance matrices to remain positive definite, the covariance estimate is left as sampled by ASREML.

Genetic correlation estimates ranged from 0.92 to 0.98 for lactations 1, 2 and 3 from the MDRR model. Heritability estimates ranged from 0.24 (lactation 2) to 0.28 (lactation 1) with the MT model, while they ranged from 0.26 (lactation 2) to 0.28 (lactation 3) with the MDRR model.

### **BCS and Dairy Form**

Correlation estimates among BCS and dairy form in different lactation periods are given in Table 11. Phenotypic correlations are moderate, ranging from  $-0.38$  to  $-0.46$ . It is possible to have an open ribbed cow that has a high level of BCS, or a tight ribbed cow with low BCS at the phenotypic level. Genetic correlation estimates are stronger (range  $-0.61$  to  $-0.72$ ) than the phenotypic correlation estimates. Bulls that sire daughters high in dairy form also tend to sire daughters that have lower BCS than average. Genetic parameters for BCS and dairy form change in a similar manner across the lactation as well. Genetic variance estimates (Figures 2 and 4) tend to be lowest in early lactation, highest in the middle of lactation, and decline toward late lactation. The genetic correlation estimates between BCS and dairy form tend to be strongest when the genetic variances for both traits are the highest.

While BCS and dairy form are not the same traits phenotypically, both contribute to angularity and have a moderate to strong genetic relationship. A cow with high angularity is open ribbed (or has a high dairy form), is free of excess fleshing, and has a flat, clean bone structure (Interbull, 2003b). The genetic correlation between dairy form

Table 11. Genetic correlation ( $r_g$ ) estimates and phenotypic correlation ( $r_p$ ) estimates between body condition score and dairy form in the following months of lactation: 1 through 2 (P1), 3 through 4 (P2), 5 through 6 (P3), 7 through 8 (P4) and 9 through 11 (P5).<sup>1</sup>

Lactation Stage	$r_g$	$r_p$
P1	-0.63	-0.38
P2	-0.67	-0.42
P3	-0.72	-0.45
P4	-0.69	-0.46
P5	-0.61	-0.45

<sup>1</sup>Standard errors for the genetic correlations ranged from 0.04 to 0.08 and standard errors for the phenotypic correlations were 0.01.



in the US and angularity in the UK is 0.89 (Interbull, 2003a). Correlations between BCS and angularity were reported to range from  $-0.47$  to  $-0.77$  in a research herd from the UK (Veerkamp and Brotherstone, 1997).

Bone quality is measured on Canadian Holsteins and contributes positively to dairy character scores (Holstein Canada, 2003). The genetic correlation between dairy character in Canada and dairy form in the US is reported to be 0.86 (Interbull, 2003a). A genetic correlation estimate between BCS and bone quality was reported to be  $-0.44$  in a pilot study in Canada (van Dorp and Boettcher, 1999). Cows with low BCS likely have less tissue surrounding the cannon bone, thus appearing to have a more refined or “flatter” bone structure in their rear legs.

Bulls that sire daughters high in dairy form tend to have daughters that are open ribbed, low in BCS and flat boned, thus appearing to be more angular. There are some differences between BCS and dairy form genetically. Body condition score was reported to be strongly correlated with strength (0.69) while dairy form was not ( $-0.11$ ) (Dechow et al., accepted). Despite the negative genetic correlation between them, both BCS and dairy form were reported to be positively correlated with stature and frame in the same study (range 0.20 to 0.27).

The increase in dairy form with lactation number (Figure 3) could be due to an increase in size as cows mature. As a cow grows and become longer, space between ribs may increase, resulting in a higher dairy form. Growth in stature and frame during first lactation could also be responsible for the general increase in dairy form across first lactation that was not observed across other lactations. Increased size with maturity does not appear to have a large impact on BCS however.

### **Predicted Transmitting Abilities**

Correlations among PTAs for BCS and dairy form in different lactations and from various models are given in Table 12. Correlations among PTAs from all models were high, indicating that all models performed consistently. Correlations among PTAs from the RPT model and all other models were 0.983 and higher for BCS, and 0.95 and higher for dairy form.

Table 12. Correlations among sire predicted transmitting abilities from repeatability (RPT) models, PTAs from lactations 1 (MT1), 2 (MT2) and 3 and higher (MT3) from multiple trait models, lactation 1 (RR1), lactation 2 (RR2) and lactation 3 (RR3) from random regression models on days in milk and lactation number, and 30 months (RR30), and 50 months (RR50) from random regression models on age at classification for body condition score (below diagonal) and dairy form (above diagonal).

	RPT	MT1	MT2	MT3	RR1	RR2	RR3	RR30	RR50
RPT		0.978	0.979	0.950	0.996	0.997	0.986	0.994	0.994
MT1	0.984		0.994	0.947	0.984	0.975	0.955	0.984	0.969
MT2	0.983	0.988		0.944	0.979	0.981	0.970	0.977	0.978
MT3	0.988	0.987	0.998		0.948	0.950	0.940	0.950	0.946
RR1	0.997	0.989	0.979	0.976		0.993	0.975	0.998	0.987
RR2	0.998	0.980	0.985	0.984	0.995		0.994	0.989	0.998
RR3	0.988	0.963	0.982	0.982	0.980	0.995		0.968	0.995
RR30	0.995	0.987	0.975	0.973	0.997	0.991	0.974		0.983
RR50	0.994	0.975	0.986	0.984	0.989	0.997	0.996	0.983	

In first and second lactation, PTAs from MT models are more highly correlated with PTAs generated with MDRR models for the corresponding lactation than with PTAs generated with RPT models. This may indicate that MDRR PTAs are slightly more accurate for lactations 1 and 2 than the RPT models. That was not true for third lactation, and MDRR models may not have fit third lactation as well as lactations 1 and 2.

Predicted transmitting abilities at 30 months of age from RRA models were more highly correlated with PTAs for first lactation from MT and MDRR models than second or third lactations. Predicted transmitting abilities at 50 months of age from RRA models were most highly correlated with second lactation PTAs from MT and MDRR models. It would be expected that PTAs for younger ages (30 months) be closer to PTAs from first lactation, while PTAs for older ages (50 months) would be closer to PTAs from lactations 2 and 3 if the RRA model fit well.

Predicted transmitting abilities between DIM 0, 70 and 305 in lactations 1, 2 and 3 from MDRR models were also generated and correlated with PTAs from RPT models (not shown). Correlations ranged from 0.977 to 0.997 for BCS and from 0.948 to 0.996 for dairy form. The RPT models also appear to be accurate for any DIM for both BCS and dairy form.

## **CONCLUSIONS**

Previously reported relationships among BCS, dairy form, reproductive performance and cow health indicate that selection for higher BCS or lower dairy form may improve reproductive performance and cow health. National genetic evaluations for BCS are not currently available, but could be generated. Body condition score has a strong genetic correlation with dairy form and BCS evaluations would only be necessary if selection for BCS is shown to improve cow health or reproductive performance beyond what is possible with selection for dairy form.

Selection in the US has been for cows with higher dairy form, and thus lower BCS, because dairy form is favorably correlated with yield. When analyzing the merit of cows in the absence of production records, at cattle shows or during linear classification for example, some preference to those cows with higher dairy form may be justified.

However, reported correlations between dairy form and production are moderate and preference for thin cows that otherwise appear equal in production to other cows should be discouraged. Preference for higher dairy form as an indicator of production is not necessary when reliable production records are available and genetic selection should be for those bulls that sire daughters high in production but low in dairy form.

Random regression models for BCS or dairy form could be used to generate PTAs for an age or DIM when heritability was highest, or be used to generate PTAs for change in BCS or dairy form. Evaluations from random regression models may be of value if a strong association were found between change in BCS or dairy form and an economically important trait, like reproductive performance. If correlations of BCS or dairy form with an important trait changed across the lactation, then evaluations from random regression models for DIM when the relationship is strongest might be of value. However, genetic correlations between BCS or dairy form at different DIM, lactations and ages are high and change in BCS and dairy form is not as heritable as the level of BCS or dairy form. Large daughter groups would be necessary to estimate accurate evaluations for change in BCS or dairy form.

The repeatability models used currently in national genetic evaluation programs in the US would appear to generate accurate PTAs for BCS or dairy form at any age or stage of lactation.

## Chapter 5

### **Correlations Among Body Condition Score from Various Sources, Dairy Form, and Cow Health from the US and Denmark**

This chapter is a slightly modified version of a paper by the same name in preparation for the Journal of Dairy Science by C. D. Dechow, G. W. Rogers, U. Sander-Nielsen, L. Klei, T. J. Lawlor, J. S. Clay, A. E. Freeman, G. Abdel-Azim, A. Kuck, and S. Schnell.

My contributions to this paper include: All data analysis, gathering and interpretation of literature and all writing of the manuscript except for editing of other authors and reviewers.

#### **ABSTRACT**

The objectives of this study were to estimate genetic correlations among body condition scores (BCS) from various sources, dairy form and measures of cow health. Body condition score and dairy form evaluated during routine type appraisal was obtained from the Holstein Association USA, Inc. A second set of BCS was obtained from Dairy Records Managements Systems (DRMS) and was recorded by producers that use PCDART dairy management software. Displaced abomasum (DA), metabolic disease, foot disease and mastitis observations were obtained from recorded veterinarian treatments in several dairy herds. Breeding values for metabolic and digestive diseases, foot and leg diseases and reproductive diseases in Denmark were also obtained. Genetic and phenotypic correlations among BCS, dairy form and cow health traits in the US were generated with sire models using ASREML. Models included fixed effects for age at calving, days in milk and contemporary group. Random effects included sire and error. Predicted transmitting abilities (PTA) for BCS and dairy form were correlated with breeding values for disease traits in Denmark. The genetic correlation estimate between BCS from DRMS and BCS from the Holstein Association USA, Inc. was 0.87. Higher BCS was significantly correlated with lower incidence of metabolic disease (-0.78) and DA (-0.72) in the US and with lower metabolic and digestive disease in Denmark (-0.25 to -0.36). Dairy form was genetically correlated with more metabolic disease (0.71) and DA (0.52) in the US, more metabolic and digestive disease (0.15 to 0.40) and more foot

and leg disease (0.43 to 0.45) in Denmark. Adjustment for protein yield PTA had a minimal effect on correlations between PTA for BCS or dairy form and disease in Denmark. Selection for higher BCS or lower dairy form with continued selection for yield may slow deterioration in cow health as a correlated response to selection for increased yield.

**(Key Words:** body condition score, dairy form, disease)

**Abbreviation Key:** **BCS** = body condition score, **BCS<sub>DRMS</sub>** = body condition scores from Dairy Records Management Systems, **BCS<sub>HOL</sub>** = body condition scores from Holstein Association USA, Inc., **DA** = Displaced abomasum, **DRMS** = Dairy Records Management Systems, **FLD** = foot and leg diseases, **MDD** = metabolic and digestive diseases, **PTAB** = predicted transmitting ability for body condition score, **PTAD** = predicted transmitting ability for dairy form, **PTAP** = predicted transmitting ability for protein yield.

## INTRODUCTION

Selection for increased milk, fat and protein yield has been successful, but is unfavorably correlated with incidences of metritis, ketosis, milk fever, cystic ovaries, lameness, mastitis and other diseases (Kadarmideen et al., 2000; Pösö et al., 1996; Tveit et al., 1992; Van Dorp et al., 1998). There also appears to be less culling on the basis of low production and more involuntary culling compared to a decade ago, which slows potential genetic progress for yield (Weigel et al., 2003). Much of this shift toward higher levels of involuntary culling is likely due to poorer cow health and reproductive performance. Failure to conceive, milk fever, displaced abomasum, ketosis and mastitis were all shown to increase the risk of culling even when the effect of disease on milk yield was considered (Gröhn et al., 1998).

Negative energy balance in early lactation is associated with increased levels of ketosis, fatty liver, displaced abomasums and other metabolic disorders (Baird, 1982; Goff and Horst, 1997). Increased negative energy balance is also associated with increased incidence of laminitis and locomotive problems (Collard et al., 2000).

Cows with high genetic merit for yield tend to have lower BCS and higher dairy form scores (Dechow et al., 2001; Short and Lawlor, 1992). Selection for higher yield is

also correlated with increased BCS loss and negative energy balance in early lactation (Berry et al., 2002; Dechow et al., 2002, Harrison et al., 1990).

Bulls that sire daughters with high dairy form in the US were reported to have daughters that were more susceptible to metabolic, reproductive and foot and leg diseases in Scandinavia (Rogers et al., 1999). Cows with higher dairy character are also reported to have more disease in Denmark (Hansen et al., 2002). The relationship between dairy form and cow health may be due to differences in BCS. The genetic correlation between dairy form and BCS in the US is reported to be -0.72 (Dechow et al., 2003). Selection for higher BCS or lower dairy form may help to increase stores of energy and decrease early lactation negative energy balance, which could improve levels of cow health.

While the phenotypic relationship between BCS and cow health has been reported in the US, the genetic relationship between BCS or dairy form and cow health in the US has not been reported.

Studies that have reported the phenotypic relationship between cow health and BCS have used BCS recorded by producers or researchers. Genetic evaluations for BCS in the US would likely come from a large national recording scheme and the genetic relationship between BCS recorded by producers and BCS recorded as part of a national recording scheme has not been reported.

The first objective of this study was to investigate the genetic relationship among BCS, dairy form and cow health measured in the US and in Denmark. The second objective was to investigate the genetic relationship between BCS that had been recorded by producers or herd consultants and BCS recorded in a large national recording scheme during linear type appraisals.

## **MATERIALS AND METHODS**

Body condition score ( $BCS_{HOL}$ ) and dairy form recorded from October of 1997 through June of 2000 were obtained from the Holstein Association USA, Inc. Body condition scores were recorded on a scale of 1 (thin) to 50 (fat) to be consistent with the scale used for linear type traits. A minimum of 20 daughters per sire and 10 cows per herd-classification visit were required and records from cows evaluated after 335 DIM,

before 24 months of age or after 60 months of age were eliminated. Classifiers that had scored BCS abnormally were eliminated using the same procedures as Dechow et al. (2003). Only the first BCS<sub>HOL</sub> record available for a cow was retained to eliminate the need for permanent environmental effects for BCS<sub>HOL</sub>. The edited data set contained records from 183,044 cows.

A second set of BCS (BCS<sub>DRMS</sub>) was obtained from Dairy Records Management Systems (DRMS) in Raleigh, NC and was recorded on a scale of 1 (thin) to 5 (fat). Body condition scores were recorded from 1990 through September of 1998. Producers and herd-consultants recorded BCS<sub>DRMS</sub> observations. Body condition scores from DRMS were recorded in one of six scoring periods: at calving, postpartum, first service, pregnancy check, before dry-off and at dry-off. Days in milk when BCS<sub>DRMS</sub> was recorded was not available. Body condition scores from each scoring period were considered a separate trait and heritabilities and correlations among BCS<sub>DRMS</sub> at all six scoring periods were reported by Dechow et al. (2001). In this study, BCS<sub>DRMS</sub> from all scoring periods were considered the same trait. Body condition scores were retained from cows that had calved between 20 and 60 months of age. A minimum of 20 daughters per sire and 10 cows per contemporary group were required. The BCS<sub>DRMS</sub> data set contained 86,854 records from 26,498 cows that were sired by 614 bulls.

Cow health data from the US was obtained from a Genex Cooperative Inc. progeny test study. Disease treatments were recorded from August of 1994 through 1999 in herds with one or more daughters of 54 progeny test bulls that had been measured for immune function (Nash et al., 2000). Observations for the following diseases were retained: displaced abomasum (DA), metabolic disease (DA, acidosis, bloat, caecal torsion, diarrhea, fatty liver, ketosis and milk fever), foot diseases (abscesses, foot rot and laminitis) and mastitis. Data for reproductive diseases (retained placenta, uterine infection and cystic ovaries) was available, but genetic variation for these diseases was near 0 and convergence was not attained for analyses including reproductive disease. Cows that had calved between 20 and 60 months of age were retained. Cows that had calved in the same herd-year-season as one or more cows with a disease record were assumed to be disease free and a minimum of 5 cows per herd-year-season was required. Only daughters of



sires with at least one daughter with a disease observation and with at least 20 daughters with  $BCS_{HOL}$  were retained. Only the first lactation available for each cow was kept. The edited US health data set contained records on 5872 cows from 408 sires.

Breeding values for metabolic and digestive diseases (MDD), reproductive diseases and foot and leg diseases (FLD) in first, second and third lactations in Denmark for 99 bulls that also have daughters with  $BCS_{HOL}$  and dairy form observations in the US were obtained from the Danish Agricultural Advisory Center (Aarhus, Denmark). A description of the diseases and procedures used to generate the breeding values are described in Principles of Danish Cattle Breeding (Danish Cattle Federation, 2002).

Body condition scores from the Holstein Association USA, Inc. were merged with  $BCS_{DRMS}$  and US health data. There was minimal overlap between the data sets. A total of 159 cows had both  $BCS_{HOL}$  and US health data and 62 cows had both  $BCS_{HOL}$  and  $BCS_{DRMS}$ . However, all cows with US health observations had a minimum of 20 paternal-half siblings with  $BCS_{HOL}$  observations and 304 sires had daughters with  $BCS_{HOL}$  and  $BCS_{DRMS}$ .

Predicted transmitting abilities for  $BCS_{HOL}$  (PTAB) and dairy form (PTAD) were generated in ASREML with the Holstein data. Sire PTAs for PTAB and PTAD were merged with breeding values for disease in Denmark. Only sires with a minimum reliability of 0.65 for  $BCS_{HOL}$  and 0.33 for disease were used. A total of 71 sires in first lactation, 68 sires in second lactation and 56 sires in third lactation met minimum reliability requirements. Official sire evaluations for protein yield (PTAP) in the US (from AIPL-USDA, Beltsville, MD) were also attained for all sires to adjust correlations for yield.

Correlations among  $BCS_{HOL}$ , dairy form,  $BCS_{DRMS}$  and US health were generated with sire models using ASREML (Gilmour et al., 2002). The general statistical model used for the analysis is as follows:

$$\mathbf{y} = \mathbf{b}_1 * \text{age}(\text{lact}) + \mathbf{cg} + \mathbf{sire} + \boldsymbol{\varepsilon}.$$

$\mathbf{y}$  is a vector of  $BCS_{HOL}$  or dairy form and either  $BCS_{DRMS}$  or a US health trait,  $\mathbf{b}_1$  is a vector of regression coefficients for age at calving in months nested with lactation number (lact),  $\mathbf{cg}$  is a vector of fixed effects for contemporary group,  $\mathbf{sire}$  is a vector of

random sire effects, and  $\epsilon$  is random error. Contemporary groups were herd-classification visit for  $BCS_{HOL}$  and dairy form and herd-year-season of calving for US health.

Contemporaries for  $BCS_{DRMS}$  were cows that had calved in the same herd, year and season and that had  $BCS_{DRMS}$  recorded in the same scoring period and lactation. Seasons of calving for US health and  $BCS_{DRMS}$  were defined as January through April, May through August, and September through December. Three generations of sire and dam were traced for all sires. Additional covariates for  $BCS_{HOL}$  and dairy form were fifth order polynomials of DIM nested within lactation  $i$ . Correlations between  $BCS_{HOL}$  and cow health were estimated with and without dairy form as a covariate and correlations between dairy form and cow health were estimated with and without  $BCS_{HOL}$  as a covariate. A permanent environment effect was included for analysis of  $BCS_{DRMS}$ .

Breeding values for disease in Denmark, PTAB and PTAD from the US were correlated. Correlations between PTAB, PTAD and the health traits in Denmark were divided by the square root of the product of the average reliabilities of the two traits to approximate genetic correlations. Rogers et al. (1999) used this method previously to estimate genetic correlations between linear type traits from the US and disease in Denmark and Sweden. Correlations between breeding values among traits generated from daughter records in different countries would be expected to result from genetic ties between the two populations only and should not be biased by shared environments among the daughter groups. Adjusting correlations between breeding values derived from separate populations for reliability then approximates genetic correlations (Calo et al., 1973).

Partial correlation estimates were generated between breeding values for disease in Denmark and PTAD after adjusting for PTAB. Likewise, partial correlation estimates were generated between breeding values for disease in Denmark and PTAB after adjusting for PTAD. Adjustment for PTAD was made by regressing breeding values for disease and PTAB on PTAD and calculating correlations among the residuals. The same procedure was used to adjust for PTAB and PTAP. Partial correlations were not adjusted for reliability.

## RESULTS AND DISCUSSION

### Body Condition Score

Heritabilities and correlations among  $BCS_{HOL}$ ,  $BCS_{DRMS}$  and dairy form are given in Table 13. The heritability estimate of  $BCS_{DRMS}$  was 0.14. Dechow et al. (2001) reported heritabilities ranging from 0.07 to 0.20 when BCS from this data set were considered a separate trait for each scoring period in lactations one through three. The heritability estimate for  $BCS_{HOL}$  was 0.21 and for dairy form was 0.24, which are nearly identical to heritability estimates reported by Dechow et al. (2003) using this data set with different edits for DIM.

The genetic correlation between  $BCS_{HOL}$  and  $BCS_{DRMS}$  was estimated to be 0.87. The phenotypic correlation estimate between  $BCS_{HOL}$  and  $BCS_{DRMS}$  was 0.44, but only 62 cows had both  $BCS_{HOL}$  and  $BCS_{DRMS}$ . Genetic correlations between dairy form and  $BCS_{HOL}$  and  $BCS_{DRMS}$  were -0.72 and -0.75, respectively. As expected,  $BCS_{HOL}$  and  $BCS_{DRMS}$  appear to be very similar traits genetically even though observations for  $BCS_{HOL}$  and  $BCS_{DRMS}$  are recorded on a different scale and come from different evaluation systems. Cows that have high BCS measured on various scales are also genetically inclined to have lower dairy form.

### US Health Data

Disease frequencies and the average heritability across all analyses for disease traits are given in Table 14. Disease frequencies ranged from 1.4% for foot diseases to 8.7% for mastitis. Average heritability estimates for disease traits ranged from 0.018 for mastitis to 0.052 for displaced abomasum. Standard errors for those heritability estimates ranged from 0.01 to 0.03.

Disease frequencies were lower than many published estimates of the frequency of cow diseases and likely underestimate the frequency of cow diseases (Collard et al., 2000; Gröhn et al., 1998; Lin et al., 1989). Because disease was recorded when a veterinarian treatment was required, many diseased cows not requiring treatment or cows treated by herd owners or managers likely went unreported. Additionally, an assumption was made that all cows without a recorded disease treatment that had calved in the same herd-year-season as a diseased cow were disease free. This assumption may not have

Table 13. Heritabilities (diagonal), genetic (above diagonal) and phenotypic (below diagonal) correlations among BCS from the Holstein Association USA, Inc. ( $BCS_{HOL}$ ), BCS from DRMS ( $BCS_{DRMS}$ ) and dairy form.<sup>1</sup>

	$BCS_{HOL}$	Dairy Form	$BCS_{DRMS}$
$BCS_{HOL}$	0.21	-0.72	0.87
Dairy Form	-0.45	0.24	-0.75
$BCS_{DRMS}$	0.44	-0.20	0.14

<sup>1</sup>Standard errors of the heritability estimates were 0.01. Standard errors of the genetic correlation estimates range from 0.02 to 0.04 and standard errors of the phenotypic correlation estimates range from 0.002 to 0.13.

Table 14. Frequency and average heritability of displaced abomasum (DA), metabolic disease, foot disease and mastitis from selected US dairy herds.<sup>1</sup>

Disease	Frequency (%)	Heritability
DA	1.9	0.052
Metabolic	3.0	0.042
Foot	1.4	0.022
Mastitis	8.7	0.018

<sup>1</sup>Standard errors of the heritability estimates range from 0.01 to 0.03.

been true for some cows, but was necessary to generate a group of non-diseased contemporaries. Displaced abomasum was the only metabolic disease recorded at a frequency greater than one percent, so was the only metabolic disease analyzed independently.

Genetic and phenotypic correlations between  $BCS_{HOL}$ , dairy form and the disease traits are given in Table 15. Standard errors for the genetic correlations are large, ranging from 0.18 to 0.66. Genetic correlation estimates among  $BCS_{HOL}$ , dairy form and DA and among  $BCS_{HOL}$ , dairy form and metabolic disease were more than twice the standard error of the genetic correlation estimates. The genetic correlation estimate between  $BCS_{HOL}$  and DA was -0.72, whereas the genetic correlation estimate between dairy form and DA was 0.52.

Phenotypic correlations between US health traits and  $BCS_{HOL}$  or dairy form were based on 159 observations. Phenotypic correlation estimates between DA and  $BCS_{HOL}$  and between DA and dairy form were -0.10 and 0.01, respectively. The genetic and phenotypic correlation estimates between metabolic disease and  $BCS_{HOL}$  were -0.78 and -0.09, respectively. The genetic correlation estimate between dairy form and metabolic disease was 0.70 and the phenotypic correlation estimate was -0.03.

Two trait sire models between health traits and  $BCS_{HOL}$  with dairy form included as a covariate for both traits were used to investigate the relationship between  $BCS_{HOL}$  and cow health after adjustment for dairy form. Likewise, correlations between dairy form and cow health were generated with two-trait sire models between health traits and dairy form with  $BCS_{HOL}$  included as a covariate. When dairy form was included in the model, the genetic correlation estimate between  $BCS_{HOL}$  and DA was -0.45, whereas the genetic correlation between  $BCS_{HOL}$  and metabolic disease was -0.49. The genetic correlation estimate between dairy form and DA was 0.50 and the genetic correlation estimate between dairy form and metabolic disease was 0.60 when  $BCS_{HOL}$  was included as a covariate.

The model including  $BCS_{HOL}$  and foot diseases failed to remain positive definite. The genetic correlation estimate between  $BCS_{HOL}$  and mastitis was 0.14. The genetic

Table 15. Correlations between body condition score ( $BCS_{HOL}$ ), dairy form and displaced abomasum (DA), metabolic disease, foot disease and mastitis.<sup>1</sup>

	$BCS_{HOL}$		Dairy Form	
	Gen	Phen	Gen	Phen
DA	-0.72*	-0.10*	0.52*	0.01
Metabolic	-0.78*	-0.09*	0.71*	-0.03
Foot	... <sup>4</sup>	... <sup>4</sup>	0.21	-0.13*
Mastitis	0.14	-0.08	-0.03	0.08
	Adjusted for Dairy Form <sup>2</sup>		Adjusted for $BCS_{HOL}$ <sup>3</sup>	
DA	-0.45	-0.07	0.50*	0.07
Metabolic	-0.49	-0.06	0.60*	0.04
Foot	... <sup>4</sup>	... <sup>4</sup>	0.21	-0.07
Mastitis	0.31	-0.04	0.08	0.14*

<sup>1</sup>Standard errors of genetic correlation estimates ranged from 0.18 to 0.66 with an average of 0.31. Standard errors of phenotypic correlation estimates ranged from 0.04 to 0.08 with an average of 0.06.

<sup>2</sup>Correlations among  $BCS_{HOL}$  and disease with dairy form included as a covariate.

<sup>3</sup>Correlations among dairy form and disease with  $BCS_{HOL}$  included as a covariate.

<sup>4</sup>Convergence to a positive definite solution failed.

\* Correlation greater than twice the standard error.

correlation estimate between dairy form and mastitis was -0.03, whereas the genetic correlation estimate between dairy form and foot diseases was 0.21.

Higher dairy form was genetically correlated with increased incidence of disease in this and other studies (Hansen et al., 2002; Rogers et al., 1999). The genetic relationship between dairy form and metabolic and digestive disease may be due, in part, to the relationship between dairy form and BCS. The genetic correlation estimate between  $BCS_{HOL}$  and dairy form was estimated to be -0.72 (Table 13). Cows with high dairy form have lower BCS, and are likely to have more severe negative energy balance in early lactation.

Genetic correlation estimates between dairy form and metabolic disease and DA were significant after adjustment for BCS, indicating that the relationship between disease and dairy form may not be due only to differences in the level of body condition. The genetic correlation estimates between BCS and metabolic disease and DA were not as strong when dairy form was included in the model.

### **Disease in Denmark**

Correlations and approximate genetic correlations of breeding values for disease in Denmark with PTAB and PTAD and are reported in Table 16. Correlations of breeding values for disease in Denmark with PTAB and PTAD adjusted for PTAB, PTAD or PTAP are reported in Table 17.

Genetic correlation estimates between PTAB and MDD and FLD were negative in lactation one through three, while genetic correlation estimates between PTAB and reproductive diseases were positive in lactation one through three. However, genetic correlation estimates were significant ( $p < 0.05$ ) only for MDD in first (-0.36) and third (-0.35) lactations. Correlations between  $BCS_{HOL}$  and MDD were negative, but not significant in first, second or third lactation after adjustment for PTAD (Table 17).

Predicted transmitting abilities for dairy form were positively correlated with reproductive disease, MDD and FLD in lactations 1, 2 and 3. Genetic correlation estimates were significant between PTAD and MDD in first lactation (0.40) and between PTAD and FLD in first (0.44), second (0.43) and third (0.45) lactations. Correlations



Table 16. Correlations and approximate genetic correlation estimates between breeding values for disease traits from Denmark and PTA for BCS or dairy form from the US.

Disease	<u>Lactation 1</u>		<u>Lactation 2</u>		<u>Lactation 3</u>	
	BCS	Dairy Form	BCS	Dairy Form	BCS	Dairy Form
	Breeding Value Correlations <sup>1</sup>					
Reproductive	0.13	0.10	0.12	0.13	0.08	0.12
Metabolic and digestive	-0.26*	0.30*	-0.18	0.11	-0.27*	0.13
Feet and Leg	-0.06	0.33*	-0.11	0.31*	-0.13	0.32*
	Approximate Genetic Correlations <sup>2</sup>					
Reproductive	0.16	0.13	0.16	0.16	0.09	0.15
Metabolic and digestive	-0.36*	0.40*	-0.25	0.15	-0.35*	0.17
Foot and Leg	-0.09	0.44*	-0.16	0.43*	-0.18	0.45*

<sup>1</sup>Product-moment correlations between breeding value for disease and PTA for BCS or dairy form

<sup>2</sup> Product-moment correlations between breeding values have been adjusted for reliability of breeding values to approximate genetic correlations.

\*p<.05

Table 17. Correlations between breeding values for disease traits from Denmark and PTA for BCS and dairy form in the US after adjusting with PTA for BCS, dairy form or protein yield.

Disease	<u>Lactation 1</u>		<u>Lactation 2</u>		<u>Lactation 3</u>	
	BCS	Dairy Form	BCS	Dairy Form	BCS	Dairy Form
	Adjusted for BCS and Dairy Form <sup>1</sup>					
Reproductive	0.20	0.20	0.21	0.20	0.16	0.17
Metabolic and digestive	-0.14	0.20	-0.14	0.01	-0.23	0
Foot and Leg	0.12	0.35*	0.05	0.30*	0.04	0.30*
	Adjusted for Protein Yield <sup>2</sup>					
Reproductive	0.14	0.08	0.14	0.13	0.09	0.11
Metabolic and digestive	-0.32*	0.35*	-0.23	0.17	-0.31*	0.19
Foot and Leg	-0.07	0.31*	-0.12	0.32*	-0.12	0.31*

<sup>1</sup>Correlations between PTA for BCS and breeding values for disease in Denmark have been adjusted for PTA dairy form. Correlations between PTA for dairy form and breeding values for disease in Denmark have been adjusted for PTA BCS.

<sup>2</sup>Correlations between PTA for BCS or dairy form and breeding values for disease in Denmark after adjustment for PTA protein yield.

\*p<.05

between PTAD and FLD were significant after adjustment for PTAB in all three lactations (range 0.30 to 0.35).

Genetic correlations between dairy form recorded in the US and disease in Denmark were estimated previously by Rogers et al. (1999). Approximate genetic correlations between dairy form and MDD and FLD were similar in magnitude to those reported here. Genetic correlation estimates between dairy form and reproductive disease were stronger, ranging from 0.61 to 0.64 (Rogers et al., 1999).

Adjustment for PTAP had a minimal affect on correlation estimates (Table 17). All correlations significant before adjustment for PTAP remained significant after adjustment for PTAP. Rogers et al. (1999) reported genetic correlation estimates between dairy form in the US and reproductive disease, FLD and MDD in Denmark that were significant after adjustment for yield. Genetic correlation estimates between dairy character and disease other than mastitis in Denmark was 0.41 before and 0.39 after adjustment for protein yield (Hansen et al., 2002). The relationship among BCS, dairy form and disease does not appear to result entirely from differences in yield. Selection to increase yield and maintain current levels of BCS or dairy form should help limit unfavorable changes in levels of cow health while yields increase.

Genetic correlation estimates for US health are based on a relatively small data set and are associated with large standard errors. Approximate genetic correlations between dairy form and  $BCS_{HOL}$  and disease in Denmark are based on a limited number of highly selected bulls that have been used worldwide. However, there are consistent patterns that indicate a relationship among dairy form,  $BCS_{HOL}$  and cow health traits. Genetic correlation estimates among dairy form,  $BCS_{HOL}$  and US health or disease in Denmark indicate that, at a given level of production, cows genetically inclined to be thin (high dairy form and low BCS) have higher levels of metabolic disease and DA. Cows genetically inclined to be thin may also be more susceptible to FLD and reproductive disease.

The relationship between dairy form,  $BCS_{HOL}$  and metabolic disease is likely due to differences in early lactation negative energy balance. Dry matter intake for early lactation cows is often inadequate to provide the energy required to support milk yield,

resulting in negative energy balance. Severe negative energy balance and excessive body fat mobilization is associated with metabolic disease (Baird, 1982; Goff and Horst, 1997). Dechow et al. (2002) reported that cows genetically inclined to have higher levels of BCS lose less BCS in early lactation. Cows genetically inclined to have higher levels of BCS appear to maintain more BCS in early lactation and likely have less severe negative energy balance in early lactation, resulting in less metabolic disease.

Negative energy balance may play a role in the relationship between dairy form and FLD observed in this study and in Rogers et al. (1999). Negative energy balance was associated with an increase in locomotive disorders, including laminitis (Collard et al., 2000). Metabolic disease may predispose cows to laminitis as well (Nocek, 1997).

The relationship between dairy form and foot and leg disease is not likely due to metabolic disorders only. High dairy form may be associated with poorer foot and leg conformation, which could predispose cows to more foot and leg disorders. The genetic correlation between dairy form and rear legs side view was 0.35 and the genetic correlation between dairy form and foot angle was  $-0.21$ , indicating that cows with higher dairy form tend to have more set to their hock and slightly lower foot angles (Dechow et al., 2003).

Dairy form was more strongly correlated with disease incidence than BCS. Correlations between BCS and disease tended to be non-significant when adjusted for dairy form, whereas genetic correlation estimates between dairy form and disease were not reduced significantly by adjustment for BCS with one exception. The correlation between dairy form in the US and MDD in Denmark declined from 0.30 (Table 16) before to 0.20 (Table 17) after adjustment for BCS. The genetic correlation between dairy form and metabolic disease in the US also declined (0.71 to 0.60), but was still significant. The relationship between dairy form and metabolic disease may be due primarily to differences among cows in BCS. However, the relationship between dairy form and foot and leg and reproductive diseases does not appear to simply be due to differences in BCS.

While BCS and dairy form are genetically similar, they are not the same trait. The genetic variance for  $BCS_{HOL}$  was reduced by 46% after adjustment for dairy form,

whereas the genetic variance for dairy form was reduced by 40% after adjustment for  $BCS_{HOL}$ . Body condition score is a new trait for classifiers and it is possible that  $BCS_{HOL}$  is scored less accurately than dairy form. This could result in a genetic correlation estimates that are underestimated between  $BCS_{HOL}$ , dairy form and health traits.

The relationship between BCS and body composition has been investigated. A correlation of 0.87 was reported between BCS and the total body fat percent of slaughtered cows of various genotypes, including dairy cows (O'Mara et al., 1998). Domecq et al. (1995) regressed BCS on various ultrasound measurements of subcutaneous fat depth from the pelvic region of Holstein cows and reported  $R^2$  values ranging from 0.36 to 0.65, indicating that BCS accurately reflects variation in fat deposition among cows in the pelvic region.

The relationship between dairy form and body composition has not been well defined, however. It is possible that dairy form is more highly correlated with differences in body fat than BCS, particularly through the front end and over the rib structure. Body condition scores are generally assigned based on a visual assessment of the pelvic region only, while dairy form is assigned based on a visual assessment of the rib structure.

## **CONCLUSIONS**

Body condition scores recorded by the Holstein Association USA, Inc. provide a large, central source of BCS that could be used to generate genetic evaluations for BCS. Body condition scores recorded by producers or herd-consultants and during linear type appraisals were highly correlated genetically.

Low body condition score and high dairy form were genetically correlated with an increase in metabolic disease and poorer cow health. Selection for higher BCS or lower dairy form would help maintain current levels of cow health while selection continues for higher milk, fat and protein yields. Including a strong positive emphasis on dairy form in the calculation of final score may encourage indirect selection for cows that are less healthy and dairy form may need to be de-emphasized in final score.

Genetic correlations between BCS and cow health were not as strong after adjustment for dairy form. Moreover, BCS and dairy form have a strong genetic correlation. It is not clear that genetic evaluations for BCS would provide valuable

genetic information beyond current dairy form evaluations. However, producers may be less reluctant to select for higher BCS than for lower dairy form because dairy form is weighted positively in final score calculations.

## **Chapter 6**

### **Body Condition Score and Dairy Form Evaluations as Indicators of Days Open in US Holsteins**

This chapter is a slightly modified version of a paper by the same name in preparation for the Journal of Dairy Science by C. D. Dechow, G. W. Rogers, L. Klei, T. J. Lawlor and P. M. VanRaden.

My contributions to this paper include: All data analysis, gathering and interpretation of literature and all writing of the manuscript except for editing of other authors and reviewers.

#### **ABSTRACT**

The objectives of this study were to estimate genetic correlations among body condition score (BCS), dairy form, yield and days open in US Holsteins and investigate the potential of using BCS or dairy form evaluations as early indicators of days open. Dairy form and BCS obtained from the Holstein Association USA, Inc. were merged with ME for milk yield and days open data from AIPL-USDA. Cows were required to be classified between 24 and 60 months of age, before 335 days in milk (DIM) and have ME milk of at least 4537 kg. A minimum of 20 daughters per sire and 10 cows per herd-classification visit (HV) or herd-year-season of calving (HYS) was required. The final data set included 159,700 records. Heritabilities and correlations among dairy form, BCS, milk yield and days open were estimated with multiple trait sire models in ASREML. Fixed effects included age at classification for dairy form and BCS, age at calving for milk yield, HV for dairy form and BCS, HYS for milk yield and days open, DIM within lactation group for dairy form and BCS and lactation group for milk yield and days open. Correlations among dairy form, BCS and days open were generated with and without a ME milk covariable. Correlations between ME milk and days open were generated with and without covariables for dairy form or BCS. Random effects included sire and error. The genetic correlation estimates of days open with dairy form, BCS and ME milk were 0.48, -0.30 and 0.38, respectively. The genetic correlation estimate between days open and dairy form was 0.38 after adjustment for ME milk, whereas the genetic correlation

between days open and BCS was  $-0.24$  after adjustment for ME milk. After adjustment for dairy form, the genetic correlation estimate between BCS and days open was 0 and the genetic correlation estimate between ME milk and days open was 0.22. Combining dairy form evaluations with direct days open evaluations for 19 recently proven bulls resulted in an average increase of 0.06 for reliability of days open evaluations. Including information on dairy form will increase the reliability of days open evaluations, but the addition of BCS evaluations did not increase reliability when dairy form observations were available.

**(Key Words:** body condition score, dairy form, fertility)

**Abbreviation Key:** AIPL-USDA = Animal Improvement Programs Laboratory at The US Department of Agriculture, BCS = body condition score, DPR = daughter pregnancy rate, HV = herd-classification visit, HYS = herd-year-season, PA = parent average, REL = reliability.

## INTRODUCTION

Fertility traits have generally been ignored in many dairy cattle selection programs because of low heritability and inaccurate recording of fertility data. Results from selection for improved fertility are thus expected to be slow. However, the genetic variation for fertility measures is substantial, indicating potential to select for improved fertility. Weigel and Rekaya (2000) reported ranges in sire breeding values for 60 day non-return rates of 16% for several California herds to 30% in several Minnesota herds. The genetic standard deviation of first service conception rate was near 0.05% in two studies (Berry et al., 2003, Veerkamp et al., 2001). The genetic standard deviation of calving interval was reported to be 7 days (Pryce et al., 2002) and 9 days (Veerkamp et al., 2001).

Unfortunately, dairy cattle fertility is unfavorably correlated with yield. Abdallah and McDaniel (2000) estimated that days open increased at a rate of 1.1 days per year from 1980 to 1993 as a correlated response to selection for increased yield in five North Carolina experimental herds. Moreover, there appears to be a trend of less voluntary culling of low producing cows, which is likely due partly to reduced fertility and slows potential genetic improvement (Weigel et al., 2003).



National genetic evaluations for daughter pregnancy rate (DPR) are now available. Daughter pregnancy rates are derived from days open records and have an estimated heritability of 0.04 (VanRaden et al., 2002). Because of the low heritability of DPR, only bulls with many daughters will have high reliability for DPR. Producer confidence may be low for DPR until second crop daughters are generated. In addition, days open cannot be recorded as early in lactation as production or type traits, resulting in further limits to reliable DPR information for recently proven bulls.

High BCS and low dairy character has been correlated with improved reproductive performance in many studies (Berry et al., 2003; Dadati et al., 1986; Dechow et al., 2001; Pryce et al., 2000; Veerkamp et al., 2001). Cows with high genetic merit for BCS have less BCS loss in early lactation, indicating that high genetic merit for BCS is associated with less severe negative energy balance (Dechow et al., 2002). Higher negative energy balance is genetically associated with an increase in days to the start of luteal activity after calving (Veerkamp et al., 2000).

Studies in Europe have indicated that selecting for higher BCS will slow deterioration in fertility as a response to selection for higher yield (Berry et al., 2003, Pryce et al., 2002). The reliability of productive life evaluations in the US has been improved by using correlated type and production evaluations to predict productive life for recently proven bulls (Weigel et al., 1998, VanRaden, 2001).

The objectives of this study were to estimate correlations among BCS, dairy form, yield and days open in the US and to investigate the potential use of BCS, dairy form or yield evaluations to increase the reliability (REL) of genetic evaluations for days open.

## **MATERIALS AND METHODS**

### **Data**

Classification data including BCS and dairy form recorded during linear type evaluation from October of 1997 through June of 2000 was obtained from the Holstein Association USA Inc. Heritability and correlation estimates among BCS, dairy form and other linear type traits using this data were previously reported by Dechow et al. (2003). A total of 728,597 classification records from 613,338 cows were included in the initial

data set. BCS was recorded on a scale of 1 (thin) to 50 (fat) and records from classifiers that had assigned BCS abnormally were removed by the same procedures as in Dechow et al. (2003). Records from cows that were classified between 24 and 60 months of age were retained and records after 335 DIM were eliminated. Only the first available classification record for each cow was retained to eliminate the need for a permanent environmental effect.

Production data including mature equivalent for 305 day milk yield (ME milk) and days open from 1997 through December 2002 was provided by the Animal Improvement Programs Laboratory at USDA (AIPL-USDA). Days open are converted to daughter pregnancy rate (DPR) in national genetic evaluations in the US (VanRaden et al., 2002), but were left as days open for this study. The original data set included 14,813,461 records from 7,149,074 cows. Cows were required to calve between 20 and 60 months of age and have ME milk of at least 4537 kg. Days open less than 25 days were eliminated. Days open greater than 250 days were set to 250, as in the national genetic evaluations for days open (VanRaden et al., 2002).

The classification data set and production data set were merged and only cows with records in both data sets in a given lactation were retained. Contemporary group effects were herd-classification visit (HV) for the classification data and herd-year-season of calving (HYS) for production data. Herd-year of calving was substituted for HYS groups with fewer than 10 cows. Three seasons were defined: January through April, May through August and September through December. A minimum of 10 cows per HYS or HV and 20 daughters per sire were required. The final data set included 159,700 cows sired by 1165 Bulls. Four generations of sires and dams were traced for all sires resulting in a pedigree file that included 2292 individuals.

## Statistical Analyses

All traits were analyzed with multiple trait sire models in ASREML (Gilmour et al., 2002). The basic statistical model used in the analyses was:

$$\mathbf{y} = \mathbf{b}_1 * \text{age}(\text{LG}) + \sum_{m=2}^6 \mathbf{b}_x * \text{DFS}^{m-1}(\text{LG}) + \mathbf{CG} + \mathbf{Sire} + \boldsymbol{\varepsilon},$$

where  $\mathbf{y}$  = a vector of length two to three including BCS, dairy form, days open or ME milk,  $\mathbf{b}_1$  = a vector of regression coefficients on age in months nested within lactation group (LG),  $\mathbf{b}_x$  = a vector of regression coefficients of order 1 through 5 on DIM within lactation group and were included for BCS and dairy form only,  $\mathbf{CG}$  = a vector of fixed effects for contemporary group,  $\mathbf{Sire}$  = a vector of random effects for sire and  $\boldsymbol{\varepsilon}$  = a vector of random errors. Three LG were defined as first lactation, second lactation and third and fourth lactations. Age was age at calving for ME milk and age at classification for BCS and dairy form. Age was not included in analysis of days open. Poor fertility is likely to increase age at calving and adjusting for age would then eliminate variance in days open that may be due to genetic differences for fertility (VanRaden et al., 2002). Contemporary groups effects were HV (n=10,807) for BCS and dairy form and HYS (n=15,916) for ME milk and days open. Fifth order polynomials of DIM nested in LG were included in analysis of BCS and dairy form and LG was included as a fixed effect for ME milk and days open. Analyses among BCS, dairy form and days open were conducted with and without ME milk as a covariable. Likewise, analyses among BCS, dairy form, ME milk and days open were conducted with and without a covariable for BCS or dairy form.

### Indirect Prediction of Days Open

The potential of using BCS, dairy form or yield evaluations as indicators of PTA for days open (PTA<sub>DO</sub>) was investigated by comparing the reliability of PTA<sub>DO</sub> (REL<sub>DO</sub>) under the following scenarios: 1) PTA<sub>DO</sub> was generated directly with daughter observations for days open, 2) PTA<sub>DO</sub> was generated indirectly (PTA<sub>DOI</sub>) with PTA for BCS (PTA<sub>BCS</sub>), dairy form (PTA<sub>DF</sub>), ME milk (PTA<sub>M</sub>), PTA<sub>BCS</sub> + PTA<sub>DF</sub> or PTA<sub>DF</sub> + PTA<sub>M</sub> and 3) PTA<sub>DO</sub> from scenario 1 combined with PTA<sub>DOI</sub> from scenario 2. Direct REL (REL<sub>dir</sub>) for PTA<sub>DO</sub>, PTA<sub>BCS</sub>, PTA<sub>M</sub> and PTA<sub>DF</sub> was calculated as:

$$REL_{dir} = n / (n + k), \text{ where } n = \text{the number of daughters and } k = (4 - h^2) / h^2.$$

The REL of PTA<sub>DOI</sub> was calculated with formulas used to calculate REL for indirect predictions of productive life with production and type data (Weigel et al., 1998). Reliability for an indirect prediction (REL<sub>ind</sub>) was calculated as:

$$REL_{ind} = \frac{Cov[TA_{DO}, TA_{ind}] [Var(TA_{ind})]^{-1} [Var(PTA_{ind})] [Var(TA_{ind})]^{-1}}{Cov[TA_{DO}, TA_{ind}] / Var(TA_{DO})}, \quad [1]$$

where TA<sub>DO</sub> = transmitting ability for days open, TA<sub>ind</sub> = a vector of transmitting abilities for the predictors of days open (PTA<sub>BCS</sub>, PTA<sub>M</sub> and PTA<sub>DF</sub>) and PTA<sub>ind</sub> = a vector of BLUP predictions of TA<sub>ind</sub>. The variance of PTA<sub>BCS</sub>, PTA<sub>DF</sub> and PTA<sub>M</sub> was calculated by multiplying the TA variance for BCS or dairy form times REL for BCS, dairy form or ME milk.

The combined REL (REL<sub>comb</sub>) can then be calculated as follows (Weigel et al., 1998):

$$REL_{comb} = \frac{REL_{dir} + REL_{ind} - 2REL_{dir}REL_{ind} \times c}{1 - REL_{dir}REL_{ind} \times c^2}, \quad [2]$$

where  $c = 1 + [DE_{both}/DE_{DO}DE_{ind}] \times [(4 - h^2_{DO}) (4 - h^2_{ind}) / (h^2_{DO} h^2_{ind})]^{0.5}$ . Cows with records for days open contribute daughter equivalents for days open (DE<sub>DO</sub>), cows with classification records contribute indirect daughter equivalents (DE<sub>ind</sub>) and cows with classification records and days open contribute to daughter equivalents both (DE<sub>both</sub>). When no daughters have both classification records and days open observations, DE<sub>both</sub> = 0 and c = 1.

The REL<sub>DO</sub> when PTA<sub>DO</sub> and PTA<sub>DOI</sub> are combined with parent average for days open (PA<sub>DO</sub>) was calculated by combining REL for PA<sub>DO</sub> with REL<sub>comb</sub> using equation [2]. Reliability for PA (REL<sub>PA</sub>) is substituted for REL<sub>ind</sub> and REL<sub>comb</sub> is substituted for

$REL_{dir}$ . If it is assumed that a young bull has no daughters for days open, and therefore does not contribute to his  $PA_{DO}$ , then  $DE_{both}$  becomes 0 and  $c = 1$  and can be dropped from equation [2]. This gives the same formula used by Harris and Johnson (1998) to approximate REL from two separate sources of information.

The expected  $REL_{PA}$  of a young bull was estimated by averaging  $REL_{PA}$  for 473 young bulls born after 1997 with daughters that had milk yield records, but no daughters for days open in national genetic evaluations for May of 2003 (AIPL-USDA, Beltsville, MD). The  $REL_{PA}$  of this population of bulls should approximate the expected  $REL_{PA}$  for bulls that are about to receive their first official proof and enter active service.

$PTA_{DOI}$  would be of most value for recently proven bulls that have few direct daughters for days open. Therefore, the number of daughters for days open, dairy form and milk yield and REL for  $PTA_{DO}$ ,  $PTA_M$  and  $PTA_{DF}$  were obtained for bulls born in 1997 or later, that had entered active service between June of 1999 and November of 1999 and that were on the High Ranking Sire Report for TPI from the Holstein Association USA, Inc. in May of 2003. This group of bulls should represent newly proven bulls that are likely to be widely used by US dairy producers. Nineteen bulls met all of the criteria listed above.

## **RESULTS AND DISCUSSION**

### **Heritabilities and Correlations**

Heritabilities and correlations among BCS, dairy form, days open and ME milk are shown in Table 18. Heritability estimates were 0.22 for BCS and 0.25 for dairy form. The genetic correlation estimate between BCS and dairy form was -0.73, whereas the phenotypic correlation estimate was -0.45. The heritability and correlation estimates among dairy form and BCS were nearly identical to those reported by Dechow et al. (2003) using a similar subset of this data.

The heritability estimates of ME milk and days open were 0.25 and 0.04, respectively, which compares to heritability estimates of 0.30 and 0.04 that are used currently in national genetic evaluations with an animal model (AIPL-USDA, 2003). The

Table 18. Heritabilities (diagonal), genetic (above diagonal) and phenotypic (below diagonal) correlations among body condition score (BCS), dairy form, ME milk and days open.<sup>1</sup>

	BCS	Dairy Form	ME Milk	Days Open
BCS	0.22	-0.73	-0.25	-0.30
Dairy Form	-0.45	0.25	0.49	0.48
ME Milk	-0.17	0.42	0.25	0.38
Days Open	-0.07	0.09	0.15	0.04

<sup>1</sup>Standard errors for the genetic correlations range from 0.02 to 0.06.

genetic correlation estimate between ME milk and days open was estimated to be 0.38, whereas the phenotypic correlation estimate between ME milk and days open was 0.15.

The genetic correlation estimates of ME milk with BCS and dairy form were -0.25 and 0.49, respectively. The phenotypic correlation estimate between ME milk and BCS was -0.17, whereas the phenotypic correlation estimate between ME milk and dairy form was 0.42. These correlation estimates are similar to previously reported estimates of genetic correlations among ME milk, BCS and dairy form. Short and Lawlor (1992) reported a genetic correlation estimate of 0.54 between lactation yield and dairy form. Estimates of the genetic correlation between BCS and milk yield range from -0.28 to -0.51 (Berry et al., 2003; Dechow et al., 2001; Veerkamp et al., 2001). Clearly, cows with high genetic merit for yield have lower BCS and higher dairy form, but the correlation is low to moderate.

The genetic and phenotypic correlation estimates between days open and BCS are -0.30 and -0.07, respectively. BCS is favorably correlated with reproductive performance in several studies. BCS appears to be most strongly correlated with the interval between calving and when luteal activity resumes, estrus is displayed or insemination occurs. Royal et al. (2002) inferred a genetic correlation estimate between BCS and the interval from calving to commencement of luteal activity as determined by milk progesterone testing to be -0.84 in the UK. This is likely due to differences in energy balance among cows that are reflected by differences in BCS. The genetic correlation between energy balance and commencement of luteal activity was estimated to be -0.60 in a research herd in The Netherlands (Veerkamp et al., 2000). Butler et al. (1981) reported that ovulation occurred at an average of 10 days after maximal daily negative energy balance, while De Vries et al. (1999) reported that total negative energy balance and maximal negative energy balance were both correlated with an increase in first observed estrus.

Harrison et al. (1990) reported higher negative energy balance in early lactation cows selected for high genetic merit for yield versus cows selected for average genetic merit. The cows selected for high genetic merit for yield did not have a significant delay in days to first ovulation, but did have a significant delay in days to first visual estrus (66 days versus 43 days). The genetic correlation between days to first heat and BCS 10

weeks in lactation was reported to be -0.49 (Pryce et al., 2001). Genetic correlation estimates between days to first service and BCS during the lactation range from -0.37 to -0.76 over a range of studies from different countries (Berry et al., 2003; Dechow et al., 2001; Pryce et al., 2000; Veerkamp et al., 2001). A delay in commencement of luteal activity or estrus are likely the underlying physiological factors for the genetic relationship between BCS and days to first service.

Higher BCS tends to be correlated with improved fertility in many studies as well, though correlations are not as strong as with days to first service. The genetic correlation estimates between BCS during the lactation and services per conception range from -0.03 to -0.42 (Berry et al., 2003; Dechow et al., 2001; Veerkamp et al., 2001). Genetic correlation estimates between BCS and first service conception were 0.34 and 0.20 (Berry et al., 2003; Veerkamp et al., 2001).

The phenotypic correlation between fertility and DIM at insemination appears to be unfavorable (positive). Dechow et al. (2002) reported a decreasing number of services per conception as days to first service increased and non-return rates were reported to increase as lactation progressed in two studies (Ravagnolo and Misztal, 2002; Weigel and Rekaya, 2000). The genetic relationship between BCS and fertility may not be as strong in many studies because cows with genetic merit for higher BCS are inseminated earlier in lactation when fertility tends to be lower.

The genetic correlation between BCS and days open in this study (-0.30) is slightly lower than reported in other studies. The genetic correlation between BCS and days to last service confirmed by a subsequent calving (which could also be considered days open) was reported to be -0.41 (Veerkamp et al., 2001). The genetic correlation between BCS and calving interval was reported to be -0.40 (Pryce et al., 2000). These correlation estimates are generally not as strong as correlation estimates between BCS and days to first service. The genetic relationship between BCS and traits like days open or calving interval is dependant upon the genetic relationship of BCS with both days to first service and fertility, which may have an unfavorable phenotypic relationship.

The genetic (0.48) and phenotypic (0.09) correlation estimates between dairy form and days open were stronger than between BCS and days open. BCS was a new trait



for Holstein classifiers in this study and it is possible that BCS is scored less accurately than dairy form, which could result in an underestimated genetic correlation between BCS and days open. However, Pryce et al. (2000) also reported a slightly stronger correlation (0.47) between angularity in the UK (similar to US dairy form) and calving interval than for BCS and calving interval (-0.40). Genetic correlations among dairyness (0.43) and dairy character (0.38) were also unfavorably correlated with calving interval in Canadian Holsteins classified between 1976 and 1983 (Dadati et al., 1986).

Dairy form may be more highly correlated with fertility than BCS. The physiological relationship among dairy form, fertility and energy balance has not been as well studied as the relationship among BCS, fertility and energy balance. It has been assumed that the relationship between dairy form and reproductive or health traits may be due to differences in BCS or energy balance. In this study, the relationship between dairy form and fertility was not explained by differences in BCS alone.

The effect of culling for yield could have an impact on genetic correlations between BCS or dairy form and days open or calving interval. Cows with high genetic merit for BCS would be expected to have genetic merit for fewer days open. However, cows with high genetic merit for BCS (or low merit for dairy form) are expected to have lower genetic merit for yield and would be at increased risk for culling for low yield. Cows culled for low production would not have records for calving interval. Some of the days open data was generated from calving intervals in this study. Approximately 6% of cows had no breeding date information, but did have a subsequent calving date (VanRaden et al., 2002). Fertile cows with no breeding dates and no subsequent calving date may have been culled for low production. However, there appears to be less culling for low production than 10 years ago (Weigel et al., 2003) and the effect of selection is probably minimal.

### **Adjustment for ME Milk**

Genetic and phenotypic correlations among BCS, dairy form and days open are reported in Table 19. The genetic correlation estimates of days open with BCS and dairy form were -0.24 and 0.38 after adjustment for ME milk, respectively. Pryce et al. (2002) reported estimated the genetic correlation between BCS and calving interval to be -0.48

Table 19. Genetic (above diagonal) and phenotypic (below diagonal) correlations among body condition score (BCS), dairy form and days open adjusted for ME milk.<sup>1</sup>

	BCS	Dairy Form	Days Open
BCS		-0.71	-0.24
Dairy Form	-0.43		0.38
Days Open	-0.05	0.03	

<sup>1</sup>Standard errors for the genetic correlations range from 0.02 to 0.06.

before and  $-0.22$  after adjustment for milk yield. Dechow et al. (2001) reported genetic correlation estimates between BCS during the lactation and days to first service ranging from  $-0.42$  to  $-0.76$  before and from  $-0.40$  to  $-0.72$  after adjustment for ME milk, respectively. The genetic correlation between energy balance and the start of luteal activity was  $-0.60$  before adjustment for yield and  $-0.49$  after adjustment for yield (Veerkamp et al., 2000). The genetic relationship between days open and BCS, dairy form or energy balance is not only the result of production differences among cows. Cows with higher BCS or lower dairy form at a given level of yield should have fewer days open, and selection to limit change in BCS or dairy form may help reduce the unfavorable correlated response in days open when selecting for yield.

### **Adjustment for BCS and Dairy Form**

Genetic correlation estimates between days open and dairy form, BCS or ME milk after adjustment for BCS or dairy form are given in Table 20. The genetic correlation between BCS and days open was 0 after adjusting for dairy form, but the genetic correlation between dairy form and BCS was 0.40 after adjustment for BCS. It would appear that differences in dairy form are more independent of differences in BCS than differences in BCS are of dairy form.

The genetic correlation estimate between ME milk and days open was 0.33 after adjustment for BCS and 0.22 after adjustment for dairy form. An unfavorable genetic relationship between ME milk and days open exists even after one adjusts for dairy form. The squared genetic correlation between ME milk and days open when not adjusted for dairy form indicates that genetic differences among cows in ME milk explain approximately 14.4% of the genetic difference among cows in days open. The squared genetic correlation between ME milk and days open is only 4.8% after adjustment for dairy form. This indicates that a majority of the unfavorable relationship between ME milk and days open is likely due to higher dairy form for cows with high genetic merit for yield.

### **Indirect Prediction of Days Open**

$REL_{DO}$  based on  $PTA_{DO}$  only,  $PTA_{DF}$  only,  $PTA_{BCS}$  only,  $PTA_M$  only and  $PTA_{DF} + PTA_M$  are shown in Figure 8. With 200 daughters,  $REL_{DO}$  was 0.08 with  $PTA_{BCS}$ , 0.14

Table 20. Genetic ( $r_g$ ) and phenotypic ( $r_p$ ) correlations between BCS and days open adjusted for dairy form, between dairy form and days open adjusted for BCS and between ME milk and days open adjusted for BCS or dairy form.<sup>1</sup>

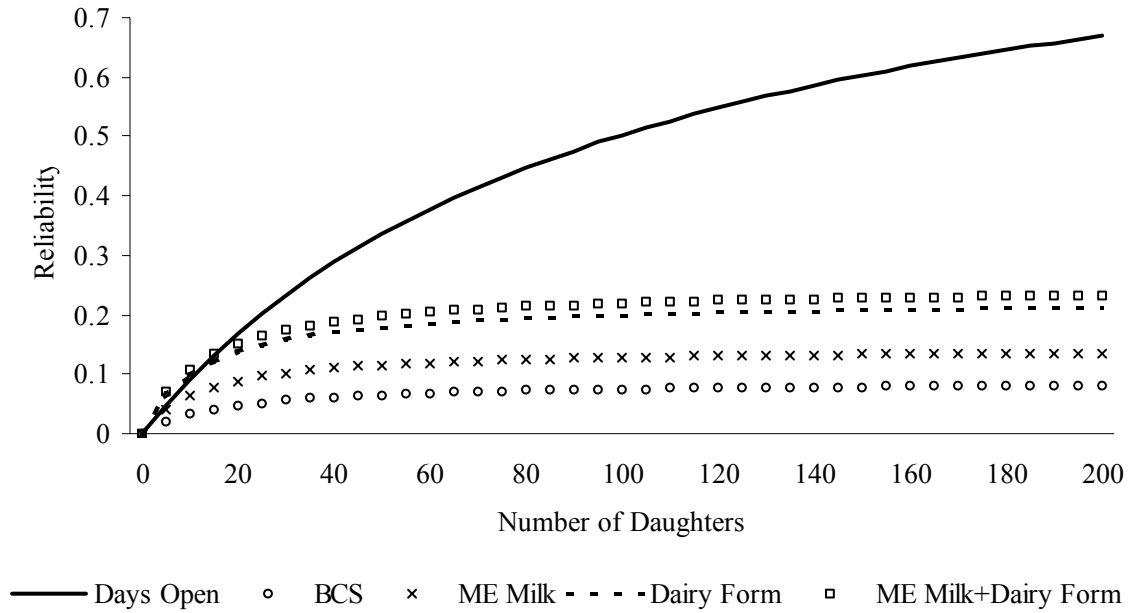
	Correlations with days open	
	$r_g$	$r_p$
BCS <sup>2</sup>	0.0	-0.04
Dairy Form <sup>3</sup>	0.40	0.06
ME Milk <sup>2</sup>	0.22	0.13
ME Milk <sup>3</sup>	0.33	0.14

<sup>1</sup>Standard errors for the genetic correlations range from 0.05 to 0.06

<sup>2</sup>Dairy form was included in the model as a covariate.

<sup>3</sup>BCS was included in the model as a covariate.

Figure 8. Reliability of PTA for days open with direct days open observations and indirect prediction of PTA for days open with BCS, dairy form, ME milk and ME milk plus dairy form.



with  $PTA_M$ , 0.21 with  $PTA_{DF}$ , 0.23 with  $PTA_{DF} + PTA_M$  and 0.67 with direct  $PTA_{DO}$ .  $REL_{DO}$  with  $PTA_{DF} + PTA_{BCS}$  was 0.006 higher (not shown) than with  $PTA_{DF}$  only. With 10 daughters or fewer,  $PTA_{DF}$  was a more reliable predictor of  $PTA_{DO}$  than the direct estimate of  $PTA_{DO}$ .

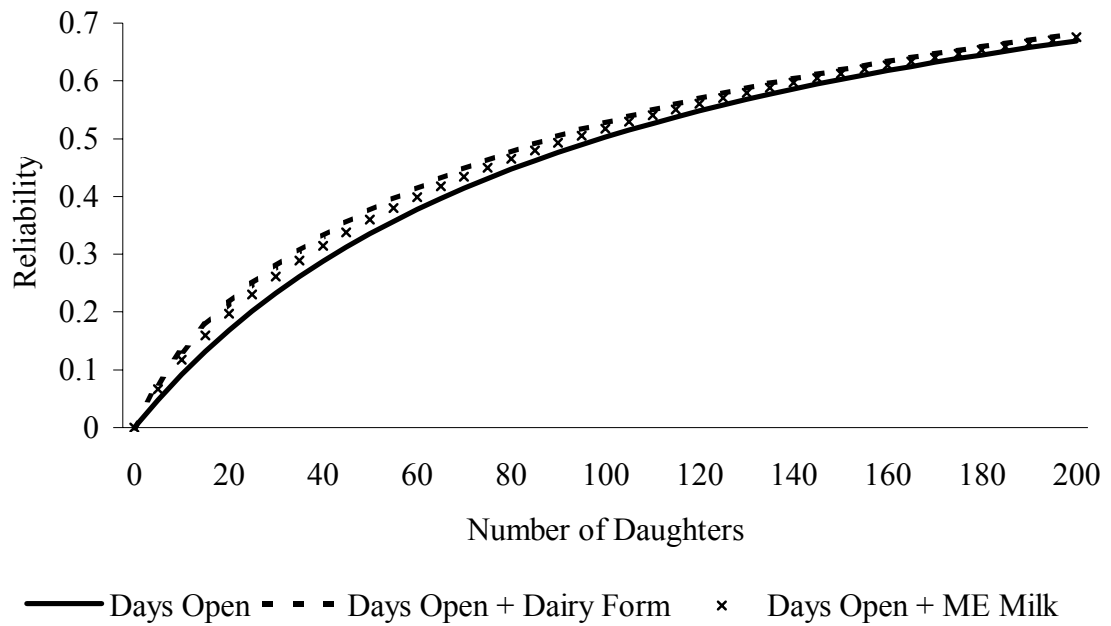
$REL_{DO}$  when  $PTA_{DO}$  is combined with  $PTA_{DOI}$  is shown in Figure 9. An equal number of daughters with days open, ME milk and dairy form records are assumed and all daughters with days open are assumed to have ME milk and dairy form records. The maximum gain in  $REL_{DO}$  when  $PTA_{DO}$  is combined with  $PTA_M$  was 0.029, whereas the maximum gain in  $REL_{DO}$  when  $PTA_{DO}$  is combined with  $PTA_{DF}$  was 0.048.

The effect of combining  $PTA_{DO}$  and  $PTA_{DOI}$  when  $PA_{DO}$  is available is shown in Figure 10. The average REL for  $PA_{DO}$  of 473 young sires that should soon have production and type proofs was 0.36. Therefore, a  $REL_{PA}$  of 0.36 was assumed for  $PTA_{DO}$ ,  $PTA_{DF}$  and  $PTA_M$ . The maximum gain in  $REL_{DO}$  was 0.023 when  $PTA_{DO}$  is combined with  $PTA_{DF}$ , 0.014 when combined with  $PTA_M$  and 0.027 when combined with  $PTA_{DF} + PTA_M$  (not shown).

Figures 9 and 10 assume an equal number of daughters for ME milk, days open and dairy form, which is not realistic. The minimum, maximum and average number of daughters for dairy form,  $PTA_{DO}$  (which is DPR in US genetic evaluations) and  $PTA_M$  for 19 recently proven bulls are given in Table 21. The average number of daughters for  $PTA_{DO}$  was 27, the average number of daughters for  $PTA_{DF}$  was 55 and the average number of daughters for  $PTA_M$  was 100. Daughter observations for days open are not available until near the end of a cow's lactation, or beginning of a subsequent lactation if derived from calving intervals. Dairy form and ME milk can be recorded earlier in lactation than days open and newly proven bulls have more daughters with dairy form and ME milk than with days open.

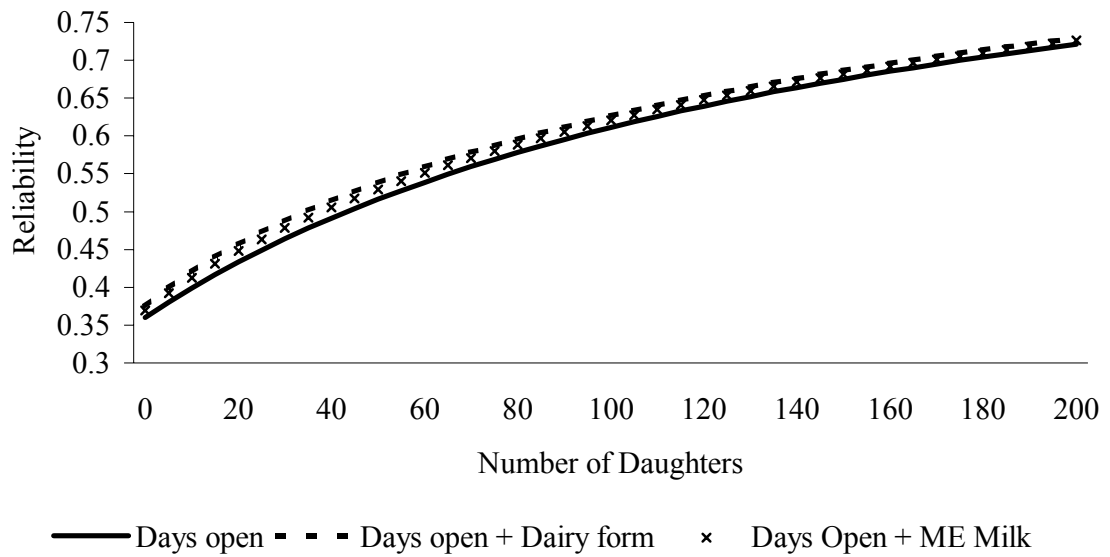
The minimum, maximum and average REL for  $PTA_{DO}$ ,  $PTA_{DF}$  and  $PTA_M$  plus the expected change in  $REL_{DO}$  when  $PTA_{DO}$  is combined with  $PTA_{DF}$ ,  $PTA_M$ , or  $PTA_{DF} + PTA_M$  are also reported in Table 21. The average  $REL_{DO}$  was 0.44. The average change in  $REL_{DO}$  if  $PTA_{DF}$ ,  $PTA_M$ , or  $PTA_{DF} + PTA_M$  is combined with  $PTA_{DO}$  is 0.06, 0.04 and 0.07, respectively. The maximum expected change in  $REL_{DO}$  was 0.08 if  $PTA_{DF}$  is

Figure 9. Reliability of PTA for days open with direct days open observations combined with ME milk, dairy form or ME milk + dairy form observations.<sup>1</sup>



<sup>1</sup>An equal number of daughters for days open and classification data is assumed and all daughters with a days open observation also have observations for ME milk and dairy form.

Figure 10. Reliability of PTA for days open with direct days open observations combined with dairy form or ME milk and parent average for days open.<sup>1</sup>



<sup>1</sup>An equal number of daughters for days open, ME milk and dairy form are assumed and all daughters with a days open observation also have observations for ME milk and dairy form.



Table 21. Minimum (Min), maximum (Max) and average (Ave) number of daughters (n) for dairy form, daughter pregnancy rate (DPR) and predicted transmitting ability for milk yield (PTAM) and official reliability (REL) for dairy form, DPR and PTAM for 19 recently proven high ranking Holstein sires. The expected change in REL for DPR when dairy form (+DF), PTAM (+PTAM), or both dairy form and PTAM (DF+PTAM) are combined with DPR is reported.

	Min	Max	Ave
DPR (n)	4	99	27
Dairy Form (n)	29	114	55
PTAM (n)	55	173	100
REL Dairy Form	0.75	0.90	0.81
REL PTAM	0.80	0.92	0.86
REL DPR	0.37	0.62	0.44
REL +DF	0.03	0.08	0.06
REL + PTAM	0.02	0.05	0.04
REL DF+PTAM	0.04	0.09	0.07

combined with  $PTA_{DO}$  and 0.09 if  $PTA_{DF} + PTA_M$  is combined with  $PTA_{DO}$  for a bull with 4 daughters with days open, 52 daughters with dairy form and 75 daughters with ME milk records.

## CONCLUSIONS

Cows genetically inclined to be thin (low in BCS and high in dairy form) have extended days open. This relationship exists even after adjustment for differences in yield and the majority of the correlated response in days open due to selection for yield is likely the result of higher dairy form and lower BCS.

Dairy form and BCS are highly correlated genetically, but dairy form was a more accurate predictor of days open than BCS. BCS may not have been scored as accurately as dairy form, or dairy form may be a more accurate predictor of energy balance than is BCS.

Genetic evaluations for dairy form or ME milk could be used to increase accuracy of days open evaluations for bulls that have few daughters with direct days open observations. The maximum increase in  $REL_{DO}$  for 19 recently proven bulls was 0.08 when direct  $PTA_{DO}$  is combined with  $PTA_{DF}$ . There appeared to be little advantage of including BCS evaluations when dairy form evaluations were available.

Dairy form was a better predictor of days open than ME milk. Combining  $PTA_{DO}$  with both  $PTA_{DF}$  and  $PTA_M$  provided a slight gain in  $REL_{DO}$  compared to using only  $PTA_{DF}$ . Yield would be weighted negatively if used as a predictor of days open, which could discourage some producers from using days open evaluations. Of the traits studied here, dairy form appears to be the most valuable early indicator of days open.

## Chapter 7

### Summary and Conclusions

Heritable variation existed for BCS that was recorded by producers and herd consultants using PCDART dairy management software. BCS recorded during the postpartum period was more highly heritable than early lactation BCS loss. Selection for higher yield increased BCS loss in early lactation. However, genetic correlations were moderate and higher levels of yield are attainable while limiting the amount BCS loss in early lactation. Increased BCS loss as a correlated response to selection occurs by lowering postpartum BCS more than BCS at calving. Increases in BCS loss and lower postpartum BCS are associated with an increase in days to first service.

Studies involving BCS recorded by producers have helped to investigate the genetic relationship among BCS, BCS loss, yield and reproductive performance. The accuracy of genetic evaluations from BCS recorded by producers using PCDART would be lower than a centralized BCS recording scheme because BCS evaluation procedures are not standardized across herds and the date when BCS is assigned is not recorded. National genetic evaluations for BCS are likely to come from BCS recorded during linear type appraisal by classifiers from the Holstein Association USA, Inc.

BCS was a new trait for classifiers and all classifiers did not record BCS normally. Edits were made to generate a BCS data set that would likely represent BCS recorded by classifiers as they become more accustomed to scoring body condition. These edits increased the heritability estimate of BCS and were genetically correlated with unedited BCS by 0.995. The heritability of edited BCS from the Holstein Association USA, Inc. was moderate (0.22).

BCS recorded by classifiers was highly correlated (0.87) with BCS recorded by producers in PCDART. Therefore, genetic evaluations could be generated with BCS from classifiers that would accurately reflect variation in BCS that is recorded and used by producers as a herd management tool.

Cows with higher BCS are genetically inclined to have low dairy form and higher strength scores. Cows with high BCS likely have more fat and tissue surrounding their ribs, causing them to appear less open ribbed and thus lower in dairy form. It is also likely that many classifiers tend to look at the general angularity of a cow when assigning dairy form scores. Cows with high BCS will look rounder and less angular and tend to receive a lower dairy form score. Those same cows likely exhibit more width through the chest resulting in higher strength scores. BCS is more independent of final score than either dairy form or strength, however.

Random regression models were applied to both BCS and dairy form to determine the effectiveness of repeatability models for both traits across different ages and stages of lactation in addition to accessing the potential to generate evaluations for change in BCS or dairy form. Evaluations from random regression models may be of value if a strong association were found between change in BCS or dairy form and an economically important trait, like reproductive performance. If correlations of BCS or dairy form with an important trait changed across the lactation, then evaluations from random regression models for DIM when the relationship is strongest might be of value.

Genetic correlations between BCS or dairy form at different DIM, lactations and ages are high and change in BCS and dairy form is not as heritable as the level of BCS or dairy form. Large daughter groups would be necessary to estimate accurate evaluations for change in BCS or dairy form. The repeatability models used currently in national genetic evaluation programs in the US would appear to generate accurate PTAs for BCS or dairy form at any age or stage of lactation.

Genetic correlations among BCS, dairy form and disease from both the US and Denmark were generated. Low body condition score and high dairy form were genetically correlated with an increase in metabolic disease and poorer cow health. The relationship between dairy form and cow health remained significant after adjustment for BCS. Selection for higher BCS or lower dairy form would help maintain current levels of cow health while selection continues for higher milk, fat and protein yields. However, evaluations for BCS or dairy form would need to be combined with some direct health evaluations to make significant progress in selection for cow health.

Higher BCS and lower dairy form were genetically associated with fewer days open (or higher daughter pregnancy rates). Combining dairy form evaluations with evaluations for days open would increase the reliability of days open evaluations for recently proven bulls. The average increase in reliability of days open was 0.06 for a sample of recently proven bulls when dairy form evaluations contributed to days open. BCS evaluations added little information when dairy form evaluations were available.

While BCS was favorably correlated genetically with cow health and reproductive performance in these studies, significant genetic effects were reduced when dairy form was included in models. Dairy form remained significantly correlated with health and reproductive performance after adjustment for BCS and was more highly heritable in these studies. Genetic evaluations for dairy form are currently available and it is not clear that BCS evaluations would generate any essential information for producers.

BCS was probably not scored as accurately as dairy form in these studies, however. More accurate BCS may result in a relationship among cow health and reproductive performance that is as strong as relationships with dairy form. If true, there are potential advantages to having BCS evaluations in addition to dairy form evaluations. Many producers and nutritional researchers are more familiar with BCS than with dairy form. Producers and researchers may be able to use BCS evaluations more effectively than dairy form evaluations simply because they understand what BCS represents. Moreover, dairy form contributes positively to final score. Producers may be less reluctant to select for higher BCS than for lower dairy form.

It is possible that dairy form is a more complete measure of body composition than BCS. The relationship between BCS and body composition has been investigated but the relationship between dairy form and body composition has not been well defined. Dairy form may be more highly correlated with differences in body fat than BCS, particularly through the front end and over the rib structure. Body condition scores are generally assigned based on a visual assessment of the pelvic region only, while dairy form is assigned based on a visual assessment of the rib structure.

Dairy form and BCS could be effective indicator traits for selection to improve cow health or improve reproductive performance. Dairy form evaluations or BCS

evaluations will need to be coupled with direct observations for cow health and reproductive performance to make significant genetic progress in cow health and fitness. Selection for higher BCS or lower dairy form will increase energy stores and improve the energy balance of cows in early lactation. Improved energy balance will result in cows that have improved reproductive performance and that are less susceptible to disease, particularly metabolic disease.

## List of References

Abdallah, J. M. and B. T. McDaniel. 2000. Genetic parameters and trends of milk, fat, days open, and body weight after calving in North Carolina experimental herds. *J. Dairy Sci.* 83:1364-1370

AIPL-USDA. 2003. Genetic Trend Estimates.  
<http://aipl.arsusda.gov/dynamic/trend/current/trndx.html>. Accessed June 11, 2003.

Baird, G.D. 1982. Primary ketosis in the high producing dairy cow: Clinical and subclinical disorders, treatment, prevention, and outlook. *J. Dairy Sci.* 65:1-10.

Bauman, D.E., and W.B. Currie 1980. Partitioning of nutrients during pregnancy and lactation: a review of mechanisms involving homeostasis and homeorhesis. *J. Dairy Sci.* 63:1514-1529.

Berry, D. P., F. Buckley, P. Dillon, R.D. Evans, M. Rath, and R.F. Veerkamp. 2002. Genetic parameters for level and change of body condition score and body weight in dairy cows. *J. Dairy Sci.* 85:2030-2039.

Berry, D.P., F. Buckley, P. Dillon, R. D. Evans, M. Rath, and R. F. Veerkamp. 2003. Genetic Relationships among Body Condition Score, Body Weight, Milk Yield, and Fertility in Dairy Cows *J. Dairy Sci.* 86:2193-2204.

Boettcher, P.J., L.B. Hansen, H. Chester-Jones, and C.W. Young. 1993. Responses of yield and conformation to selection for milk in a designed experiment with a control population. *J. Dairy Sci.* 76:267-274.

Butler, W.R., R.W. Everett, and C.E. Coppock. 1981. The relationships between energy balance, milk production and ovulation in postpartum Holstein cows. *J. Anim. Sci.* 53:742-748.



- Calo, L.L., R.E. McDowell, L.D. VanVleck and P.D. Miller. 1973. Genetic aspects of beef production among Holstein-Friesians pedigree selected for milk production. *J. Anim. Sci.*, 37:676-682.
- Collard, B. L., P. J. Boettcher, J. C. Dekkers, D. Petitclerc, and L. R. Schaeffer. 2000. Relationships between energy balance and health traits of dairy cattle in early lactation. *J. Dairy Sci.* 83:2683-2690.
- Dadati, E., B.W. Kennedy and E.B. Burnside. 1986. Relationships between conformation and calving interval in Holstein cows. *J. Dairy Sci.* 69:3112-3119.
- de Vries, M. J., and R. F. Veerkamp. 2000. Energy balance of dairy cattle in relation to milk production variables and fertility. *J. Dairy Sci.* 83:62-69.
- de Vries, M. J., S. Van Der Beek, L. M. T. E. Kaal-Lansbergen, W. Ouweltjes, and J. B. M. Wilmink. 1999. Modeling of energy balance in early lactation and the effect of energy deficits in early lactation on first detected estrus postpartum in dairy cows. *J. Dairy Sci.* 82:1927-1934.
- Danish Cattle Federation. 2002. Principles of Danish Cattle Breeding. Available at: <http://www.lr.dk/kvaeg/diverse/principles.pdf>. Accessed June 11, 2003.
- Dechow, C.D., G.W. Rogers, and J.S. Clay. 2001. Heritabilities and correlations among body condition scores, production traits, and reproductive performance. *J. Dairy Sci.* 84: 266-275.
- Dechow, C.D., G.W. Rogers, and J.S. Clay. 2002. Heritability and correlations among body condition score loss, body condition score, production and reproductive performance. *J. Dairy Sci.* 85: 3062-3070.

- Dechow, C.D., G.W. Rogers, L. Klei, and T.J. Lawlor. 2003. Heritabilities and correlations among body condition score, dairy form and selected linear type traits. *J. Dairy Sci.* 86:2236-2242.
- Domecq, J. J., A. L. Skidmore, J. W. Lloyd, and J. B. Kaneene. 1997. Relationship between body condition scores and conception at first artificial insemination in a large dairy herd of high yielding Holstein cows. *J. Dairy Sci.* 80:113-120.
- Domecq, J. J., A. L. Skidmore, J. W. Lloyd, and J. B. Kaneene. 1995. Validation of body condition scores with ultrasound measurements of subcutaneous fat of dairy cows. *J. Dairy Sci.* 78:2308-2313.
- Enevoldsen C., and T. Kristensen. 1997. Estimation of body weight from body size measurements and body condition scores in dairy cows. *J Dairy Sci.* 80:1988-1995.
- Falconer, D. S., and T. F. C. Mackay. 1996. page 316 *in* Introduction to Quantitative Genetics. 4<sup>th</sup> Edition. Longman, Inc. New York, NY.
- Gallo L, P. Carnier, M. Cassandro, R. Dal Zotto, and G. Bittante. 2001. Test-day genetic analysis of condition score and heart girth in Holstein Friesian cows. *J Dairy Sci.* 84:2321-2326.
- Garnsworthy, P.C., and G.P. Jones. 1987. The influence of body condition at calving and dietary protein supply on voluntary food intake and performance in dairy cows. *Anim.Prod.* 44: 347-353.
- Gillund, P., O. Reksen, Y.T Gröhn, and K. Karlberg. 2001. Body condition related to ketosis and reproductive performance in Norwegian dairy cows. *J. Dairy Sci.* 84: 1390-1396.

Gilmour, A.R., B.R. Cullis, S.J. Welham, and R. Thompson. 2000. ASREML Reference Manual.  $\beta$  version. <ftp://ftp.res.bbsrc.ac.uk/pub/aar/>. Accessed Mar. 25, 2002.

Gilmour, A.R., B.R. Cullis, S.J. Welham, and R. Thompson. 2002. ASREML Reference Manual. <ftp://ftp.res.bbsrc.ac.uk/pub/aar/>. Accessed July 10, 2003.

Goff, J. P. and R. L. Horst. 1997. Physiological changes at parturition and their relationship to metabolic disorders. *J. Dairy Sci.* 80:1260-1268.

Gröhn, Y. T., S. W. Eicker, V. Ducrocq, and J. A. Hertl. 1998. Effect of diseases on the culling of Holstein dairy cows in New York State. *J. Dairy Sci.* 81:966-978.

Hansen, L. B. 2000. Consequences of selection for milk yield from a geneticist's viewpoint. *J. Dairy Sci.* 83:1145-1150.

Hansen L.B., J.B. Cole, G.D. Marx, and A.J. Seykora. 1999. Productive life and reasons for disposal of Holstein cows selected for large versus small body size. *J Dairy Sci.* 82:795-801.

Hansen M., M.S. Lund, M.K. Sorensen, and L.G. Christensen. 2002. Genetic parameters of dairy character, protein yield, clinical mastitis, and other diseases in the Danish Holstein cattle. *J Dairy Sci.* 85:445-452.

Harris, B. and D. Johnson. 1998. Approximate reliability of genetic evaluations under an animal model. *J. Dairy Sci.* 81:2723-2728.

Harrison, R. O., S. P. Ford, J. W. Young, A. J. Conley, and A. E. Freeman. 1990. Increased milk production versus reproductive and energy status of high producing dairy cows. *J. Dairy Sci.* 73:2749-2758.

Holstein Association USA Inc. 2002. Sire Summaries. August 2002.

Holstein Canada. 2003. Dairy character.

<http://www.holstein.ca/English/TC/dairychar.asp>. Accessed Jan. 7, 2003.

Interbull. 2003a. Estimated genetic parameters: Appendix II.

<http://www.interbull.slu.se/conform/framesida-conf.htm>. Accessed Jan. 7, 2003.

Interbull. 2003b. Service documentation Holstein conformation.

[http://www.interbull.slu.se/service\\_documentation/Conformation/framesida-conformation.htm](http://www.interbull.slu.se/service_documentation/Conformation/framesida-conformation.htm). Accessed Jan. 7, 2003.

Jamrozik, J., L. R. Schaeffer and J. C. Dekkers 1997. Genetic evaluation of dairy cattle using test day yields and random regression model. *J. Dairy Sci.* 80:1217-1226.

Jensen J. 2001. Genetic evaluation of dairy cattle using test-day models. *J Dairy Sci.* 84:2803-2812.

Jones, H.E., I.M.S. White, and S. Brotherstone. 1999. Genetic evaluation of Holstein Friesian sires for daughter condition-score changes using a random regression model. *Animal Science.* 68:467-475.

Jones, W. P., L. B. Hansen, and H. Chester-Jones. 1994. Response of health care to selection for milk yield of dairy cattle. *J. Dairy Sci.* 77: 3137-3152.

Kadarmideen, H. N., R. Thompson and G. Simm. 2000. Linear and threshold model genetic parameters for disease, fertility and milk production in dairy cattle. *Animal Science.* 71:411-419.

Kirkpatrick M., D. Lofsvold, and M. Bulmer. 1990. Analysis of the inheritance, selection and evolution of growth trajectories. *Genetics*. 124:979-993.

Koenen E.P.C., and A.F. Groen. 1998. Genetic evaluation of body weight of lactating Holstein heifers using body measurements and conformation traits. *J Dairy Sci*. 81:1709-1713.

Koenen, E.P.C., R.F. Veerkamp, P. Dobbelaar, and G. De Jong. 2001. Genetic analysis of body condition score of lactating Dutch Holstein and Red-and-White heifers. *J. Dairy Sci*. 84: 1265-1270.

Lin H.K., P.A. Oltenacu, L.D. Van Vleck, H.N. Erb and R.D. Smith. 1989. Heritabilities of and genetic correlations among six health problems in Holstein cows. *J. Dairy Sci*.72:180-186.

Loeffler, S. H., M. J. de Vries, and Y. H. Schukken. 1999. The effects of time of disease occurrence, milk yield, and body condition on fertility of dairy cows. *J. Dairy Sci*. 82:2589-2604.

Meyer, K. 1998. DFREML: version 3.0  $\beta$ . <http://agbu.une.edu.au/~kmeyer/dfreml.html>. Accessed Mar. 25, 2002.

Misztal I., T. Strabel, J. Jamrozik, E.A. Mantysaari, T.H. Meuwissen. 2000. Strategies for estimating the parameters needed for different test-day models. *J Dairy Sci*. 83:1125-1134.

Misztal, I., K. Weigel and T.J. Lawlor. 1995. Approximation of Estimates of (Co)variance Components with Multiple-Trait Restricted Maximum Likelihood by Multiple Diagonalization for More Than One Random Effect. *J Dairy Sci*. 78:1862-1872.

Misztal, I., T.J. Lawlor, T.H. Short, and P.M. VanRaden. 1992. Multiple-trait estimation of variance components of yield and type traits using an animal model. *J Dairy Sci.* 75:544-551.

Nash, D. L., G. W. Rogers, J. B. Cooper, G. L. Hargrove, J. F. Keown, and L. B. Hansen. 2000. Heritability of clinical mastitis incidence and relationships with sire transmitting abilities for somatic cell score, udder type traits, productive life, and protein yield. *J. Dairy Sci.* 83:2350-2360.

Nocek, J. E. 1997. Bovine acidosis: implications on laminitis. *J. Dairy Sci.* 80:1005-1028.

O'Mara, F. M., S. E. Williams, J. D. Tatum, G. G. Hilton, T. D. Pringle, J. W. Wise and F. L. Williams. Prediction of slaughter cow composition using live animal and carcass traits. *J. Anim Sci.* 76:1594-1603.

Philipsson, J., G. Banos and T. Arnason. 1994. Present and future uses of selection index methodology in dairy cattle. *J. Dairy Sci.* 77:3252-3261.

Pösö J. and E. A. Mäntysaari. 1996. Genetic relationships between reproductive disorders, operational days open and milk yield. *L. Prod. Sci.* 46:41-48.

Pryce, J.E., M.P. Coffey, and G. Simm. 2001. The relationship between body condition score and reproductive performance. *J. Dairy Sci.* 84:1508-1515.

Pryce, J.E., M.P. Coffey, and S. Brotherstone. 2000. The genetic relationship between calving interval, body condition score and linear type and management traits in registered Holsteins. *J. Dairy Sci.* 83:2664-2671.

- Pryce, J. E., M. P. Coffey, S. H. Brotherstone, and J. A. Woolliams. 2002. Genetic relationships between calving interval and body condition score conditional on milk yield. *J. Dairy Sci.* 85:1590-1595.
- Ravagnolo, O. and I. Misztal. 2002. Effect of Heat Stress on Nonreturn Rate in Holsteins: Fixed-Model Analyses. *J. Dairy Sci.* 85:3101-3106.
- Rogers, G. W., G. Banos, and U. Sander-Nielsen. 1999. Genetic correlations among protein yield, productive life, and type traits from the United States and diseases other than mastitis from Denmark and Sweden. *J. Dairy Sci.* 82:1331-1338.
- Rogers, G. W., G. Banos, U. S. Nielsen, and J. Philipsson. 1998. Genetic correlations among somatic cell scores, productive life, and type traits from the United States and udder health measures from Denmark and Sweden. *J. Dairy Sci.* 81:1445-1453.
- Royal, M. D., J. E. Pryce, J. A. Woolliams and A. P. F. Flint. 2002. The Genetic Relationship between Commencement of Luteal Activity and Calving Interval, Body Condition Score, Production, and Linear Type Traits in Holstein-Friesian Dairy Cattle. *J. Dairy Sci.* 85:3071-3080.
- Short, T.H., and T.J. Lawlor. 1992. Genetic parameters of conformation traits, milk yield, and herd life in Holsteins. *J. Dairy Sci.* 75:1987-1998.
- Short, T.H., T.J. Lawlor and K.L. Lee. 1991. Genetic parameters for three experimental linear type traits. *J. Dairy Sci.* 74:2020-2025.
- Treacher, R.J., I.M. Reid, and C.J. Roberts. 1986. Effect of body condition score at calving on health and performance of dairy cows. *Anim. Prod.* 43:1-6.

Tsuruta S., I. Misztal, L. Klei, T.J. Lawlor. 2002a. Analysis of age-specific predicted transmitting abilities for final scores in Holsteins with a random regression model. *J. Dairy Sci.* 85:1324-1330.

Tsuruta, S., I. Misztal, T.J. Lawlor, and L. Klei. 2002b. Estimation of changes of genetic parameters over time for type traits in Holsteins using random regression models. Proc. 7<sup>th</sup> World Congr. Genet. Appl. Livest. Prod., Montpellier, France.

Tveit, F. Lingaas, M. Svendsen, and O. V. Sjaastad. 1992. Etiology of acetonemia in Norwegian cattle. 1. Effect of ketogenic silage, season, energy level, and genetic factors. *J. Dairy Sci.* 75: 2421-2432.

Uribe, H. L.R. Schaeffer, J. Jamrozik, and T.J. Lawlor. 2000. Genetic evaluation of dairy cattle for conformation traits using random regression models. *J. Anim. Breed. Genet.* 117:247-259.

Van Dorp, R., and P. Boettcher. 1999. Technical report: Genetic parameters of mobility, body condition score, and conformation traits.  
<http://cgil.uoguelph.ca/pub/pbpapers/gebtc99-4.htm>. Accessed Jan. 7, 2003.

Van Dorp, T. E., J. C. Dekkers, S. W. Martin, and J. P. Noordhuizen. 1998. Genetic parameters of health disorders, and relationships with 305-day milk yield and conformation traits of registered Holstein cows *J. Dairy Sci.* 81: 2264-2270.

VanRaden, P.M. 2001. Methods to combine estimated breeding values obtained from separate sources. *J. Dairy Sci.* 84(E. Suppl.):E47-E55.



VanRaden, P.M., A. Sanders, M. Tooker, B. Miller and D. Norman. 2002. Daughter pregnancy rate evaluation of cow fertility. AIPL Research Report: DPR1 (11-02). Available at: [http://aipl.arsusda.gov/reference/fertility/DPR\\_rpt.htm](http://aipl.arsusda.gov/reference/fertility/DPR_rpt.htm). Accessed June 11, 2001.

Veerkamp R. F., J. K. Oldenbroek, H. J. Van Der Gaast, and J. H. J. Van Der Werf. 2000. Genetic correlation between days until start of luteal activity and milk yield, energy balance, and live weights. *J. Dairy Sci.* 83:577-583.

Veerkamp R.F., C.L. Gerritsen, E.P.C. Koenen, A. Hamoen, and G. De Jong. 2002. Evaluation of classifiers that score linear type traits and body condition score using common sires. *J Dairy Sci.* 85:976-983.

Veerkamp R.F., E.P.C. Koenen, and G. De Jong. 2001. Genetic correlations among body condition score, yield, and fertility in first-parity cows estimated by random regression models. *J Dairy Sci.* 84:2327-2335.

Veerkamp, R. F. 1998. Selection for economic efficiency of dairy cattle using information on liveweight and feed intake: a review. *J. Dairy Sci.* 81:1109-1119.

Veerkamp, R.F., and S. Brotherstone. 1997. Genetic correlations between linear type traits, food intake, live weight and condition score in Holstein Friesian dairy cattle. *Anim. Sci.* 64:385-392.

Waltner, S. S., J. P. McNamara, and J. K. Hillers. 1993. Relationships of body condition score to production variables in high producing Holstein dairy cattle. *J. Dairy Sci.* 76:3410-3419.

Weigel, K. A. and R. Rekaya. 2000. Genetic parameters for reproductive traits of Holstein cattle in California and Minnesota. *J. Dairy Sci.* 83:1072-1080.

Weigel, K. A., R. W. Palmer, and D. Z. Caraviello. 2003. Investigation of Factors Affecting Voluntary and Involuntary Culling in Expanding Dairy Herds in Wisconsin using Survival Analysis. *J. Dairy Sci.* 86:1482-1486.

Weigel, K. A., T. J. Lawlor, Jr, P. M. Vanraden, and G. R. Wiggans. 1998. Use of linear type and production data to supplement early predicted transmitting abilities for productive life. *J. Dairy Sci.* 81:2040-2044.

Wildman, E. E., G. M. Jones, P. E. Wagner, R. L. Boman, H. F. Troutt, and T. N. Jr. Lesch. 1982. A dairy cow body condition scoring system and its relationship to selected production characteristics. *J. Dairy Sci.* 65:495-501.

## **Vita**

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