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

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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 bivariate 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 nonproduction traits were analyzed with and without mature equivalent 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: 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 (Butler et al., 1981; Baird, 1982; Loeffler et al., 1999; de Vries and Veerkamp, 2000). BCS is a subjective measure of body tissue reserves and is commonly used to monitor energy balance during the lactation (Wildman et al., 1982).

Genetic parameters for BCS have been reported by several authors (Veerkamp, 1998; Jones et al., 1999; Dechow et al., 2001; Koenen et al., 2001). 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 (Pryce et al., 2000; Dechow et al., 2001; 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 (Rogers et al., 1999; Hansen et al., 2002).

Direct estimates of the heritability of body condition score loss (BCSL) and the genetic relationship among

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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 DIM 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 DIM when postpartum BCS was recorded is likely to be slightly less than the average DFS. The average DFS 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.

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

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

¹Services per conception used in genetic analyses.

²Services per conception used to investigate the phenotypic relationship between SPC and DFS.

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 4536 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 reproduc-

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
1st Lactation						
Calving	17,316	3.18	25.29	...	2325	188
Postpartum	10,728	2.91	25.30	...	1806	178
First service	5828	2.87	26.08	...	1384	177
Pregnancy check	12,405	2.96	25.87	...	2003	205
Before dry off	9639	3.12	26.26	...	1781	204
Dry off	9214	3.31	25.26	...	1620	155
BCS Loss ¹	7424	0.30	24.97		1381	115
2nd Lactation						
Calving	13,937	3.07	39.03	421.51	2097	203
Postpartum	8308	2.82	39.14	425.17	1654	177
First service	4562	2.81	39.83	422.12	1193	177
Pregnancy check	8865	2.92	39.36	420.90	1600	210
Before dry off	6328	3.19	39.96	425.78	1373	196
Dry off	8105	3.45	38.78	415.41	1617	171
BCS loss ¹	6092	0.29	38.81	423.79	1317	125

¹Body condition score loss is defined as BCS at calving minus postpartum BCS.

tive 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, 9656 cows had BCS available in one scoring period only, 6524 in two scoring periods, 6430 in 3 scoring periods and 5,167 in four or more scoring periods. Body condition scores were available for 6496, 5300, 5287 and 3853 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 postpar-

tum 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 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 7424 cows in first lactation and 6092 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 bivariate 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:

$$y_{ij} = \mathbf{b} \times \text{age} + \mathbf{hys}_i + \text{animal}_j + \mathbf{e}_{ij}$$

where

y = a vector of BCSL and one of the following:
BCS at calving or postpartum, ME milk,
ME fat, ME protein, DFS, or SPC,

age = age at calving in months,

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

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

animal $_j$ = j th random animal effect, and

$\mathbf{e}_{ij}$ = 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 5390 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 DIM when postpartum BCS was recorded was not known, DIM was not included in the model. However, DIM 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 DIM 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:

$$y_i = b_1 \times \text{age} + \mathbf{hys}_i + \sum_{b=2}^5 \text{DFS} + \mathbf{e}_i$$

where

y = SPC,

age = age at calving in months,

b_1 = regression coefficient on age at calving
in months,

b_2 to b_5 are regression coefficients on polynomials

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.¹

	Genetic correlations		Phenotypic correlations	
	First lactation	Second lactation	First lactation	Second lactation
Calving ²	-0.15	-0.29	0.55	0.53
Calving ^{2,4}	-0.17	-0.11	0.55	0.54
Calving ³	-0.26	-0.48	NA	NA
Calving ^{3,4}	-0.24	-0.44	NA	NA
Postpartum ²	-0.72	-0.89	-0.69	-0.64
Postpartum ^{2,4}	-0.70	-0.91	-0.68	-0.62
Postpartum ³	-0.81	-0.56	NA	NA
Postpartum ^{3,4}	-0.99	-0.64	NA	NA

¹Approximate 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.

²Correlations derived using all available BCS and BCS loss observations. Cows with BCS loss observations also had BCS observations.

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

⁴ME Milk included as a covariable.

of order 1 to 4 for DFS,
 hys_i = i th fixed effect for herd-year-season of calving, and
 e_i = 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 ($P > 0.24$).

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. 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

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

	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

¹Approximate 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.

around 175 DIM. 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 esti-

mates in all three studies tend to be highest for BCS measured in consecutive months or stages of lactation.

A second factor likely contributing to the low heritability for BCSL in this study was the inability to account for the DIM 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, an increase in 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

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.¹

	Genetic correlations		Phenotypic correlations	
	First lactation	Second lactation	First lactation	Second lactation
DFS	0.68	0.44	0.09	0.06
DFS ²	0.52	0.29	0.08	0.05
SPC	0.20	-0.21	0.02	-0.01
SPC ²	0.16	-0.46	0	-0.03

¹Approximate 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.

²ME milk included as covariate in model.

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

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 lac-

tation 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.

BCSL 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.

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