Genetic Relationship Between First-Lactation Body Energy and Later-Life Udder Health in Dairy Cattle

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ABSTRACT

Weekly body condition score (BCS) and live weight records were used to calculate energy content (EC) and cumulative effective energy balance (CEEB) for 508 Holstein-Friesian cows in their first lactation. Cows were raised on an experimental farm and had calved between 1991 and 2000. Energy content was an estimate of the actual energy level of a cow at any given stage of lactation, whereas CEEB was associated with the total body energy content as defined by accumulated weekly energy balance changes since the onset of lactation. Genetic evaluations were computed for the 3 body energy traits (BCS, EC, and CEEB) for each week of first lactation. Random regression models were used to assess the association between first-lactation weekly genetic evaluations for body energy and monthly test-day log-transformed SCC, clinical mastitis, and other udder problems in the first 3 lactations. There was a significant effect of at least one body energy trait at any stage of first lactation past wk 3 on SCC in the first 3 lactations. Maximum genetic correlation estimates were $-0.18 (\pm 0.04)$ between wk-16 BCS and SCC in the first 2 lactations, $-0.18 (\pm 0.04)$ between wk-11 EC and SCC in the first 2 lactations, and $-0.17 (\pm 0.07)$ between wk-6 CEEB and SCC in the first 2 lactations. The effect of body energy traits on clinical mastitis was, in general, nonsignificant; nevertheless, moderate genetic correlations were estimated, ranging from $-0.05 (\pm 0.07)$ to $-0.25 (\pm 0.15)$. The effect of body energy traits on udder problems other than mastitis was negligible in all cases. Results suggest that, amongst the traits studied here, BCS, EC, and CEEB in the first 3 to 4 mo of lactation 1 had the greatest genetic association with SCC and mastitis in first, second, and, to a lesser extent, third lactations.

Key words: energy trait, udder health, dairy cattle

INTRODUCTION

Cows require sufficient energy intake to support their physiological functions, namely to produce milk and grow while reproducing and maintaining metabolic balance and good health. When energy intake is not sufficient, cows are in negative energy balance, which may lead to undesirable health conditions. Collard et al. (2000) reported increased digestive and locomotive problems because of prolonged periods in negative energy balance. Goff and Horst (1997) found that insufficient energy intake was associated with metabolic disorders. Adverse effects of unfavorable energy status on follicular development (Beam and Butler, 1998) and ovarian activity (Kendrick et al., 1999; Reist et al., 2000; Veerkamp et al., 2000) have also been reported.

Various body energy measures have been proposed in the literature (Collard et al., 2000; Veerkamp et al., 2000; Coffey et al., 2001, 2002; Banos et al., 2005). Coffey et al. (2001) calculated measures based on changes in live weight and BCS throughout a cow's first lactation. Such measures are desirable because they are attainable in a commercial dairy cattle population. Body condition scoring may be incorporated into a routine cow classification program, as is currently practiced in the United Kingdom, and live weight, if unavailable, can be predicted from linear conformation traits (Koenen and Groen, 1998; Coffey et al., 2003).

Mastitis remains the costliest disease afflicting dairy cattle. In the United Kingdom, direct selection to reduce mastitis incidence is currently not possible due to lack of credible field data. Somatic cell count concentration in milk is used as a proxy for mastitis incidence because of the established genetic relationship between the 2 traits (Emanuelson, 1988; Shook, 1993; Philipsson et al., 1995; Mrode and Swanson, 2003).

Unfavorable body energy status might lead to elevated SCC and mastitis incidence, thereby compromising udder health. In fact, significant genetic correlation estimates between BCS and clinical mastitis have been documented (Lassen et al., 2003; Dechow et al., 2004). However, the genetic relationship between other body energy traits and udder health is not known.

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Table 1. Descriptive statistics of live weight, BCS, energy content (EC), cumulative effective energy balance (CEEB), milk SCC, and natural log-transformed SCC (LSCC)

Trait	No. of records	No. of cows	No. of lactations	Mean	SD	Minimum	Maximum
Live weight (kg)	16,520	508	1	553	55	355	860
BCS	16,520	508	1	2.57	0.43	1.00	4.00
EC (MJ)	16,520	508	1	4,634	837	2,106	8,896
CEEB (MJ)	16,520	508	1	77	990	-3,677	4,339
SCC (1000/mL)	8,549	399	3	134	356	1.2	7,131
LSCC	8,549	399	3	10.97	1.12	6.91	15.78

The objective of this study was to examine the genetic relationship between body energy traits measured throughout first lactation and udder health traits in first through third lactations, in dairy cattle.

MATERIALS AND METHODS

Body Energy Traits

A total of 16,520 weekly live weight and BCS records of 508 first-lactation Holstein-Friesian cows were obtained from the Langhill Dairy Cattle Research Centre in Scotland (Table 1). These cows had calved between 1991 and 2000 and participated in feed and selection trials in which they had been grouped according to diet (high vs. low concentrates offered in a TMR) and genetic merit (sired by high vs. average merit bulls).

Weekly live weight and BCS records were used to calculate body lipid and protein weights using the formulas proposed by the US National Research Council based on the Cornell Nutrient Management Planning System (NRC, 2001). Estimated body lipid and protein weights were then combined to calculate energy content (EC). This trait relates to the absolute level of energy that the body of a cow contains on each day of measurement, regardless of what the energy intake and use had been on previous days. The calculation procedure is presented in detail in Appendix 1; descriptive statistics of EC are shown in Table 1.

Changes in body lipid and protein weights during the lactation were used to derive effective energy balance based on the system of Emmans (1994). This system describes the amount of energy processed when lipid and protein weights change. Furthermore, the system differentiates between cases of weight gain and loss. Cumulative effective energy balance (CEEB) was calculated based on the cumulative lipid and protein change since the beginning of lactation. This trait relates to total body energy as it accumulates throughout lactation. Calculation details are presented in Appendix 1 and descriptive statistics for CEEB are shown in Table 1.

First-lactation BCS, EC, and CEEB were defined as the 3 body energy traits of interest in this study. A

genetic analysis was conducted for weekly BCS, EC, and CEEB records using Model 1. Each trait was analyzed separately.

$$Y_{ijklmn} = YS_i + G_j + F_k + a_1 \cdot \text{age} + a_2 \cdot \text{phol} + \sum_{n=0}^4 b_n P_n W_m + \sum_{n=0}^4 c_{ln} P_n W_m + \sum_{n=0}^1 p_{ln} P_n W_m + e_{ijklmn} \quad [1]$$

where Y_{ijklmn} = BCS, EC, or CEEB record of cow l in week m of first lactation, YS_i = fixed effect of year-season of calving i , G_j = fixed effect of selection line j , F_k = fixed effect of feeding group k , a_1 = linear regression coefficient on age at calving (age), a_2 = linear regression coefficient on percentage of North American Holstein genes (phol), W_m = week m of first lactation, b_n = fixed regression coefficient associated with the overall curve, c_{ln} = random regression coefficient associated with the additive genetic value of cow l , p_{ln} = random regression coefficient associated with the permanent environment of cow l , P_n = n th Legendre polynomial of week m (n = degree of Legendre polynomial), and e_{ijklmn} = random residual term.

Complete cow pedigree information available at the Langhill Dairy Cattle Research Centre was included to account for genetic relationships between animals. In this respect, 5,758 animals were included in the analysis.

Genetic variances for each week of first lactation were calculated using the (co)variance estimates for the cow additive genetic effect and corresponding polynomial solutions. Breeding values and reliabilities were also estimated for all animals, for each week of lactation.

Depending on lactation stage, 7 measurement error classes were defined as follows: wk 1 to 2, 3 to 4, 5 to 8, 9 to 12, 13 to 16, 17 to 20, and ≥ 21 . Different residual variances were estimated per measurement error class but residual variance was assumed homogeneous within class. Residual covariances were assumed to be zero.

Table 2. Description of clinical mastitis (CM) and udder problems other than mastitis (UPOM) incidence in the first 3 lactations combined

	CM	UPOM
No. of cows with 3 lactations completed	315	315
No. of cows with at least 1 diagnosis	80	22
Proportion of cows afflicted at least once, %	25.4	7.0
Total number of diagnoses	118	25
Average number of diagnoses per cow	1.48	1.14
SD of diagnoses per cow	0.94	0.14
Maximum number of diagnoses per cow	6	3

Variance component and effect solutions were obtained with the ASREML software package (Gilmour et al., 2002).

Udder Health Traits

Monthly records of natural log-transformed SCC of cows in their first 3 lactations were obtained from the Langhill Dairy Cattle Research Centre in Scotland. Cows were required to have first-lactation data to be included in the analysis. Records of cows that also had live weight and BCS records and, consequently, EC and CEEB measures were kept for 8,549 monthly test-day records of 399 cows (Table 1).

Cases of clinical mastitis (CM) were routinely recorded at the Langhill Dairy Cattle Research Centre. A file of cows diagnosed with mastitis during their first 3 lactations was obtained, complete with date of treatment and lactation number. This file was combined with a list of cows that had completed 3 lactations and were never treated for mastitis. This edit was required to compare animals that had been afflicted with mastitis to animals that had the opportunity for infection during the same period of time but had remained healthy. Overall, 315 cows with BCS, EC, and CEEB records had completed the first 3 lactations, of which 80 (25.4%) had been treated at least once for CM. Descriptive statistics related to CM incidence are given in Table 2.

Two CM traits were defined. The first was the total number of cases per cow during the first three lactations (one record per animal, with values ranging from 0 to 6). The second was a binary trait (0/1) with repeated observations per cow. In this case, each lactation was divided into 30-d intervals. A cow reportedly treated in any interval was assigned a CM record of value 1 for this interval; otherwise the assigned value was 0.

A file including incidence and treatment of udder problems other than mastitis (UPOM) was also obtained from the Langhill Dairy Cattle Research Centre. These problems included teat contusion or blockage, teat or udder injury, amputated teat, edema, treatment

of dry cows having exudates from their teats, bloody milk, hard quarters, and difficult milking. The same kind of edits and trait definitions as for CM were applied here. Table 2 shows descriptive statistics for UPOM incidence.

Genetic Relationship Between Body Energy and Udder Health Traits

Model 2 was used to determine the genetic relationship between body energy and udder health traits.

$$Y_{ijklmno} = (YS_i + G_j + F_k + a_1 \cdot age + a_2 \cdot phol + \sum_{n=0}^2 d_n M + a_3 \cdot P + \sum_{n=0}^4 b_n P_n D_m + \sum_{n=0}^4 c_{ln} P_n D_m)_o + e_{ijklmno} \quad [2]$$

where $Y_{ijklmno}$ = test-day log-transformed SCC or monthly CM or UPOM record (0/1) of cow l in day or month m of lactation o , d_n = regression coefficients on milk yield (M) on day m of lactation o (n = degree of regression), a_3 = linear regression coefficient on genetic evaluation (P) of cow l for BCS, EC, or CEEB in any week of first lactation weighted by the corresponding reliability, and D_m = day or month m of lactation o . The other effects were as in Model 1. Time unit (D) was days for SCC and months (30-d intervals) for CM and UPOM. Milk yield was only included in the analysis of SCC. Preliminary analysis including milk yield on day closest to date of mastitis incidence revealed no significant effect. Weekly genetic evaluation P for energy traits and corresponding reliability had been derived from Model 1. Model 2 was fitted separately for each combination of udder health (SCC, CM, UPOM) and body energy (BCS, EC, CEEB) traits.

In Model 2, the regression on the genetic evaluation for a body energy trait represented a statistic prediction of future udder health from these traits. Sequential analyses were conducted to derive predictions of SCC, CM, and UPOM in the first 3 lactations from all weekly first-lactation genetic evaluations for BCS, EC, and CEEB. In each case, first-lactation udder health records were included only if they were subsequent to the week of genetic evaluation for body energy. For example, when the genetic evaluation fitted in Model 2 was for wk 4, first-lactation SCC records had to be after d 28, whereas CM and UPOM records had to be after the first month (30-d interval) of lactation. Adjusted F -statistics were considered as determinants of significance effects of body energy traits on udder health.

Regressions of SCC, CM, and UPOM on genetic evaluations for BCS, EC, and CEEB were used to calculate

the genetic correlation between these groups of traits, using the following formula:

$$r_G = b \cdot \frac{\sigma_1}{\sigma_2} \quad [3]$$

where r_G = the estimated genetic correlation, b = the regression coefficient calculated from Model 2, and σ_1 and σ_2 = the genetic standard deviation estimates for body energy and udder health traits, respectively. Genetic standard deviations for BCS, EC, and CEEB in each week of first lactation were estimated with Model 1. Genetic standard deviation estimates for SCC, CM and UPOM were obtained by running Model 2 without the regression on body energy genetic evaluation, including pedigree relationships among cows. The resulting genetic correlation would pertain to any first-lactation week of body energy and udder health in subsequent productive life (up to the end of third lactation).

Standard errors of estimated genetic correlations were based on the formula:

$$V(r_G) \equiv r_G^2 \cdot \left[\frac{V(b)}{b^2} + \frac{V(\sigma_1^2)}{4 \cdot \sigma_1^4} + \frac{V(\sigma_2^2)}{4 \cdot \sigma_2^4} \right] \quad [4]$$

where $V(r_G, b, \sigma_1, \text{ or } \sigma_2)$ = the standard error of the estimate squared; other effects as defined in Equation 3.

Estimation of genetic correlations with the above method was considered a practical alternative to conducting bivariate analyses of longitudinal data (weekly energy traits and monthly udder health traits). It was not obvious how the residual structure could be properly modeled in such case; computational aspects would also become an issue.

The chosen method of estimating genetic correlation, which may be sensitive to the genetic variance estimates assumed, has been used in different studies by Brotherstone and Hill (1991), Pryce et al. (2000), and Banos et al. (2004). Given the amount of data and pedigree depth as well as models considered in the present study, these genetic variance estimates are considered robust.

In addition to Model 2, an alternative analysis was conducted for CM and UPOM in which they were defined as the total number of episodes in a cow's productive life (here defined as the first 3 lactations). The model considered for this analysis included the fixed effects of selection line, feeding group, age at first calving, year-season of first calving, and regression on weekly genetic evaluation of the cow for a body energy trait weighted by the corresponding reliability.

RESULTS AND DISCUSSION

Energy Traits

The 3 body energy traits were analyzed with models including fourth-degree Legendre polynomials of week of lactation for the random animal genetic effect. In this case there were 5 parameters to be estimated: intercept (degree 0), linear (degree 1), quadratic (degree 2), cubic (degree 3), and quartic (degree 4). Initially, polynomials of degrees 0 to 4 were sequentially fitted and the log-likelihoods were compared with Schwartz's Bayesian information criterion, which accounts for data set size and number of parameters to be estimated. In each case, inclusion of a higher degree significantly ($P < 0.05$) increased the log likelihood. Increasing the polynomial degree beyond 4 did not result in a significantly larger log likelihood value.

A first-degree (linear) regression on the random permanent environment effect was fitted in this study. Efforts to increase the degree of this regression while including a fourth-degree animal genetic regression led to failure to converge, probably due to limited data set size. The only situation in which a higher (second degree) regression on the permanent environment could be fitted was when the degree of the polynomial for the animal genetic effect decreased to 2. However, the significantly ($P < 0.05$) reduced log likelihood of this model did not justify its usage for the final analysis. Therefore, the chosen model included a fourth-degree random regression on the animal genetic effect and a first-degree random regression on the permanent environment effect.

Banos et al. (2005) fitted the same degree of random (animal genetic and permanent environment) and fixed polynomials in a study of total body energy content. Coffey et al. (2001, 2003) and Banos et al. (2004), while studying BCS and other energy traits, fitted the same degree fixed and third-degree random (animal genetic) polynomials in their respective analyses. However, Coffey et al. (2001) managed to fit a third-degree polynomial for both the animal genetic and permanent environment effect.

Seven measurement error classes were defined according to lactation stage and a different residual variance was estimated per measurement error class. In all cases, individual class estimates differed significantly ($P < 0.05$) from each other. In general, estimates were highest in the first 4 wk of lactation and decreased gradually afterwards, before increasing slightly toward the end of lactation. Coffey et al. (2001) and Banos et al. (2005) reported similar findings.

Genetic analysis of weekly BCS cow records revealed the same picture that has been reported in previous studies (Coffey et al., 2001; Banos et al., 2004). Genetic

Figure 1. Estimated fixed curve of daily energy content (EC, $\square$) and cumulative effective energy balance (CEEB, $\triangle$) by week of first lactation.

variance and heritability estimates increased with lactation stage; weekly heritability estimates ranged from 0.39 to 0.84, in accordance with Coffey et al. (2001) who conducted a genetic analysis with a similar data set. These heritability estimates are expected to be high because they are based on data from a single experimental farm where cows were raised in a controlled environment. Results might also be somewhat inflated because permanent environment was fitted only as a first-degree (linear) regression. For this reason, parts of the permanent environmental variance might have been inadvertently added to the estimated additive genetic and residual variances; however, it is not possible to know the extent of this bias.

Estimated fixed curves of first-lactation EC and CEEB, derived from the genetic analyses of these traits, are shown in Figure 1.

Energy content decreased until wk 10 of first lactation, coinciding with peak milk production, and increased thereafter. Genetic variance and heritability estimates increased with lactation stage; weekly heritability estimates ranged from 0.46 to 0.88.

As defined in this study, EC describes the absolute level of energy that a cow is expected to have on a given day of lactation, as calculated from her estimated body lipid and protein levels on that day. Because it is based on her level of BCS and live weight, EC can be calculated nationally for all classified cows. Linear conformation traits and body condition are scored once in all first-lactation cows participating in the scheme. Live weight can then be predicted from angularity, chest width, body depth, and stature; Coffey et al. (2003) estimated that the correlation between actual and predicted live weight is 0.92. Just like BCS, across the trajectory, EC may be evaluated nationally with a sire model, in which individual daughter records are considered as repeated measures of the same sire. Therefore, EC is amenable to a genetic evaluation at the national level.

Cumulative energy balance decreased during the first 7 wk of lactation and increased afterwards (Figure 1). As with BCS and EC, genetic variance estimates increased with lactation stage and weekly heritability estimates ranged from 0.19 in early lactation to 0.95 in late lactation stages. These estimates are high because they are based on data from a single experimental farm. Banos et al. (2005) used the same data to examine a very similar trait (termed total body energy content in that study), and reported heritability estimates between 0.25 and 0.94 in the various stages of lactation. The scientific literature offers no other estimates for this trait. However, another study of related traits based on experimental farm data has also reported high heritability estimates of 0.48 to 0.61 for body weight, 0.33 for energy balance, and 0.61 for DM intake (Veerkamp et al., 2000).

Cumulative effective energy balance is based on changes in weekly body lipid and protein weights as calculated from changes in BCS and live weight. Therefore, this trait relates to body energy as it accumulates over lactation. This is the reason that Banos et al. (2005) termed it total body energy content. The difference with that work is that, in the present study, updated equations were used for the prediction of body protein and lipid weights, developed by the US National Research Council (NRC, 2001). Banos et al. (2005) had used the relatively long-standing equations proposed by the UK Agricultural Research Council (Emmans, 1994; Coffey et al., 2001). In both studies, however, the effective energy system of Emmans (1994) was used to convert protein and lipid weight change to energy balance. In general, the old equations yielded slightly more variable results than the newer ones.

Unlike EC, CEEB is based on BCS and live weight change; therefore, it cannot be calculated for individual cows in the national herd that are classified only once in their lifetime. It can, however, be calculated at the sire level based on genetic evaluations for BCS and live weight changes across the progeny group of a sire whose daughters are classified at different lactation stages. Coffey et al. (2003) and Banos et al. (2004) demonstrated the feasibility of repeated measure analyses per sire having daughters with single records.

Genetic Association Between Body Energy and Milk SCC

Figure 2 illustrates the effect of BCS, EC, and CEEB on SCC in the first 2 lactations. This effect is quantified by the adjusted *F*-statistic, which is the ratio of the adjusted mean squares due to the energy trait over the residual mean squares. The numerator mean squares was first adjusted for all other fixed effects in Model 2.

Figure 2. Adjusted F -statistic associated with the regression of milk SCC in the first 2 lactations on weekly first-lactation BCS (●), daily energy content (□), and cumulative effective energy balance (△) genetic proofs; significance threshold value of F is 3.8415 ($\alpha = 0.05$).

Because weekly genetic evaluations for the 3 energy traits were fitted sequentially, there was one F -statistic associated with each week of lactation. In all cases, there was a single numerator degree of freedom and, practically, infinite denominator degrees of freedom. Therefore, a genetic evaluation for BCS, EC, or CEEB was deemed to have a significant ($P < 0.05$) effect on SCC if the associated F -statistic exceeded the value of 3.8415. Figure 2 suggests that all BCS genetic evaluations after wk 7 had a significant impact on SCC in the first 2 lactations, with wk 15 having the largest effect. The latter coincides with the time BCS reached its nadir in first lactation.

Early lactation EC and CEEB genetic evaluations in first lactation also had a significant ($P < 0.05$) effect on SCC in the first 2 lactations, with wk 11 and 7, respectively, being the most informative (Figure 2). As with BCS, these weeks correspond to times when either trait reached its lowest point in first lactation (Figure 1), suggesting that the level to which a cow loses body energy may largely determine her propensity to clinical and subclinical udder infection as manifested by SCC in the first 2 lactations.

Figure 3 shows the same F -statistics relating to the effect of BCS, EC, and CEEB on SCC in the first 3

Figure 3. Adjusted F -statistic associated with the regression of milk SCC in the first 3 lactations on weekly first-lactation BCS (●), daily energy content (□), and cumulative effective energy balance (△) genetic proofs; significance threshold value of F is 3.8415 ($\alpha = 0.05$).

Figure 4. Genetic correlation estimates between milk SCC in the first 2 lactations and weekly first-lactation BCS (●, SE = 0.02 to 0.04), daily energy content (□, SE = 0.03 to 0.04), and cumulative effective energy balance (△, SE = 0.04 to 0.07).

lactations. Here, again, genetic evaluations in early lactation (wk 8 to 18) appear to have the greatest impact on SCC in later life. The effect, however, is dampened compared with Figure 2, by the inclusion of third-lactation SCC records. Looking separately at the effect of first-lactation body energy evaluations on third-lactation SCC only revealed a nonsignificant ($P > 0.05$) association between them. This is the reason for the reduced body energy effect on SCC in first 3 compared with first 2 lactations. There may be various explanations for this observation. Distance between first and third compared with first and second lactations is most likely to play a role. In addition, looking at each lactation separately may introduce bias due to data selection, because cows with third lactation constitute a selected group that has avoided voluntary or involuntary culling. In fact, only 62% of all first-lactation cows also had a third lactation. Furthermore, third-lactation cows are considered to have reached, or be very close to reaching, full maturity. Energy that is no longer expended to support growth is partitioned to other functions, including milk yield. This indicates that body energy may be a different trait in different lactations. In fact, Banos et al. (2005) reported genetic correlations between first- and third-lactation total body energy content, a trait very similar to CEEB, as low as 0.45.

Genetic correlation estimates between body energy in various first-lactation stages and SCC in the first 2 and 3 lactations are shown in Figures 4 and 5, respectively. These correlation estimates originate from regressions whose significance levels were presented in Figures 2 and 3, respectively. Consequently, the strongest correlations were observed for weeks near those with the largest F -statistics. Maximum genetic correlation estimates between first-lactation body energy content and first 2 lactations SCC were $-0.18 (\pm 0.04)$, $-0.18 (\pm 0.04)$, and $-0.17 (\pm 0.07)$ for BCS, EC, and CEEB in wk 16, 11, and 6, respectively (Figure 4, Table 3). Consistent with smaller values for F -statistics, such estimates with SCC in the first 3 lactations were lower,

Figure 5. Genetic correlation estimates between milk SCC in the first 3 lactations and weekly first-lactation BCS (●, SE = 0.02 to 0.04), daily energy content (□, SE = 0.03 to 0.04), and cumulative effective energy balance (△, SE = 0.04 to 0.06).

Figure 6. Genetic correlation estimates between mastitis incidence in the first 3 lactations and weekly first-lactation BCS (●, SE = 0.07 to 0.17), daily energy content (□, SE = 0.10 to 0.17), and cumulative effective energy balance (△, SE = 0.15 to 0.20).

maxima being $-0.11 (\pm 0.03)$ for BCS (wk 15), $-0.11 (\pm 0.04)$ for EC (wk 11), and $-0.11 (\pm 0.06)$ for CEEB (wk 6). Table 3 summarizes the maximum genetic correlation estimates between weekly body energy traits and SCC in various lactation subsets. Looking at SCC in each lactation separately, maximum genetic correlation estimates with body energy traits were significantly negative in first (Table 3) and second lactation (-0.22 ± 0.06 , -0.22 ± 0.06 , and -0.18 ± 0.07 for BCS, EC, and CEEB, respectively) but they were practically zero in third lactation, in accordance to the nonsignificant *F*-statistics discussed above.

Data used in this study were from a single experimental farm where cows were raised under a controlled environment. It is possible that these cows were not challenged as much by infectious agents as cows in commercial herds. Indeed, SCC measured here were somewhat lower compared with field data collected in the United Kingdom, as reported by Booth (1993) and Mrode and Swanson (2003).

The above results suggest that cows with high BCS, EC, and CEEB in early first lactation are expected to have low SCC in later life.

Genetic Association of Body Energy with CM and UPOM

Because of low frequency of these diseases, clinical cases in the first 3 lactations were analyzed jointly. Separate analyses were first attempted by lactation

subset, as was done with SCC. However, in all such cases results were not significantly different from zero ($P > 0.05$).

Adjusted *F*-statistics of the regression of CM incidence in the first 3 lactations, expressed as repeated 0/1 measures, on genetic evaluations for first-lactation body energy traits were, in general, nonsignificantly different from zero ($P \geq 0.05$). This suggests that body energy traits may not have as strong genetic association with later-life mastitis as they did with SCC. Despite this result, genetic correlation estimates between weekly genetic evaluations for first-lactation BCS, EC, or CEEB and CM incidence were derived and are shown in Figure 6. These estimates ranged from -0.05 to -0.25 for BCS, between -0.02 and -0.17 for EC, and from -0.07 to -0.21 for CEEB. Genetic evaluation for BCS in wk 26 of first lactation had the highest genetic correlation with CM (-0.25 ± 0.15). The highest genetic correlation between EC and CM was in wk 26 (-0.17 ± 0.16), whereas for CEEB it was in wk 6 (-0.21 ± 0.20). Albeit nonsignificantly different from zero ($P > 0.05$), these results indicate that well-conditioned animals with positive energy balance in first lactation could be more resistant to CM in later life. The estimates are consistent with those of Lassen et al. (2003) who reported a genetic correlation of -0.16 between first-lactation BCS and CM in Danish Holsteins. Dechow et al. (2004) reported genetic correlations between BCS and CM of -0.93 , based on a relatively small data set, and correla-

Table 3. Maximum weekly genetic correlation estimates (SE in parentheses) of first-lactation BCS, energy content (EC), and cumulative effective energy balance (CEEB) with milk SCC in the first 3 lactations

Lactation	BCS		EC		CEEB	
	Week	Estimate	Week	Estimate	Week	Estimate
First	16	$-0.12 (0.03)$	11	$-0.13 (0.04)$	6	$-0.17 (0.07)$
First and second	16	$-0.18 (0.04)$	11	$-0.18 (0.04)$	6	$-0.17 (0.07)$
First, second, and third	15	$-0.11 (0.03)$	11	$-0.11 (0.04)$	6	$-0.11 (0.06)$

tions between US genetic evaluations for BCS and Danish evaluations for CM of -0.25 .

Nonsignificance in most parameters describing the association between body energy traits and CM may be the combined result of fewer data compared with SCC, the binary nature of the trait, and the relatively low frequency of CM in the experimental farm on whose data this study was based. In fact, the proportion of cows that had been afflicted at least once in 3 lactations was 25.4% of all 315 cows that had completed all 3 lactations. This statistic, however, accumulates repeated incidence on the same cow across lactation. Looking at each lactation separately, mastitis incidence was 7.0, 7.0, and 15.5% for first-, second-, and third-lactation cows, respectively. This was an experimental farm with an adequately controlled environment. Higher mastitis frequency than the one observed here is expected in most commercial herds. Such data, however, are not available in the United Kingdom. Elsewhere, Ødegård et al. (2003) and Heringstad et al. (2003), working with different field data, reported a clinical mastitis frequency of 15 and 23.3%, respectively, among first-lactation Norwegian cattle. Nash et al. (2000) reported frequency of 22 and 27% of first- and second-lactation cows, respectively, in certain US herds. Lassen et al. (2003) reported 22% incidence amongst first-lactation Holstein cows in selected Danish herds. In Sweden, data from the national herd revealed mastitis incidences of 10.4, 12.1, and 14.9% in the first 3 lactations of Holstein cows, respectively (Carlén et al., 2004). Clearly the definition of a mastitis case may differ in various studies; however, there is ample evidence that frequencies observed in this experimental farm may not be indicative of mastitis incidence in the commercial population. Therefore, it could be speculated that the nonsignificant results obtained here might actually turn significant in environments with average mastitis frequency.

In the present study, CM incidence was expressed as a string of 0s and 1s across a cow's lactation. Lactations were divided into 30-d intervals and each was assigned a value of 1 if a cow was diagnosed with mastitis or 0 otherwise. This data structure aimed at capturing the repeated nature of mastitis incidence in an environment of potentially continuous challenge by infectious agents. Heringstad et al. (2003) considered a similar trait for the analysis of mastitis data. However, the frequency of 1s, indicating a mastitis episode, was low (just over 1%) in the present study. An alternative analysis was also conducted, where clinical mastitis was defined as the total number of episodes in a cow's productive life (first 3 lactations). Linear regressions on body energy genetic evaluations were nonsignificantly different from zero ($P > 0.05$) in all cases.

The association between any energy traits and UPOM was always negligible, whether the latter was defined as a repeated measure of 0s and 1s or a total number of incidence cases in a cow's first 3 lactations. Adjusted F -statistics were far below the threshold value ($P > 0.05$) and genetic correlation estimates were practically zero. This result is likely due to the very low frequency of these udder problems in the data set considered in the present study.

Final Remarks

Ongoing efforts to reduce mastitis in genetic improvement programs might be more effective if clinical mastitis were routinely recorded and evaluated, and selected for directly. This, however, is not explicitly happening in most countries. Furthermore, recording clinical mastitis does not account for subclinical cases. Udder health-related traits are being widely considered as mastitis indicators to alleviate these problems. Milk SCC is by far the most widespread used udder health-related trait, in this matter.

The utility of the results of this study may be viewed in terms of additional (marginal) gains that can be expected when energy traits are considered together with SCC in a selection scheme to improve udder health. These gains can be calculated based on estimates of the phenotypic and genetic parameters of each trait and the correlation among individual traits, using classical selection index theory (Van Vleck, 1979). For example, given the parameters estimated in the present study, genetic progress in SCC can be expected to be 0.6% higher when selection applies on both SCC and BCS compared with selection applied only on SCC. To derive the above figure, a genetic correlation of -0.18 was used between the 2 traits, which was the maximum estimate in this study and relates to BCS in wk 16 and SCC in the first 2 lactations.

Similarly, genetic progress in mastitis can be viewed as the correlated response to selection based on SCC alone or SCC together with energy traits. This will also be a function of phenotypic and genetic parameters, and the correlation between mastitis and the predictor traits. To illustrate this point, and using parameters estimated in this study, genetic progress in mastitis may be expected to be 1.9% higher when selection is based on both SCC and BCS compared with selection applied only on SCC. To derive the above figure, a genetic correlation of -0.25 was used between BCS and mastitis, which was the maximum estimate in the present study. A genetic correlation of 0.75 between SCC and mastitis was assumed in this example (Philipsson et al., 1995).

In summary, results from this study suggest that modest marginal gains can be expected in udder health when energy traits are considered in a selection program. An additional benefit may emanate from the fact that these correlations associate energy traits measured in early life (early first lactation) with udder health in later life.

Similar experiences have been gained in studies of the correlation between BCS and other economically important characteristics such as cow fertility. In fact, Pryce et al. (2002) showed that single-trait selection for BCS would result in a 3.3-d reduction in calving interval. Admittedly, the genetic correlation between energy traits and fertility is higher than the correlation between energy and udder health. As with fertility, however, the benefits of correlated response in udder health will be based on the relatively higher heritability of the selection trait (energy) and their statistically significant genetic correlation, as well as the fact that energy traits can be available early in a cow's life.

Furthermore, inclusion of energy traits in an overall selection index can be considered, if appropriate genetic parameters are available. This would have an overall favorable effect, not only on udder health but also on other functional traits (Pryce et al., 2002; Dechow et al., 2004).

In the present study 3 energy traits were considered. Two of them (EC and CEEB) were partly derived from the third (BCS). Therefore, correlations among them are expected to be high. In fact, BCS proofs (genetic evaluations) had a correlation of 0.89 and 0.74 with EC and CEEB proofs, respectively. Correlation between EC and CEEB proofs was 0.77. These were unadjusted pooled estimates across weekly genetic evaluations for the 3 traits. Energy content and CEEB were functions of BCS but also of live weight, meaning they may carry additional information related to cows' growth and maintenance requirements as manifested by the latter.

According to results from this study, all 3 traits have similar correlations with SCC and mastitis and would be equally useful in a program aimed at reducing cow predisposition to mastitis and elevated SCC in later life. Preference is given first to BCS and then to EC because they can be directly recorded under commercial conditions on sire daughters at different stages of lactation. Even if a cow is only recorded once, the observations can be viewed as repeated measures of the same sire and evaluated using a random-regression sire model. This would yield sire genetic evaluations at different stages of lactation, from which the most informative can be used in a selection program.

CONCLUSIONS

Results from the present study suggest that genetic evaluations for BCS, daily energy content, and cumula-

tive effective energy balance calculated in the first 3 to 4 mo of the first lactation have a significant genetic correlation with SCC in the first and second and, to a lesser extent, third lactations. Cows with the genetic propensity for high BCS, increased energy content, and positive energy balance in early first lactation are expected to have reduced SCC in later life, suggesting fewer mastitis infections. The genetic association between body energy traits and clinical mastitis, albeit statistically nonsignificant, presented an additional suggestion of the role body energy can play as an auxiliary indicator of the genetic predisposition of future udder health. This may support the idea of including energy traits in an overall index to offset potentially negative side effects of selection for yield on health and other functional traits in dairy cattle.

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REFERENCES

- Banos, G., S. Brotherstone, and M. P. Coffey. 2004. Evaluation of body condition score measured throughout lactation as an indicator of fertility in dairy cattle. *J. Dairy Sci.* 87:2669–2676.
- Banos, G., S. Brotherstone, and M. P. Coffey. 2005. Genetic profile of total body energy content of Holstein cows in the first three lactations. *J. Dairy Sci.* 88:2616–2623.
- Beam, S. W., and W. R. Butler. 1998. Energy balance, metabolic hormones, and early postpartum follicular development in dairy cows fed prilled lipid. *J. Dairy Sci.* 81:121–131.
- Booth, J. M. 1993. Mastitis in the 1990s. *Cattle Pract.* 1:125–131.
- Brotherstone, S., and W. G. Hill. 1991. Dairy herd life in relation to linear type traits and production 1. Phenotypic and genetic analyses in pedigree type classified herds. *Anim. Prod.* 53:279–287.
- Carlén, E., E. Strandberg, and A. Roth. 2004. Genetic parameters for clinical mastitis, somatic cell score, and production in the first three lactations of Swedish Holstein cows. *J. Dairy Sci.* 87:3062–3070.
- Coffey, M. P., G. C. Emmans, and S. Brotherstone. 2001. Genetic evaluation of dairy bulls for energy balance traits using random regression. *Anim. Sci.* 73:29–40.
- Coffey, M. P., G. Simm, and S. Brotherstone. 2002. Energy balance profiles for the first three lactations of dairy cows estimated using random regression. *J. Dairy Sci.* 85:2669–2678.
- Coffey, M. P., G. Simm, W. G. Hill, and S. Brotherstone. 2003. Genetic evaluations of dairy bulls for daughter energy balance profiles using linear type scores and body condition score analyzed with random regression. *J. Dairy Sci.* 86:2205–2212.

- Collard, B. L., P. J. Boettcher, J. C. M. Dekkers, D. Petclerc, and L. R. Schaeffer. 2000. Relationships between energy balance and health traits of dairy cattle in early lactation. *J. Dairy Sci.* 83:2683–2690.
- Dechow, C. D., 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. 2004. Correlations among body condition scores from various sources, dairy form, and cow health from the United States and Denmark. *J. Dairy Sci.* 87:3526–3533.
- Emanuelson, U. 1988. Recording of production diseases in cattle and possibilities for genetic improvements: A review. *Livest. Prod. Sci.* 20:89–106.
- Emmans, G. C. 1994. Effective energy: A concept of energy utilization applied across species. *Br. J. Nutr.* 71:801–821.
- Gilmour, A. R., B. J. Gogel, B. R. Cullis, S. J. Welham, and R. Thompson. 2002. ASREML User Guide. Release 1.0. VSN International Ltd., Hemel Hempstead, UK.
- Goff, J. P., and R. L. Horst. 1997. Physiological changes at parturition and their relationship to metabolic disorders. *J. Dairy Sci.* 80:1260–1268.
- Heringstad, B., Y. M. Chang, D. Gianola, and G. Klemetsdal. 2003. Genetic analysis of longitudinal trajectory of clinical mastitis in first-lactation Norwegian cattle. *J. Dairy Sci.* 2676–2683.
- Kendrick, K. W., T. L. Bailey, A. S. Garst, A. W. Pryor, A. Ahmadzadeh, R. M. Akers, W. E. Eyestone, R. E. Pearson, and F. C. Gwazdauskas. 1999. Effects of energy balance on hormones, ovarian activity, and recovered oocytes in lactating Holstein cows using transvaginal follicular aspiration. *J. Dairy Sci.* 82:1731–1740.
- 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.
- Lassen, J., M. Hansen, M. K. Sørensen, G. P. Aamand, L. G. Christensen, and P. Madsen. 2003. Genetic relationship between body condition score, dairy character, mastitis, and diseases other than mastitis in first-parity Danish Holstein cows. *J. Dairy Sci.* 86:3730–3735.
- Mrode, R. A., and G. J. T. Swanson. 2003. Estimation of genetic parameters for somatic cell count in the first three lactations using random regression. *Livest. Prod. Sci.* 79:239–247.
- 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.
- NRC. 2001. Nutrient Requirements of Dairy Cattle. 7th ed. Natl. Acad. Press, Washington, DC.
- Ødegård, J., G. Klemetsdal, and B. Heringstad. 2003. Genetic improvement of mastitis resistance: Validation of somatic cell score and clinical mastitis as selection criteria. *J. Dairy Sci.* 86:4129–4136.
- Philipsson, J., G. Ral, and B. Berglund. 1995. Somatic cell count as a selection criterion for mastitis resistance in dairy cattle. *Livest. Prod. Sci.* 41:195–200.
- 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.
- Reist, M., A. Koller, A. Busato, U. Kupfer, and J. W. Blum. 2000. First ovulation and ketone body status in the early postpartum period of dairy cows. *Theriogenology* 54:685–701.
- Shook, G. E. 1993. Genetic improvement of mastitis through selection on somatic cell count. *Vet. Clin. North Am. Food Anim. Pract.* 9:563–581.
- Van Vleck, L. D. 1979. Notes on the theory and application of selection principles for the genetic improvement of animals. 2nd ed. Cornell University Press, Ithaca, NY.
- 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.

APPENDIX

Calculation of Energy Traits

Weekly live weight and BCS records were used to calculate body lipid and protein weights using the formulas proposed by the US National Research Council based on the Cornell Nutrient Management Planning System (NRC, 2001). The following equations were used:

$$\text{Body lipid weight (kg)} = (0.037683 \times \text{BCS}) \times (\text{empty body weight})$$

$$\begin{aligned} \text{Body protein weight (kg)} = & [0.200886 \times (0.0066762 \times \text{BCS})] \\ & \times (\text{empty body weight}) \end{aligned}$$

where empty body weight is in kilograms and BCS is expressed on a 1 to 9 scale. In our data, cows were scored on a 1 to 5 scale (BCS5) and that was converted to the 1 to 9 scale (BCS9) using the following formula:

$$\text{BCS9} = (\text{BCS5} - 1) \times 2 + 1.$$

The empty body weight was calculated as a function of live weight and number of days the cows was assumed to be pregnant (d) as follows:

$$\begin{aligned} \text{Empty body weight} = & [(\text{Live weight}) \times 0.96] \\ & - (0.7312 \times \exp(0.02 \times d - 0.0000143 \times d \times d)) \times 0.851. \end{aligned}$$

In the above equation, a calf birth weight of 40 kg is assumed. Furthermore, cows were assumed to have conceived on d 110 postpartum.

Estimated body lipid and protein weights were combined to predict daily energy content (in MJ) using the following formula (NRC, 2001):

$$\begin{aligned} \text{EC (MJ)} = & [(9.4 \times (\text{Body lipid weight})) \\ & + [5.7 \times (\text{Body protein weight})] \times 4.1868. \end{aligned}$$

Changes in body lipid and protein weights during the lactation were used to derive effective energy balance based on the system proposed by Emmans (1994). This system describes the amount of energy processed when lipid and protein weights change. Accordingly, 56 MJ of energy are assumed to be needed for 1 kg of lipid gain and 39.6 MJ of energy are yielded for 1 kg of

Table A1. Calculation of daily energy content (EC in MJ) and cumulative effective energy balance (CEEB in MJ) based on live weight (LWT), BCS, predicted body lipid (FAT), and protein (PRT) weights, cumulative changes in body lipid (FCH) and protein (PCH) weights, and effective energy associated with FCH (EEF) and PCH (EEP), for 4 separate days postpartum (DPP)

DPP	LWT, kg	BCS	FAT, kg	PRT, kg	EC, MJ	FCH, kg	PCH, kg	EEF, MJ	EEP, MJ	CEEB, MJ
d 12	555	4	68.3	79.0	4,537					
d 19	540	4.5	74.8	75.4	4,742	6.5	-3.6	361.8	-48.7	313.1
d 26	545	4	67.1	77.5	4,491	-1.2	-1.4	-48.9	-19.3	-68.2
d 33	550	3.5	59.2	79.7	4,235	-9.1	0.8	-359.8	39.0	-320.8

lipid loss. Corresponding figures of energy required or released for 1 kg of protein gain or loss are 50 and 13.5 MJ, respectively. These coefficients were used to calculate effective energy processed by changes in lipid and protein weights in consecutive records. Cumulative effective energy balance was calculated based on the

cumulative lipid and protein change since the beginning of lactation. This trait relates to total body energy as it accumulates throughout lactation.

Table A1 illustrates an example of the calculation procedure for 4 records of the same animal taken 12, 19, 26, and 33 d postpartum.