

Negative Influence of High Maternal Milk Production Before and After Conception on Offspring Survival and Milk Production in Dairy Cattle

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ABSTRACT

There is a paucity of studies on the effect of intrauterine conditions on subsequent progeny performance in dairy cows. Using a large national data set on Irish Holstein-Friesian dairy cows, the objective of this study was to determine if intrauterine conditions, quantified by a maternal genetic variance component, significantly affected milk production, age at first calving, calving interval, somatic cell score (natural logarithm of somatic cell count) and survival in first-, second-, and third-parity female offspring. Maternal genetic variance for each trait in each parity was estimated in a linear mixed model which included, other than fixed effects, direct additive genetic, maternal genetic, cytoplasmic and permanent environmental effect of the dam, and residual component. A covariance was also estimated between the direct additive and maternal genetic components where possible. Because calves in Irish dairy herds are separated from dams at birth, a significant maternal genetic variance (with all other random effects in the model) indicates a prepartum maternal effect. A significant maternal genetic variance was estimated for 305-d milk yield in first and third lactation, somatic cell score in first lactation, and survival to second lactation from 188,144 lactations on 80,881 animals. Where estimated, a negative correlation existed between the direct additive and maternal genetic components. Regression of maternal mixed model solutions on dam milk production at different stages relative to conception revealed that greater milk yield pre-conception and during gestation was associated with reduced survival and milk yield and greater somatic cell count in the progeny. This study suggests that offspring survival and performance are affected by prepartum conditions that offspring experience as an oocyte, embryo, or fetus, one of which is mediated

through milk production (or factors related to milk production) of the dam.

Key words: thrifty, Barker hypothesis, fetal origin, dairy cattle

INTRODUCTION

The effect of intrauterine environmental conditions on postnatal performance of offspring has been discussed for many decades (Everitt, 1968). A substantial amount of evidence, based primarily on epidemiological studies of human data, suggests that perturbations during fetal life are associated with hypertension, vascular dysfunction, dyslipidemia, and insulin resistance in adulthood (Barker, 1995; Godfrey et al., 1997). Nevertheless, studies are also available that refute this hypothesis in humans (Stanner et al., 1997).

The hypothesis suggests that if intrauterine conditions are poor (e.g., poor nutrition), then the fetus becomes adapted through altered gene expression (Breier, 2006) to maximize the uptake and utilization of the nutrients available. One potential epigenetic mechanism is the silencing of gene expression through DNA methylation or histone acetylation (Langley-Evans, 2006). An animal exposed to a poor intrauterine environment will exhibit a competitive advantage if exposed to a similarly poor environment in adulthood; this has been referred to as the “thrifty phenotype hypothesis” or the Barker hypothesis (Hales and Barker, 1992). However, favorable postnatal conditions can challenge the individual’s homeostatic mechanisms and lead to the development of deleterious metabolic conditions such as hypertension, dysfunction, dyslipidemia, and insulin resistance.

Several studies in sheep (Heasman et al., 1999; Hawkins et al., 2000; Ford et al., 2007; Tygesen et al., 2007) have imposed alternative nutritional treatments at different stages of gestation and investigated the effect on subsequent male and female progeny performance. Similar studies have also been undertaken in rats (Woodall et al., 1996; Lesage et al., 2004). Although the conclusions of the animal model studies vary with

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the type, severity, and timing of the nutritional intervention, nutritionally challenged dams generally confer a restriction on fetal growth, as well as greater predisposition of the subsequent offspring to adulthood hypertension, hyperinsulinism, and dyslipidemia (Heasman et al., 1999; Hawkins et al., 2000; Ford et al., 2007) as well as bone density and mean relative wall thickness (Tygesen et al., 2007).

Although Roche et al. (2006) reported a significant effect of dam BCS and BW on the secondary sex ratio in New Zealand dairy cattle, the authors are aware of only one study (Pryce et al., 2002) that has attempted to quantify the influence of maternal environment on subsequent progeny performance in dairy cattle. Pryce et al. (2002) reported no significant effect of dam parity or milk production, DMI, or BCS in the first or second 13 wk postpartum on subsequent female progeny reproductive performance. However, their data set was limited in size and diverse management systems were not represented.

To address the question of whether the environment to which a developing female fetus is exposed can affect its subsequent postnatal performance, the objective of this study was to quantify the effects of intrauterine conditions on performance indicators in first, second, and third lactation dairy females using a large national database.

MATERIALS AND METHODS

Data

Calving dates for 2,776,684 cows up to fourth calving were extracted from the Irish Cattle Breeding Federation database. Following the removal of cows with no identified sire or dam, 1,048,120 cows remained. Only cows born as singleton calves and calving for the first time between 1990 and 2006 were retained, bringing the number of cows to 924,379. Breed fraction of each animal is recorded in the Irish Cattle Breeding Federation database in increments of 1/32. Only cows of at least 27/32 parts Holstein-Friesian from Holstein-Friesian sires (all 32 parts known) and Holstein-Friesian dams (at least 24/32 parts known) were retained so that heterosis and recombination loss effects could be accurately accounted for in the analysis. Heterosis and recombination loss were calculated for each animal as described by Akbas et al. (1993):

$$\text{heterosis} = P_S(1 - P_D) + P_D(1 - P_S)$$

$$\text{recombination loss} = P_D(1 - P_D) + P_S(1 - P_S),$$

where P_S and P_D are the proportion Holstein in the sire and dam, respectively.

In Ireland, calving assistance is scored on a scale of 1 to 4 as follows: 1 = no assistance/unobserved; 2 = slight assistance; 3 = severe assistance; 4 = veterinary assistance (including caesarean). Animals that did not have any information on calving assistance were allocated a separate code to facilitate their inclusion in the analysis. To avoid potential errors in recorded calving date, median age at calving per parity was calculated and records from animals calving ± 1 yr from the median were removed; a total of 507,705 animals remained. Lactations in which a multiple birth occurred were removed, as were lactations in which embryo transfer occurred. Only lactations in which the animal calved within the herd where it was born were retained; this was done to minimize the effect of herd in which progeny was producing being absorbed in the effect of the dam. Furthermore, only cows born in the months of December, January, February, March, and April were retained, which represents the predominant system of milk production in Ireland.

Dam lineage was determined for each animal by tracing back through its female pedigree to a founder female. Up to 16 generations were traced back and only dam lines with a least 3 cows were retained to facilitate the accurate estimation of dam lineage effects; a total of 15,766 dam lines were identified. An iterative algorithm was invoked to remove animals from herd-year contemporary groups, sire daughter groups, and sire grand-daughter groups of less than 4. This was iterated 5 times. Finally, only (grand)progeny of sires with (grand)progeny in at least 3 herd-years were retained. These edits were used to obtain an accurate estimate of the contemporary group, direct additive genetic, and maternal genetic effect.

Following all edits, 188,144 first-parity to third-parity lactation records from 80,881 animals remained; 170,623 of these lactations had information on milk production and SCC. The 80,881 animals were from 61,579 different dams; 15,781 animals appeared in the data set as both dams and progeny. The natural logarithm of SCC was used to normalize the distribution and is referred to herein as SCS. Calving intervals outside the range of 300 to 600 d were set to missing and survival from lactation i to lactation $i + 1$ was allocated a value of 1 if the animal recalved within this time frame, otherwise zero. Survival was set to missing if an animal's last calving date was less than 600 d from the herd's last test-day record. Survival in the present study was defined, using the aforementioned criteria of acceptable calving interval, as whether the animal survived to lactation i given that it calved at least once.

Estimation of (Co)variance Components

Estimation of (co)variance components using a multitrait analysis was not computationally feasible;

therefore, (co)variance components were estimated for each trait separately within parity using a linear model in ASREML (Gilmour et al., 2007) as follows:

$$\mathbf{Y} = \mathbf{Xb} + \mathbf{Z}_1\mathbf{a} + \mathbf{Z}_2\mathbf{m} + \mathbf{Z}_3\mathbf{c} + \mathbf{Z}_4\mathbf{p} + \mathbf{e}$$

where \mathbf{Y} is a vector of the phenotypic performance for each trait within each parity separately; \mathbf{b} is a vector of fixed effects (which differed by trait analyzed; for age at first calving the effects included in the model were herd-year of birth interaction, year of birth-month of birth interaction, heterosis, recombination, and Holstein percentage; for all other traits the effects included in the model were herd-year of calving interaction, year of calving-month of calving interaction, heterosis, recombination, Holstein percentage, age at calving, and whether calving difficulty was experienced at calving; heterosis, recombination, Holstein percentage, and age at first calving were all treated as continuous variables with nonlinear effects also tested for significance); \mathbf{a} is a vector of animal; \mathbf{m} is a vector of dam of animal; \mathbf{c} is a vector of dam lineage; \mathbf{p} is a vector of permanent environment effect of the dam; and \mathbf{e} is a vector of residuals. The \mathbf{X} and \mathbf{Z} matrices are incidence matrices linking the vectors of fixed and random effects, respectively, to the vector of observations (i.e., \mathbf{Y}).

Expectations of effects and variances were as follows:

$$\mathbf{E} \begin{bmatrix} \mathbf{Y} \\ \mathbf{a} \\ \mathbf{m} \\ \mathbf{c} \\ \mathbf{p} \\ \mathbf{e} \end{bmatrix} = \begin{bmatrix} \mathbf{Xb} \\ \mathbf{0} \\ \mathbf{0} \\ \mathbf{0} \\ \mathbf{0} \\ \mathbf{0} \end{bmatrix}$$

$$\mathbf{V} \begin{bmatrix} \mathbf{a} \\ \mathbf{m} \\ \mathbf{c} \\ \mathbf{p} \\ \mathbf{e} \end{bmatrix} = \begin{bmatrix} \mathbf{A}\sigma_a^2 & \mathbf{A}\sigma_{a,m} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{A}\sigma_{a,m} & \mathbf{A}\sigma_m^2 & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{I}\sigma_c^2 & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{I}\sigma_p^2 & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{I}\sigma_e^2 \end{bmatrix}$$

where \mathbf{A} is the numerator relationship matrix among all animals with or without phenotypes; σ_a^2 is the direct additive genetic variance; σ_m^2 is the maternal genetic variance; σ_c^2 is the cytoplasmic variance; σ_p^2 is the dam permanent environmental variance; σ_e^2 is the residual variance; and $\sigma_{a,m}$ is the direct-maternal genetic covariance. Significance of each of the random effects was determined by removing the random effect from the model and comparing the likelihood of the full and reduced model using the Akaike's information criterion

(AIC). No covariance between the direct additive genetic and maternal genetic effect was estimated if either component failed to exhibit a variance. When estimating the significance of the maternal component, the AIC of the full model was compared with the AIC of the model without a maternal genetic variance and covariance with the direct additive genetic component.

Description of Maternal Factors Implicated in Progeny Performance

To elucidate the extent of the association between dam milk production and progeny performance, milk test-day records from the lactations of dams immediately before the birth of the progeny were extracted from the national database. Days postcalving to conception for each dam were calculated as the date of birth of the progeny less a standard gestation length of 282 d. Average milk yield, milk solids yield, and milk fat and protein concentrations were calculated for each trimester of pregnancy. Milk fat to protein ratio has been suggested as an indicator of negative energy balance and thus, average milk fat to protein ratio within each time period was also calculated; a high fat to protein ratio is indicative of negative energy balance (Grieve et al., 1986). The variables previously described were also calculated for dam milk test-dates in the 30 d before conception.

For traits in which a significant maternal genetic variance was identified, 1 progeny per dam was randomly selected and the analysis undertaken once more; 61,579 animals from 61,579 dams were included in the mixed model analysis. Dam solutions from the mixed model equations were deregressed by dividing by their respective reliability and regressed on each of the dam milk performance measures before conception and during gestation. Of the 61,579 dams that had solutions from the mixed model analysis, up to 28,690 also had information on milk production before and/or during pregnancy.

RESULTS

Summary statistics of the progeny variables investigated are presented in Table 1. Mean and variation in milk yield and SCS increased with parity. There was a minimal effect of parity on calving interval. Based on the edits used in the present study, only 55% of animals that calved at least once calved for the fourth time. This is likely to be an underestimate, because the editing criteria applied in the present study ensured that lactations from cows calving in a herd different to that in which they were born were removed and survival was set to missing if a cow failed to recalve within 600 d.

Table 1. Number of records (n), mean and standard deviation (SD) for age at first calving, milk yield, calving interval, survival, and SCS in the first 3 parities

Trait	Parity	n	Mean	SD
Age at first calving (d)		80,881	800	111.2
Milk yield (kg)	1	80,129	5,831	1,258.6
	2	54,270	6,736	1,401.9
	3	36,224	7,130	1,444.3
Calving interval (d)	1	61,066	383	47.5
	2	41,570	383	47.1
	3	27,989	381	46.3
Survival (%)	1	74,242	0.86	
	2	62,447	0.70	
	3	54,258	0.55	
SCS (units)	1	78,398	4.459	0.800
	2	53,534	4.529	0.852
	3	35,926	4.688	0.896

Variance components for the different traits analyzed are summarized in Table 2. All direct additive genetic effects were significant, and direct heritability estimates varied from 0.01 (survival to parity 3) to 0.34 (milk yield in first-parity animals). Maternal genetic variance was only different ($P < 0.05$) from zero for milk yield in first- and third-parity progeny, survival to second lactation, and SCS in first-parity progeny.

Maternal genetic variance for milk yield increased with parity number. Despite its statistical significance, however, the maternal genetic variance accounted for less than 1% of the phenotypic variation. Furthermore, the coefficient of variation accounted for by the maternal genetic component was less than 3%. The permanent environmental effect of the dam did not affect progeny performance. Maternal lineage had a significant effect only on milk yield in third-parity animals and SCS in second-parity animals, explaining less than 1% of the phenotypic variation in both traits. The ratio of the maternal genetic variance to the direct additive genetic variance were mostly less than 5%, the exceptions being survival in first (23%) and second (8%) lactation. Where estimated, the correlations between the direct additive genetic component and the maternal genetic component were all negative (Table 2).

Summary statistics for dam performance before conception and during each trimester of pregnancy are outlined in Table 3. Table 4 summarizes the association between dam milk production at different stages relative to conception and the maternal genetic solutions from the mixed models equations. Nonlinear associations were not evident. The association with offspring

Table 2. Standard deviations of each of the components estimated and correlation (r) between the direct and maternal effects¹

Trait	Parity	Standard deviation					r
		Additive genetic	Maternal	Lineage	Permanent environment	Residual	
Age at first calving (d)		11.26*** (0.019)	0.00 ^{NS} (0.000)	3.81 ^{NS} (0.002)	6.66 ^{NS} (0.007)	80.62 (0.972)	—
Milk yield (kg)	1	461.87*** (0.342)	65.51** (0.007)	0.00 ^{NS} (0.000)	0.00 ^{NS} (0.000)	637.34 (0.651)	-0.46**
	2	514.92*** (0.305)	71.33 ^{NS} (0.006)	71.04 ^{NS} (0.006)	58.14 ^{NS} (0.004)	769.32 (0.680)	-0.18 ^{NS}
	3	485.45*** (0.231)	87.85* (0.008)	99.48* (0.010)	0.00 ^{NS} (0.000)	875.97 (0.752)	-0.42 ^{NS}
Calving interval (d)	1	6.06*** (0.020)	0.00 ^{NS} (0.000)	1.66 ^{NS} (0.002)	0.00 ^{NS} (0.000)	41.97 (0.978)	—
	2	7.26*** (0.028)	0.00 ^{NS} (0.000)	0.00 ^{NS} (0.000)	1.78 ^{NS} (0.002)	42.46 (0.970)	—
	3	6.98*** (0.027)	0.86 ^{NS} (0.000)	1.97 ^{NS} (0.002)	0.00 ^{NS} (0.000)	42.21 (0.971)	0.00 ^{NS}
Survival (%)	1	0.05*** (0.024)	0.02* (0.006)	0.00 ^{NS} (0.000)	0.04 ^{NS} (0.014)	0.32 (0.957)	-0.68*
	2	0.06*** (0.032)	0.02 ^{NS} (0.003)	0.02 ^{NS} (0.002)	0.00 ^{NS} (0.000)	0.33 (0.963)	-0.99*
	3	0.04** (0.013)	0.00 ^{NS} (0.000)	0.02 ^{NS} (0.003)	0.00 ^{NS} (0.000)	0.34 (0.984)	—
SCS (units)	1	0.26*** (0.127)	0.05** (0.005)	0.00 ^{NS} (0.000)	0.04 ^{NS} (0.003)	0.69 (0.865)	-0.22*
	2	0.26*** (0.117)	0.00 ^{NS} (0.000)	0.06* (0.006)	0.00 ^{NS} (0.000)	0.71 (0.877)	—
	3	0.29*** (0.132)	0.05 ^{NS} (0.003)	0.05 ^{NS} (0.000)	0.08 ^{NS} (0.009)	0.74 (0.856)	-0.12 ^{NS}

¹The proportion of phenotypic variance attributed to each of the individual components is presented in parentheses below each value.

*** $P < 0.001$; ** $P < 0.01$; * $P < 0.05$; NS = not significantly different from zero.

Table 3. Number of dam records (n), mean and standard deviation (SD) for dam milk production before conception and during each trimester of pregnancy

Time period	Trait	n	Mean	SD
Before conception	Milk yield (kg)	21,993	28.1	6.17
	Fat concentration (%)	21,993	3.57	0.601
	Protein concentration (%)	21,993	3.22	0.266
	Fat:protein ratio	21,993	1.11	0.195
	Milk solids yield (kg)	21,993	3.2	0.70
First trimester	Milk yield (kg)	27,434	24.2	5.14
	Fat concentration (%)	27,434	3.61	0.475
	Protein concentration (%)	27,434	3.33	0.232
	Fat:protein ratio	27,434	1.08	0.129
	Milk solids yield (kg)	27,434	2.8	0.57
Second trimester	Milk yield (kg)	27,534	17.4	4.42
	Fat concentration (%)	27,534	4.05	0.577
	Protein concentration (%)	27,534	3.62	0.308
	Fat:protein ratio	27,534	1.12	0.131
	Milk solids yield (kg)	27,534	2.1	0.51
Third trimester	Milk yield (kg)	17,102	12.8	4.60
	Fat concentration (%)	17,102	4.41	0.715
	Protein concentration (%)	17,102	3.77	0.429
	Fat:protein ratio	17,102	1.17	0.154
	Milk solids yield (kg)	17,102	1.6	0.56

performance as measured by the strength of the correlation and the absolute value of the regression coefficient tended to be greatest for dam performance in the second and third trimesters of pregnancy. Nonetheless, all correlations between dam phenotypic performance and

progeny performance, measured by the maternal genetic solutions, were weak; the absolute correlations were all <0.19. The absolute correlation with dam performance was stronger for first-parity progeny milk yield compared with third-parity progeny milk yield.

Table 4. Regression coefficients (b) (with respective standard errors in parentheses) of maternal solution from the mixed model equations on dam milk production and correlations (r) between maternal solutions and dam milk production

Time period	Trait	Milk yield in first parity		Milk yield in third parity		Survival to second parity ¹		SCS in first parity ¹	
		b	r	B	r	b	r	b	r
Before conception	Milk yield (kg)	-7.0 (0.38)	-0.12	-6.5 (0.65)	-0.07	-0.6 (0.10)	-0.05	1.2 (0.25)	0.03
	Fat concentration (%)	40.3 (3.91)	0.07	32.5 (6.71)	0.03	-3.3 (0.99)	-0.02	-11.4 (2.60)	-0.03
	Protein concentration (%)	58.9 (8.84)	0.05	-37.8 (15.16)	-0.02	-36.3 (2.21)	-0.11	-3.7 (5.87) ^{NS}	0.00
	Fat:protein ratio	85.2 (12.05)	0.05	125.9 (20.61)	0.04	14.2 (3.03)	0.03	-32.5 (7.98)	-0.03
	Milk solids yield (kg)	-52.7 (3.33)	-0.11	-57.1 (5.73)	-0.07	-8.9 (0.84)	-0.07	8.9 (2.22)	0.03
First trimester	Milk yield (kg)	-8.8 (0.40)	-0.13	-8.2 (0.69)	-0.07	-0.3 (0.10)	-0.02	1.6 (0.28)	0.03
	Fat concentration (%)	68.4 (4.38)	0.09	66.9 (7.50)	0.05	-4.0 (1.13)	-0.02	-31.3 (2.98)	-0.06
	Protein concentration (%)	141.5 (8.94)	0.10	63.7 (15.38)	0.03	-27.8 (2.3)	-0.07	-39.7 (6.09)	-0.04
	Fat:protein ratio	130.3 (16.21)	0.05	209.9 (27.72)	0.05	13.3 (4.17)	0.02	-83.5 (11.00)	-0.05
	Milk solids yield (kg)	-66.5 (3.64)	-0.11	-68.2 (6.25)	-0.07	-5.9 (0.94)	-0.04	9.3 (2.48)	0.02
Second trimester	Milk yield (kg)	-13.5 (0.47)	-0.17	-16.3 (0.81)	-0.12	-1.5 (0.12)	-0.07	4.5 (0.32)	0.08
	Fat concentration (%)	72.6 (3.60)	0.12	64.5 (6.19)	0.06	-0.6 (0.93) ^{NS}	0.00	-37.5 (2.43)	-0.09
	Protein concentration (%)	136.5 (6.75)	0.12	83.5 (11.61)	0.04	-14.1 (1.73)	-0.05	-49.4 (4.57)	-0.07
	Fat:protein ratio	155.2 (15.96)	0.06	207.0 (27.3)	0.05	20.5 (4.08)	0.03	-113.9 (10.74)	-0.06
	Milk solids yield (kg)	-101.0 (4.06)	-0.15	-135.2 (6.98)	-0.12	-15.3 (1.04)	-0.09	32.1 (2.76)	0.07
Third trimester	Milk yield (kg)	-13.9 (0.58)	-0.18	-18.3 (1.00)	-0.14	-1.9 (0.15)	-0.10	4.8 (0.39)	0.09
	Fat concentration (%)	58.6 (3.76)	0.12	50.3 (6.45)	0.06	-1.1 (0.96) ^{NS}	-0.01	-27.7 (2.50)	-0.08
	Protein concentration (%)	97.8 (6.27)	0.12	86.4 (10.76)	0.06	-2.2 (1.59) ^{NS}	-0.01	-42.3 (4.17)	-0.08
	Fat:protein ratio	101.1 (17.56)	0.04	86.8 (30.01)	0.02	-0.2 (4.43) ^{NS}	0.00	-55.5 (11.64)	-0.04
	Milk solids yield (kg)	-103.7 (4.8)	-0.16	-147.4 (8.25)	-0.14	-18.1 (1.22)	-0.11	34.7 (3.21)	0.08

¹Coefficients and standard errors are expressed as 10⁶.

^{NS}Coefficients not significantly ($P > 0.05$) different from zero.

Irrespective of period relative to conception, increased dam milk yield was associated with reduced progeny milk yield in first and third parity, reduced survival to second parity, and increased SCS in first parity; associations between dam milk solids yield and progeny performance followed a similar trend. Increased milk fat concentration in dams was associated with greater milk yield, reduced survival, and reduced SCS in the progeny; a similar trend was observed for milk protein concentration although the sign of the association was not always consistent. Increased milk fat to protein ratio in the dam before conception and during gestation was associated with either greater milk yield, greater survival, and reduced SCS, or the effects were not significantly different from zero.

DISCUSSION

Considerable discussions are ongoing on the impact of fetal programming on subsequent progeny performance, with most research being undertaken using epidemiological databases on humans (Barker, 1995; Godfrey et al., 1997) and to a lesser extent experimentally induced nutritional treatments in sheep (Heasman et al., 1999; Ford et al., 2007; Tygesen et al., 2007) and rodents (Stewart et al., 1980; Woodall et al., 1996; Lesage et al., 2004). The objective of this study was to investigate whether a significant proportion of the variation in measures of performance, health, fertility, and survival in dairy cows can be attributed to the dam and the conditions provided by her to the developing fetus. Results from this study suggest that prenatal conditions experienced by the fetus can affect subsequent performance and health. The majority of the maternal genetic variation in progeny performance was due to factors other than the dam milk production variables investigated in the present study. Mean performance parameters and heritability estimates were similar to those reported elsewhere on Irish dairy cows (Olori et al., 2002; Berry et al., 2004).

The maternal genetic component can be partitioned into the direct additive genetic contribution, the cytoplasmic effect, the maternal genetic effect additional to her direct additive genetic contribution, and repeatability. Cytoplasmic effects may be due to the maternal inheritance of mitochondria or other cytoplasmic components. They are transferred directly from dam to offspring (Boursot and Bonhomme, 1986) via the ovum and, assuming homogeneity of the cytoplasmic component within dam and no mutation, all female offspring of a dam will receive identical copies. The significant cytoplasmic variance estimated in the present study for milk production, albeit only in third lactation, is not surprising given the involvement of mitochondria in

lactation (Jones and Rosano, 1972). The ratio of the cytoplasmic variance to the phenotypic variance reported in the present study (<1%) is slightly lower than reported elsewhere for milk production (Bell et al., 1985; Schutz et al., 1992). Bell et al. (1985) reported that cytoplasmic effects accounted for 2% of the total variation in mature-equivalent milk yield in first-lactation US Holsteins; however, Bell et al. (1985) only included lines with at least 5 females compared with at least 3 in the present study, which may have affected the estimated variance component. Nonetheless, in contrast to the results from the present study, Bell et al. (1985), using a model including sire as a fixed effect, reported a significant cytoplasmic effect in US Holsteins for days open, a trait very similar to calving interval as assessed in the present study. However, when both sire and maternal grandsire were included as fixed effects in the model, the cytoplasmic variance component only approached significance. The authors are unaware of any study that has attempted to quantify the cytoplasmic variance of SCS. However, Roughsedge et al. (2000) failed to report any significant cytoplasmic effect on udder type traits that have been shown to be correlated with SCS (Berry et al., 2004). Nevertheless, given the small proportion of phenotypic variance explained by the cytoplasmic effects relative to that explained by the direct additive genetic effects, the impact of including cytoplasmic effects in genetic evaluations for these traits will be minimal.

The maternal genetic component as estimated in beef populations (Albuquerque and Meyer, 2001) generally refers to postnatal mothering ability of the dam. In Ireland, however, the newborn calf from a dairy cow is generally separated immediately from the dam. Therefore, it seems logical to assume that the maternal genetic variance estimated using a linear model also containing a cytoplasmic and transmittable nuclear genetic component, encompasses the dam's genetic and environmental components excluding any direct additive genetic effects that are transmitted directly to her progeny (i.e., it represents the preconception and/or intrauterine effect of the dam on subsequent progeny performance). The significance of the maternal genetic effect (excluding cytoplasmic effects) and covariance with the direct additive genetic effect for milk yield is in contrast, however, to previous results from US Holsteins in which no significant effect was reported (Schutz et al., 1992). Nonetheless, in agreement with the present study, Albuquerque et al. (1998) documented that additive maternal genetic effects accounted for 0.8 to 1.0% of the phenotypic variance in milk yield in primiparous and multiparous dairy cattle; however, a positive covariance between the direct additive and additive maternal effects was reported in that

study, which is in contrast to the negative covariance reported in the present study. Less is known about the maternal impact on survival and udder health in dairy cows. In the present study, a significant maternal genetic variance for survival to second lactation persisted even after adjusting for milk yield and SCS in the model as fixed effects, implying that the effect of prenatal conditions on survival was independent of the potential effect of milk yield and SCS on culling.

In agreement with the present study, Pryce et al. (2002) reported no difference in daughter age at first calving and interval from calving to first insemination (a trait similar to calving interval) among dams fed either high or low concentrate diets. Similarly, the regression coefficient of daughter fertility on dam BCS, DMI, and milk production did not differ significantly from zero (Pryce et al., 2002). However, those authors did not investigate the effect of dam environment on progeny milk production, SCS, or survival.

The effect of intrauterine conditions on milk yield in the present study is at odds with the view that prenatal mammary development is autonomous and is therefore not likely to be affected by external factors (Robinson et al., 1999) as well as the compensatory ability of the mammary gland during lactation being able to compensate for earlier insults (Knight, 1997). Nonetheless, Knight and Sorensen (2001) suggest that deficient ductular development in early fetal life may affect secretory tissue mass because the secretory cells proliferate on the ducts. Additionally, postnatal mammatogenesis may itself be affected by prenatal intrauterine conditions. A more likely physiological pathway for the effect of prenatal conditions on milk production is that perturbations may alter the energy partitioning mechanisms in the fetus that persist in the adult. For example, changes in homeostatic mechanisms in offspring exposed to adverse intrauterine conditions may predispose the individual to a more conservative metabolism, storing body tissue in adult life in anticipation of future potential adverse conditions (i.e., the individual may partition more energy toward the anabolism of body reserves rather than toward milk production).

Despite the significant maternal genetic variation for progeny performance observed in the present study, the proportion of variation in progeny performance explained by dam milk production was small, after accounting for the direct additive and cytoplasmic effects as well as fixed effects in the model. The stronger association with progeny performance observed for milk production in mid- to late-gestation suggests that perturbations during the latter 2 trimesters of pregnancy may have a greater impact on subsequent progeny performance than dam performance in early gestation or immediately before conception. In cattle, most of the major

developmental changes in the developing fetal mammary have occurred by mid gestation; thickening of the ectoderm occurs by d 35, teat canals have developed by 3 mo, and the excretory duct system is complete after the second trimester (Sejrsen, 1994). This further substantiates that the influence of prenatal conditions on subsequent adult performance may be through factors other than mammatogenesis.

The antagonistic direct maternal genetic correlation indicates a difficulty in improving both components simultaneously. Simultaneous selection for 2 traits in the same direction will lead to a negative genetic correlation. This phenomenon occurs because alleles with a favorable effect on both traits (or a favorable effect on 1 trait but neutral effect on the other) become fixed in the population, whereas alleles with an unfavorable effect on both traits (or an unfavorable effect on 1 trait but neutral effect on the other) are eliminated from the population. However, alleles with a favorable effect on 1 trait and unfavorable effect on the other traits will remain segregating in the population. Elimination of the alleles affecting both traits in the same direction (or neutral effect) will theoretically result in a negative correlation assuming that the original correlation is primarily due to pleiotropy and that the selection horizon is long enough for the alleles to become fixed in the population (Sheridan and Barker, 1974). Holstein dairy cattle have been aggressively selected for greater milk production for several decades. However, milk production is a major component of dairy cow energy balance (Berry et al., 2006). Higher-producing cows tend to be in greater negative energy balance, and in Ireland most cows are in negative energy balance or slightly positive energy balance in early pregnancy (Berry et al., 2006). Hence, selection for greater milk production in dairy cows may lead to greater metabolic stress during pregnancy with subsequent deleterious repercussions for progeny performance (i.e., a negative correlation). This hypothesis is substantiated by the negative association between dam milk yield at different stages relative to conception and subsequent progeny milk yield in first and third parities.

The large number of traits analyzed ($n = 13$) and (co)variance components estimated per trait ($n \leq 7$) suggests that the identification of significant maternal genetic variance for some traits may be an artifact of multiple testing. For example, dam explained a significant proportion of the variation in survival to second lactation, whereas no significant effect was observed for survival to third or fourth lactation despite the part-whole relationship between the survival traits; the maternal genetic variance for survival to third lactation approached ($P = 0.08$) significance. Nonetheless, the strength of the significance of some of the maternal

effects suggests that indeed the estimated variance component is real. Because a multitrait analysis was not possible, selection among animals, especially in higher parities, was not accounted for and may also bias the estimated variance components. Furthermore, the lack of significant maternal genetic variation in some traits may be due to the difficulty in partitioning of the total variance among its components. This is particularly true when the cytoplasmic and maternal genetic random effects are both included in the model. Stewart et al. (1980), using data from a study on rats maintained on marginal protein deprivation for 12 generations, reported that the effect on subsequent adult disease can persist for 3 generations. This multigenerational effect may have entered the cytoplasmic component in the present study.

CONCLUSIONS

Significant maternal genetic variation was observed in milk production, SCS, and survival to second lactation. In Irish dairy farming production systems the calf is generally removed immediately postcalving from the cow, suggesting that prenatal factors (i.e., effects on the oocyte before fertilization or intrauterine conditions to which the developing fetus is exposed) affect the subsequent performance of the offspring.

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