

The Importance of Maternal Lineage on Milk Yield Traits of Dairy Cattle¹

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ABSTRACT

Maternal lineage effects on milk yield traits, considered indicative of cytoplasmic inheritance, were evaluated with animal models. Cattle were from a selection experiment begun in 1968. Maternal pedigrees were traced to the first female member in the *Holstein-Friesian Herdbook*; purchased cows entering the herd, considered foundation females, were assigned to maternal lineage groups. All models accounted for year-season of calving, parity, and selection lines. Maternal lineage effects were included in a repeated records model with cow effects and preadjustment for sire and maternal grandsire transmitting abilities. Maternal lineage accounted for 5.2, 4.1, and 10.5% of phenotypic variation of preadjusted records of milk yield, fat yield, and fat percentage, respectively. Maternal lineage was evaluated as a fixed effect in an animal model including random animal and permanent environmental effects. Maternal lineage significantly affected fat percentage but not milk yield. Maternal genetic (nuclear) effects and their covariance with additive animal effects did not significantly account for additional variation nor did they influence maternal lineage estimates. Maternal lineage affected calculated net energy of milk but was not important for SNF yield or concentration. Maternal lineage influenced fat per-

centage, energy concentration, and, to a lesser extent, fat yield in milk of dairy cattle.

(Key words: maternal lineages, cytoplasmic inheritance, mitochondrial inheritance, variance components)

Abbreviation key: MEFAT = mature equivalent fat yield, MEMILK = mature equivalent milk yield, mtDNA = mitochondrial DNA, TV = transmitting value.

INTRODUCTION

Recent studies (1, 11) have demonstrated the existence of maternal lineage effects on yield and reproduction of dairy cattle that may be indicative of cytoplasmic inheritance. Because mitochondria are transmitted only from female parents to ensuing offspring (6), mitochondrial DNA (mtDNA) is a probable source of cytoplasmic inheritance.

Mitochondrial DNA sequence is known to differ among dairy cattle (8). Koehler (14) used restriction enzymes to detect 11 polymorphisms among maternal lineages, and two additional polymorphisms occurred within lineages. Lindberg (15) sequenced the entire D-loop region of mtDNA and identified 48 sites of nucleotide substitution plus one deletion and two variable-length regions among lineages.

Evidence of maternal lineage effects comes from two recent studies (1, 11). In a study of 4461 cows, representing 102 cytoplasmic lineages, Bell et al. (1) showed that 2.0, 1.8, 1.8, and 3.5% of variation in milk yield, fat yield, 3.7% FCM yield, and fat percentage, respectively, was explained by cytoplasmic lineage. The authors concluded that cytoplasmic origin was a significant source of variation in yield traits of dairy cattle. Huizinga et al. (11) attributed 10% of variation in milk, fat, and protein yields and 13% of variation in milk economic returns to cytoplasmic components. By using

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field data from 36 lineages in 28 herds, Ron (1989, personal communication) attributed 3% of variation in milk and fat yields to cytoplasmic effects.

Some evidence suggests that cytoplasmic effects may not be important. Considering only additive effects, Kennedy (12) simulated a closed population similar in size to that used by Bell et al. (1). Kennedy's work (12) showed that analyses that ignore covariances between observations, such as was done by Bell et al. (1), can lead to spurious *F* test results. By using regression analysis of daughter-dam and granddaughter-granddam records, Reed and Van Vleck (21) concluded that cytoplasmic effects accounted for no variation in either milk and fat yields or fat percentage. Correction for environmental effects, however, was made only for daughter records. Thus, dam and granddam records were assumed to be subject to the same environmental effects as daughter records, an assumption not likely to be valid in field data. Kirkpatrick and Dentine (13) proposed a different model, which gave an alternative explanation to Reed and Van Vleck's (21) conclusion. They concluded that observations were consistent with the existence of a positive maternal effect, cytoplasmic inheritance, and additive nuclear genetic effects.

Additive maternal effects cause genetic differences among dams, exhibited as strictly environmental influences with regard to offspring performance (28). It is unclear whether additive maternal effects influence yield traits in dairy cattle. Maternal genetic effects are present in beef cattle, for which genetic mothering ability influences preweaning growth of calves (29). In contrast, dairy calves generally do not nurse their dams, so additive maternal effects would be caused by intrauterine environment. In 1960, Brumby (4) reported maternal genetic differences of 8 to 14% of total variance in milk yield, but he admitted the difficulty of separating the effects of additive genetic maternal differences from the effects of cytoplasmic differences.

The ability to separate maternal influences into their cytoplasmic and additive genetic components by animal models has been demonstrated (24). By using simulated data and true or incorrect models containing additive direct, additive maternal, cytoplasmic, and error variances, Southwood et al. (24) con-

cluded that certain animal models can be used to partition variation caused by these components.

The objective of the present study was to determine the extent of maternal lineage effects, which are indicative of cytoplasmic inheritance, on milk yield traits in a herd of dairy cattle with known molecular variation in mtDNA (14, 15).

MATERIALS AND METHODS

Cattle studied were part of a selection experiment begun at Iowa State University's Breeding Research Herd in 1968. Foundation females were mated to Holstein AI sires with high or average transmitting abilities for milk to form two divergent genetic lines. A description of the design of this breeding experiment is presented by Bertrand et al. (3). Records initiated through 1986 were included. At that time, the herd consisted of 150 milking cows, which differed by 1304 kg of milk per lactation between high and average lines.

The genetic backgrounds of the original members of the herd were diverse. The 158 foundation females were purchased from 38 Iowa Holstein breeders. Maternal heritage was verified by tracing maternal lineage to the first female member recorded in the *Holstein-Friesian Herdbook* (26), resulting in the 133 registered females being assigned to 81 separate maternal lineages. Only records with unusual circumstances, such as those initiated by abortion or those with serious mastitis, were excluded. Lineages with only one member with usable information were excluded also; thus, 53 maternal lineages from 105 foundation cows were studied. Of these, 19 had members only in the high yield line, 15 only in the average yield line, and 19 in both lines. Foundation females were, on average, 19 generations removed from their matriarchs first recorded in the herdbook. Inbreeding was negligible in this herd.

Mature equivalent (twice daily milking, 305-d lactation) milk (MEMILK) and fat (MEFAT) yields and fat percentage were the yield traits considered. A sire PD plus one-half of the maternal grandsire PD model, similar to Model 3 of Bell et al. (1), was used to analyze yield traits for maternal lineage effects:

$$Y_{imn} = \mu + YS_i + b_1(\text{age}) + b_2(\text{age}^2) + b_3(\text{TV}) + b_4(\text{GTO}) + \text{ML}_m + e_{imn} \quad [1]$$

where Y_{imn} is MEMILK, MEFAT, or fat percentage record of cow n in maternal lineage m calving in year-season of calving i (seasons were October to April and May to September, and some early years were combined because of too few records); b_1 and b_2 are linear and quadratic regressions on age at calving to account for specific herd effects because mature equivalent adjustments are on a regional basis; b_3 is regression on estimated transmitting value (TV = 1982 PD value of the sire plus one-half of the 1982 PD value of the maternal grandsire of cow n); b_4 is regression on the number of generations to the maternal lineage origin; ML_m is effect of maternal lineage m ; and e_{imn} is residual. All effects except maternal lineage and residual were considered fixed. Variance of maternal lineages was estimated using the RANDOM option of the general linear models procedure of SAS [SAS PROC GLM; (22)]. Estimates are according to Henderson's method 3 (10). Because foundation females were purchased from other herds, and because sire and maternal grandsire PD values could not always be obtained, records of foundation females were excluded from this part of the study. Only cows from subsequent generations were used. Separate analyses were conducted for first and second parity with Model [1].

Also, a repeated records model was used to estimate variance components based on as many as seven records per cow. Records were preadjusted for sire plus one-half of the maternal grandsire 1982 PD values to account for a portion of additive nuclear contributions.

The repeated records model was

$$Y_{ijklm} = \mu + YS_i + P_j + b_1(\text{age}) + b_2(\text{age}^2) + \text{ML}_k + C(\text{ML})_{l:k} + e_{ijklm} \quad [2]$$

where Y_{ijklm} is record m in year-season i and parity j of cow l in maternal lineage k . Effects are as in Model [1], and $C(\text{ML})_{l:k}$ is the effect of cow l nested in maternal lineage k . In this model, maternal lineage, cow, and residual were treated as random effects. Expectations of

maternal lineage, cow, and error effects were zero, and variance among maternal lineages was $\text{var}(\text{ML}) = \text{I}\hat{\sigma}_{\text{ML}}^2/\hat{\sigma}_e^2$ and among cows was $\text{var}(C) = \text{I}\hat{\sigma}_c^2/\hat{\sigma}_e^2$, where $\hat{\sigma}_e^2$ is error variance and I is an identity matrix.

Restricted maximum likelihood estimates of variances of maternal lineage and error and solutions for maternal lineages were by an expectation-maximization algorithm (16). Convergence was declared when change in all estimates expressed as a percentage was less than 1×10^{-4} (16). Inclusion of cow effects, in addition to preadjustment for sire plus one-half the maternal grandsire 1982 PD values, accounted for a portion of additive nuclear differences.

Detailed animal models, including all known additive genetic covariances among related individuals, have been proposed (10). Recent computing methods have made animal model analyses feasible (2, 17, 19). Records of all cows in the herd, including foundation females, were analyzed according to this animal model:

$$Y_{ijklmno} = \mu + YS_i + P_j + \text{ML}_k + L_l + G(L)_{m:l} + \text{PE}_n + a_n + e_{ijklmno} \quad [3]$$

Effects in the model are as previously defined except that L_l is the "high" or "average" sire selection line; $G(L)_{m:l}$ is sire birth-year group m nested in selection line l ; PE_n is the permanent environmental effect of animal n with a record; and a_n represents the additive genetic effect of animal n .

For purposes of testing the hypothesis that maternal lineage effects differ, maternal lineages were considered fixed in Model [3]. Permanent environment, animal, and residual effects were considered random and independently distributed with zero expectations. Variance among permanent environments was $\text{var}(\text{PE}) = \text{I}\hat{\sigma}_{\text{PE}}^2/\hat{\sigma}_e^2$, where $\hat{\sigma}_e^2$ is error variance. Variance among animals was $\text{var}(a) = \text{A}\hat{\sigma}_a^2/\hat{\sigma}_e^2$, where A is the numerator relationship matrix. For cows with records, A was complete back to sires and dams of foundation cows, and relationships among sires and paternal grandsires of AI bulls represented by daughters with records were included.

Based on Model [3], a derivative-free REML procedure (17) was used to estimate variance components for permanent environmental and animal effects. The procedure uses a simplex or polytope method to evaluate explicitly the maximum log-likelihood. Convergence was declared when variance of function values in the simplex was less than 1.0×10^{-5} (17). Used in this way, Model [3] corresponds to Model [2] of Meyer (17).

Because animal models are usually of large order, conventional tests of significance, requiring elements of variance-covariance matrices and, hence, direct inversion, are often unfeasible. Thus, an alternative test based on mixed model conjugate normal equations was developed to determine the significance of maternal lineage effects (7, 9). This procedure results in an approximate test, which is exact when estimated variance ratios are true. Variance ratios for random effects in the model used variance component estimates from analyses with Model [3]. Because of concern about increased bias when using variance ratios estimated from the same data (10), variance ratios based on heritabilities of .2, .2, and .5 and repeatabilities of .5, .5, and .7 (G. R. Wiggans, 1990, personal communication) for MEMILK, MEFAT, and fat percentage, respectively, were also used.

For both sets of variance ratios, genetic groups were fixed sire birth-year groups. Both sets of variance ratios were also used in testing maternal lineage effects in models that differed from Model [3] in definition of genetic groups. Genetic groups were defined according to Westell et al. (27), and unknown parent groups accounted for an additional genetic trend not explained by known additive genetic relationships. Thus, four tests of significance were conducted.

Variance component estimation with Model [3] was expanded to include variance caused by additive maternal genetic effects and covariance between additive animal and maternal genetic effects. The model used was

$$Y_{ijklmnop} = \mu + YS_i + P_j + ML_k + L_l + G(L)_{m:l} + PE_n + A_n + M_o + e_{ijklmnop}, \quad [4]$$

where effects and assumptions are as in Model [3], except M_o is the effect of dam o. Maternal

genetic effects also had expectation of zero. Variance among maternal genetic effects was $\text{var}(M) = A\hat{\sigma}_M^2/\hat{\sigma}_e^2$, and covariance between animal and maternal genetic effects was $\text{cov}(a,M) = A\hat{\sigma}_{a,M}/\hat{\sigma}_e^2$, where A and $\hat{\sigma}_e^2$ are as previously defined. Model [4b] differed from Model [4] by considering additive animal and maternal genetic effects to be uncorrelated [$\text{cov}(a,M) = 0$]. Priors for Model [4] used variance estimates resulting from analysis with Model [3] for animal and permanent environmental components. Maternal genetic variance (m^2) and covariance between animal and maternal genetic component ratios were set near zero.

RESULTS AND DISCUSSION

Number of cows and overall means for the three yield traits for first and second parity are in Table 1. Numbers of records in subsequent parities decreased quickly, and results from later parities are not reported. Means increased from first to second parity for MEMILK and MEFAT but remained nearly constant for fat percentage. Increases may have resulted from mature equivalent age factors not being exact for a single herd. Culling of cows in first parity also may have contributed to increased means in second parity. After culling for involuntary reasons, voluntary culling was based on transmitting ability for milk. Any culling was without regard to maternal lineage. Standard deviations of traits were nearly identical in both parities.

The results in Table 2 are based on Model [1]. Year-season of calving and regression on sire plus one-half of the maternal grandsire 1982 PD values were highly significant ($P \leq .01$) for all traits in both parities. Linear or quadratic regressions on age were not significant for any trait. As expected, F values for regressions on age were smaller for MEMILK and MEFAT because they were already age-adjusted. Regressions on generations to origin were not significant in either parity. Bell et al. (1) obtained similar results. Hence, generations to origin were not considered in subsequent analyses. The effect of maternal lineage (Table 2) was highly significant ($P \leq .01$) for fat percentage in both parities and MEFAT in first parity and was significant ($P \leq .05$) for

TABLE 1. Means and effects of maternal lineage (ML) on yield traits from Model [1].

Trait ¹	Cows	Overall mean	SD	ML $P > F$	σ_{ML}^2/σ_e^2	Range ²
Parity 1						
MEMILK, kg	664	7643	1721	.020	.041	5493
MEFAT, kg	661	271	60	.002	.058	195
Fat, %	662	3.62	.41	.001	.084	1.24
Parity 2						
MEMILK, kg	409	8360	1718	.170	.028	3258
MEFAT, kg	407	299	57	.017	.072	148
Fat, %	409	3.64	.42	.001	.140	1.12

¹MEMILK = Mature equivalent milk yield, MEFAT = mature equivalent fat yield.

²Range of maternal lineage solutions.

MEFAT in second parity and MEMILK in first parity.

Ratios of estimates of maternal lineage variance to error variance are presented in Table 1. Ratios for MEMILK and fat percentage were greater than those previously reported from similar models using actual (1) or simulated (12) records. This finding suggests that maternal lineage may account for an appreciable portion of residual variance in models not considering its influence. Ranges of maternal lineage least squares means from Model [1] are also presented in Table 1. Ranges for all traits were much greater than one phenotypic standard deviation. Moreover, ranges for all three traits were greater than those reported in previous work (1), even though fewer cytoplasmic lineages were represented in this study.

Table 3 has numbers of records, cows, and lineages used in the repeated records Model [2] with preadjustment for sire plus one-half of the maternal grandsire 1982 PD values for each of the three traits. There was an average of 2.4 records per cow and 12.6 cows per cytoplasmic lineage. Resulting variance components for cytoplasmic lineage, cows within lineage, and residuals also are in Table 3. Ideally, 1982 PD values might have been regressed to account for herd level and herd variance during preadjustment. Such regression coefficients, however, could not be determined accurately for later parities; thus, 1982 PD values were used as additive adjustments.

Ratios of variance components from repeated records analysis are in Table 4. Variance caused by cytoplasmic lineage accounted

TABLE 2. F Statistics and residual mean squares for Model [1].

Source	Parity 1				Parity 2			
	df	MEMILK ¹	MEFAT	Fat, %	df	MEMILK	MEFAT	Fat, %
Year-season	27	2.43**	1.87**	1.71*	26	2.81**	1.47†	1.88**
Age at calving								
Linear	1	.01	.23	.31	1	.51	.03	2.10
Quadratic	1	.01	.24	.37	1	.37	.01	1.98
Transmitting value	1	30.63**	29.80**	106.66**	1	22.18**	12.14**	84.79**
Generations to origin	1	.12	.53	1.61	1	.24	.14	.02
Maternal lineage	52	1.47*	1.67**	1.97**	49	1.21	1.53*	2.03**
Residual mean square	580	2,236,085	3082	.12	329	1,623,664	2439	.10

¹MEMILK = Mature equivalent milk yield, MEFAT = mature equivalent fat yield.

† $P > F \leq .10$.

* $P > F \leq .05$.

** $P > F \leq .01$.

TABLE 3. Distribution of records in repeated records Model [2] and variances for maternal lineage, cows within lineage, and error.

Trait ¹	(n)	Cows	Lineages	σ_{ML}^2	σ_C^2	σ_e^2
MEMILK	1595	669	53	126,385	1,258,207	1,039,727
MEFAT	1595	669	53	161	2442	1300
Fat, %	1593	667	53	.0146	.0556	.0388

¹MEMILK = Mature equivalent milk yield, MEFAT = mature equivalent fat yield.

for 4 to 10% of phenotypic variance after removal of a portion of additive nuclear effects. Phenotypic variance was defined as the sum of maternal lineage, cow, and residual variances, as listed in Table 3. The ratio of cytoplasmic to residual variance ranged from 12 to 38%. This ratio was much greater than that from the analysis using Model [1] or from previous reports (1, 12). One explanation is that inclusion of cow effects in the model with repeated measures decreased residual variance and inflated the ratio of maternal lineage to residual variance. Perhaps ratios of maternal lineage to phenotypic (5.2 to 10.5%) or to cow variance (6.6 to 17.2%) are more stable measures of the importance of maternal lineage. Even after inclusion of cow effects, appreciable differences exist among maternal lineages, especially for fat percentage.

Estimates of variance components from Model [3], including animal and permanent environment, are in Table 5. Phenotypic variance is the sum of animal, permanent environmental, and error variance for Model [3]. Animal variances as a ratio to total phenotypic variances (heritabilities) from Model [3] are in Table 6. Heritabilities for milk and fat yields are in the range of recent estimates for Holsteins (20, 23). Heritability of fat percentage was slightly higher than recent reports (23) but similar to the estimate by deJager and Kennedy (5). Ratios of permanent environmental variance to phenotypic variance are also in Table 6.

Variance estimates from Model [4], which includes maternal genetic effects and covariance between additive animal and maternal genetic effects, are also in Table 5. Ratios of maternal genetic variance and covariance to phenotypic variance from this model are also in Table 6. Maternal genetic ratio was small for milk and fat yields, but it was .065 for fat

percentage. Inclusion of maternal genetic and covariance terms in Model [4] decreased the portion of variance previously partitioned to additive genetic effects (Model [3]) for milk and fat percentage. Total phenotypic variance explained by each model was nearly identical. The covariance ratio of .0599 was not readily explained. It may be a result of sampling error. Confounding between additive and maternal effects could produce covariance among errors of estimates.

Model [4] was reanalyzed assuming no covariance between additive direct and maternal genetic components. Results of this model (Model [4b]) are in Tables 5 and 6. Use of likelihood ratio tests (18) showed that covariance terms were not significantly different from zero. Log-likelihoods for models including a maternal genetic component were actually smaller than those for the model with only animal and permanent environmental components. Maternal genetic effects and covariances were not important in this study.

Because mtDNA is passed from female to offspring with no segregation, inclusion of maternal lineage effects (indicative of mtDNA) as fixed effects in a mixed model is arguably appropriate. Maternal lineages are exactly

TABLE 4. Variance ratios of REML solutions for maternal lineages from Model [2].

Trait ²	σ_{ML}^2/σ_P^2	σ_{ML}^2/σ_e^2	σ_{ML}^2/σ_C^2	σ_C^2/σ_P^2
MEMILK	.052	.121	.100	.519
MEFAT	.041	.124	.066	.626
Fat, %	.105	.376	.172	.614

$$^1\sigma_P^2 = \sigma_{ML}^2 + \sigma_C^2 + \sigma_e^2$$

²MEMILK = Mature equivalent milk yield, MEFAT = mature equivalent fat yield.

TABLE 5. Variance estimates with or without maternal genetic effects.

Trait and model ¹	Animal (A)	Permanent environment (PE)	Maternal genetic (M)	Covariance (Cov)	Phenotype	Error
MEMILK, kg²						
Model [3]	575,951	736,359			2,292,447	980,138
Model [4]	323,483	811,067	59,609	138,660	2,314,464	981,646
Model [4b]	583,040	734,214	14		2,296,631	979,362
MEFAT, kg²						
Model [3]	1130	1009			3434	1295
Model [4]	1179	989	1	-14	3448	1293
Model [4b]	1130	992	1		3423	1299
Fat, %						
Model [3]	.0944	.0242			.1554	.0368
Model [4]	.0863	.0223	.0101	-.0004	.1551	.0369
Model [4b]	.0846	.0234	.0098		.1546	.0368

¹ Model [3] includes A and PE; Model [4] includes A, PE, M, and Cov(A,M); and Model [4b] includes A, PE, and M. MEMILK = Mature equivalent milk yield, MEFAT = mature equivalent fat yield.

duplicated in offspring of the same lineage. Southwood et al. (24) included cytoplasmic effects as random when reporting the ability of animal models to partition them from maternal genetic effects. Small maternal genetic variance ratios from Model [4] help to clarify the question of "whether reported values are true estimates of cytoplasmic variance or due to random fluctuations of other maternal genetic effects," posed by Southwood et al. (24).

Distributions of maternal lineage solutions for MEMILK, MEFAT, and fat percentage treated as fixed effects are in Figure 1. Most,

but not all, solutions fell within one phenotypic standard deviation of zero. Solutions were from -1715 to 1219 kg, -77 to 77 kg, and -.51 to .39% for MEMILK, MEFAT, and fat percentage, respectively, in this herd. Fixed maternal lineage solutions were very nearly identical under Models [3] or [4] for MEMILK, MEFAT, and fat percentage and further supported the conclusion that maternal lineage effects are not caused by unaccounted nuclear maternal genetic differences because their inclusion did not change differences among maternal lineage solutions.

TABLE 6. Ratio of parameter estimates to phenotypic variance $\times 100\%$ with or without maternal genetic effects.

Trait and model ¹	Animal (A) (h ²)	Permanent environment (PE)	Maternal genetic (M)	Covariance (Cov)
MEMILK				
Model [3]	25.12	32.12		
Model [4]	13.98	35.04	2.58	5.99
Model [4b]	25.39	31.97	.00	
MEFAT				
Model [3]	32.90	29.40		
Model [4]	34.20	28.68	.04	-.41
Model [4b]	33.01	28.99	.04	
Fat, %				
Model [3]	60.80	15.60		
Model [4]	55.62	14.35	6.50	-.23
Model [4b]	54.70	15.16	6.36	

¹ Model [3] includes A and PE; Model [4] includes A, PE, M, and Cov(A,M); and Model [4b] includes A, PE, and M. MEMILK = Mature equivalent milk yield, MEFAT = mature equivalent fat yield.

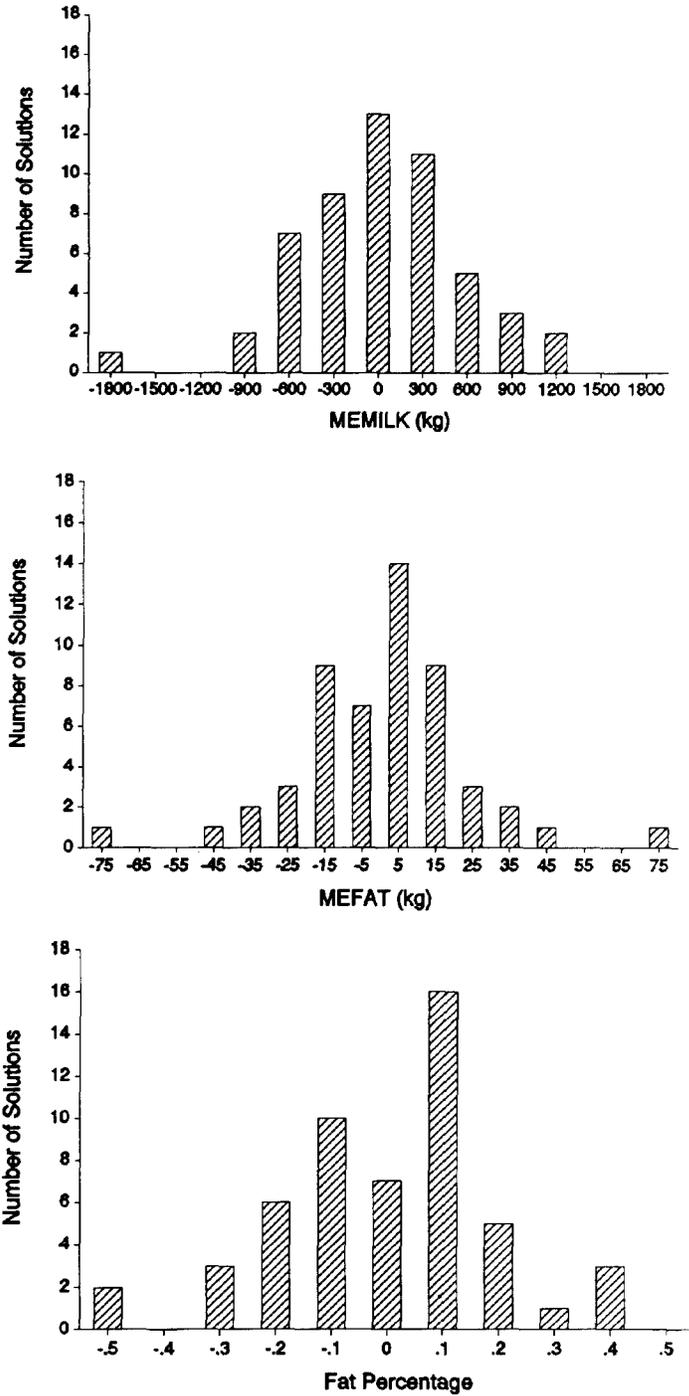


Figure 1. Distribution of maternal lineage solutions. MEFAT is mature equivalent fat yield, and MEMILK is mature equivalent milk yield. Numbers on horizontal axis are center points of discrete classes of equal length. For example, (□), (■), (●), (○), (▲), (▼), (△), (▽), 0 for MEMILK represents solutions between -150 and 150, and 300 represents solutions between 150 and 450.

TABLE 7. Tests of significance of maternal lineage effects.¹

	Variance ratio source							
	DFREML (from data)				Population ⁴ (from national evaluations)			
	Trait ²	F	P > F	MSE ³	Trait	F	P > F	MSE
Fixed sire groups ⁵	MEMILK	1.04	.397	4,763,298	MEMILK	1.17	.188	5,121,185
	MEFAT	1.08	.325	6293	MEFAT	1.47	.017	7131
	Fat, %	1.38	.039	.0367	Fat, %	1.71	.001	.0400
Westell groups ⁶	MEMILK	.94	.597	4,762,1546	MEMILK	1.09	.308	5,125,973
	MEFAT	1.03	.416	6274	MEFAT	1.39	.035	7103
	Fat, %	1.48	.015	.0371	Fat, %	1.82	.001	.0404

¹Degrees of freedom for maternal lineages = 52.

²MEMILK = Mature equivalent milk yield, MEFAT = mature equivalent fat yield.

³MSE = Mean squared error.

⁴Variance ratios were based on heritabilities of .2, .2, and .5 and repeatabilities of .5, .5, and .7 for MEMILK, MEFAT, and Fat, %, respectively.

⁵Residual degrees of freedom = 1829.

⁶Residual degrees of freedom = 1845.

Tests of significance of maternal lineage effects are in Table 7. For Model [3], with variance ratios estimated from these data, *F* values were 1.04, 1.08, and 1.38 for MEMILK, MEFAT, and fat percentage, respectively, and were significant only for fat percentage. Because all sires were from outside this herd, effects of maternal lineages on yield traits also were tested in Model [3] but with variance ratios more typical of values used on a national basis (G. Wiggans, 1990, personal communication). The *F* values (Table 7) were somewhat greater, and associated probability values were much smaller. Small changes in *F* values affect probability values greatly with many degrees of freedom. The *F* values were influenced appreciably by the use of different variance

ratios. Significant maternal lineage effects were observed for MEFAT and fat percentage.

Effects of maternal lineages also were tested applying Westell grouping strategies to account more completely for genetic similarities among base cows and selected AI sires (27) in conjunction with both sets of variance ratios described. Probability levels associated with *F* values did not differ greatly under either grouping scheme for the same variance ratios. In all instances, maternal lineage effects on fat percentage were significant. When variance ratios like those from national evaluations were used, maternal lineages also significantly influenced MEFAT.

Because fat is the component containing the most energy in milk and was significantly

TABLE 8. Variance estimates of SNF and calculated energy in milk.¹

Trait	Animal ² (A)	Permanent environment ² (PE)	Phenotype	Error
SNF, kg ²	4390 (23.09)	6425 (33.79)	19,011	8196
SNF, % ²	.0618 (58.51)	.0037 (3.53)	.1056	.0401
Energy, kcal/kg ²	1308 (60.82)	347 (16.12)	2150	496
Lactation energy, Mcal × 10 ²	3307 (26.75)	3948 (31.94)	12,361	5105

¹Model [3] includes A and PE.

²Ratios to phenotypic variance × 100% are in parentheses.

TABLE 9. Tests of significance for effects on SNF and calculated energy in milk from Model [3].^{1,2}

Trait	δ_p	F	P > F
SNF, kg	138	1.00	.475
SNF, %	.325	1.02	.444
Energy, kcal/kg	46	1.42	.028
Lactation energy, Mcal \times 10	111	1.03	.423

$${}^1\delta_p = (\delta_A^2 + \delta_{PE}^2 + \delta_e^2)^{1/2}$$

²Model [3] includes animal and permanent environment.

influenced by maternal lineages, which are considered indicative of mtDNA, perhaps effects of maternal lineage are exhibited through differences in efficiencies of conversion of precursors to milk fat by the cow. Lactose and protein are also energy-containing components in milk, but only information for SNF was complete for this study. Milk net energy, as reported in Table 8, was calculated according to Tyrrell and Reid (25) as follows:

$$\text{net energy} = 41.84(\text{fat \%}) + 22.29(\text{SNF \%}) - 25.58.$$

Lactation net energy in milk was calculated by multiplying net energy by MEMILK yield.

Variance components of random effects in Model [3] for SNF, SNF percentage, milk energy, and lactation energy are in Table 8. Heritability of SNF was smaller than MEFAT, but heritability of SNF percentage was nearly the same as for fat percentage. The ratio of permanent environmental to phenotypic variance was much smaller for SNF percentage than for fat percentage. Variance ratios for net energy in milk were nearly identical to those for fat percentage, perhaps because fat percentage receives the highest weight in calculation of milk net energy. Possibly for a similar reason, variance ratios for lactation net energy in milk were similar to those for MEMILK. The F statistics and associated probability levels are in Table 9. Maternal lineages significantly affected energy concentration in milk from this herd of dairy cattle.

CONCLUSIONS

Maternal lineage effects, considered indicative of cytoplasmic inheritance that is likely

related to mtDNA, were significant for fat percentage, net energy of milk, and, to a lesser extent, MEFAT yield. Maternal lineages did not significantly affect MEMILK, SNF, SNF percentage, or lactational energy of milk. Because SNF traits are composites of lactose, proteins, and minerals, future work is warranted to examine these milk constituents separately.

Unaccounted maternal genetic effects or their covariance with additive animal effects did not seem important as an explanation for maternal lineage effects. Variance components for maternal genetic and covariance terms were not significantly different from zero. Animal models with variance components only for animal and permanent environment were most appropriate for this analysis. Models with complete additive relationships should eliminate most concerns that maternal lineage effects could be caused by spurious additive genetic effects.

Several possible consequences of maternal lineage effects on traits of economic importance are foreseen. Because mitochondria are transferred to offspring via dam only, there has been no exploitation of potential gains from selection of more efficient genotypes. First, maternal lineage differences could be employed in embryo transfer programs to choose donor and recipient females to produce replacement heifers. Second, adjustment for maternal lineage when selecting potential bulldams could increase the accuracy of predicting a son's breeding value. Finally, current cloning techniques in dairy cattle involve transfer of cells to enucleated ova, without regard to cytoplasmic content. Potential exists for increasing performance by enucleating ova from females with inferior nucleus genes but from superior maternal lineages.

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