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GENETICS AND BREEDING

Variances of Direct Genetic Effects, Maternal Genetic Effects, and Cytoplasmic Inheritance Effects for Milk Yield, Fat Yield, and Fat Percentage[1]

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ABSTRACT

Milk yield, fat yield, and fat percentage during the first three lactations were studied using New York Holsteins that were milked twice daily over a 305-d, mature equivalent lactation. Those data were used to estimate variances from direct and maternal genetic effects, cytoplasmic effects, sire by herd interaction, and cow permanent environmental effects. Cytoplasmic line was traced to the last female ancestor using DHI records from 1950 through 1991. Records were 138,869 lactations of 68,063 cows calving from 1980 through 1991. Ten random samples were based on herd code. Samples averaged 4926 dams and 2026 cytoplasmic lines. Model also included herd-year-seasons as fixed effects and genetic covariance for direct-maternal effects. Mean estimates of the effects of maternal genetic variances and direct-maternal covariances, as fractions of phenotypic variances, were 0.008 and 0.007 for milk yield, 0.010 and 0.010 for fat yield, and 0.006 and 0.025 for fat percentage, respectively. Average fractions of variance from cytoplasmic line were 0.011, 0.008, and 0.009 for milk yield, fat yield, and fat percentage. Removal of maternal genetic effects and covariance for maternal direct effects from the model increased the fraction of direct genetic variance by 0.014, 0.021, and 0.046 for milk yield, fat yield, and fat percentage; little change in the fraction was due to cytoplasmic line. Exclusion of cytoplasmic effects from the model increased the ratio of additive direct genetic variance to phenotypic variance by less than 2%. Similarly, when sire by herd interaction was excluded, the ratio of direct genetic variance to phenotypic variance increased 1% or less.

(**Key words**: restricted maximum likelihood, variance components, Holsteins, cytoplasmic effects)

INTRODUCTION

Maternal effects have been defined as any influence from a dam on its offspring, excluding the effects of directly transmitted genes that affect performance of the offspring (16). Biological mechanisms to explain maternal effects include cytoplasmic inheritance, intrauterine and postpartum nutrition provided by the dam, antibodies and pathogens transmitted from dam to offspring, and maternal behavior (11).

Differences in heritability estimates obtained by daughter-dam regressions and paternal half-sib correlations have been considered as an indication of maternal effects for milk yield (7, 27, 28).

According to Willham (31), in mammals, environmental variation in the offspring is partially due to genetic variation of some other traits from the dams, such that quantitative traits can be influenced by two genetic components, animal genotype (direct genetic effect) and dam genotype (maternal genetic effect). There are indications that maternal genetic effects are not important for yield traits of dairy cattle (19, 29), although estimates of variance components for these effects are not abundant.

Some evidence suggests that maternal lineage effects, considered as cytoplasmic line effects, can affect yield and reproductive traits of dairy (2, 12, 19) and beef (23) cows. A significant effect of cytoplasmic inheritance on milk yield also was described for beef cows (23). Reed and Van Vleck (17), comparing daughter-dam and granddaughter-grandam regressions, found no evidence of cytoplasmic effects on milk yield, fat yield, or fat percentage. However, Kirkpatrick and Dentine (14) presented an alternative model to explain the results of Reed and Van Vleck (17), concluding that those results were consistent with the presence of cytoplasmic inheritance. Kennedy (13) simulated a data file without cytoplasmic effects and found results that were consistent

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with those described by Bell et al. (2) for actual data from similar models. Kennedy (13) concluded that additive genetic covariances that were not accounted for by the model may produce spurious cytoplasmic effects and suggested that an animal model analysis could separate cytoplasmic effects from additive genetic effects. Also using simulation, Southwood et al. (22) demonstrated that animal models can be used to partition variation from additive direct effects, maternal genetic effects, and cytoplasmic effects. Data used by Tess et al. (23) were reanalyzed using an animal model with cytoplasmic lines as fixed effects, and no evidence of cytoplasmic effects was found (24). Also using an animal model to analyze the fat-corrected milk yield of Holsteins, Faust et al. (9) found that cytoplasmic lines differed by as much as 1447 to 1846 kg and by 669 to 769 kg of milk when these lines contained ≥5 and ≥20 females, respectively. Faust et al. (9) suggested that the results of Bell et al. (2) were not due to unaccounted for genetic effects as suggested by Kennedy (13). Schutz et al. (19), working with animal models and records from an experimental herd, found significant effects of cytoplasmic line on milk fat percentage. In a preliminary report of the present study, Van Vleck et al. (26) concluded that cytoplasmic line did not account for much of the variation in milk and fat yields. Finally, Boettcher and Gibson (4) found that cytoplasmic effects accounted for 0.41%, 0.34%, and 0.47% of total variance for milk yield, fat yield, and fat percentage using 245,510 first lactations of Canadian Holstein cows with an animal model.

The objective of the present paper was to quantify the contribution of additive direct and maternal genetic effects and cytoplasmic line effects to phenotypic variance of milk yield, fat yield, and fat percentage using large samples of field data.

MATERIALS AND METHODS

The data consisted of milk yield, fat yield, and fat percentage during the first three lactations of New York Holstein cows that were milked twice daily over a 305-d, mature equivalent lactation. Unregistered cows and cows with records from <240 DIM were deleted.

Cytoplasmic line (i.e., animals having the same cytoplasmic origin) was determined by tracing female paths to the last female ancestor using DHI records from 1950 through 1991. The analyses used 138,869 lactation records from 68,063 cows calving from 1980 through 1991. All herds had to have cows calving in at least 3 yr and had to have a minimum of 100 cows. First lactation yield was required for all cows, and later lactations were used only if the previous one was recorded. Four seasons of calving were defined: January to March, April to June, July to September, and October to December. The data were assigned randomly to 10 samples based on the herd code. Structure of samples and overall means for the three traits are presented in Table 1.

TABLE 1. Summary of structure of data for 10 samples of herds.

Data	Mean average	Maximum	Minimum
	(no.)		
Records	13,887	16,563	11,760
Animals in $\mathbf{A}^{-1}$	10,565	12,343	8912
Mixed model equations	34,159	40,263	28,682
Dams	4927	5761	4151
Sires	1174	1332	1081
Sire by herd interaction	2668	3186	2196
Dam line	2026	2541	1638
Cows	6806	8023	5678
Herd-year-season	1530	1908	1292
Means			
Milk, kg	9022	9305	8844
Fat, kg	327	334	323
Fat, %	3.6	3.7	3.6

The data were analyzed using the derivative-free REML (10, 20) algorithm developed by Boldman et al. (5) and Boldman and Van Vleck (6). A complete animal model was described as

$$\mathbf{y} = \mathbf{X}\beta + \mathbf{Zg} + \mathbf{Mm} + \mathbf{Ps} + \mathbf{Dc} + \mathbf{Wpe} + \mathbf{e} \quad [1]$$

where $\mathbf{y}$ = vector of observations, β = vector of fixed effects (herd-year-seasons), $\mathbf{g}$ = vector of additive direct genetic effects of animals, $\mathbf{m}$ = vector of additive maternal genetic effects, $\mathbf{s}$ = vector of sire by herd interaction effects, $\mathbf{c}$ = vector of cytoplasmic effects, $\mathbf{pe}$ = vector of permament environmental effects, $\mathbf{e}$ = vector of residual effects, and $\mathbf{X}$, $\mathbf{Z}$, $\mathbf{M}$, $\mathbf{P}$, $\mathbf{D}$, and $\mathbf{W}$ = incidence matrices that associate the appropriate effects to $\mathbf{y}$. For this model, $E[\mathbf{y}] = \mathbf{X}\beta$, $E[\mathbf{g}] = 0$, $E[\mathbf{m}] = 0$, $E[\mathbf{s}] = 0$, $E[\mathbf{c}] = 0$, and $E[\mathbf{e}] = 0$; $Var[\mathbf{g}] = \mathbf{A}\sigma^2_g$, $Var[\mathbf{m}] = \mathbf{A}\sigma^2_m$, $Cov[\mathbf{g},\mathbf{m}'] = \mathbf{A}\sigma_{gm}$, $Var[\mathbf{s}] = \mathbf{I}_{NS}\,\sigma^2_s$, $Var[\mathbf{c}] = \mathbf{I}_{NC}\,\sigma^2_c$, $Var[\mathbf{pe}] = \mathbf{I}_{NP}\,\sigma^2_{pe}$, and $Var[\mathbf{e}] = \mathbf{I}_N\,\sigma^2_e$, NS = number of sire by herd effects, NC = number of maternal lines, NP = number of cows, N = number of records, $\mathbf{A}$ = numerator relationship matrix, and $\mathbf{I}$ = identity matrix. The vectors $\mathbf{g}$ and $\mathbf{m}$ are not correlated with vectors $\mathbf{s}$, $\mathbf{c}$, $\mathbf{pe}$, and $\mathbf{e}$.

Another five single-trait animal models were fitted: Model [2] was as Model [1] but excluding cytoplasmic effects, Model [3] was as Model [1] but excluding additive maternal effects and covariance between ad-

TABLE 2. Mean (±SE) estimates of fractions of phenotypic variance and empirical standard errors for milk yield, fat yield, and milk fat percentage and phenotypic variance (σ^2) from 10 samples using Model [1].

	Milk yield				Fat yield				Fat percentage			
Fraction[1,2]	$\bar{X}$	SE	Maximum	Minimum	$\bar{X}$	SE	Maximum	Minimum	$\bar{X}$	SE	Maximum	Minimum
g^2	0.278	0.008	0.318	0.232	0.595	0.013	0.656	0.533	0.281	0.009	0.341	0.250
m^2	0.008	0.003	0.029	0.000	0.006	0.004	0.043	0.000	0.010	0.005	0.045	0.000
gm	0.007	0.006	0.041	–0.010	0.025	0.006	0.053	–0.007	0.010	0.006	0.038	–0.019
sh^2	0.018	0.003	0.038	0.007	0.017	0.004	0.034	0.002	0.020	0.004	0.041	0.007
c^2	0.011	0.004	0.034	0.000	0.009	0.002	0.023	0.000	0.008	0.003	0.024	0.000
pe^2	0.246	0.006	0.274	0.216	0.173	0.009	0.204	0.132	0.236	0.006	0.274	0.213
e^2	0.432	0.005	0.456	0.403	0.175	0.003	0.189	0.160	0.435	0.004	0.451	0.417
σ^2	1724	25	1901	1636	1561	21	1682	0.533	2304	33	2473	2149

[1]g = Additive direct genetic effect, m = additive maternal genetic effect, gm = covariance direct and maternal genetic effects, sh = sire by herd interaction effect, c = cytoplasmic line effect, p = permanent environmental effect, and e = residual.

[2]Phenotypic variances: (kilograms)^2/1000 for milk, kilograms squared for fat, and (percentage)^2 × 10,000 for fat percentage.

ditive direct and maternal effects, Model [4] was as Model [3] but excluding sire by herd interactions effects, Model [5] was as Model [3] but excluding cytoplasmic effects, and Model [6] was as Model [5] but excluding sire by herd interaction effects.

Convergence was assumed when the simplex variance reached $<10^{-6}$. All analyses were restarted to check for the occurrence of local maxima until the log-likelihood did not change beyond the first decimal. Estimates of variance components of the samples for each model were averaged and, assuming that the samples were not correlated, empirical standard errors were calculated.

RESULTS AND DISCUSSION

Mean values for variance and covariance components as ratios of phenotypic variances from Model [1] are in Table 2. For all traits, the variance for additive maternal genetic effects varied from 0.8 to 1% of phenotypic variance, and the covariance between maternal and direct genetic effects varied from 0.7 to 2.5%. Thus, these effects did not contribute importantly to phenotypic variance. Using different pairs of cousins, Van Vleck and Hart (29) estimated variances for additive direct and maternal effects and dominance direct and maternal genetic effects; covariances were estimated for direct maternal effects for milk yield. The authors (29) concluded that only additive genetic effects were important for this trait (38% of total variation). The current estimates are smaller than those that were credited to Brumby in Schutz et al. (19) for maternal genetic effects on milk yield (8 to 14% of total variance). Schutz et al. (19), using an animal model, also estimated larger values for ratios of variances for maternal genetic effects to phenotypic variances for milk yield (2.58%) and fat percentage (6.5%) and for covariances between maternal and direct effects for milk yield (5.99%) than the estimates in the present work. However, these estimates were not significantly different from zero. Maternal genetic effects have been described in domestic mammals such as swine (18, 21) and beef cattle (3, 15) and represent an environmental effect on the growth of offspring from birth to weaning. However, dairy calves are separated from their dams at birth so that the influence of the dam would be only through intrauterine environment.

Cytoplasmic line effects do not seem to be important for milk yield, fat yield, or fat percentage, although estimates obtained from some samples reached 3.4, 2.4, and 2.3% of phenotypic variance for milk yield, fat yield, and fat percentage, respectively. Mean values for ratios, however, were 0.011, 0.008, and 0.009 in the same order. These results are similar to those described by Boettcher and Gibson (4) for milk yield, fat yield, and fat percentage for the first lactation of Canadian Holsteins, also using an animal model and Gibbs sampling (about 0.5% of total variation). Larger estimates have been described in the literature for Holsteins (2, 19). Bell et al. (2), using 4461 first lactations from Holstein cows and a set of models including cytoplasmic effects as fixed, concluded that cytoplasmic effects significantly influenced milk yield, fat yield, and fat percentage and were responsible, respectively, for 2.0, 1.8, and 3.5% of total variation. Using similar models, Huizinga et al. (12) analyzed 290 first lactations of Holstein Friesian and Dutch Friesian crossbred cows and reported that cytoplasmic effects contributed 6.0, 5.0, 1.0, and 13.0% of the phenotypic variance of milk yield, fat percentage, fat yield plus protein yield, and milk returns (net income per lactation), respectively. Using simulation, Kennedy (13) found indications that those results could be due to drift variance of additive

TABLE 3. Fractions[1] of phenotypic variance (± empirical SE) and phenotypic variance [σ^2; (kilograms)^2/1000] for milk yield using six animal models.

Model	g^2	SE	m^2	SE	gm	SE	sh^2	SE	c^2	SE	p^2	SE	e^2	SE	σ^2	SE
[1]	0.278	0.008	0.008	0.003	0.007	0.006	0.018	0.003	0.011	0.004	0.246	0.006	0.432	0.005	1724	25
[2]	0.284	0.008	0.011	0.004	0.013	0.004	0.018	0.003			0.244	0.006	0.431	0.005	1725	25
[3]	0.298	0.010					0.017	0.003	0.012	0.003	0.242	0.007	0.432	0.005	1726	25
[4]	0.308	0.009							0.011	0.003	0.248	0.007	0.433	0.005	1724	26
[5]	0.319	0.007					0.016	0.003			0.235	0.007	0.430	0.005	1730	25
[6]	0.326	0.007									0.242	0.007	0.432	0.005	1727	25

[1]g = Additive direct genetic effect, m = additive maternal genetic effect, gm = covariance direct and maternal genetic effects, sh = sire by herd interaction effect, c = cytoplasmic line effect, p = permanent environmental effect, and e = residual.

genetic effects that was not taken into account by the model. More recently, Schutz et al. (19) studied cytoplasmic effects using data from an experimental herd. For repeated records that had been preadjusted for sire and one-half of maternal grandsire PTA and including cow and cytoplasmic effects as random effects in the model, Schutz et al. (19) found that cytoplasmic effects accounted for 5.2, 4.1, and 10.5% of phenotypic variances for milk yield, fat yield, and fat percentage, respectively. Those same researchers included cytoplasmic effects as fixed effects in an animal model with additive genetic direct effects, additive genetic maternal effects, and permanent environmental effects as random and concluded that cytoplasmic effects significantly affected fat percentage but not milk yield. The current estimates, in addition to being smaller than those reported, did not show larger fractional cytoplasmic effects for fat percentage than for milk and fat yields as described by Bell et al. (2) and Schutz et al. (19). According to Kennedy (13), this larger influence of cytoplasmic effects on fat percentage was not a result of the role of mitochondria in fatty acid synthesis as proposed by Bell et al. (2), but rather was a result of the larger heritability estimates for fat percentage than for milk yield. In this case, ignored additive genetic effects could have a larger effect on variances that were due to cytoplasmic line for this trait than for milk yield.

As already reported by Dimov et al. (8), also using New York data, estimates of variance components from sire by herd effects as ratios of phenotypic variances were between 0.017 and 0.020, which was much smaller than that used for national genetic evaluations of 0.14 (25, 30), but similar to those shown in Table 2. Banos and Shook (1), with a sire model, estimated that sire by herd interaction accounted for 1.84, 2.11, and 3.00% of total variation of milk yield in the first, second, and third lactations, respectively.

Estimates of heritability and repeatability for milk yield, fat yield, and fat percentage are similar to those reported recently (8, 19).

Variance components for milk yield, fat yield, and fat percentage as ratios of phenotypic variances for the six models are presented on Tables 3, 4, and 5, respectively. For milk yield (Table 3), for each effect dropped from the model, estimates of heritability of direct genetic effects increased by 0.006 to 0.014. The greatest increase was from Model [2] to [3] (i.e., including cytoplasmic effects and excluding genetic maternal effects and covariance between direct and maternal genetic effects in the model). The same pattern was observed for fat yield and fat percentage (Tables 4 and 5). For each effect dropped from the model, estimates of heritability of direct genetic effects increased by 0.004 to 0.021 for fat yield and by 0.006 to 0.046 for fat percentage.

TABLE 4. Fractions[1] of phenotypic variance (± empirical SE) and phenotypic variance [σ^2; (kilograms)^2] for fat yield using six animal models.

Model	g^2	SE	m^2	SE	gm	SE	sh^2	SE	c^2	SE	p^2	SE	e^2	SE	σ^2	SE
[1]	0.281	0.009	0.010	0.005	0.010	0.006	0.020	0.004	0.008	0.003	0.236	0.006	0.435	0.004	2304	33
[2]	0.285	0.009	0.011	0.005	0.014	0.005	0.020	0.004			0.235	0.007	0.435	0.004	2305	33
[3]	0.306	0.009					0.019	0.003	0.009	0.003	0.231	0.007	0.435	0.004	2308	34
[4]	0.316	0.008							0.010	0.002	0.239	0.007	0.436	0.004	2304	33
[5]	0.323	0.007					0.018	0.004			0.225	0.007	0.434	0.004	2312	34
[6]	0.332	0.006									0.233	0.007	0.435	0.004	2308	33

[1]g = Additive direct genetic effect, m = additive maternal genetic effect, gm = covariance direct and maternal genetic effects, sh = sire by herd interaction effect, c = cytoplasmic line effect, p = permanent environmental effect, and e = residual.

TABLE 5. Fractions[1] of phenotypic variance (± empirical SE) and phenotypic variance [σ^2; (percentage)^2 × 10,000] for fat percentage using six animal models.

Model	g^2	SE	m^2	SE	gm	SE	sh^2	SE	c^2	SE	p^2	SE	e^2	SE	σ^2	SE
[1]	0.595	0.013	0.006	0.004	0.025	0.006	0.017	0.004	0.009	0.002	0.173	0.009	0.175	0.003	1561	21
[2]	0.601	0.013	0.007	0.004	0.031	0.004	0.017	0.004			0.171	0.009	0.175	0.004	1566	22
[3]	0.647	0.011					0.015	0.001	0.012	0.008	0.152	0.010	0.174	0.003	1569	21
[4]	0.653	0.011							0.012	0.002	0.160	0.010	0.175	0.003	1567	20
[5]	0.668	0.011					0.014	0.004			0.144	0.010	0.174	0.003	1573	21
[6]	0.674	0.011									0.152	0.010	0.174	0.003	1571	20

[1]g = Additive direct genetic effect, m = additive maternal genetic effect, gm = covariance direct and maternal genetic effects, sh = sire by herd interaction effect, c = cytoplasmic line effect, p = permanent environmental effect, and e = residual.

Exclusion of cytoplasmic line from the model (Model [1] to [2] and Model [4] to [5]) increased estimates of heritability of direct genetic effects by 0.006 and 0.010 for milk yield, 0.004 and 0.007 for fat yield, and 0.006 and 0.015 for fat percentage, respectively. Estimates of additive maternal genetic variance and estimates of covariance between direct and maternal effects, as ratios of phenotypic variances (Model [1] to [2]), increased, respectively, 0.003 and 0.006 for milk yield, 0.001 and 0.004 for fat yield, and 0.001 and 0.006 for fat percentage. These results show that cytoplasmic effects do not seem to make important contributions to phenotypic variation of these three traits, and estimates of variance from cytoplasmic lines do not seem to be confounded with estimates of variance from additive genetic effects.

The removal of additive genetic maternal effects and covariance between direct and maternal genetic effects from the model (Model [2] to [3]) increased estimates of heritability of direct genetic effects by 0.014, 0.021, and 0.046 for milk yield, fat yield, and fat percentage, respectively. Some confounding between direct and maternal genetic effects should be expected because the dam that contributes the maternal genetic effect also transmits half of her genetic value for direct effects to her daughter. Some cows do not have lactating daughters, and many sires do not have any lactating granddaughters. Ratios of cytoplasmic to phenotypic variance increased 0.001 for milk and fat and 0.003 for fat percentage when maternal genetic effects and covariance between maternal and direct effects were excluded from the model (Model [1] to [3]). The inclusion of maternal genetic effects in the model did not change the estimates of variance of cytoplasmic effects, which is in agreement with results reported by Schutz et al. (19).

Regardless of the model, fractions of variance because of sire by herd interaction effects and residual variance to phenotypic variance were similar for the three traits. Removal of effects of sire by herd interaction from the model (Model [3] to [4] and Model [5] to [6]) increased the estimates for fractions of additive direct genetic variance by a maximum of 0.01 of phenotypic variance. Banos and Shook (1) compared two models, with and without sire by herd interaction, and reported that the heritability of milk decreased after sire by herd interaction was included.

As already pointed out by Van Vleck et al. (26), it is not clear whether the small fractions obtained for additive maternal genetic effect and cytoplasmic effect are estimating real differences caused by these effects. Negative estimates for variance components are outside the parameter space for REML procedures; hence, small nonzero estimates can arise even when the parameter is zero. Analyses including nonsense variables in the model, such as the last digit from the animal or sire identification number, could help to clear up this point, but the computational effort to perform such an analysis may not be worthwhile.

CONCLUSIONS

Additive maternal genetic effects and covariance between maternal and direct genetic effects do not seem to make important contributions to the phenotypic variances of milk yield, fat yield, and fat percentage, probably because the only environmental influence of the dams on their calves is from conception to birth.

Ratios of variances from cytoplasmic effects to phenotypic variance were consistently small and were similar for the three traits. Inclusion or not of additive maternal genetic effects and covariances between maternal and direct effects in the model did not change the contributions of cytoplasmic line effects to phenotypic variances.

Each effect dropped from the model (cytoplasmic, maternal genetic, covariance between direct and maternal, and sire by herd interaction) increased estimates of direct heritability by about 0.01.

Whether these small values arose just because REML estimates were forced to be in the parameter space or whether they measured real effects is not clear. Because the contribution of maternal genetic effects and cytoplasmic effects to phenotypic variance is small, these effects are probably not important to genetic evaluations.

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