Influence of Nonadditive Effects on Estimation of Genetic Parameters in Dairy Cattle

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ABSTRACT

A population was simulated having progeny that descended from sires and dams with various fractions of genes from two breeds. Additive breed effects and nonadditive effects from breed crosses were simulated.

Data on performance were analyzed using mixed models that accounted for fixed additive genetic group effects and random sire effects. Three additive models, with genetic groups defined according to 1) breed composition of the progeny, 2) breed composition of the sire and the dam, or 3) linear regression on breed fraction of the progeny, were compared with a nonadditive model with a linear regression on breed fraction, heterozygosity and recombination in the genome of the progeny. Variance components were estimated using restricted maximum likelihood.

Additive genetic variance and heritability were overestimated for an additive model with progeny groups. Additive models gave estimates for breed difference, group effects, and breeding values that were not equal to true additive genetic values. Breed differences were overestimated when sire groups were used. Estimators for each parameter were unbiased with the nonadditive model.

INTRODUCTION

Due to increased exchange of semen and embryos, dairy cattle data often result from a mixture of genes from different populations. Therefore, the observed genetic effects in the resulting population might not be solely additive and may contain nonadditive genetic effects. In some crossbreeding programs, the objective is explicitly to estimate or to use the nonadditive genetic variation. However, nonadditive variance is not often used explicitly in dairy cattle breeding. Moreover, models for analysis of data from crossbred dairy populations often consider only additive genetic effects. Ignoring nonadditive variance might bias predictions of breeding values, which also may not be of minimum prediction error variance. Estimators of variance components and additive genetic parameters might be biased as well.

Genetic variation in crossbred populations usually is estimated from within breed variance, i.e., predictions of random effects are adjusted for fixed breed-group effects. Recently, estimates for heritability of milk production traits in crossbred populations, using mixed models with breed-group effects (12, 20, 22), were higher than published values from pure breeds (7, 10). Among other factors, nonadditive effects might have caused an increase in heritability estimates.

Experimental evidence for nonadditive effects in dairy production traits has been reviewed by several authors, [e.g., (14, 19)]. Estimates of heterosis, defined as the relative deviation of the F_1 mean from the midparent mean, for first lactation 305-d milk yield varied from −1.7 to 8.2%. Recent estimates for percentages of heterosis were 10% for Holstein Friesian × Red Danish (4) and 2% for Swedish Friesian × Swedish Red and White (2).

There are some examples of a linear relationship between degree of heterozygosity and heterosis (15). In most data from farm animals, however, heterosis in F_1 crosses is more than twice that in F_2 crosses (19). Such a deviation from linearity between degree of heterozygosity and observed heterosis can be due to epistatic

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effects, which are often estimated as recombination loss (8). There is a considerable variation in estimates of recombination loss for milk production, ranging from −12% (4) to −.4% (2). Crossbreeding parameters might be hard to estimate, particularly from field data, and well-designed experiments are required, at minimum (3, 18).

In this paper, the impact of heterosis and recombination loss on the estimation of additive genetic parameters was studied for data from crossbred populations. Computer simulation was used for a comparison of estimates from different models with known values. A half-sib structure, typical for estimation of variances in dairy populations, was considered.

MATERIALS AND METHODS

Simulation of Data

A dairy population from a combination of two breeds, Dutch Friesian (FH) and Holstein-Friesian (HF), was simulated. Cows originated from matings of different genetic groups on the basis of the fraction of HF genes.

Records were simulated according to the following model:

$$Y_{ijkmn} = \mu + h_i + ys_j + g_{s_k} + g_{d_m} + NA_{km} + a_{kmn} + e_{ijkmn}$$

where μ is the population mean of reference breed FH; h_i and ys_j are fixed environmental effects of herd and year season with i = 1,..,150 and j = 1,..,4; g_{s_k} and g_{d_m} are fixed additive effects of breed group of sire and of breed group of dam with k = 1,..,9 and m = 1,..,9; NA_{km} is the fixed nonadditive effect of interaction of sire group k by dam group m; a_{kmn} is the additive genetic effect of the cow making the record; and e_{ijkmn} is a residual effect. Sires and dams were genetically unrelated; only relations between sires and daughters were considered.

Heritability (h^2) was .30 and phenotypic variance (σ_p^2) was 518,400; μ was fixed at 6000 kg. A pseudorandom value for each of herd was sampled from N[0, .1 σ_p^2] and for each year-season effect, from N[0, .025 σ_p^2], corresponding to assumed fractions of the observed variance in milk production that is due to herds and year-seasons. The additive effect of the cow (a_{kmn}) was .5 * a_{sire} + $\sqrt{(.75)}$ * a_n, where a_{sire} and a_n were sampled from N[0, σ_a^2] and e_{ijkmn} was sampled from N[0, σ_e^2]. $\sigma_a^2 = h^2\ \sigma_p^2$ is additive genetic variance and $\sigma_e^2 = (1 - h^2)\ \sigma_p^2$ is residual variance.

Breed difference (HF − FH) was 800 kg. Additive genetic group effects were linearly related to fraction of HF genes of animals from that group. Let two parents be mated with fraction of HF genes being p_s for sire and p_d for dam where $p_s = (k - 1)/8$ for sires in group k and $p_d = (m - 1)/8$ for dams in group m. Group effects were .5 * p_s * 800 for sire group and .5 * p_d * 800 for dam group. The fraction HF genes (p_p) in the progeny was equal to [(p_s + p_d)/2].

Nonadditive effects (NA_{km}) originate either through dominance effects, from interactions between HF and FH genes within loci, or epistatic effects from interactions between loci. It is assumed that a very large number of loci contribute to the genetic variance with no linkage in segregation. Coefficients for the heterosis effect (HET) were derived from the degree of heterozygosity of the animals (3). Thus, heterosis represents dominance effects as well as 50% of additive by additive effect that is confounded with dominance. The coefficient for heterosis (b_{HET}) was [$p_s(1 - p_d) + p_d(1 - p_s)$].

Dickerson (1) defined recombination (REC) loss as a deviation from linear relation of performance with heterosis, such that "the coefficient of REC [loss] describes the average fraction of independently segregating pairs of loci in gametes from both parents which are expected to be nonparental combinations." The coefficient for a recombination effect (b_{REC}) was derived from the heterozygosity of the parental gametes, representing a within-gamete epistatic effect as [$p_s(1 - p_s) + p_d\ (1 - p_d)$]. Hill (6) argued that Dickerson's definition can be ambiguous, and he prefers an explicit parametrization with dominance effects and additive by additive effects (epistatic effects). Assuming that recombination refers to 50% of the

TABLE 1. Additive (A) and nonadditive[1] (NA) effects (kg) for different combinations of sire and dam groups for structure I.

Group of dam	% HF[2] Genes	Effect	Group of sire (% HF genes)		
			1 (0%)	5 (50%)	9 (100%)
1	0	A	0	200	400
		NA	0	80	320
5	50	A	200	400	600
		NA	80	0	80

[1]Heterosis = 5%, recombination loss = -5%.

[2]HF = Holstein-Friesian.

epistatic effects (with no linkage), however, the parametrization used by Dickerson (1) defines appropriately the sum of dominance and epistatic effects. Effects of heterosis and recombination were simulated with the level varying at 0, 5, and 10% of the phenotypic mean of the two breeds.

Three data structures were simulated with particular sire group by dam group combinations. For structure I, additive and nonadditive effects for each combination of sire and dam group are in Table 1. Matings were such that nonadditive effects were unequal within groups of progeny that had equal additive effect. Hence, additive effects and nonadditive effects were not confounded. In structure I, data were distributed equally over sire group by dam group combinations. A second structure was created to represent an actual mating situation in a gene importing country. Structure II (Table 2) was based on Dutch data from 399,383 crossbred cows (Dutch Friesian × Holstein Friesian) calving between 1983 and 1986 (21). Structure III (Table 3) represented a future generation of cows, which sires were distributed over groups according to the inseminations in 1987 in The Netherlands. Dams were distributed over groups according to the distribution of progeny that resulted from matings in structure II.

For each mating structure, a data set was simulated for 100 sires having progeny in a number of herds. Total number of records per herd averaged 33, with a standard deviation of 17. Progeny group size was 50 and alternative sizes were 25 and 100. For each herd, daughters were randomly assigned to dam groups and to sire groups according to the distribution of the structure. Such a design was sampled 10 times. To reduce sampling variance, 10 replications within each design resulted in 10 × 10 repetitions for each alternative. Using one design for several samples was attractive computationally.

Models for Evaluation

Additive models A1, A2, and A3 varied in strategy to account for fixed genetic effects. In A1, fixed group effects (g_p) were defined according to breed composition of the progeny making the record. In A2, two breed groups

TABLE 2. Distribution of data over sire and dam groups[1] for structure II.

Group of dam	Group of sire					
	1	5	7	8	9	Total
1	.251	.059	.034	.010	.495	.849
5	.004	.003	0	0	.014	.021
7	.015	.008	.006	0	.090	.119
8	0	0	0	0	.007	.007
9	0	0	0	0	.004	.004
Total	.270	.070	.040	.010	.610	1.000

[1]Group number i corresponds to (i - 1) * 12.5% Holstein-Friesian genes.

TABLE 3. Distribution of data over sire and dam groups[1] for structure III.

Group of dam	Group of sire					
	1	5	7	8	9	Total
1	.034	.030	.016	.009	.158	.247
2	0	0	0	0	.005	.005
3	.004	.008	.004	0	.055	.071
4	0	.005	.003	0	.033	.041
5	.012	.057	.031	.018	.385	.503
6	0	0	0	0	.016	.016
7	0	.010	.006	.003	.074	.093
8	0	0	0	0	.016	.016
9	0	0	0	0	.008	.008
Total	.050	.110	.060	.030	.750	1.000

[1]Group number i corresponds to (i – 1) * 12.5% Holstein-Friesian genes.

were defined according to the breed composition of the sire (g_s) and of the dam (g_d). Model A1 will be referred to as progeny group model and A2 as parent group model. Model A3 accounted for breed differences by a linear regression of performance on breed composition of progeny (p_p). In addition, a complete genetic model (nonadditive) was used that included a regression on p_p and on coefficients for heterosis and recombination in the progeny. Models can be represented as:

model A1:

$$Y_{ijkn} = HYS_i + g_{p_j} + s_k + r_{ijkn}$$

model A2:

$$Y_{ijkmn} = HYS_i + g_{s_j} + g_{d_m} + s_{jk} + r_{ijkmn}$$

model A3:

$$Y_{ijkn} = HYS_i + b_1 * p_{p_j} + s_k + r_{ijkn}$$

model NA:

$$Y_{ijkn} = HYS_i + b_1 * p_{p_j} + b_2 * b_{HET} + b_3 * b_{REC} + s_k + r_{ijkn}$$

Fixed herd-year-season (HYS) effects were used to account for environmental effects. Effects of HYS interactions were not simulated. Rather, HYS subclasses were used to create subclass sizes as is common in sire evaluation programs. Sire and residual effects were taken as random with variances var(s) = .25 * $I_s\sigma_a^2$ and var(r) = I_n (σ_e^2 + .75σ_a^2).

Variance components for s and r were estimated using REML (11). Solutions were computed for fixed additive group effects from the same model using REML estimates for the variance components. Breeding values were computed from model A2 as $2(g_{s_j} + s_{jk})$ or from model A1 as $(g_p + 2s_k)$, where g_p is the solution for progeny group with the same fraction of HF genes as the sire.

RESULTS

Genetic Parameters

Estimates of variance components and heritability were obtained for each model. Results for structure I are in Table 4, by levels of HET and REC averaged over 100 repetitions.

When nonadditive effects were absent, models showed similar results in estimates of variances and heritability. Hence, differences in expectations of Y_{ijkmn} due to fixed additive genetic effects were accounted for by each model; the parent group model was equivalent to the simulated model, and the progeny group model reflected the genetic mean of animals making the record. A linear regression on p_p (models A3 and NA) also accounted appropriately for differences due to breed effects.

TABLE 4. Estimates for variance components (σ_a^2,σ_e^2) and heritability (h^2) with additive (A1, A2, A3) and nonadditive (NA) models for analysis of different levels of heterosis (HET) and recombination (REC) effects simulated for structure I.

Model[1]	HET	REC	σ_a^2	σ_e^2	h^2
	(%)				
A1	0	0	148,543	479,780	.287
	10	0	196,263	480,569	.370
	5	-5	208,168	480,573	.390
	10	-5	270,781	481,009	.493
	10	-10	395,279	481,117	.681
A2	0	0	148,904	479,768	.287
	5	-5	148,640	484,100	.285
	10	-10	148,380	496,976	.277
A3	0	0	148,576	479,766	.287
	5	-5	195,545	484,932	.365
	10	-10	328,522	498,578	.565
NA	0	0	148,791	479,791	.287
	5	-5	148,791	479,791	.287
	10	-10	148,791	479,791	.287
Sampled values			151,955	479,220	.293

[1]A1 = Progeny group, A2 = parent group, A3 = regression group.

When nonadditive effects were different from zero, the progeny group model (model A1) gave biased estimates for additive genetic variance and heritability (Table 4). Overestimation of σ_a^2 and h^2 was relatively small for levels of heterosis and recombination of 5% and smaller. Bias increased with increasing level of heterosis and recombination. Estimates of σ_e^2 did not increase. Hence, the progeny group model accounted for nonadditive genetic effects by other effects in the model.

Estimates for additive variance were not biased using the parent group model (A2). In A2, however, the estimate of residual variance was higher resulting in a slight underestimation of heritability. A model with linear regression on p_p (A3) yielded an overestimation of both genetic and residual variance. Model NA accounted for all additive and nonadditive effects and showed unbiased estimates for each parameter at each level of nonadditive effect.

Structure I is an example of a balanced situation. To make inferences toward "real life"

TABLE 5. Estimates for variance components (σ_a^2 and σ_e^2) and heritability (h^2) with additive (A) and nonadditive (NA) models for analysis for structure II and III.[1]

Structure	Model	σ_a^2	σ_e^2	h^2
II	A1	160,430	480,281	.308
	A2	156,139	481,161	.300
	A3	245,806	484,272	.449
	NA	156,574	479,669	.301
III	A1	184,720	480,559	.350
	A2	154,825	481,225	.297
	A3	210,494	482,100	.393
	NA	155,344	480,143	.299

[1]Heterosis = 5%, recombination loss = -5%.

TABLE 6. Solutions[1] from different models for genetic groups of progeny(g_p), sire(g_s), or dam(g_d) and average predicted sire effects ($s_{j..}$) within sire groups for structure I.[2]

		Group solutions				Average sire effects		
Model	Effect	i = 3	i = 5	i = 7	i = 9	$s_{1..}$	$s_{5..}$	$s_{9..}$
A1	g_{p_i}	301	497	511	. . .	–45	–80	121
A2	$2g_{s_i}$	. . .	409	. . .	1113	0	0	0
A2	$2g_{d_i}$	. . .	235	. . .	. . .			
A3	$b_1{}^* p_{p_i}$	173	347	520	694	–65	–40	103
NA	$b_1{}^* p_{p_i}$	197	394	591	787	–2	2	0
Additive values		200	400	600	800	2	6	0

[1] $g_{p_1} = g_{s_1} = g_{d_1} = 0$.

[2]Heterosis = 5%, recombination loss = –5%.

situations, structures II and III were investigated, reflecting a first and second generation of cows in a country that imports semen. Table 5 gives estimates for variance components for data structures II and III, for moderate levels of heterosis and recombination effects. With structure II, each model except A3 gave estimates for variance components that were close to simulated values. Differences in heritability estimates were small as well. Differences between models were larger for structure III; models A1 and A3 gave considerable bias in estimates of additive genetic variance and heritability.

Estimation of Genetic Effects

Sire effects are usually interpreted as additive genetic effects within populations, and group effects as additive genetic differences between populations. Therefore, solutions for sire effects and for group effects were considered to be biased when they also contained nonadditive effects. Solutions were determined for a moderate level of nonadditive effects (5% for heterosis and –5% for recombination). Group solutions for animals with 0% HF genes were restricted to zero. Hence, additive genetic effects were expressed relative to the FH population.

Table 6 gives group solutions and average sire effects for structure I. Sire effects were averaged for groups of sires with equal breed composition. In the progeny group model (A1), sires were crossclassified with groups. Sire effects and group effects were each biased by nonadditive effects. Nonadditive effects were partly accounted for by solutions for groups,

TABLE 7. Solutions[1] from different models for genetic groups of progeny(g_p), sire(g_s), or dam(g_d) and average predicted sire effects ($s_{j..}$) within sire groups for structures II.[2]

		Group solutions				Average sire effects		
Model	Effect	i = 3	i = 5	i = 7	i = 9	$s_{1..}$	$s_{5..}$	$s_{9..}$
A1	g_{p_i}	320	688	647	760	–10	–77	15
A2	$2g_{s_i}$	. . .	528	940	1380	0	0	0
A2	$2g_{d_i}$	–4	34	10	158			
A3	$b_1{}^* p_{pi}$	172	343	515	686	–183	–115	94
NA	$b_1{}^* p_{pi}$	198	397	595	793	0	–6	0
Additive values		200	400	600	800	2	–20	–8

[1] $g_{p_1} = g_{s_1} = g_{d_1} = 0$.

[2]Heterosis = 5%, recombination loss = –5%.

TABLE 8. Solutions[1] from different models for genetic groups of progeny(g_p), sire(g_s), or dam(g_d) and average predicted sire effects ($s_{j..}$) within sire groups for structures III.[2]

Model	Effect	Group solutions i = 3	i = 5	i = 7	i = 9	Average sire effects $s_{1..}$	$s_{5..}$	$s_{9..}$
A1	g_{p_i}	355	583	583	668	-64	-165	36
A2	$2g_{s_i}$	. . .	561	877	1220	0	0	0
A2	$2g_{d_i}$	-12	16	115	256			
A3	$b_1 * p_{pi}$	75	151	226	301	-302	157	48
NA	$b_1 * p_{pi}$	193	387	580	773	-7	-11	3
Additive values		200	400	600	800	-10	-22	1

[1] $g_{p_1} = g_{s_1} = g_{d_1} = 0$.

[2]Heterosis = 5%, recombination loss = -5%.

but residual bias caused overprediction of effects from 100% HF sires. Sire effects from other groups were underestimated. Hence, variance between sires was biased upward. The deviation of group solution from additive genetic value was largest for the 50% HF group (Table 6). The group effect for 100% HF cows was biased downward due to overestimation of the effects of their sires.

In model A2, sires are nested within sire groups. Predictions of sire effects within groups were unbiased because sires were equally distributed over dam groups. Group solutions contained additive effects as well as average nonadditive effects (Table 6). Nonadditive effects not accounted for by groups contributed to an increase only in the residual variance. Solutions for the group of 100% HF sires considerably overestimated the additive genetic effect. The group effect of 0% HF dams was overestimated, i.e., group solution for 50% HF dams was lower than expected additive value (Table 6).

Model A3 gave biased estimates of group effects as well as biased prediction of sire effects. The estimate for linear regression of performance on p_p (b_1) was 697 for structure I, which is lower than true additive genetic value (Table 6).

The nonadditive model yielded estimates for group effects and sire effects that were close to true additive genetic effects. Solutions for group effects (Table 6) were derived from the estimate for b_1.

Results for structures II (Table 7) and III (Table 8) were comparable with those from structure I. Estimators from the nonadditive model were empirically unbiased. Regression on portion of HF genes was 793 for structure II and 773 for structure III. The estimate for HET was averaged over repetitions, 321 for structure II and 318 for structure III with empirical standard errors 5.8 and 5.4. Estimates for the REC effect were -332 for structure II and -302 for structure III with empirical standard errors 15.0

TABLE 9. Effect of progeny group size (ND) on bias in estimation of additive genetic parameters with model A1 for structure I.[1]

ND	Parameters σ_a^2	h^2	Group effects g_{p_3}	g_{p_5}	g_{p_7}	Sire effects $s_{1..}$	$s_{5..}$	$s_{9..}$
25	195,708	.369	310	525	565	-25	-70	92
50	208,168	.390	301	502	514	-45	-85	127
100	223,044	.415	297	464	453	-70	-95	160

[1]Heterosis = 5%, recombination loss = -5%.

and 8.7. Simulated values were 320 for HET and −320 for REC effect.

The effect of size of the progeny groups on bias is given for model A1 (Table 9). A greater fraction of nonadditive effects was assigned to the random sire effects with increasing progeny group size. Bias in estimation of additive genetic variances increased with more progeny per sire. Bias in estimation of group effects for progeny with 25% HF and 50% HF, which is due to nonadditive effects, was smaller and bias for the 75% HF progeny group, which is due to overestimation of 100% HF sires, was larger with increasing number of progeny.

DISCUSSION

Nonadditive models have been proposed in the literature, including those with random nonadditive genetic effects due to dominance variation within breeds [e.g., (5)]. Those effects were not considered in this study because they are likely to be less important than fixed nonadditive effects due to interactions between breeds. Another simplification of the simulated model was the assumption of equal genetic variances across populations and across crossbred groups before genetic equilibrium was reached. Hence, additive genetic variation was expected to be homogeneous, i.e., differences in gene frequency were small. This is justifiable for two closely related breeds with no inbreeding and a trait that is determined by an infinite number of loci. However, it may be worthwhile to investigate models that account for heterogeneity of additive genetic variance across crossbred groups.

More family information is often used in sire evaluation by incorporating numerator additive genetic relationships between sires. Pollak and Quaas (17) have pointed out the association of relationships with genetic groups, i.e., groups can account for selection differentials between generations not accounted for by relationships. Those groups, however, are defined on the basis of time to account for short-term selection. Differences between populations or breeds are based on long-term selection and group effects reflect effects of gene substitution and gene interaction. Hence, use of genetic relationships between generations is not expected to change the interpretation of group effects. However, bias in random effects, due to not accounting for nonadditive effects, may decrease when information from relatives is used across genetic groups of sires.

An alternative strategy for analysis of data from crossbred populations could be the use of an animal model so that additive genetic variance would be estimated from the random additive values of animals. Correction for genetic means could be by groups that are defined on a basis of fraction of HF genes of animals making the records. However, an additive animal model would not account for different nonadditive effects within groups, so that additive genetic variance would likely be overestimated.

In this simulation study, distribution of sire's progeny over dam groups was balanced and distribution of sire and dam groups over environmental effects was random, i.e., all herds have dams with on average equal breed composition. Meyer (13) showed that environmental effects might account for differences between genetic groups when groups are partially confounded with environmental effects.

Heterosis levels in crossbred populations, in particular in crosses between different strains of Friesian cattle, might be small in temperate climates (2, 14). However, in addition to levels of HET and REC, the genetic structure of the data influences bias in estimating genetic variance and breeding values. Bias depends on the distribution of data over sire group by dam group combinations where sires are imported from other populations, a progeny group model gives more bias in estimation of additive genetic variance as dams have higher fraction of HF genes.

European countries recently have been interested in making comparisons of genetic merit between and within dairy cattle populations. Philipsson (16) mentioned some problems of comparing breeding stock internationally, such as possible bias due to selective mating and to special treatment of progeny. Bias in prediction due to nonadditive effects was not considered. However, accounting for nonadditive effects affects international comparisons of breeding stock. Estimating additive genetic values with a sire group model gives maximum bias through comparison of bull groups of the pure breeds, and mating them with dams from one of the breeds. Breed difference would be estimated as twice the group difference; hence, overestimation would be equal to twice the HET. The

impact of nonadditive effects on sire ranking, based on total genetic effect, is dependent upon the heterogeneity of the cow population. However, a correct sire evaluation procedure should specify the components of the total genetic merit.

CONCLUSION

Low levels for HET and REC affected estimators of additive genetic variance in crossbred populations. Predictions of breeding values and estimation of breed difference were considerably biased with additive models. It is necessary to estimate HET and REC loss in actual populations to assess the problem of bias in genetic evaluations.

REFERENCES

1 Dickerson, G. E. 1973. Inbreeding and heterosis in animals. Page 54 *in* Proc. Anim. Breeding Genet. Symp. in Honor of Dr. J. L. Lush. Am. Soc. Anim. Sci. and Am. Dairy Sci. Assoc. Champaign. IL.

2 Ericson, K., and B. Danell. 1986. Crossbreeding effects between Swedish dairy breeds for production and reproduction traits. Page 17 *in* Proc. 37th Annu. Mtg. Eur. Assoc. Anim. Prod.

3 Fimland, E. A. 1983. Methods of estimating the effect of heterosis. Z. Tierz. Zuchtungsbiol. 100:3.

4 Gjøl-Christensen, L., and J. Pedersen. 1988. Krydsning af malkekvaegracer. 650 Beretning fra Statens Husdyrbrukforsøg, København.

5 Henderson, C. R. 1985. Best linear unbiased prediction of nonadditive genetic merits in noninbred populations. J. Anim. Sci. 60:111.

6 Hill, W. G. 1982. Dominance and epistasis as components of heterosis. Z. Tierz. Zuchtungsbiol. 99:161.

7 Hill, W. G., M. R. Edwards, M. K. Ahmed, and R. Thompson. 1983. Heritability of milk yield and composition at different levels and variability of production. Anim. Prod. 36:59.

8 Kinghorn, B. 1980. The expression of recombination loss in quantitative traits. Z. Tierz. Zuchtungsbiol. 97:138.

9 Kinghorn, B. 1982. Genetic effects in crossbreeding. I: Models of merit. Z. Tierz. Zuchtungsbiol. 99:59.

10 Maijala, K., and M. Hanna, 1974. Reliable phenotypic and genetic parameters in dairy cattle. Proc. 1st World Congr. Genet. Appl. Livest. Prod. Madrid. 1:541.

11 Meyer, K. 1986. Restricted maximum likelihood estimation of genetic parameters-in practice. Proc. 3rd World Congr. Genet. Appl. Livest. Prod. 12:454.

12 Meyer, K. 1987. Estimates of variances due to sire × herd interactions and environmental covariances between paternal half sibs for first lactation dairy production. Livest. Prod. Sci. 17:95.

13 Meyer, K. 1987. Mixed model analysis when genetic and environmental groups are closely linked. Z. Tierz. Zuchtungsbiol 104:321.

14 McAllister, A. J. 1986. The role of crossbreeding in breeding programs for intensive milk production in temperate climates. Proc. 3rd World Congr. Genet. Appl. to Livest. Prod., Lincoln, Vol IX:47.

15 McGloughin, P. 1980. The relationship between heterozygosity and heterosis in reproductive traits of mice. Anim. Prod. 30:69.

16 Philipsson, J., 1987. Standards and procedures for international genetic evaluations of dairy cattle. J. Dairy Sci. 70:418.

17 Pollak, E. J., and R. L. Quaas. 1983. Definition of group effects in sire evaluation models. J. Dairy Sci. 66:1503.

18 Schulte-Coerne, H., and D. Boie. 1986. Heterosis in crosses between European and American dairy breeds. Page 21 *in* Proc. 37th Annu. Mtg. Eur. Assoc. Anim. Prod.

19 Sheridan, A. K. 1981. Crossbreeding and heterosis. Anim. Breed. Abstr. 49:131.

20 Van der Werf, J.H.J. A. E. van Veldhuizen and S. Korver. 1987. Relationship between young bull performance and dairy performance of progeny. S. Korver, G. Averdunk, and B. B. Andersen, ed. Performance testing of AI bulls for efficiency and beef production in dairy and dual purpose breeds. Eur. Assoc. Anim. Prod. Publ. 34, Pudoc, Wageningen, Neth.

21 Van der Werf, J.H.J., and W. de Boer. 1989. Estimation of genetic parameters in a crossbred population of Dutch Black and White dairy cattle. J. Dairy Sci. 72:2615.

22 Wilmink, H., and F. de Graaf. 1986. Genetic parameters for 305-days yield in first lactation and adjustment of Net Guilder Index (Dutch with English summary). Rep. 86-676, Dutch Royal Cattle Syndicate, Arnhem.