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Use of inbred sires to exploit epistatic variance (short communication)

Dedicated to Prof. Dr. Peter Glodek on the occasion of his 70th birthday

Abstract

The genetic variance of inbred sires is increased and variance due to additive x additive effects proportionately more than that due to additive ones. In case of inbreeding by mating sire to daughters the time interval elite sire – son needs not to be elongated so that the genetic progress could be increased by this procedure. This pertains even more to the procedure

where inbred sires are produced from sib mating. Use of such sires should increase the genetic gain via the sire son path by about 10 %

Key Words: genetic variance, epistatic variance, inbred sires, genetic gain

Zusammenfassung

Titel der Arbeit: Ausnutzung epistatischer Varianz durch ingezüchtete Vatertiere (Kurzmitteilung)

Die genetische Varianz ist bei ingezüchteten Vatertieren vergrößert und die durch additiv x additive Wirkungen verursachte Varianz relativ mehr als die durch additive Wirkungen. Auch das Zeitintervall Elitevatertier-Sohn braucht nicht verlängert zu sein, wenn ersteres rasch an die besten der Prüftöchter gepaart wird. Dies gilt natürlich noch mehr, wenn Inzuchtvatertiere aus Geschwisterpaarung hervorgehen. Der genetische Fortschritt entlang des Vater-Sohn Pfades soll durch Verwendung solcher Vatertiere um etwa 10 % zu steigern sein

Schlüsselwörter: Genetische Varianz, epistatische Varianz, Inzuchtvatertiere, genetischer Fortschritt

Epistasis receives considerable attention in analysis of crosses but comparatively little in that of within population variation and hardly any in exploitation of the latter by selection. This is in no small degree attributable to difficulties in estimating epistatic effects. Nevertheless, several papers deal with this (SHERIDAN and RANDALL, 1977; FAIRFUL and GOWE, 1987; TEMPELMANN and BURNSIDE, 1990; HOESCHELE, 1991; FUERST and SÖLKNER, 1994; MISZTAL et al., 1995; SZWACZKOWSKI, 1999) and report considerable epistatic influence on performance and fertility. QTLs have shown to be involved in epistatic influence, eg. on lung tumor susceptibility in mice (FIJNEMAN et al., 1986) and epistatic interactions between QTLs cause nearly one third of the total variance of maternal performance in F2 of mouse lines (PERIPATO et al., 2002). GRAML (1994) found interaction between casein and the lactoglobuline loci to cause 7, 24,and 45 % of the genetic variance between the haplotypes, for serum protein, casein and fat content, respectively.

One problem in recognizing epistasis within populations is that the average effect of an allele encompasses also its average influence on effects of other loci, i.e. some epistasis (CHEVERUD and ROUTMAN, 1995). However, inbreeding makes epistatic effects more evident as of course shown by SEWALL WRIGHT and extended by COCKERHAM (1954). HILL (1982) and KINGHORN (1982) have shown its contribution to heterosis.

We found, among halfsib groups by inbred sires, the variance to be increased to an extent which made epistatic influence very probable, both in cattle for dairy performance (PIRCHNER et al., 1989), and for layers (PIRCHNER and GRAML, 2003). The variance between non-inbred progeny groups of such inbred sires relative to that between groups of non-inbred sires is increased, that within such groups decreased: In case of laying performance this is particularly evident for more complex traits like feed efficiency but lacking for more "simple" traits as egg size e.g. In Table 1 it is shown that the variance between additive effects of progeny groups by inbred sires is increased but that due to additive x additive interaction relatively much more. Where epistatic variance has been estimated, its exploitation is suggested by considering the "specific combining ability" of sires, i.e. by choosing sire- maternal grandsire combinations which have proofed well. However, no reference is made to consider epistatic effects in selection other than those which are included in average additive effects. The over proportional increase of epistatic variance among inbreds suggests to use inbred sires in selection to exploit epistatic effects in addition to average gene effects. The breeding success by selection among progeny tested bulls will be

$$D_g = i r s_g$$

i = selection intensity in standard deviations, r the correlation between breeding value and progeny average and s_g the genetic standard deviation

$$r^{2} = \frac{n \left[h_{A}^{2} \left(1+F \right) / 4 + h_{AA}^{2} \left(1+F \right)^{2} / 16 \right]}{1 + (n-1) \left[h_{A}^{2} \left(1+F \right) / 4 + h_{AA}^{2} \left(1+F \right)^{2} / 16 \right]}$$
$$s_{g}^{2} = h_{A}^{2} / 4 \left(1+F \right) + h_{AA}^{2} / 16 \left(1+F \right)^{2}$$

Table 1
Variance components between sires (Varianzkomponenten zwischen Vatertieren)

	$\mathbf{F} = 0$	F = 1/8	F = 1/4
E(V _A)	V _A /4	$(1+F)V_A/4 = 9/8V_A/4$	5/4 V _A /4
E(V _{AA})	V _{AA} /16	$(1+F)^2 V_{AA}/16 = 81/64 V_{AA}/16$	25/16 V _{AA} /16

F=inbreeding coefficient

In Table 2 correlations between progeny averages and breeding values, genetically exploited variance and increase in breeding values are compared for bulls with no inbreeding, with 1/8 and with $\frac{1}{4}$ inbreeding. Two heritabilities were assumed for additive effects, three for epistatic effects AxA, and two progeny group sizes, 25 and 50. The rather low values for heritabilities were chosen since it appears likely that highly heritable traits have "little room" for epistatic effects (eg. LEAMY et al., 2002) The improvement in genetic gain from using inbred sires is not unexpectedly greater at lower additive heritabilitis. Yet there is little difference between the two progeny group sizes. However, the more highly inbred sires (F=1/4) lead to more genetic gain than those with 1/8 F. This is also expected with more complex gene interactions.

For comparing the gain from using such sires the generation interval ought to be considered. Both parent-offspring and fullsib mating could lead to offspring with F=1/4 but uniparous animals exclude any selection among sisters while parent offspring matings permit pairing of sires with highly selected daughters. In case of cattle, the bull could be mated to his best daughters originating in his testing period and no or little prolongation of the time span sire-son and thus of the generation interval needs to occur. In some countries test mating is also practiced to identify gene carriers and this could be utilized for the practice here suggested. Inbred sons out of fullsib matings would not entail any prolongation of the generation interval. Halfsib matings to produce sons with F=1/8 would of course permit strong selection among mating partners also in uniparous animals.

Already in 1923 SEWALL WRIGHT argued that the relationship between phenotype and genotype is inadequately described by our simple polygenic model. Rather this relationship depends in a fundamental way on epistasis. Its utilisation deserves more attention and the approach suggested here may provide one way to achieve this.

$h_A^2 h_{AA}^2$	n	F	r	Sg	Dg	$d(F-F_0)$	d%
.1 .1 25 50	25	0	.668	.177	.118		
		1/8	.694	.190	.132	.014	12
		1/4	.719	.216	.155	.023	15
	50	0	.786	.177	.139		
		1/8	.807	.190	.151	.012	9
		1/4	.825	.216	.178	.027	15
.2 .2 25	25	0	.791	.25	.198		
		1/8	.812	.268	.218	.02	10
		1/4	.831	.286	.238	.09	9
	50	0	.877	.25	.219		
		1/8	.892	.268	.239	.02	9
		1/4	.904	.286	.259	.02	8
.1 .2	25	0	.702	.194	.136		
		1/8	.731	.21	.153	.02	12
		1/4	.756	.225	.170	.02	11
4	50	0	.813	.194	.157		
		1/8	.835	.21	.175	.02	11
		1/4	.853	.225	.192	.02	10
.2 .4	25	0	.818	.274	.224		
		1/8	.841	.296	.249	.02	8
		1/4	.859	.319	.274	.02	10
	50	0	.896	.274	.246		
		1/8	.91	.296	.269	.02	9
		1/4	.911	.319	.291	.02	7

Table 2

Correlations with breeding value (r), its standard deviation (s_g) and predicted gain D_g (Korrelation mit Zuchtwerten, deren Standardabweichungen und geschätzter Zuchtfortschritt)

 $D_g = i x r x sg$

 $d(F-F_0) = difference in D_g$ between use of inbred and noninbred sires

d% Difference in Dg relative to genetic gain with use of noninbred sires

References

CHEVERUD, J.M.; ROUTMAN, E. J.:

Epistasis and its contribution to genetic variance components. Genetics **139** (1995), 144-1461 COCKERHAM, C.C.:

An extension of the concept of partitioning hereditary variance for analysis of covariances among relatives when epistasis is present. Genetics **39** (1954), 859

FAIRFUL, R.W.; GOWE, R. S.:

Dominance and epistasis in heterosis of WL strain crosses. Can. J. Anim. Sci. 67 (1987), 663-680

FIJNEMAN, R.I.A.; DE VRIES, S. S.; JANSEN, R.C.; DEMONT, P.:

Complex interactions of new QTL Sluc 1, Sluc 2, Sluc 3, and Sluc 4, that influence the susceptibility to lung cancer in the mouse. Nature Genet. **14** (1986), 464-470

FUERST, CH.; SÖLKNER, J.:

Additive and non-additive genetic variance for milk yield, fertility and lifetime performance traits of dairy cattle. J. Dairy Sci. **77** (1994), 1114-1125

GRAML, R.:

Expression der Milchproteingene. Habil.thesis, TU München, 1994

HILL, W.G.:

Dominance and epistasis as components of heterosis. J. Anim. Breed. Gen. **99** (1982), 161-168 HOESCHELE, I.:

Additive and nonadditive genetic variance in female fertility of Holsteins. J. Dairy Sci. **74** (1991), 1743-1752

LEAMY, L.: ROUTMAN, E.; CHEVERUD, J.:

An epistatic genetic basis for fluctuating asymmetry of mandible size in mice. Evolution **56** (2002), 642-653

MISZTAL, I. et al.:

Dominance and epistatic effects in genetic evaluation of farm animals. Anim.Sci.papers reports. 13 (1995), 251-265

PERIPATO, A.C., et al.:

Quantitative trait loci for maternal performance for offspring survival in mice. Genetics **162** (2002), 1341-1353

PIRCHNER, F.; REINHARDT, F; GOTTSCHALK, A.:

Die Verwendung der Inzucht zur Steigerung des Selektionsfortschrittes. Züchtungskunde 61 (1989), 6-13

PIRCHNER, F.; GRAML, R.:

Increase of genetic variance by inbred sires. Arch. Geflügelk. 67 (2003), 179-181

SHERIDAN, A.K., RANDALL, M.C.:

Heterosis for egg production in WL-Australorp crosses. Brit. Pty. Sci. 18 (1977), 69-78

SZWACZKOWSKI, T.:

Additive and additive by additive genetic variability of productive traits in laying hens. J. Anim. and Feed Sci. 8 (1999), 191-201

TEMPELMANN, R.J.; BURNSIDE, E.B.:

Additive and nonadditive genetic variation for production traits in Canadian Holsteins, J. Dairy Sci. **73** (1990), 2206-2213

WRIGHT, S.:

The role of mutation, inbreeding, crossbreeding, and selection in evolution. Proc. 6^{th} Int. Con. Genet. 1 (1923), 356-366

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