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Analysis of Inheritance of the Number of Kernel Rows in Maize

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With 6 figures

The primary components of the total grain yield per plant in corn are the number of ears per plant, kernel weight, number of kernel rows and number of kernels per row; consequently the study of the inheritance of kernel-row number has besides the theoretical importance also a practical one.

ANDERSON (1944) emphasized: "Ear row number is a good example for a quantitative character which is easy to record exactly but difficult to analyse". The complicated inheritance may be connected with the intricated evolution of the ear by mutation (WEATHERWAX, 1935), fasciation (WHITE, 1948), introgression (MANGELSDORF, 1961) and also artificial selection.

EAST described as early as 1910 the segregation of the kernel-row number and he was the first who attempted to explain this phenomenon on the line of Mendelian inheritance, but we have not a clear picture about it as yet. In spite of the slight influence of the environment on the kernel-row number (EMER-

Table 1. *The number of individuals counted (n) and the mean kernel row number ($\bar{X}$) in generations and crosses.*

Cross	F_1		F_2		B_1		B_2		F_3		Total
	n	$\bar{X}$	n	$\bar{X}$	n	$\bar{X}$	n	$\bar{X}$	family	individuals	n
P40 × P18	147	28,603	313	29,385	206	35,210	211	22,333	17	584	1461
P40 × P12	149	22,548	321	22,045	202	31,318	208	16,305	8	269	1149
P40 × P8	154	15,220	304	17,823	211	27,773	220	10,878	10	336	1225
P40 × P4	156	12,005	311	12,595	205	24,788	203	7,983	10	331	1206
P18 × P12	152	15,835	309	14,890	208	16,760	203	14,203	10	340	1212
P18 × P8	154	12,493	332	12,508	211	14,663	209	9,780	10	354	1260
P18 × P4	149	10,880	303	10,693	192	14,098	205	7,600	10	316	1165
P12 × P8	151	10,713	307	10,318	199	11,675	197	8,573	10	335	1189
P12 × P4	147	8,610	307	8,570	195	10,958	211	7,110	10	302	1162
P8 × P4	149	7,392	295	7,098	194	7,795	209	5,495	8	246	1093
Total	1508	144,299	3102	145,925	2023	195,038	2076	110,260	103	3413	12122

SON and SMITH, 1950; ALEXANDER, 1952; GWYNN, 1959; SCHREIBER and STANBERRY, 1962; ANDERSON and CHOW, 1963), the attempts to detect linkages between the number of kernel-rows and some qualitative characters failed (KEMPTON, 1924; LINDSTROM, 1929, 1931; TAVČAR, 1935; BURDICK, 1951), and while TAVČAR obtained a monogenic segregation in crosses between four and eight rowed lines, BURDICK got some indications for two complementary genes. The kernel-row number of the lowest inserted ears of the F_1 was significantly lower under glasshouse conditions than those of the highest one, but in the parents the differences were smaller and he assumed: "... the within plant environments may give phenotypic expression to one genome at one time or to the other genome at another".

The averages of the F_1 surpassed the midparent values in some instances relevant (KEMPTON, 1924; LENG, 1954), but in others they lay slight above or beneath it and it was remarkable that pure lines with the same number of kernel rows ($\bar{X} = 12$) almost possessed different genotypes for kernel-row number (EMERSON and SMITH, 1950).

In the polygenic inheritance the nature of the gene effects is important. Partial dominance was detected by GARDNER, HARVEY, COMSTOCK and ROBINSON (1953), while HYER (1960) found complete dominance, but with a very big standard error and significant epistatic effects. The epistatic effects were significant in some of the crosses (BAUMAN, 1959), yet the additive and the dominance effects seemed to have more importance (GAMBLE, 1957, 1962 b) and the relevant role of the additive effects is also supported by the rather big coefficients of heritability (WARNER, 1952; DANIEL und VÁRÓCZY, 1959 b).

Material and methods

General part

The experimental material used in this study consisted of five self-developed lines, P40 ($n = 133$; $\bar{X} = 38{,}96$; $V = 22{,}73$), P18 ($n = 155$; $\bar{X} = 17{,}32$; $V = 1{,}47$), P12 ($n = 111$; $\bar{X} = 11{,}97$; $V = 1{,}16$), P8 ($n = 153$; $\bar{X} = 8{,}06$; $V = 0{,}12$) and P4 ($n = 148$; $\bar{X} = 4{,}07$; $V = 0{,}16$), together with their F_1, F_2 (Fig. 1), B_1 ($F_1 \times P_1$), B_2 ($F_1 \times P_2$) progenies and F_3 families (Table 1). The P4 — a selfed line of an eight-rowed variety — and one of the fraternal lines of the P40 — a type with fasciated ears — was already used in our earlier experiments (DANIEL, 1956).

Field experiments were made at Martonvásár in 1958 and 1959, respectively, in an 4×4 "Latin rectangle" after MUDRA (1952) although DARBY and GILBERT (1958) stated that it neglects certain restrictions in the randomization. The F_3 families were produced in 1958, thus they could only be used in the trials in 1959, therefore these data were taken in consideration only in the analyses based on the variances within generations.

Observations were taken on single plant basis; the number of kernel rows of all the ears was recorded, but in this experiment only the data of the uppermost ears have been used. The variances within combinations have been computed and tested for years, but the difference was not significant ($t_{34} = 1{,}06$). The variances within the non segregating generations were analysed separately. The combinations showed significant differences at the 1% level, but not the years and the parents versus F_1. The heterogeneity of the combined data of these two years was also highly significant by the homogeneity test of BARTLETT ($Chi^2_{14} = 1419$). The very small differences between the two years and between the parents and F_1 suggested the probability that the variances and the averages were connected; their correlation coefficient proved to be significant ($r = +0{,}88$; $t_{13} = 6{,}68$) indeed. To minimize the genotype — environment interactions, after uniting the replications, the data were transformed according to WRIGHT (1952) to logarithms on basis 10 (lg), but even so the variance analysis ($P = 0{,}05-0{,}01$) and the BARTLETT test ($Chi^2_{14} = 213$) proved the heterogeneity of the variances, the correlation, however, was not significant ($r + - 0{,}33$; $t_{13} = 1{,}26$). After that the computations were undertaken on both scales and the error variances of averages of the different generations were separately estimated with the partition of years — reciprocs — remainder, and the same for the parents partitioning in years and remainder.

Estimation based on the variances within the generations

The test of the scale and the additive ($\Sigma d^2 = D$), the dominance ($\Sigma h^2 = H$) and the error variances (E_1 = individual, E_2 = average) were computed after MATHER (1949) for each combination with the method of the least squares equations applying the following partitioning, where E_2 was inserted in the equation system in the form of $1/n\ E_1$:

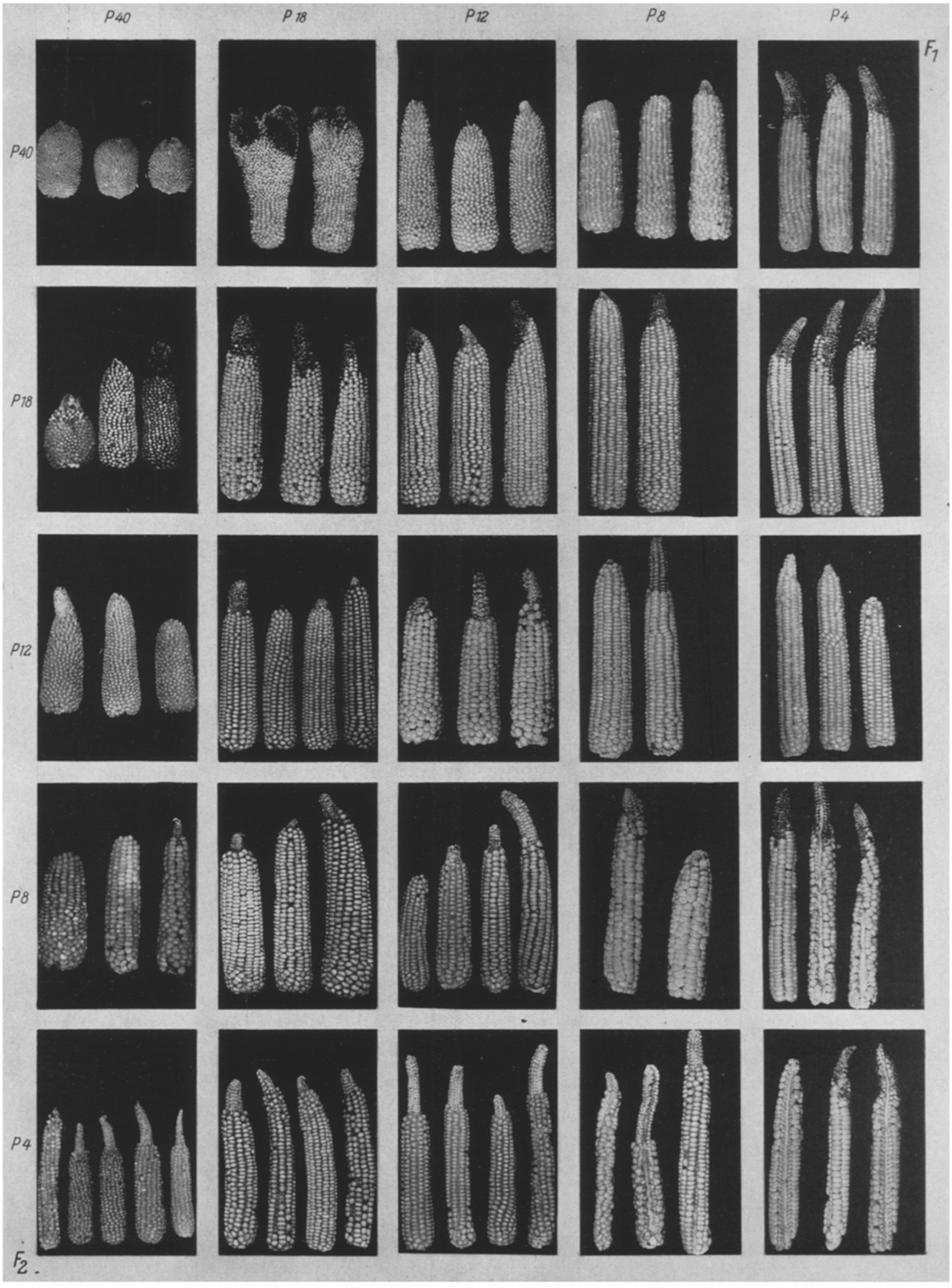


Fig. 1. Photographs of some characteristic ears of the parents, of the first and the second generation of the 5 × 5 diallel crosses.

		D	H	E_1	E_2
V_{P1}	1958			1	
	1959			1	
V_{P2}	1958			1	
	1959			1	
V_{F1}	1958			1	
	1959			1	
V_{F2}	1958	0,5	0,25	1	
	1959	0,5	0,25	1	
$V_{B1}+V_{B2}$	1958	0,5	0,5	2	
	1959	0,5	0,5	2	
V_{F3}	1959	0,25	0,125	1	
$V_{\bar{F}3}$	1959	0,5	0,0625		1
$W_{F2/\bar{F}3}$	1959	0,5	0,125		

The number of effective factors using the additive variance [$k_D = (P_1 - P_2)^2/4D$], and the degree of dominance ($\bar{a} = \sqrt{H/D}$) was estimated on normal and lg scale, where it was possible, and the most probable factor number (K) was also given on the basis of these data (Table 2).

Diallel crosses

Based first of all on papers of HAYMAN, there were accomplished with L_1 (P and F_1), L_2 (P and F_2) and L_B [P and $(B_1 + B_2)/2$] diallels the graphic solution (HAYMAN, 1954 b, 1958 a; JINKS, 1954, 1956) using the data averaged over years (Figs. 2, 3), and with L_1 the statistical analysis (HAYMAN, 1954 a) for the complete diallels, and only on lg scale the statistical analysis for the P18 — P12 — P8 diallel (Table 3), and the graphic solution with the P18 — P12 — P8 — P4, and P18 — P12 — P8 diallels too (Fig. 4). It was also tested the heterogeneity of the differences of the covariances and the variances of the arrays ($W_r - V_r$) and computed the correlation coefficients of the parents with the sum of the covariances and variances of their arrays [$r_{yr-(Wr+Vr)}$] with L_1, L_2 and L_B for the complete diallels, and only with L_1

Table 2. *Components of genetic variation (D = additive variance; H = dominance variance; E = error variance), the number of factors estimated with the additive variances (k_D), the degree of dominance ($\bar{a}$) and the supposed number of factors (K).*

Cross	Normal scale					Lg scale × 100					K
	D	H	E	k_D	$\bar{a}$	D	H	E	k_D	$\bar{a}$	
P40 × P18	28,365	20,177	10,387	4,21	0,8	0,956	0,122	0,177	3,22	0,4	3
P40 × P12	5,448	33,514	8,979	33,65	2,5	0,599	0,606	0,188	10,77	1,0	11
P40 × P8	12,922	29,152	8,096	18,62	1,5	2,729	−2,113	−0,350			18
P40 × P4	5,274	26,782	6,858	58,13	2,3	2,782	−2,084	−0,642			
P18 × P12	1,016	5,234	1,546	6,74	2,2	0,114	0,360	0,131	5,39	1,8	5
P18 × P8	2,866	4,150	1,065	7,36	1,6	0,187	0,619	0,196	14,86	1,8	15
P18 × P4	1,741	4,483	1,232	24,92	1,6	0,379	0,782	0,207	26,05	1,5	26
P12 × P8	1,890	2,177	1,182	2,06	1,1	0,084	1,050	0,282	9,29	3,5	9
P12 × P4	−1,228	7,384	0,868			0,623	−0,565	0,257			
P8 × P4	0,445	5,104	0,558	8,94	3,4	−0,193	3,000	0,332			9?

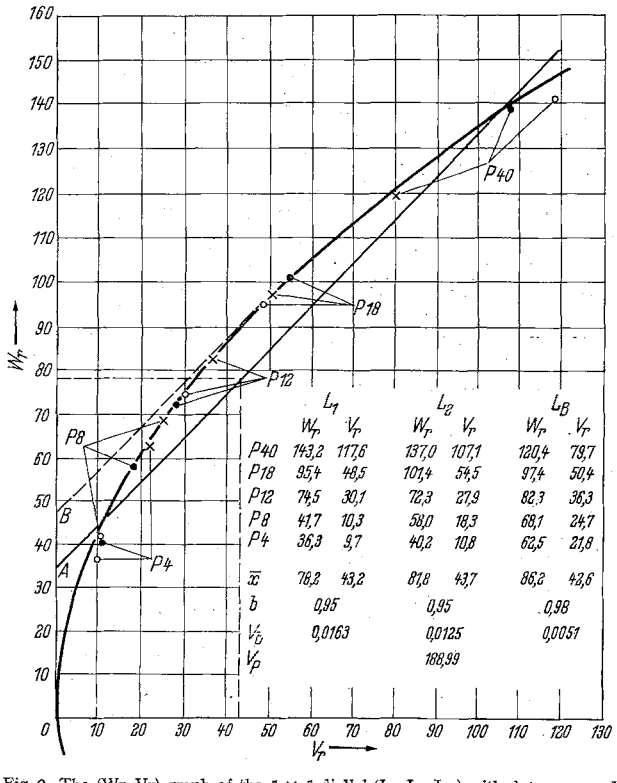


Fig. 2. The (Wr, Vr) graph of the 5 × 5 diallel (L_1, L_2, L_B) with data averaged over years; normal scale (○ = L_1; ● = L_2; × = L_B).

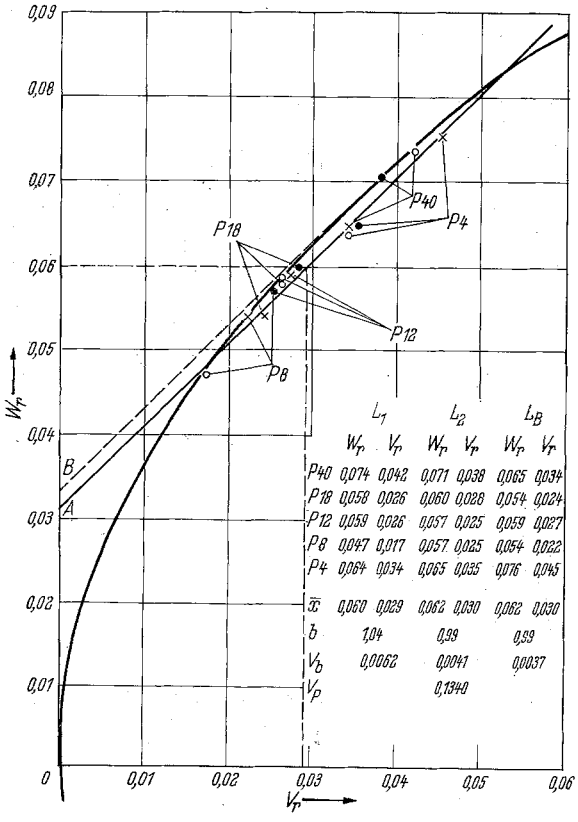


Fig. 3. The (Wr, Vr) graph for the 5 × 5 diallel (L_1, L_2, L_B) with data averaged over years; lg scale (○ = L_1; ● = L_2; × = L_B).

for the P40 — P18 — P12 — P8, P40 — P18 — P8 — P4, P18 — P12 — P8 — P4, and P18 — P12 — P8 diallels (Table 4). Moreover there were plotted the W_r and V_r data from L_1 against those from L_2 and L_B, further those from L_2 against those from L_B too (Fig. 5, 6).

With the data of L_1, L_2 and L_B, the parameters D $[\Sigma d_i^2 (1 - w_i^2)]$, F $[2 \Sigma d_i h_i w_i (1 - w_i^2)]$, H_1 $[\Sigma h_i^2 (1 - w_i^2)]$, H_2 $[\Sigma h_i^2 (1 - w_i^2)^2]$ and h^2 $[\Sigma h_i (1 - w_i^2)]$ ($w_i = u_i - v_i$ = the difference between the frequencies of the parents with the positive and the negative allele, respectively, in the i locus), were estimated by the years on the basis of the least squares equations for the whole and for the P18 — P12 — P8 diallels (Table 5). Considering that the error variances were extraordinarily small with negligible differences between the generations, common values were established for E_0, E_1, E_2 and E_B and the following system of equations devised:

$$32 \{V_{0L0} + 1/2 (W_{0L01} + W_{0L02} + W_{0L0B}) + 1/4 (V_{1L1} + V_{0L1} + W_{1L12} + W_{0L12} + W_{1L1B} + W_{0L1B} + V_{1L2} + V_{0L2} + V_{1LB} + V_{0LB}) - 1/4 n^2 [(7 n + 11) n - 1] E\} = 76 D - 22 F + 10 H_1 - 5 H_2.$$

$$64 \{1/4 (W_{0L01} + V_{1L1} + V_{0L1}) + 3/16 (W_{1L12} + W_{0L12} + W_{1L1B} + W_{0L1B}) + 1/8 (W_{0L02} + V_{1L2} + V_{0L2} + W_{0L0B} + V_{1LB} + V_{0LB}) - 1/8 n^2 [(4 n + 11) n - 1] E\} = 44 D - 27 F + 16 H_1 - 8 H_2.$$

$$128 \{1/4 (V_{1L0} + V_{0L1}) + 1/8 (W_{1L12} + W_{0L12} + W_{1L1B} + W_{0L1B}) + 1/16 (V_{1L2} + V_{0L2} + V_{1LB} + V_{0LB}) - 1/8 n^2 [(3 n + 5) n - 1] E\} = 40 D - 32 F + 26 H_1 - 13 H_2.$$

$$256 \{1/4 V_{0L1} + 1/8 (W_{0L12} + W_{0L1B}) + 1/16 (V_{0L2} + V_{0LB}) - 1/8 n^2 (3 n - 1) E\} = 40 D - 32 F + 26 H_1 - 26 H_2.$$

$$16 \{(m_{L1} - m_{L0})^2 + 1/4 [(m_{L2} - m_{L0})^2 + (m_{LB} - m_{L0})^2 + (m_{L1} - m_{L2})^2 + (m_{L1} - m_{LB})^2] - 2/n^2 (n - 1) E\} = 5 h^2.$$

Solving the equation system, the corresponding data of the two years were averaged and the standard deviations computed on the basis of their differences; e. g. $D = 1/2 (D_{1958} + D_{1959}) \pm \sqrt{1/2 (D_{1958} - D_{1959})^2}$ (Table 5).

Estimation based on the averages of the generations

The scale test of MATHER (1949) was improved by CAVALLI (1952), ANDERSON and KEMPTHORNE (1954) and HAYMAN (1958 b, 1960). Making use of the test, GAMBLE (1962 a, b) studied the inheritance of some quantitative characters in corn with the populations corresponding to our experiments and the analyses were accomplished on the basis of his formulas (Table 6).

Results

The scaling test after MATHER showed in all combinations both in the normal scale and in the lg scale greater or smaller deviations from additivity and consequently the estimation on the basis of individual variances was not full satisfactory (Table 2). In spite of the fact, that D, H and E are square values, they took up several times negative sign, as well as the dominance variances showed remarkable magnitude and consequently this manifested itself in the relative great value of the average degree of dominance ($\bar{a}$).

The number of active factors estimated in lg scale on the basis of additive effects was fairly consistent: P18 × P12 + P12 × P8 = P18 × P8 (5,39 + 9,29 ≈ 14,86), while P40 × P18 and P40 × P12 — P18 × P12 did not much differ from each other (3,22 and 10,77 — 5,39 = 5,38). The P18 × P4 and the P40 × P18 crosses provided sufficiently identical data in the two scales used.

According to the means and variances of the very few F_3 families by the single combinations, but in total 103, the F_2 parent of none of the F_3 family examined had been of identical genotype for kernel-row number with each of the two correspondent parents used in the crosses. The average of one of the F_3 families of the P12 × P8 cross was near to the P12 parent, and that of one of the F_3 families of the P40 × P18 cross to the P18 parent, but the variance within the F_3 families surpassed in both cases the corresponding values of the respective parental lines (2,13 and 1,16; 4,90 and 1,47). The extraordinary small differences shown in the means of the F_3 families of the P12 × P4, and in the P18 × P4 crosses were surprisingly remarkable (the range was in the former 8,96 and 7,66, whereas in the latter was 10,96 and 8,33). In the combination P8 × P4 even the average of the F_3 family with the highest values scarcely surpassed the average of the F_1 (7,57 and 7,39), whereas its variance was much greater than that of P8 or P4 (1,16; 0,12 and 0,16).

The graphic solution of the complete diallel cross on normal scale (Fig. 2) showed the presence of disturbing effects in spite of the regression coefficients with values near to 1. The points corresponding to the single areas were placed along the parabola, the data of the L_2 and L_B differed greatly and their regression lines did not overlap each other. In L_B the values corresponding to the P4 and the P40 parents diverged very much from the values expected on the basis of L_2 and L_1. There was some partial dominance in the whole diallel, but this was rather small as compared to the additive effects ($\overline{AB}/\overline{OB}$). The order of dominance for the parents was: P4 > P8 > P12 > P18 > P40.

The graph of W_{r1} against W_{r2} and V_{r1} against V_{r2} also showed the presence of epistasis, however, not the graph W_{r1} against W_{rB}, and V_{r1} against V_{rB}; the value of the regression coefficient of the epistasis free diallel is 0,5. The regression coefficients of W_{r2} against W_{rB} and V_{r2} against V_{rB} showed also not the expected values of 1 (Fig. 5).

On lg scale the regression coefficients of the complete diallels coincided excellently with the expected value of 1, the regression lines for L_2 and L_B cover each other and lay between the regression line of L_1 and the tangent of the parabola. As compared to the additive effects, the dominance effects proved to be very small and the order of the parents was:

P8 > P12 ≈ P18 > P4 > P40; in L_2 P8 = P12, and in L_B P40 > P4 (Fig. 3).

The graph of W_{r1} against W_{r2} and V_{r1} against V_{r2} did likewise not show disturbing interactions, but the graph of W_{r1} against W_{rB} and V_{r1} against V_{rB}, as well as that of W_{r2} against W_{rB} and V_{r2} against V_{rB} showed unequivocally that the values corresponding to the P4 parent were too high on the L_B axis. The main role of the additive effects as compared to the dominance effects was evident too (Fig. 6).

The variance analysis of the complete diallel (Table 3) detected a highly significant additive effect on both scales. The dominance effect and the asymmetrical distribution of the genes in the parental lines were also significant. The difference between the reciprocals and the residual part (error) were not significant.

The graphical L_1 analysis achieved by the omission of single parents and their combinations revealed changes in the order of dominance. The order of dominance corresponding to the single diallels were

Table 3. *The analysis of variance of the 5 × 5 diallel, and in lg scale that of the P18 — P12 — P8 diallel (L_1).*

	DF	Normal scale		Lg scale				
		Variance ratio	Mean square	Variance ratio	Mean square	DF	Mean square	Variance ratio
a	4	679,8113	8572,65^{++}	0,547064	4144,42^{++}	2	0,0846260	2203,80^{++}
b_1	1	21,9586	276,91^{++}	0,011873	89,95^{++}	1	0,0044220	115,20^{++}
b_2	4	39,7925	501,80^{++}	0,007441	56,37^{++}	2	0,0001100	2,86
b_3	5	37,6045	474,21^{++}	0,007039	53,33^{++}			
b	10	36,9151	465,51^{++}	0,007683	58,20^{++}	3	0,0015470	40,30^{++}
c	4	0,1499	1,89	0,000125	0,95	2	0,0000505	1,32
d	6	0,1746	2,20	0,000046	0,35	1	0,0003740	9,74^{+}
								6,03
t	24	128,7518		0,094443		8	0,0217961	
B	1	1,7113		0,001824		1	0,0001230	
Ba	4	0,0447		0,000084		2	0,0000025	
Bb_1	1	0,2850		0,000281		1	0,0000150	
Bb_2	4	0,0543		0,000166		2	0,0000820	
Bb_3	5	0,0369		0,000025				
Bb	10	0,0687		0,000107		3	0,0000597	
Bc	4	0,1295		0,000204		2	0,0000305	
Bd	6	0,0866		0,000156		1	0,0000620	
Bt	24	0,0793		0,000132		8	0,0000384	
X^2	5	3,44		5,08		4	3,80	

$^{+}$ Significant difference from zero at 5% level; $^{++}$ Significant difference from zero at 1% level; X^2 From BARTLETT's test for heterogeneity of the error variances.

Table 4. *The analysis of variance of the differences between the covariances (W_r) and the variances (V_r) of the arrays, and the correlation coefficients of the parents with the sums of the variances and covariances of their arrays ($r_{yr-(Wr+Vr)}$).*

Source of variation			DF	Normal scale		Lg scale × 10^5	
				Mean square	Variance ratio and correlation coeff.	Mean square	Variance ratio and correlation coeff.
L_1		Years	1	14,8938	6,27*	0,2110	2,81
		Arrays	4	201,1096	84,62^{++}	0,3862	5,15*
		Error	4	2,3767	$r_{yr-(Wr+Vr)} = +0,99^{++}$	0,0750	$r_{yr-(Wr+Vr)} = +0,14$
L_2		Years	1	0,4452	0,24	0,1190	23,80^{++}
		Arrays	4	131,1917	70,71^{++}	0,1206	24,12^{++}
		Error	4	1,8553	$r_{yr-(Wr+Vr)} = +0,98^{++}$	0,0050	$r_{yr-(Wr+Vr)} = +0,33$
L_B		Years	1	0,0137	0,02	0,0046	0,15
		Arrays	4	17,1736	25,52^{++}	0,0544	1,77
		Error	4	0,6731	$r_{yr-(Wr+Vr)} = +0,97^{++}$	0,0307	$r_{yr-(Wr+Vr)} = -0,54$
L_1	P40 P18 P12 P8	Years	1	12,6464	3,43	0,2530	7,83*
		Arrays	3	164,0446	44,53^{+}	0,2330	7,21*
		Error	3	3,6837	$r_{yr-(Wr+Vr)} = +0,96^{++}$	0,0323	$r_{yr-(Wr+Vr)} = +0,93^{++}$
L_1	P40 P18 P8 P4	Years	1	16,9990	2,39	0,3650	2,17
		Arrays	3	309,8490	43,55^{++}	0,5633	3,35
		Error	3	7,1143	$r_{yr-(Wr+Vr)} = +0,99^{++}$	0,1683	$r_{yr-(Wr+Vr)} = -0,06$
L_1	P18 P12 P8 P4	Years	1	0,2440	10,56*	0,0192	0,41
		Arrays	3	0,0225	0,97	0,0937	1,99
		Error	3	0,0231	$r_{yr-(Wr+Vr} = +0,35$	0,0471	$r_{yr-(Wr+Vr)} = -0,89^{++}$
L_1	P18 P12 P8	Years	1	0,0051	0,35	0,00228	4,75
		Arrays	2	0,0761	5,28	0,00058	1,21
		Error	2	0,0144	$r_{yr-(Wr+Vr)} = -0,39$	0,00048	$r_{yr-(Wr+Vr)} = -0,96^{++}$

* Significant difference from zero at 10% level; $^{+}$ Significant difference from zero at 5% level; $^{++}$ Significant difference from zero at 1% level.

on normal scale: P8 > P12 > P18 > P40; P4 > P8 > P18 > P40; P8 > P4 > P18 > P12; P8 > P18 > P12,
on lg scale: P8 > P12 > P18 > P40; P8 > P18 > P4 > P40; P18 > P8 > P12 > P4; P18 > P12 > P8.

The variances of the differences between the covariances and variances of the arrays (V_{Wr-Vr}) or the correlation coefficients of the average of the parents with the sum of the covariances and variances of their arrays [$r_{yr-(Wr+Vr)}$], or both showed the presence of epistasis for almost all the diallels examined (Table 4) in spite of the regression lines with angles near 45° (the extreme values for b were 0,95 and 1,04).

On normal scale the V_{Wr-Vr} were very significant with the exceptions of the P18 — P12 — P8 — P4 and the P18 — P12 — P8 diallels, but by these the $r_{yr-(Wr+Vr)}$ were not significant ($P > 0,2$).

On lg scale the probability of V_{Wr-Vr} was generally far greater than on normal one, but even then it was quite near to the accepted limitvalue of 0,05, with three exceptions. The L_B with a probability for V_{Wr-Vr} more than 0,2, but for $r_{yr-(Wr+Vr)}$ with only 0,1—0,2, showed also not a clear picture. The F_1 diallels P18 — P12 — P8 — P4, and P18 — P12 — P8 figured only insignificant V_{Wr-Vr} and significant $r_{yr-(Wr+Vr)}$ complying with the requirements of the diallel free from epistasis; the minus sign of the correlation coefficients signifies the increase in the number of the dominant genes with the increase of the number of kernel rows.

The P18 — P12 — P8 — P4 diallel showed distinctly that the genotype P4 differed essentially from the other three parental genotypes and disturbed the analysis of their dominance conditions (the mean values $W_r + V_r$ of the two years by the parental lines are: P18 = 0,033; P12 = 0,042; P8 = 0,039 and P4 = 0,081). The P18 — P12 — P8 diallel, however, showed a clear picture; the dominance in contrast to the additive effects was again unimportant and the sequence was unquestionable P18 > P12 > P8. HAYMAN's test showed a significant additive variance, a scarcely reliable dominance variance and an insignificant asymmetry in the gene distribution (Fig. 4; Table 3).

The statistical analysis of the whole diallel as well as of the P18 — P12 — P8 diallel including the P, F_1, F_2 and B populations (Table 5) supported the graphic estimation. The additive variances had a decisive part in both diallels and on both scales; the dominance variances were relatively so low in spite of their significance in the whole diallel, that further analysis based on the dominance effects could not be regarded as full of value. This was the cause, that in the P18 — P12 — P8 diallel on normal scale we obtained $H_2 > H_1$ and $H_2/4H_1 > 0,25$, but the differences were not significant.

The analysis made on the basis of the averages of the single generations (Table 6) also confirmed the decisive role of the additive effects. In the half of all combinations the sign of dominance was minus, and it was significant only in a third of all the combinations where it was always positive. The additive × additive and dominance × dominance interactions were somewhat greater, whereas the additive × dominance interactions were small. Sequence: a > dd > aa > d > ad.

When the estimated additive effects on lg scale in the crosses P18 × P12, P18 × P8 and P12 × P8,

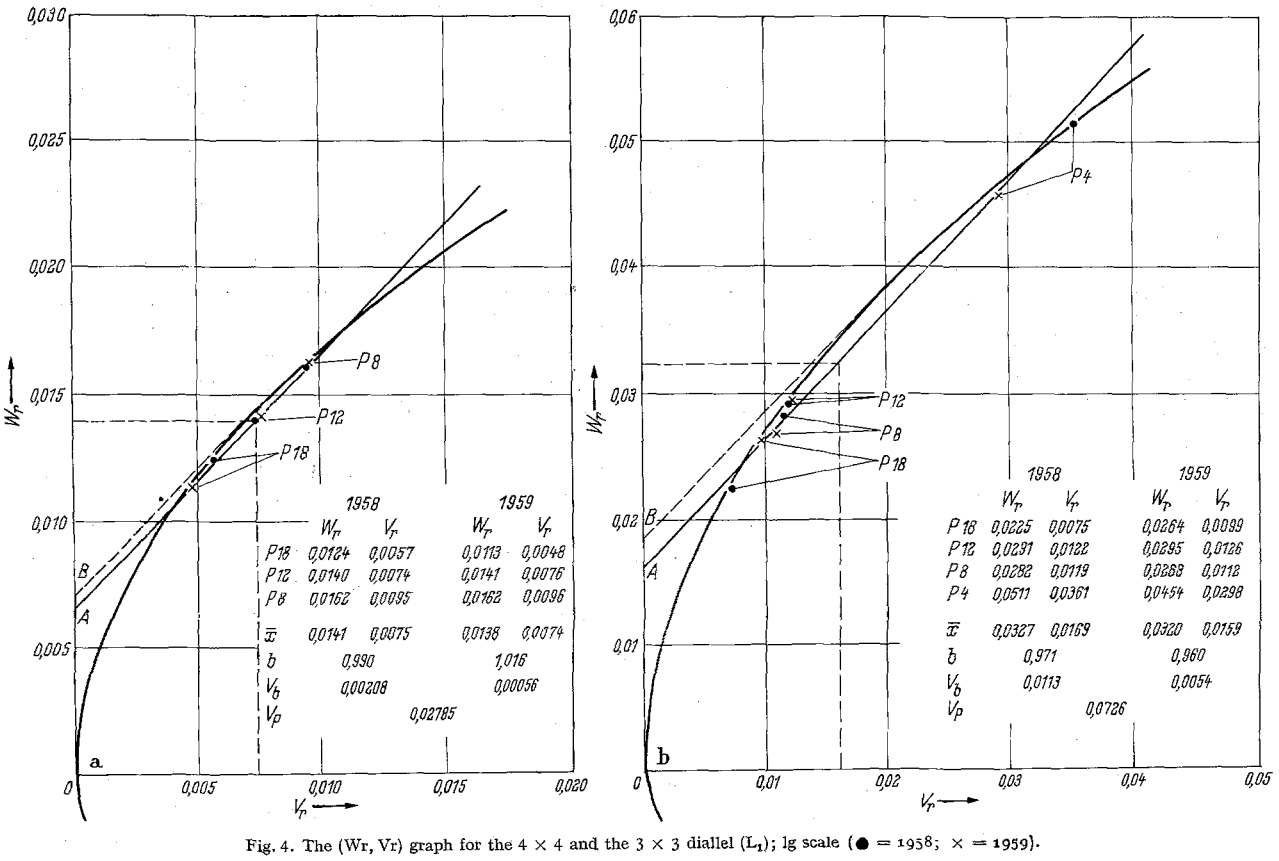


Fig. 4. The (Wr, Vr) graph for the 4 × 4 and the 3 × 3 diallel (L_1); lg scale (● = 1958; × = 1959).

Table 5. *The genetic characteristics of the 5 × 5 and the 3 × 3 diallel with their standard errors*

Item	The 5 × 5 diallel		The 3 × 3 diallel	
	Normal scale	Lg scale	Normal scale	Lg scale
D	188,220 ± 1,348	0,13346 ± 0,00263	21,267 ± 1,197	0,02790 ± 0,00113
F	70,001 ± 4,037	0,02906 ± 0,00752	−1,459 ± 0,510	−0,00047 ± 0,00093
H_1	62,649 ± 12,128	0,01271 ± 0,00403	1,633 ± 1,671	0,00205 ± 0,00225
H_2	47,205 ± 7,785	0,00882 ± 0,00034	1,683 ± 1,062	0,00141 ± 0,00024
h^2	7,620 ± 1,826	0,00443 ± 0,00129	0,538 ± 0,001	0,00199 ± 0,00036
$H_2/4\ H_1$	0,189 ± 0,006	0,17855 ± 0,06330	0,366 ± 0,212	0,23560 ± 0,22987
ā	0,575 ± 0,053	0,30721 ± 0,15096	0,252 ± 0,147	0,25861 ± 0,27410

Table 6. *Mean estimates of the mean (m), additive (a), dominance (d), additive × additive (aa), additive × dominance (ad) and dominance × dominance (dd) gene effects based on the means of the P, F_1, F_2, B_1 and B_2 populations.*

Cross	Scale	m	a	d	aa	ad	dd
P40 × P18	Normal	29,3850++	12,8775++	−2,0150	−2,4550	1,9550	0,9000
	Lg	1,4605++	0,1955++	−0,0282	−0,0718+	0,0200	0,0336
P40 × P12	Normal	22,0450++	15,0125++	4,0675++	7,0650++	1,4725	− 6,1250++
	Lg	1,3343++	0,2853++	0,0804++	0,0614+	0,0313	− 0,0920+
P40 × P8	Normal	17,8225++	16,8950++	−2,3400	6,0100	1,3800	− 5,7300
	Lg	1,2365++	0,4082++	−0,0772	−0,0124	0,0659+	− 0,0737
P40 × P4	Normal	12,5950++	16,8050++	5,5900++	15,1600++	−0,7050	−13,5400++
	Lg	1,0850++	0,4972++	0,2114++	0,2316++	0,0074	− 0,4567++
P18 × P12	Normal	14,8900++	2,5575++	3,5775++	2,3650++	−0,0600	− 3,3750++
	Lg	1,1690++	0,0725++	0,1042++	0,0662++	−0,0060	− 0,1044++
P18 × P8	Normal	12,5075++	4,8825+	−1,3000	−1,1450	0,2900	2,5400
	Lg	1,0910++	0,1767+	−0,0392	−0,0654	0,0099	0,0943
P18 × P4	Normal	10,6925++	6,4975++	0,8525	0,6250	0,0900	− 0,9550
	Lg	1,0210++	0,2723++	0,0659	−0,0454	0,0365	− 0,0857
P12 × P8	Normal	10,3775++	3,1025++	−0,3325	−1,0150+	1,1274+	2,0050+
	Lg	1,0075++	0,1337++	−0,0097	−0,0454	0,0454+	0,0933+
P12 × P4	Normal	8,5700++	3,8475++	2,4300++	1,8550++	−0,1225	− 4,6999++
	Lg	0,9275++	0,1943+	0,1262+	0,0386	0,0468	− 0,2411++
P8 × P4	Normal	7,0985++	2,3000++	−0,5790	−1,8140++	0,3049+	1,9440++
	Lg	0,8395++	0,1735++	−0,0343	−0,1438++	0,0260+	0,1666++

+ Significant difference from zero at 5% level. ++ Significant difference from zero at 1% level.

which crosses correspond to the epistasis free 3 × 3 diallel, and of the P18 × P4 cross (no relevant interactions) were divided with the number of the effective factors, quite similar values were obtained. Furthermore, when the average additive variance for a single factor was computed for the above crosses by taking use of the additive effects obtained from the variances within the families as well as from the averages of the families, the data agreed satisfactorily. For the P40 × P18 and P40 × P12 crosses the data are not so consistent (Table 7).

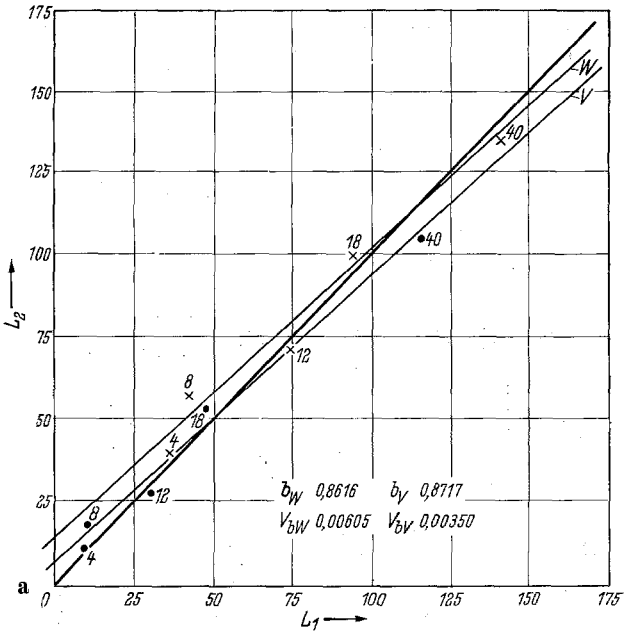
Table 7. *The additive variance corresponding to one active factor (d^2) estimated with the additive variance (D) from the within generation variances and with the non squared additive effects (a) from the means of the generations; lg scale.*

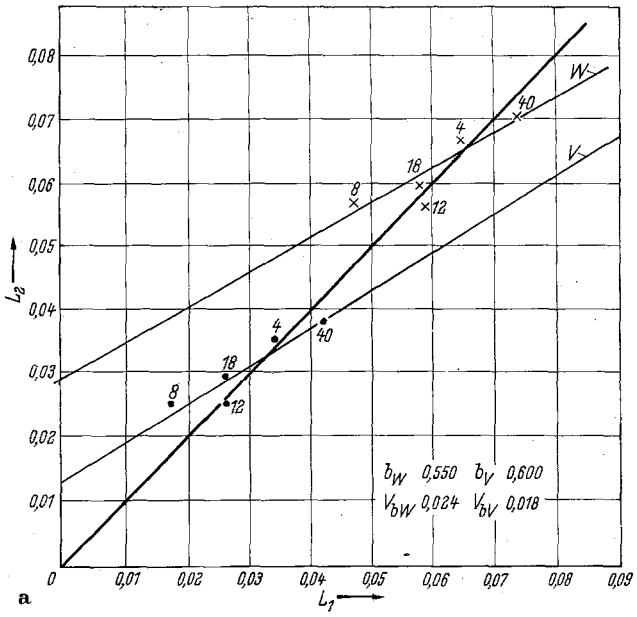
Item	k_D from table 2	D from table 2	d^2_D	a from table 6	a/k_D	d^2_a
P18 × P12	5,39	0,00114	0,000212	0,0725	0,01345	0,000181
P18 × P8	14,86	0,00187	0,000126	0,1767	0,01189	0,000141
P18 × P4	26,05	0,00379	0,000145	0,2723	0,01045	0,000109
P12 × P8	9,29	0,00084	0,000090	0,1337	0,01439	0,000207
$\bar{X}$	13,90	0,00191	0,000143	0,1638	0,01255	0,000160
P40 × P18	3,22	0,00956	0,002969	0,1955	0,06071	0,003686
P40 × P12	10,77	0,00599	0,000556	0,2853	0,02649	0,000702
$\bar{X}$	7,00	0,00778	0,001763	0,2404	0,04360	0,002194

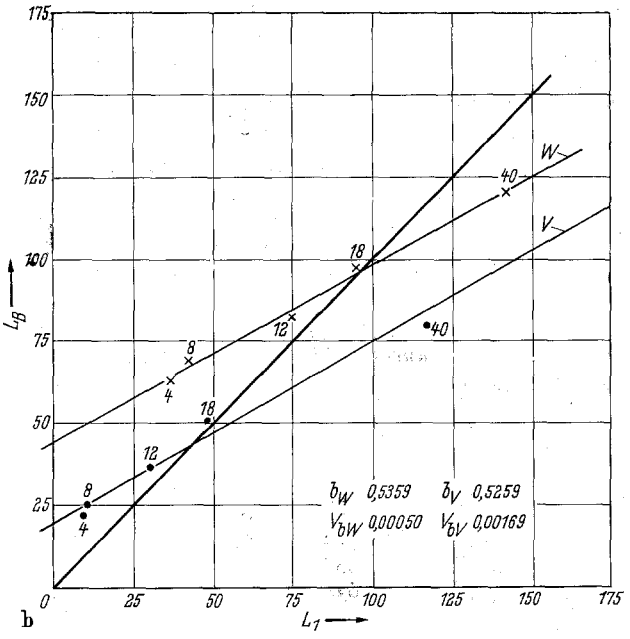
Discussion

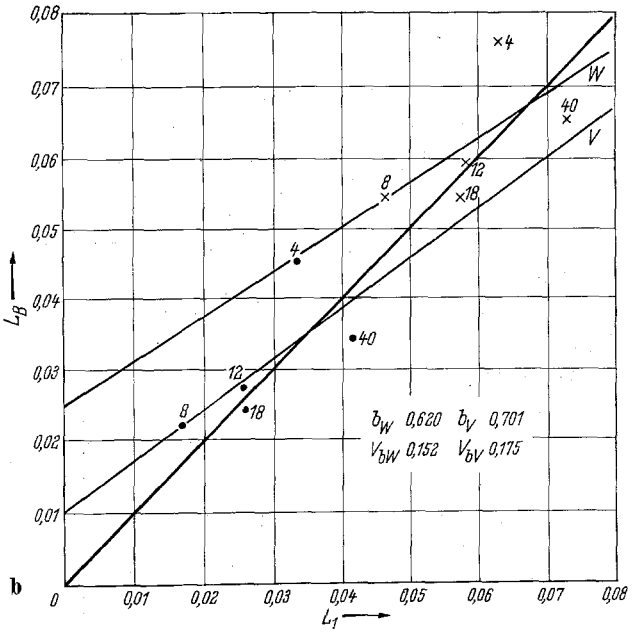
The literature indicated the extraordinarily complicated inheritance of the number of kernel rows in maize, nevertheless the environmental variances were almost negligible. It is true that in our experiments it was not possible to find a suitable common scale for the material containing only five, however very different parents and their progenies. The variances within generations of the non segregating progenies did not prove homogenous neither on normal scale nor on lg scale, but averages and variances showed significant positive correlation on normal scale, while they showed insignificant negative correlation on lg scale, and consequently a greater change of scale was not justified. GILBERT (1961) emphasized that variances were more sensitive to less differences from the additivity (inappropriate scale) than averages are, in contrast to MATHER (1949) who stated that minor differences from the additivity do not cause significant error. Our experiments showed also between the analysis undertaken on different scales often relevant differences (Table 2, 4; Fig. 2, 3); however, while at the estimations based on variances within generations only three combinations showed approximately identical values, in the diallel crosses based on variances and covariances of averages, the general aspect was sufficiently uniform and a great difference appeared only in order of the dominance, the analysis applying the means (Table 6) presented quite completely the same aspect in both scales.

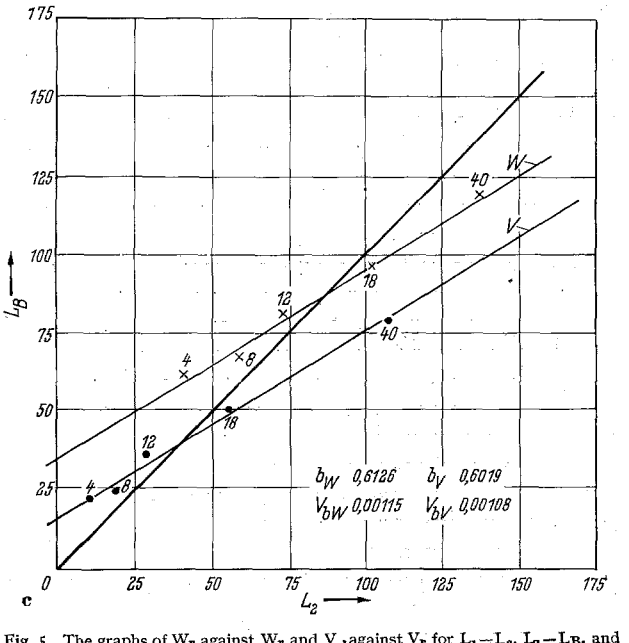
Most information is expected from the analysis of the individual variances of single generations











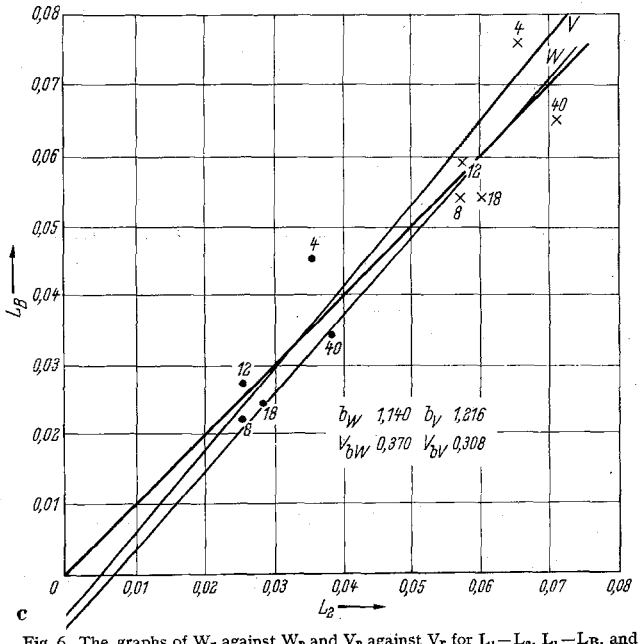


Fig. 5. The graphs of W_r against W_r and V_r against V_r for L_1-L_2, L_1-L_B, and L_2-L_B with the 5 × 5 diallel; normal scale.

Fig. 6. The graphs of W_r against W_r and V_r against V_r for L_1-L_2, L_1-L_B, and L_2-L_B with the 5 × 5 diallel; lg scale.

when it is possible to make it, however, it can be done only in simple cases. MATHER (1949) employing the data of EMERSON and EAST on the inheritance of kernel rows of maize, obtained minus values for the dominance variances. WARNER (1952) manipulated with the data of about 3000 plants to establish the coefficient of heritability by lg scale, though he used only F_2 and B.

It must be emphasized that in our experiment the average number of individuals for each combination (P, F_1, F_2, F_3 and B) were only about 1300. Both the small number of individuals and the inappropriate scale might have contributed to the fact that we could not obtain the expected results. The dominance variances were very high in both the scales and taking into consideration that the averages of the F_1 were in each combination between the values of the two parents, and in most of the cases closely approaching the mid-parent value, the high values obtained were not real. The additive effects on lg scale were sufficiently consistent in combinations appropriate for analysis and in these the estimated number of active factors (Table 2) except the P40 crosses, agreed with the additive effects estimated by the means of the families (Table 6) giving the same additive variance for one factor in the two estimations with satisfactory proximity (Table 7). Although the maize has ten linkage groups, the high numbers for the factors were not exaggerated as compared to figures 20—40 for oil content (SPRAGUE and BRIMHALL, 1949) and to that of 22 for protein (FREY, 1949). The difference of one, respectively two pairs of genes with complementary action as reported by TAVČAR (1935) and BURDICK (1948, 1951) in their crosses between lines with 8 and 4 kernel rows, was not confirmed in this material; the fact that none of the 8 F_3 families studied was identical for kernel-row genes to any of the two parental lines, supported also our suggestions. In the other crosses the situation was the same too, and from the 103 families of the whole experiment, no one had the identical genotype for kernel-row number with one of the 5 parental lines. Moreover, the extraordinarily high mutation rate of the number of kernel rows in diploids of monoploid origin (3,6 on 100 gametes) found by SPRAGUE, RUSSEL and PENNY (1960), as well as the failure to establish any linkage between the number of kernel rows and monogenic characters, made it very probable, that this character is controlled by many genes.

The diallel analysis introduced by HAYMAN and JINKS gave a good general information on the basis of the L_1 too; the extension to the L_2 and L_B naturally strengthens the gain of information, however, the analysis becomes less efficient, because by the L_2 and L_B the multiplicator of H_1 and H_2 is $^1/_{16}$, and that of F is $^1/_8$, in contrast with the multiplicator of $^1/_4$ for both in the L_1. Furthermore the parents are the same in all the three diallels, complicating the calculation of significance. The plus gain of information in the diallel analysis obtained with the F_2 and B generations is not proportional to the surplus of labour and time required by the production of the more advanced generations. Plotting against the data from L_1 and L_2, L_1 and L_B, as well as that of L_2 and L_B also offered valuable informations on both the dominance conditions and the epistasis effects, but this may also be solved on the basis of L_1 (Table 4). In the present experiment the P18 — P12 — P8 diallel offered a good example, in which the data were estimated on the basis of L_1 graphically, while with the jointed L_1, L_2 and L_B by the least square equations; the $^1/_4$ D and $^1/_4$ H show a good agreement (0,006963 and 0,006975; 0,000559 and 0,000513).

The analysis on normal scale was never free from disturbing effects (Table 4), thus the dominance order showed by it was not true. Only the P18 — P12 — P8 diallel on lg scale offered a completely clear aspect and showed the definite dominance of the greater number of kernel rows, although the partial dominance with a value $\bar{a} = 0{,}26$ was wery low. The small disturbance in the order of dominance in the P18 — P12 — P8 — P4 diallel (Fig. 4) is presumably due to the interaction of the P8 and P4. The P40 line with fasciated ears resulted in a quite different situation. The points corresponding to the array of this parent in spite of its high number of kernel rows were the most distant from the origo. A very pronounced interaction was between P40 and P4; the $r_{yr-(Wr+Vr)}$ values in L_1 on lg scale were: for the whole diallel $r = +\ 0{,}14$; for P40 — P18 — P12 — P8, $r = +\ 0{,}93$, and for P18 — P12 — P8 — P4, $r = -\ 0{,}89$ (Table 4). The inheritance of fasciation of this line has been studied in earlier experiments (DANIEL and VÁRÓCZY, 1959 a) and it seemed very probable that the factor or factors for fasciation interact with the factors controlling the kernel-row number. Further study on inheritance of fasciation is in progress to elucidate the phenomenon.

Besides of the many advantages, of course, the diallel method has some disadvantages too, e. g. the estimation of the significance with partially independent data. However, such difficulties may be avoided when the estimations are based on replications or years, as it was done in this experiment. The critique, that the restricted number of lines selected from a population for the diallel cross do not represent appropriately the population under study, or that the omission of the parents of arrays showing epistasis is not justified (GILBERT, 1958), did not hold here, because this study was not extended to populations, but only to certain lines. A further objection can be raised from the mathematical point of view that the 3×3 diallel is too small, however, the additive effects playing the most important role in our system, were in every case significant with such a probability, that even this cannot be a serious disadvantage. Diallels of this extent were also used by others; JOSHI, RAMANUJAM and PILLAY (1961) demonstrated the suitability of the method for the selection of the parents in a breeding programme in a 3×3 diallel, and recently MURTY, MURTY and PAVATE (1962) estimated four quantitative characters of tobacco from a 4×4 diallel.

The separation of gene-effects on the basis of the averages of single generations gave additional data to the diallel crosses (Table 6). Besides the major additive effects, a significant epistasis had been shown in the half of the combinations, which is also essential from the breeder's point of view.

Conclusions

Joining the results obtained by the three methods adapted, it can be concluded that in agreement with EMERSON and SMITH (1950) the pattern of inheritance of kernel-row number in maize is complicated, however, it can be explained by its evolution.

In our experiments performed under normal conditions, the environmental variances were weak, as it was to be expected after the publications and in accordance with GWYNN (1959) there was no considerable genotype-environment interaction. The number of factors controlling the formation of the kernel-row number was very great and probably in most combinations more than, or equal with the number of haploid chromosomes; the data of TAVČAR (1935) and BURDICK (1948, 1951) were not confirmed in these experiments. The epistasis disturbed the analysis in some extent and probably the linkage too, but on lg scale the dominance variance for one factor approached very well the 0,00015 value in the crosses of the epistasis free diallel and in the P18 × P4 cross if computations were made with the number of factors computed on the basis of individual variances and the additive effects estimated by using the individual variances as well as the averages. In accordance with GARDNER, HARVEY, COMSTOCK and ROBINSON (1953), the degree of dominance computed by the diallel analysis is very small and the high values for the dominance variance obtained after the estimation of the variances within generations may be due to linkages and epistasis. Besides the additive effects significant epistasis were also detected in some of the crosses supporting the data of GAMBLE (1957, 1962b), BAUMAN (1959) and HYER (1960). In spite of their little importance, the dominance effects were also significant for some of the crosses and the parents with higher kernel-row number possessed the greater number of dominant factors too.

In contrast to the dominant unpaired spicelet of the ear with monogenic inheritance (HEPPERLY, 1949; WILCOX, 1951), the two anomalous forms in our experiment were recessive with complicated pattern of inheritance. Although one line was 40-rowed and the other one 4-rowed, the points of both approached in the diallel analysis the point corresponding to the full recessive, i. e. the upper point of intersection of the regression line with the parabola. The 40-rowed line is a fasciated type and the factor or factors for fasciation presumably mask the effect of the true kernel-row number genes.

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Summary

The inheritance of the number of kernel rows in maize was studied in a two years experiment made by 5 × 5 complete diallel crosses, including the P, F_1, F_2 and B generations. In the second year of the experiment some of the F_3 families was also involved, although in a limited number for each single combination, and the estimation of these data was accordingly performed.

The material including two extreme ($\bar{X} = 39$, and 4), and three intermediate ($\bar{X} = 17$, 12, and 8) lines, showed a complicated inheritance of the number of kernel rows; the lg scale proved to be more suitable than the normal scale, as this was also not satisfactory, therefore the analyses were undertaken on both scales. The estimation made on the basis of variances within the single generations gave unjustified high dominance variances. In the combinations suitable for valuation, the number of the effective factors taking part in the manifestation of the character was sufficiently consistent, though rather great; presumably it often surpasses the number of haploid chromosomes of maize. In the analysis made by the method of HAYMAN and JINKS, the P18 — P12 — P8 diallel alone, which include the intermediate lines, and only on lg scale proved to be free from effects disturbing the analysis. Additive effects are characteristic for the inheritance of the number of kernel rows and their dominance effects are slight ($\bar{a} = 0{,}26$). The order of dominance is: P18 > P12 > P8; when the P4 parent was also involved in the analysis, this showed a definite recessivity, already disturbed the order of dominance of the other lines. Among the parental lines P40, a fasciated type, contained the most recessive factors on both scales, in spite of the high number of kernel rows; the factor or factors for fasciation probably mask the effect of the true kernel-row number genes. The result of the analysis made with the averages of single generations on normal and on lg scale showed a quite good agreement and a decisive role of the additive effects was found beside the significant epistasis effects. The additive × additive and the dominance × dominance interactions were rather relevant, the dominance effects of less importance, while the additive × dominance interactions were very small. On the basis of both the individual variances and the averages, the dominance variance for one factor was sufficiently uniform in the combinations suitable for analysis.

Zusammenfassung

Untersuchungen zur Vererbung der Kornreihenzahl beim Mais

In einem zweijährigen Versuch, der die P-, F_1-, F_2- und B-Rückkreuzungsgenerationen umfaßt, wurde die Vererbung der Kornreihenzahl beim Mais anhand von vollständigen 5 × 5 Diallelkreuzungen geprüft. Im zweiten Versuchsjahr wurde für jede Kombination noch eine beschränkte Anzahl von F_3-Familien in die Prüfung einbezogen. Das Material, das aus 2 Linien mit extremen Kornreihenzahlen ($\bar{X} = 39$ und 4) und 3 Linien mit mittlerer Kornreihenzahl ($\bar{X} = 17$, 12 und 8) besteht, zeigte eine kompliziertere Vererbung des Merkmals. Für die Berechnungen erwies sich die logarithmische Skala geeigneter als die normale arithmetische Skala; da auch sie nicht immer befriedigte, wurden die Analysen auf der Grundlage beider Skalen durchgeführt. Die Varianz- und Kovarianzanalysen der einzelnen Generationen ergaben unerwartet hohe Dominanzvarianzen. Die Zahl der an der Manifestierung des Merkmals beteiligten Faktoren erwies sich in den bewertbaren Kombinationen als genügend übereinstimmend, sie

ist allerdings sehr hoch und überschreitet vermutlich oft die Zahl der haploiden Mais-Chromosomen. Bei der nach der Methode von HAYMAN und JINKS durchgeführten Analyse erwiesen sich nur die Diallelkreuzungen P18 — P12 — P8, die die Linien mit mittlerer Kornreihenzahl umfassen, und zwar nur nach der logarithmischen Berechnung, als unbeeinflußt durch teilweise auftretende Störungen. Für die Vererbung der Kornreihenzahl ist additive Wirkung charakteristisch, die Dominanzwirkung ist gering ($\bar{a} = 0{,}26$). Die Dominanzfolge ist P18 > P12 > P8. Der in die Untersuchung einbezogene P4-Elter zeigte eine deutliche Rezessivität und durchbrach die Dominanzfolge der übrigen Linien. Unter den Elternlinien enthielt P40, ein fasziierter Typ, nach beiden Berechnungsarten die meisten rezessiven Faktoren trotz hoher Zahl an Kornreihen. Wahrscheinlich verdecken der oder die Faktoren für Fasziation die Wirkung der Gene für Kornreihenzahl. Die Untersuchung, die mit den Durchschnittswerten der einzelnen Generationen auf normaler und logarithmischer Basis durchgeführt worden ist, erbrachte ausreichend gute Übereinstimmung. Neben signifikanten Epistasiewirkungen wurde die entscheidende Rolle der Additivität festgestellt. Die Wechselwirkungen additiv × additiv und dominant × dominant waren ziemlich hoch, die letzteren etwas geringer, die Wechselwirkungen additiv × dominant erwiesen sich als sehr klein. Sowohl auf der Grundlage von Einzelvarianzen als auch auf der von Durchschnittswerten zeigte die Dominanzvarianz für einen Faktor eine genügende Uniformität in den zur Untersuchung geeigneten Kombinationen.

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