GENETIC VARIATION IN DIPLOID *DACTYLIS*

III. PANICLE, SPIKELET AND FLORET CHARACTERS

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1. INTRODUCTION

IN this final paper of a series describing the pattern of genetic variation to be found in some subspecies populations of diploid *Dactylis*, the 9×9 diallel cross previously described (Parker, 1968 *a, b*) is analysed for a series of panicle characters, some of which have been replicated within the plant to study the reliability of the genetic information provided by a single measurement.

2. MATERIALS AND METHODS

The same subspecies numbers as before are used in the tables and graphs (fig. 1) and the characters considered are:

- (i) Panicle length (mm.).
- (ii) Number of florets per spikelet.
- (iii) Spikelet length (mm.).
- (iv) Lemma length. Measured on a Prior binocular microscope; micrometer units used in table = mm. $\times 2$.
- (v) Lemma awn length, measured as (iv).
- (vi) Lemma apex notch depth, measured as (iv).

The first three panicles to emerge on each plant were measured separately and analysed as genotype replicates (*i.e.* intra-plant replicates) in order to estimate the extent of variation in gene expression due to environmental and developmental changes within the plant. The two characters, florets per spikelet and spikelet length, were similarly treated, using two genotype replicates.

The extended Hayman's analysis of variance for these three characters is similar to that used by Broadhurst and Jinks (1966). Analysis of the results summed over genotype replicates gives the variation within samples. For the main effects items to be significant, they must be significantly greater than all three interaction items, however as the block interactions were the largest in all cases, main effects were tested against these. The block and genotype replicate interactions were tested against the appropriate residual items in each case.

The variance (Vr) covariance (Wr) analysis developed by Jinks (1954) was also used in the genetic assessment of the populations. Both this analysis and the parental means were further analysed using the method proposed by Allard (1956); this method gives an assessment of the stability of three parameters, namely, additive effects, dominance effects and epistatic effects.

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The analysis of parental means tests for additive genetic effects; constancy of these additive components of variation can be detected by the parents $\times$ genotype replicates item, significance indicating interaction, and non-significance indicating constancy of expression in the different genotype replicates.

A good test for the constancy of additive components of variation being available from analysis of parental means, this component is minimised in the analysis of Wr and Vr by dividing them by the variance of the parents (Vp) occurring in the same block. This improves the prospects of detecting dominance $\times$ genotype replicate interactions by placing comparisons between them on similar scales. The $W'r/Wr$ graph is also used to detect the order of dominance of the parents, it is much less affected by genetic disturbances than the Wr/Vr graph and virtually undisturbed by the level of inbreeding (Hayman, 1958). The $W'r/Wr$ graph differs from the Wr/Vr however, in that it is more obviously affected by asymmetry of gene distribution, and indicates this whether the genes are correlated or not (Hayman, 1958). However, as both graphs are disturbed by both these effects to some extent it is not always possible to separate the causes of the disturbance with complete precision. With gene symmetry the regression of $W'r$ on Wr is a straight line of slope $+0.5$, when gene asymmetry occurs, parents with common genotypes will fall above the line of slope $+0.5$, parents with different or relatively rare genotypes will fall below it. Both male and female points were put on the Wr/Vr graphs when the Hayman's analysis indicated the presence of significant consistent reciprocal differences, but if the reciprocal regressions were not significantly different, the joint line was fitted. The environmental component of variation (E_2) derived from the between blocks total interaction mean square from the Hayman's analysis of variance, and appropriately calculated for Wr and Vr (Jinks, 1954) was also obtained, but only in two characters was the non-heritable variation large enough to noticeably alter the level of dominance indicated by the uncorrected Wr/Vr graph. In these graphs the corrected ordinates and abscissae are put in as broken lines.

3. RESULTS

(i) *Panicle length*

The mean lengths of each genotype replicate are presented in table 1, and Hayman's analysis of variance in table 2. The block interactions are homogeneous ($\chi^2_{(5)} = 7.8$; $P = 0.3$) so individual main items were tested against the common interaction item (Bt). There are large and highly significant additive genetic differences between the populations (item a) and some dominance variation (item b). All three components of dominance are significant (b_1) indicating the presence of directional dominance (b_2) gene asymmetry, and (b_3) inconsistent dominance interactions. The items of both block and replicate interactions are all significant (apart from $\text{Reps} \times c$), when tested against the items of the residual, the main replicates interaction is also significant. The replicates $\times a$ interaction stands out as being quite large, but this does not alter the significance of the main (a) item.

The Wr/Vr graphs (fig. 1) show that the array points of all three genotype replicates fall on the same line, analysis of variance showed no significant differences between the regressions so a common line is fitted ($b = 1.142 \pm 0.110$; $P = 0.001$), this cuts the Wr axis positively, indicating a low

level of dominance. The correlation of parental means on $(Wr + Vr)$, ($r = 0.745^*$) shows this to be directional for small panicle. The $W'r/Wr$ graph gives a similar distribution of points, the regression ($b = 0.441 \pm 0.041$; $P = 0.001$) is not significant from $+0.5$.

TABLE 1

Mean panicle length (mm.) for the first (top line) second (centre line) and third (bottom line) genetic replicates

	1	2	3	4	5	6	7	8	9
1.	94.97 87.96 87.77	103.21 87.46 77.36	150.26 105.14 94.75	120.49 99.07 86.47	148.01 112.93 102.92	110.91 93.76 91.20	142.35 103.67 83.57	133.75 93.60 76.55	121.40 101.17 87.94
2.	79.08 74.78 61.03	34.01 33.78 34.14	80.72 62.40 55.67	65.85 57.67 51.39	88.26 78.60 79.37	59.26 57.42 52.78	76.15 46.72 46.31	77.21 57.60 47.03	84.71 59.32 54.15
3.	134.47 90.24 84.38	82.67 71.60 61.47	118.54 86.89 83.57	156.42 115.51 97.01	167.54 126.64 114.68	138.54 109.57 100.28	127.35 90.12 80.29	142.71 93.50 84.14	154.55 114.58 96.76
4.	108.46 95.49 82.26	68.06 58.51 57.22	160.79 129.61 111.43	110.49 99.47 99.34	156.17 113.31 100.61	133.43 112.37 97.28	126.43 77.69 69.12	137.62 107.70 90.80	144.09 104.52 94.13
5.	133.35 111.21 101.53	72.07 80.74 78.64	174.93 127.33 115.10	146.71 113.18 104.29	187.81 147.68 138.35	170.22 133.39 125.82	144.71 102.34 90.80	168.57 114.61 100.75	166.62 134.67 116.07
6.	118.85 95.74 89.54	66.60 63.50 64.08	125.26 95.67 86.90	117.67 107.99 101.41	158.77 131.52 118.62	145.58 122.47 123.26	124.24 89.21 81.35	156.60 101.58 87.60	148.79 114.55 93.93
7.	133.28 103.28 84.87	83.76 60.17 55.33	120.78 81.60 66.45	133.13 90.41 89.16	154.18 118.06 96.39	112.79 79.83 75.60	102.89 67.74 55.82	137.02 94.56 80.45	139.26 108.89 87.12
8.	141.04 92.62 86.96	90.58 64.14 49.33	139.21 91.53 70.59	130.56 110.45 95.98	160.92 124.17 104.45	132.32 106.50 96.86	123.43 84.09 82.03	158.79 87.57 68.92	175.79 99.94 93.11
9.	127.58 88.14 81.01	88.65 57.19 57.07	149.05 104.20 78.70	150.53 104.97 90.17	166.47 128.92 106.50	136.18 103.87 92.21	128.11 95.24 81.42	195.64 110.42 94.67	121.11 83.23 2.84

Analysis of parental means (table 3a) gives a highly significant mean square for parents, indicating that they have different additive and/or epistatic effects. The significant genotype replicates mean square shows that the expression of the additive genes differed between replicates, this is no doubt related to the different times of initiation, and different growth periods of the panicle primordia. The mean square for the parents $\times$ genotype replicates interaction is large and highly significant, this provides evidence for the interaction of additive and/or additive $\times$ additive gene effects with

genotype replicates. There is also a significant interaction between parents and blocks. The order of parental panicle size is:

Large				Small						
G.R.	I	5	6	9	8	4	1	3	7	2
	II	5	6	8	4	3	1	9	7	2
	III	5	6	4	3	8	9	1	7	2

TABLE 2

Combined Haymans analysis of variance including genotype replicates, for panicle length, number of florets per spikelet, and spikelet, and spikelet length

Item	(a) Panicle length			(b) No. of Florets			(c) Spikelet length	
	D.F.	M.S.	V.R.	D.F.	M.S.	V.R.	M.S.	V.R.
<i>a</i>	8	45233.82	186.75***	8	10.43	28.84***	20.4802	77.90***
<i>b</i> ₁	1	1770.38	7.31**	1	6.04	14.71***	5.0126	19.07***
<i>b</i> ₂	8	1861.19	7.68***	8	1.18	3.27***	1.4808	5.63***
<i>b</i> ₃	27	1167.20	4.82***	27	2.16	5.97***	1.3656	5.19***
<i>b</i>	36	1338.18	5.52***	36	2.05	5.67***	1.4925	5.68***
<i>c</i>	8	403.40	1.66	8	0.77	2.13*	0.2957	1.12
<i>d</i>	28	242.42	—	28	0.57	1.59	0.3744	1.42
Total	80			80				
Blocks	2	2920.04	2.68	2	1.74	4.84	0.7836	2.36
<i>Ba</i>	16	230.65	2.10*	16	0.34	4.64**	0.2005	6.77***
<i>Bb</i> ₁	2	795.11	15.81*	2	0.05	—	0.0852	3.52
<i>Bb</i> ₂	16	111.52	2.71**	16	0.36	5.11**	0.2997	11.02***
<i>Bb</i> ₃	54	201.61	2.74**	54	0.39	16.88***	0.2074	5.34***
<i>Bb</i>	72	202.63	3.08***	72	0.38	8.42***	0.2245	6.27***
<i>Bc</i>	16	290.92	4.77***	16	0.26	5.32**	0.2185	6.28***
<i>Bd</i>	56	282.51	6.31***	56	0.38	15.12***	0.3428	24.31***
<i>B</i> total	160	242.22		160	0.36		0.2629	
Total	242			242				
Replicates	2	117061.64	107.53**	1	7.13	18.35	5.0251	15.17
<i>Ra</i>	16	2399.22	21.86***	8	0.07		0.0429	1.45
<i>Rb</i> ₁	2	393.51	7.82*	1	0.02		0.0567	2.34
<i>Rb</i> ₂	16	162.73	3.96**	8	0.05		0.0236	
<i>Rb</i> ₃	54	174.48	2.37**	27	0.03	1.42	0.0141	
<i>Rb</i>	72	177.95	2.71**	36	0.03		0.0174	
<i>Rc</i>	16	78.55	1.29	8	0.02		0.0170	
<i>Rd</i>	56	70.14	1.57***	28	0.03	1.19	0.0193	1.3/
<i>R</i> total	160	352.40		80	0.03		0.0206	
Blocks ×								
Replicates	4	1088.60		2	0.39		0.3313	
<i>B × R × a</i>	32	109.73		16	0.07		0.0296	
<i>B × R × b</i> ₁	4	50.29		2	0.41		0.0242	
<i>B × R × b</i> ₂	32	41.08		16	0.07		0.0272	
<i>B × R × b</i> ₃	108	73.54		54	0.02		0.0388	
<i>B × R × b</i>	144	65.68		72	0.04		0.0358	
<i>B × R × c</i>	32	60.93		16	0.05		0.0348	
<i>B × R × d</i>	112	44.78		56	0.02		0.0141	
<i>B × R</i> total	320	62.30		160	0.04		0.0275	
Total	484			242				
Grand total	726			484				

P* = 0.05; *P* = 0.01; ****P* = 0.001.

FIG. 1.— Wr/Vr and $W'r/Wr$ graphs for panicle length. Numbers are as follows; 1. smithii; 2. juncinella; 3. lusitanica; 4. aschersoniana; 5. parthiana; 6. himalayensis; 7. ibizensis; 8. santai; 9. castellata.

The most variable parents appear to be *castellata* (9), *lusitanica* (3) and *santai* (8), they are also the ones that change position most on the *Wr/Vr* graph.

In the analysis of variance of *Wr* and *Vr* (table 3b), only three mean squares are of interest; the significance of that for genotype replicates suggests

TABLE 3a
Analysis of variance of parental means

Item	D.F.	M.S.	V.R.
Parents	8	10584.74	189.41***
Blocks	2	156.40	2.80 N.S.
Genotype replicates	2	9385.28	167.95***
Blocks $\times$ Gen. reps.	4	177.51	3.18*
Parents $\times$ Gen. reps.	16	632.59	11.32***
Parents $\times$ blocks	16	197.54	3.53**
Error	32	55.88	
Total	80		

TABLE 3b
*Analysis of variance of *Wr* and *Vr* for panicle length*

Item	D.F.	M.S.	V.R.
Arrays	8	20.78	36.15***
Genetic replicates	2	9.51	16.54***
Dominance	1	59.84	104.13***
G.r's $\times$ arrays	16	1.31	2.28**
G.r's $\times$ dominance	2	1.10	1.92 N.S.
Arrays $\times$ dominance	8	0.43	
G.r's $\times$ dom $\times$ arrays	16	0.09	
Error	108	0.57	
Total	161		

*P = 0.05; **P = 0.01; ***P = 0.001.

a change in mean dominance and/or the relative proportion of dominant and recessive effects within the plants. The mean square for genotype replicates $\times$ arrays tests the constancy of the mean level of dominance for each parent over the three measurements, this is highly significant, indicating that the positions of the parental arrays altered relative to each other, either up or down the regression line, in different genotype replicates. The ranking of the parents in order of decreasing dominance was:

Dominant											Recessive		
G.R.	I	2	1	7	3	4	8	9	6	5			
	II	1	2	7	8	4	3	5	9	6			
	III	1	2	7	4	9	8	3	5	6			

The genotype replicates $\times$ dominance mean square is non-significant, indicating that the regression lines for the three measurements on the same plant occupy the same positions. These results suggest that both additive gene effects and dominance relationships among various parental populations were affected by intra-plant variability, however, the shifts in dominance that occurred among individual parental populations varied at random

around the mean so that the mean level of dominance over all arrays was not altered.

(ii) *Number of florets per spikelet*

The two genotype replicates were taken from different areas of the panicle, one from the lower part of a basal glomerulus, the other from the same area of a glomerulus taken from the centre of the head.

TABLE 4

Mean number of florets per spikelet for the first (top line) and second genetic replicates

	1	2	3	4	5	6	7	8	9
1.	5.39 5.51	4.22 4.58	6.53 6.69	6.03 6.08	7.22 7.33	6.25 6.36	6.42 6.42	5.97 6.30	4.44 4.53
2.	4.69 4.78	4.20 4.51	5.75 5.92	5.58 6.17	5.60 5.62	5.44 5.78	5.14 5.56	5.05 5.30	4.58 4.92
3.	6.25 6.25	5.86 6.00	5.44 6.14	5.39 5.81	5.64 5.92	5.14 5.22	5.08 5.44	5.64 5.69	4.61 4.81
4.	5.47 6.05	5.08 5.39	6.22 6.53	5.25 5.55	6.28 6.68	5.14 5.67	5.26 5.69	4.87 4.89	5.09 5.26
5.	5.72 5.72	5.58 6.03	4.86 5.14	6.44 6.56	6.25 6.50	5.31 5.69	6.40 6.40	5.86 5.86	5.14 5.39
6.	5.64 5.86	5.30 5.61	5.50 5.83	5.64 5.85	5.38 5.67	4.99 5.25	5.28 5.54	5.36 5.58	4.53 4.64
7.	6.30 6.44	5.33 5.69	4.48 4.61	4.97 5.39	5.63 5.60	5.20 5.47	4.47 4.89	5.07 5.44	4.72 4.89
8.	5.64 5.89	4.92 5.05	5.83 6.03	4.83 5.07	5.67 5.92	5.53 5.78	5.24 5.69	4.33 4.36	4.78 4.97
9.	4.25 4.39	4.67 4.97	4.49 4.91	5.11 5.36	5.19 5.61	5.17 5.33	5.00 5.33	4.44 4.64	4.60 4.71

The means of the two genotype replicates are presented in table 4, and Hayman's analysis of variance in table 2. The block interaction variances were homogenous ($\chi^2_{(5)} = 3.9$; $P = 0.7$) therefore all items of the main effects were tested against the common interaction term (Bt). There is significant additive variation between the populations (item a), also significant dominance variation (item b). All the components of (b) are significant, the largest (b_1), indicating the presence of directional dominance with lesser indications of gene asymmetry (b_2) and inconsistent dominance interactions (b_3). There is also a small (c) item suggesting the presence of some slight consistent reciprocal differences. All of the individual block interactions except for $B \times b_1$ were significant when tested against the residual items, however, none of the replicate interactions were significant, neither were the main block and main replicate interactions, although the latter is large. It appears from this analysis that variation between blocks is of greater importance than that within genotypes.

The Wr/Vr graph (fig. 2) shows a wide scatter of points which do not fall on any significant regression line; this indicates some form of non-allelic

interaction. The distribution of points suggests that this may be of the complementary type. The correction of ordinate and abscissa for the environmental component (E_2) reduces the level of dominance only slightly. The $W'r/Wr$ graph (fig. 2) does have a significant regression line in both replicates, these are not significantly different from each other, therefore, the joint regression is fitted ($b = 0.316 \pm 0.090$; $P = 0.01$).

FIG. 2.— $W'r/Vr$ and $W'r/Wr$ graphs for number of florets per spikelet. Corrected ordinate and abscissa included as dashed lines.

Analysis of parental means (table 5a) shows that there are significant differences in the expression of additive genes between both replicates and blocks, with some interaction between them. The mean number of florets is different in the two replicates. The order of parental spikelet size is:

	Large					Small				
G.R. I	5	3	1	4	6	9	7	8	2	
G.R. II	5	3	4	1	6	7	9	2	8	

TABLE 5a

Analysis of variance of parental means

Item	D.F.	M.S.	V.R.
Parents	8	2.854	30.96***
Blocks	2	0.417	4.52*
Genotype replicates	1	1.047	11.36**
Blocks $\times$ Gen. reps.	2	0.263	2.85*
Parents $\times$ Gen. reps.	8	0.059	—
Parents $\times$ blocks	16	0.143	—
Error	16	0.092	—
Total	53		

*P = 0.05; **P = 0.01; ***P = 0.001

TABLE 5b

Analysis of variance of W_r and V_r for number of florets per spikelet

Item	D.F.	M.S.	V.R.
Genetic replicates	1	36.56	5.28*
Arrays	8	61.77	8.92***
Dominance	1	260.77	37.68***
Genetic replicates $\times$ arrays	8	7.67	1.11 N.S.
Genetic replicates $\times$ dominance	1	1.32	—
Arrays $\times$ dominance	8	21.93	3.17**
Gen. reps. $\times$ arrays $\times$ dominance	8	1.36	—
Error	71	6.92	—
Total	107		

*P = 0.05; **P = 0.01; ***P = 0.001.

Analysis of W_r and V_r (table 5b) shows that there is a significant mean square for genotype replicates, indicating a change in the proportion of dominant and recessive effects within the plant; the significance of the dominance $\times$ array mean square indicates that epistatic effects are important, these may also have contributed to the genotype replicate effects.

The ranking of the parents is:

	Dominant						Recessive		
G.R. I	1	7	8	2	4	5	3	6	9
G.R. II	1	4	2	8	3	7	5	9	6

(iii) *Spikelet length*

The data are presented in table 6, and Hayman's analysis of variance in table 2. Bartlett's test showed the block interactions to be homogeneous ($\chi^2_{(5)} = 5.25$; $P = 0.7$), therefore all the main items were tested against the common block interaction (Bt). There is significance additive variation between the parental populations (item a), also significant dominance variation (item b). The largest component of (b) is (b_1) indicating a directional dominance component, with some indications of gene asymmetry (b_2), and inconsistent dominance interactions (b_3), with this character however, the more important genetic effects appear to be additive.

The Wr/Vr graph (fig. 3) shows the combined regression line ($b = 0.960 \pm 0.087$; $P = 0.001$), which cuts the Wr axis positively, this indicates that dominance is incomplete. The lack of correlation between the parental means and $(Wr + Vr)$ shows that this dominance must be ambi-directional.

TABLE 6

Mean spikelet length (mm.) for the first (top line) and second genetic replicates

	1	2	3	4	5	6	7	8	9
1.	5.12 5.03	4.85 5.16	6.87 7.05	6.53 6.66	7.07 7.15	6.42 6.76	6.22 6.32	6.50 6.46	5.07 5.17
2.	5.19 5.26	5.30 5.41	6.39 6.78	6.53 6.76	6.58 6.62	6.53 6.82	5.67 6.05	6.10 6.35	5.60 5.86
3.	6.50 6.60	6.85 7.15	6.86 7.18	7.47 7.83	7.16 7.40	6.56 6.64	6.19 6.47	7.21 7.35	6.35 6.42
4.	6.31 6.66	6.25 6.46	7.98 8.22	7.89 8.19	7.86 8.19	6.86 7.36	6.70 6.96	6.65 6.67	6.54 6.72
5.	5.91 5.96	6.70 6.93	6.65 6.88	8.08 8.11	7.80 8.08	6.71 7.01	6.97 7.09	6.96 7.08	6.40 6.61
6.	6.35 6.41	6.62 6.94	7.06 7.30	7.11 7.37	6.79 7.15	6.53 6.50	6.21 6.44	6.74 7.04	5.79 5.98
7.	6.32 6.51	6.10 6.18	5.83 5.95	6.59 6.79	6.56 6.62	6.37 6.68	5.01 5.12	5.85 6.15	5.97 6.17
8.	6.21 6.31	6.13 6.28	7.54 7.80	6.68 6.88	6.83 6.97	6.75 6.90	5.76 6.06	5.43 5.54	6.44 6.64
9.	5.15 5.32	5.83 6.02	6.20 6.64	6.64 6.94	6.57 6.91	6.50 6.62	6.05 6.48	6.18 6.39	5.67 5.84

The $W'r/Wr$ graph (fig. 3) has a similar distribution of points and a regression ($b = 0.349 \pm 0.073$; $P = 0.001$) which is significantly different from the theoretical line of $+0.5(t_{(7)} = 0.01)$. This displacement of all the array points below the theoretical line suggests the occurrence of gene asymmetry.

Analysis of parental means (table 7a) shows that the parental genes have different additive effects which differ between replicates, but there are no significant differences in the expression of dominance effects between replicates, although there is a parents $\times$ blocks interaction which is highly significant, indicating differences of additive gene effects over blocks.

TABLE 7a

Analysis of parental means

Item	D.F.	M.S.	V.R.
Parents	8	8.362	269.74***
Blocks	2	0.064	2.06 N.S.
Genotype replicates	1	0.270	8.71**
Blocks $\times$ Gen. replicates	2	0.032	1.03 N.S.
Parents $\times$ Gen. replicates	8	0.029	—
Parents $\times$ blocks	16	0.227	7.32***
Error	16	0.031	
Total	53		

* $P = 0.05$; ** $P = 0.01$; *** $P = 0.001$.

FIG. 3.— Wr/Vr and $W'r/Wr$ graphs for spikelet length.

TABLE 7b

Analysis of variance of W_r and V_r for spikelet length

Item	D.F.	M.S.	V.R.
Genetic replicates	1	0.445	—
Arrays	8	9.501	7.95***
Dominance	1	19.746	16.53***
Gen.reps. $\times$ arrays.	8	0.156	—
Gen.reps. $\times$ dom.	1	0.008	—
Arrays $\times$ dom.	8	1.140	0.95 N.S.
Gen.reps. $\times$ arrays $\times$ dom.	8	0.041	—
Error	71	1.195	—
Total	107		

*P = 0.05; **P = 0.01; ***P = 0.001.

The analysis of W_r and V_r (table 7b) shows that there are differences in dominance between the parents (Arrays M.S.) also that mean dominance is not complete (Dominance M.S.). There are no significant differences in the expression of dominance effects between replicates.

(iv) *Lemma length*

Genotype replicates were not recorded in this, or the following characters, means are presented in table 8, and Hayman's analysis in table 9. Bartlett's

TABLE 8

Mean lemma length (mm. $\times$ 2)

	1	2	3	4	5	6	7	8	9
1.	6.63	8.30	9.07	9.60	8.78	8.90	8.05	8.48	7.96
2.	8.11	8.83	9.34	10.31	9.72	10.18	9.21	9.34	9.10
3.	9.89	10.19	11.39	11.60	10.84	10.73	9.55	10.67	10.78
4.	9.71	10.58	11.16	12.64	11.30	11.58	10.42	10.84	10.64
5.	8.17	10.54	10.97	11.78	11.07	10.85	9.86	10.22	10.00
6.	9.28	10.80	10.70	11.72	10.90	10.94	9.39	10.61	10.47
7.	7.90	9.72	9.36	10.49	9.44	9.56	7.96	9.08	9.08
8.	8.33	9.83	10.82	10.96	9.75	10.46	9.00	8.88	10.37
9.	8.42	9.55	10.30	10.80	10.17	10.31	9.34	10.83	9.53

test shows the error mean square to be homogeneous ($\chi^2_{(5)} = 4.5$; $P = 0.5$), therefore all items were tested against the common error item (Bt). There are significant additive genetic differences between the parental populations (item a) also a significant dominance component (item b). The (c) item indicating the presence of consistent reciprocal differences is also significant. The size of the (a) item suggests however that most of the genetic variation is additive.

TABLE 9
Hayman's analysis of variance for lemma length

Item	D.F.	M.S.	V.R.
<i>a</i>	8	32.586	187.27***
<i>b</i> ₁	1	0.671	3.86 N.S.
<i>b</i> ₂	8	0.843	4.84***
<i>b</i> ₃	27	0.517	2.97**
<i>b</i>	36	0.594	3.41***
<i>c</i>	8	0.655	3.76**
<i>d</i>	28	0.114	0.65 N.S.
Total	80		
<i>B</i>	2	0.395	2.27
<i>Bt</i>	160	0.174	
Total	242		

*P = 0.05; **P = 0.01; ***P = 0.001.

The Wr/Vr graph (fig. 4) is fitted with a joint regression line ($b = 1.051 \pm 0.168$; $P = 0.001$) as the reciprocal regressions are not significantly different ($P = 0.20$). The regression cuts the Wr axis positively, indicating incomplete dominance. The lack of correlation between the parental means and $(Wr + Vr)$ shows this to be ambidirectional. The $W'r/Wr$ graph also shows a similar distribution of parental points, the regression ($b = 0.0373 \pm 0.041$; $P = 0.001$) is significantly different from $+0.5$ ($t_{(7)} = 0.01$).

(v) *Awn length*

The means are presented in table 10 and Hayman's analysis in table 11. The error mean squares are homogenous ($\chi^2_{(5)} = 4.7$; $P = 0.5$), therefore all items were tested against the common error (Bt). There is significant additive genetic variation between the populations (item *a*), also significant dominance variation (item *b*). All the components of (*b*) are significant,

TABLE 10
Mean awn length (mm. $\times$ 2)

	1	2	3	4	5	6	7	8	9
1.	0.64	1.12	1.06	1.13	0.95	0.93	0.82	0.76	0.80
2.	1.07	1.38	1.20	1.08	1.17	1.41	1.41	1.17	1.19
3.	1.19	1.32	1.58	1.28	1.25	1.38	1.11	1.20	1.34
4.	0.98	1.18	1.45	1.22	1.05	1.05	1.12	0.96	1.11
5.	0.72	1.48	1.20	1.22	1.24	1.17	1.22	1.01	1.05
6.	1.09	1.61	1.35	1.01	1.24	1.44	1.07	0.84	1.22
7.	0.75	1.48	1.12	1.19	1.03	1.00	0.97	1.05	1.11
8.	0.68	1.34	1.26	1.02	0.92	1.13	1.02	1.43	1.32
9.	0.89	1.24	1.22	1.19	1.15	1.17	1.17	1.37	0.96

the largest (b_1) indicating the presence of directional dominance, (b_2) gene asymmetry, and (b_3) inconsistent dominance interactions. There is also a small (*c*) item suggesting the presence of some consistent reciprocal effects.

FIG. 4.— W_r/V_r and W'_r/W_r graphs for lemma length.

TABLE 11

Hayman's analysis of variance for awn length

Item	D.F.	M.S.	V.R.
<i>a</i>	8	0.720	42.35***
<i>b</i> ₁	1	0.133	7.82**
<i>b</i> ₂	8	0.101	5.94***
<i>b</i> ₃	27	0.080	4.70***
<i>b</i>	36	0.086	5.06***
<i>c</i>	8	0.034	2.00*
<i>d</i>	28	0.022	1.29 N.S.
Total	80		
<i>B</i>	2	0.005	
<i>Bt</i>	160	0.017	
Total	242		

P* = 0.05; *P* = 0.01; ****P* = 0.001.

The Wr/Vr graph (fig. 5) shows no significant regression, both male and female points are scattered across the graph in a random manner, indicating that the disturbance is due to non-allelic interaction. The correction of the ordinate and abscissa for the environmental component (E_2) reduces the level of dominance expressed only slightly. The $W'r/Wr$ graph which is relatively undisturbed by non-allelic interaction, shows a significant joint regression ($b = 0.316 \pm 0.108$; $P = 0.05$), with the array points of *castellata* (9) and *lusitanica* (3) some distance below the theoretical line, suggesting that these two populations have genes not present in the other samples.

(vi) *Lemma apex*

The character is portrayed in figure 6, means are presented in table 12, and Hayman's analysis in table 13. Each main item was tested against its own error mean square, as these were heterogeneous ($\chi^2_{(5)} = 39.4$; $P = 0.001$). There is significant additive genetic variation between the parental populations (item *a*), also significant dominance variation (item *b*). Only two components of dominance are significant, those indicating gene asymmetry (*b*₂) and inconsistent dominance interactions (*b*₃). Item (*d*) is also significant, indicating the presence of inconsistent reciprocal differences.

TABLE 12

Lemma apex notch (mm. $\times$ 2)

	1	2	3	4	5	6	7	8	9
1.	0.035	0.140	0.193	0.107	0.146	0.088	0.164	0.108	0.257
2.	0.193	0.330	0.295	0.084	0.257	0.150	0.174	0.219	0.204
3.	0.204	0.241	0.257	0.208	0.174	0.285	0.215	0.226	0.240
4.	0.064	0.064	0.166	0.093	0.080	0.061	0.031	0.087	0.079
5.	0.244	0.208	0.211	0.058	0.196	0.076	0.200	0.114	0.149
6.	0.035	0.264	0.222	0.065	0.060	0.122	0.096	0.137	0.096
7.	0.185	0.219	0.210	0.044	0.249	0.098	0.236	0.169	0.314
8.	0.088	0.185	0.254	0.047	0.133	0.143	0.203	0.460	0.262
9.	0.160	0.235	0.250	0.104	0.159	0.097	0.237	0.280	0.199

FIG. 5.— Wr/Vr and $W'r/Wr$ graphs for awn length, corrected ordinate and abscissa included as dashed lines.

TABLE 13

Hayman's analysis of lemma apex notch

Item	D.F.	M.S.	V.R.
<i>a</i>	8	0.1164	64.31***
<i>b</i> ₁	1	0.0617	9.82 N.S.
<i>b</i> ₂	8	0.0294	13.86***
<i>b</i> ₃	27	0.0111	3.97***
<i>b</i>	36	0.0166	6.04***
<i>c</i>	8	0.0009	0.57 N.S.
<i>d</i>	28	0.0036	2.30**
Total	80		
Block	2	0.0009	
<i>Ba</i>	16	0.0018	
<i>Bb</i> ₁	2	0.0063	
<i>Bb</i> ₂	16	0.0021	
<i>Bb</i> ₃	54	0.0028	
<i>Bb</i>	72	0.0027	
<i>Bc</i>	16	0.0016	
<i>Bd</i>	56	0.0016	
Total	242		

P* = 0.05; *P* = 0.01; ****P* = 0.001.

The *Wr/Vr* graph (fig. 7) shows a significant joint regression ($b = 0.956 \pm 0.118$; *P* = 0.001), which cuts the axes at their intersection, this indicates full dominance for the character. There is no correlation between the parental means and (*Wr* + *Vr*) therefore dominance is ambi-directional.

FIG. 6.—Lemma, showing apex forms.

The population of subspecies sample *santai* (8) stands out as having the highest number of recessive genes, also the deepest apical lobe, the rest of the population points are concentrated toward the lower (dominant) end of the graph. The *W'r/Wr* graph (fig. 7) shows a similar distribution of points, the regression line ($b = 0.218 \pm 0.015$; *P* = 0.001) deviates significantly from the theoretical ($t_7 = 0.001$), this is caused entirely by the population

sample of *santai* (8), which indicates that the population has a large number of genes not distributed throughout the other population samples considered.

FIG. 7.— Wr/Vr and $W'r/Wr$ graphs for lemma apex.

4. DISCUSSION

(i) *Stability of genetic replicates*

The data for panicle length show that there is a significant variation of additive gene expression in the parental populations in successively produced

heads, caused by micro-environmental and developmental effects. This variability is reflected in the response of the progeny of these particular parents, which from their position on the Wr/Vr graphs appear to be the populations containing the more heterozygous and recessive gene combinations, *i.e.* stability tends to be dominant as shown by Paxman (1956) and Broadhurst and Jinks (1966). The presence of morphological variability did not however, alter the overall level of dominance in the arrays, only the relative expression of the proportion of dominant and recessive genes in the parents concerned, depending upon the panicle selected. The degree of dominance expressed is incomplete, and directional for short panicles. This is not a geographical trend, for although some northern populations do have longer panicles, this is only one aspect of floral adaptation to a cooler, damper, climate.

The number of florets per spikelet also show differences due to different environmental effects and/or developmental variations occurring between the two genotype replicates, these differences are not very great in the parental populations, but assume more importance in the F_1 progenies, particularly those of *ibizensis* (7) suggesting either a disturbed polygenic balance or a more rapid rate of differentiation for this character. The distribution of the array points of the two genotype replicates indicates that considerable non-allelic interaction, probably of the complementary type, is occurring, giving a pattern of spurious overdominance. Spikelet length shows no significant within plant differences of gene expression, and the pattern of inheritance is also much easier to interpret than that of the previous character. Dominance is incomplete and ambi-directional, indicating that selection has presumably been favouring an intermediate optimum in each population, although it is possible that dominance could be in opposite directions in different populations giving an over-all picture of ambi-directional dominance when they are crossed.

In the characters discussed above, the stability of gene expression, *i.e.* the response to fluctuations of both the internal and external environment, varies between different populations. This physiological developmental threshold determining gene expression within the individual also differs with the characters concerned; in *Dactylis* therefore, as in *Lycopersicon esculentum* (Williams, 1960) and *Nicotiana rustica* (Paxman, 1956), this variation is specific to individual characters in relation to particular environmental influences. Both the above authors showed that unrelated characters within a plant had unrelated stabilities; Paxman (1956) also showed that in *Nicotiana*, characters closely related developmentally (flower parts) did possess related stabilities. This is not so in *Dactylis* with regard to the two spikelet characters, for mean floret numbers varies significantly, appearing to experience competition (probably due to density effects) depending upon position in the panicle, while mean spikelet size does not show this effect.

(ii) *Inheritance pattern of correlated characters*

Table 14 shows the phenotypic correlations between the characters studied within the nine parental populations. There is a large and highly significant correlation between the number of florets and spikelet length in most populations, and weak correlations between spikelet length and lemma length, and the number of florets and lemma length in the populations of

santai (8) and *castellata* (9). Whereas the Wr/Vr graph for number of florets shows some form of non-allelic interaction, spikelet length shows incomplete dominance, however the scatter of points on the two graphs is similar, and the developmental association could be caused either by pleiotropism or linkage. If the latter is the case, then this linkage has been selected against in the population of *parthiana* (5) where there is no statistically significant relationship.

The pattern of inheritance for lemma length shows incomplete and ambidirectional dominance, with a large consistent difference between the reciprocals. The positive phenotypic correlation between lemma and awn length is present only in two-thirds of the populations used, and is apparently an ecological rather than a developmental relationship, as the populations lacking this correlation may have either very short or very long awns.

TABLE 14
Intra-population correlations for the parents

Populations Characters	1	2	3	4	5	6	7	8	9
Panicle L./ Spikelet L.	-0.02	-0.57*	-0.34	0.42	0.20	-0.06	0.41	-0.38	-0.20
Panicle L./ Lemma L.	-0.06	0.53*	-0.15	0.70***	0.09	0.12	-0.14	-0.07	0.23
Panicle L./ Awn L.	-0.43	0.65**	-0.03	0.08	0.03	0.10	0.01	-0.08	0.04
No. Florets/ Spikelet L.	0.90***	0.91***	0.85***	0.79***	-0.28	0.62**	0.77***	0.96***	0.75***
No Florets/ Lemma L.	0.13	-0.48*	0.11	0.17	0.27	0.42	0.46	0.47*	0.47*
No. Florets/ Awn L.	0.30	-0.49*	0.02	0.14	-0.21	0.31	-0.08	0.24	0.43
Spikelet L./ Lemma L.	0.41	-0.44	0.07	0.29	0.22	0.42	0.38	0.49*	0.53*
Lemma L./ Awn L.	0.12	0.63**	0.24	0.54*	0.55*	0.65**	0.53*	0.78***	0.26

There is also no relation between the genetic systems controlling lemma and awn length, the pattern of inheritance of awn length is disturbed by both consistent reciprocal differences and non-allelic interaction, however, the probability is that the interaction is of the duplicate type. The lemma apex character, which was included because of its emphasis in some classifications of the genus, appears to be simply inherited and shows full dominance. Selection has been stabilising and most genes for deeply notched apex, which are recessive, are asymmetrically distributed in population 8 (*santai*).

Little direct evidence for the genetic control of correlated characters is available, although Cooper (1960) concluded from his study of two populations of *Lolium perenne*, that linkage was responsible. It is possible that both pleiotropic gene effects and linkage of polygenic systems controlling different characters are effective in *Dactylis*, although linked polygenes are probably the more likely explanation (Mather, 1943).

(iii) *Conclusions*

The characters considered above, and in the previous papers (Parker, 1968 *a, b*) were selected for an experimental study in breadth to analyse the

nature of genetic differences between divergent populations within a species. The various subspecies populations of *Dactylis* can be distinguished morphologically, but analysis of hybrids shows that they cannot be consistently and clearly distinguished in genetical terms with regard to characters other than those closely associated with reproductive fitness. Fisher's fundamental theorem of natural selection states that the rate of change of fitness in a population is proportional to the additive genetic variance remaining in it (Fisher, 1930). Therefore the population which has reached equilibrium with its present environment should have little or no additive genetic variance in characters closely connected with fitness, although genotypes may vary

TABLE 15

Expressions of dominance, cytoplasmic effects and nuclear/cytoplasmic interactions for all characters used in the study. c only = c significant; c = d, c and d equally significant; c > d, c is significantly greater than d, but both are significant

Character	Wr/Vr Analysis	Hayman's Analysis
1. Seed set	Spurious overdominance, gene asymmetry	F ₁ c; F ₂ —
2. Seed germination	Spurious overdominance, gene asymmetry	F ₁ c; F ₂ —
3. Fertility index	Directional overdominance, gene asymmetry	F ₁ c; F ₂ —
4. Growth rate	Complementary gene interaction	c = d
5. Internode length	Incomplete directional dominance	c = d
6. Leaf six area	Incomplete directional dominance; some interaction	c only
7. Tiller number	Incomplete ambi-directional dominance	c = d
8. Emergence date	Incomplete directional dominance	c = d
9. Flag leaf area	Incomplete ambi-directional dominance	c = d
10. Summer leaf area	Incomplete directional dominance	c only
11. Leaf dry weight	Gene interaction	c only
12. Chlorophyll content (a)	Incomplete ambi-directional dominance	c > d
Chlorophyll content (b)	Incomplete ambi-directional dominance	c > d
13. Panicle length	Incomplete directional dominance	—
14. Floret number	Complementary gene interaction	c only
15. Spikelet length	Incomplete ambi-directional dominance	—
16. Lemma length	Incomplete ambi-directional dominance	c only
17. Awn length	Duplicate gene interaction	c only
18. Lemma apex	Complete ambi-directional dominance; gene asymmetry	d only

with regard to their over-all fitness. On the other hand, where moderate changes of the phenotype can have no effect on reproductive fitness, the hereditary variance will be almost entirely additive, the results (table 15) show, within experimental limits, that this occurs in *Dactylis*. The characters concerned with fertility (nos. 1, 2, 3, table 15) present a pattern of genetic variation expressing spurious overdominance. In the interpopulation hybrids there is obviously considerable disturbance of the relational balance of the polygenic systems controlling fertility, which appear to be independently balanced, with natural selection acting directionally towards high viability within each population. Some other characters of apparent importance with regard to viability and fertility of the parental populations in the different ecological niches they occupy, such as growth rate, number of florets, and awn length (nos. 4, 14, 17, table 15) also show gene interaction and dominance. The effect of natural selection has been to change these characters in different directions, being initially disruptive, then stabilising towards

the optimum favoured in each environment. This has resulted in the present genetic polymorphism. The remaining eleven characters under consideration are controlled by polygenic systems of low dominance, and low interaction, on which natural selection has acted sometimes in a stabilising manner, sometimes directionally, these characters apparently have only a slight effect on viability.

The large amount of consistent and inconsistent interaction which occurs throughout most of the analyses is of interest, for the parents although perennials, reproduce by seed and live in fairly open or disturbed habitats. There is clearly strong selection for mutual balance of parental genes and cytoplasm in each environment, for both consistent and inconsistent reciprocal effects are apparent in the F_1 plants through to the adult stage. Analysis of the seed characters (Parker, 1968a), also analysis of some other characters in the F_2 generation (Parker, unpub.), show that these effects do not go beyond the F_1 .

The consistent reciprocal effect is dependent on differences in the embryonic environment which are probably due to maternal nuclear products in the cytoplasm, the other effect is a differential interaction between the F_1 hybrid nucleus and the parental cytoplasm in various crosses. Consistent effects in the F_1 are a fairly common phenomenon, not only in hybrids between reproductively isolated populations, but also hybrids in sexually reproduced perennial cultivars such as *Lucerne* (Lamprecht, 1964), they are not however expressed by all individual characters, some genes are apparently insensitive to the cytoplasmic milieu. This also applies to the analysis of within-plant variability, this shows clearly that in some characters only, and in some parents only, there was an environmental effect which was largely one of increasing or reducing the differences in expression of dominance of the various genes, this variation is presumably adaptive in the original habitats of the populations concerned. This study shows that large, usually non-allelic, genetic differences are found only where the measured characters are apparently directly involved with viability and "fitness". Thus these spatially isolated populations have accumulated adaptive genetic differences often as separate components of sets of dominant genes with complementary and duplicate interactions, and it is probable that these interactions are also the cause of the incipient reproductive isolation observed (Parker and Borrill, 1968). It is apparent that the stresses of natural selection on the various major morphological and physiological characters of the nine subspecies populations have, during the period of their isolation, considerably altered their fundamental genetic characteristics.

5. SUMMARY

1. The investigation of the genetic variation present in some populations of diploid *Dactylis* was concluded with a study of panicle and floret characters.

2. Analysis of the hybrids showed that the pattern of variation was largely additive, with some characters controlled by polygenic systems characterised by large amounts of non-allelic interaction.

3. Analysis of genotype replicates showed that some characters were affected by non-heritable variation to a certain degree.

4. The results are discussed, and some conclusions drawn from the complete set of analyses.

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