Inheritance of Leaf and Head Resistance of Winter Wheat to *Septoria nodorum* in a Diallel Cross

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We gratefully acknowledge the technical assistance of Heidi Buch, P. Streckeisen, and Rosmarie Zäch, as well as valuable discussions with G. Popow.
Accepted for publication 25 February 1987.

ABSTRACT

Fried, P. M., and Meister, E. 1987. Inheritance of leaf and head resistance of winter wheat to *Septoria nodorum* in a diallel cross. Phytopathology 77:1371-1375.

A diallel analysis of four parents of similar heading date and plant height but differing in resistance to *Septoria nodorum* on leaves and heads (as estimated by area under the disease progress curves) showed that the genetic variation is mainly additive. Statistical tests for the nonadditive variation indicated that dominance for susceptibility was significant, but that interaction between nonallelic genes was not. No cytoplasmic effects could be detected. Heritability estimates based on parents and F_1's varied between 0.48 and 0.68. Estimates based on the standard-unit method were 0.30 and 0.26 for flag leaves and heads, respectively. Evidence for independent segregation of the genes controlling head and leaf resistance could be found by correlation analysis between F_2's and F_3's.

Additonal key words: breeding for disease resistance, Septoria nodorum blotch.

Glume blotch of wheat caused by *Phaeosphaeria nodorum* (Müller) Hedjaroude (teleomorph of *Staganospora nodorum* (Berk.) Cast & Germ.; synonym *Septoria nodorum* Berk.) is a widespread wheat disease. It may reduce yield up to 50% (1,3,12,20). Plant breeders and pathologists have worked extensively to incorporate resistance in new cultivars. Disease resistance, however, has often been found to be highly correlated with late-maturing, tall cultivars (4,6,18,21). Scott et al (19) concluded that resistance to glume blotch is under polygenic control and that pleiotropy could explain the genetic association of resistance with height and ear emergence. Brönnimann (5) found high heritability values (66%) for tolerance to the disease. Genotypes were considered tolerant when yield reduction was small after inoculation. Heritabilities in the same range (63% and 52%, respectively) were reported by Rosielle and Brown (17) for Septoria scores on flag leaves and heads. A simpler mode of inheritance of seedling resistance to *S. nodorum* was shown by Frecha (7), who suggested that in the cultivar Atlas 66 resistance is determined by a single dominant gene.

In selecting for resistance to glume blotch of wheat, we (9,10) repeatedly identified genotypes with different quantitative resistance levels on leaves and heads, respectively. This may, of course, have consequences on the breeding scheme and on selection of both parents and offspring. We therefore made an effort to investigate the genetic base of these two characteristics. For this, four breeding lines representing all combinations of leaf and head resistance were selected. The lines were of almost equal height and nearly identical heading date to overcome the mentioned parental association between resistance to Septoria and plant height and/or time of ear emergence (19). These lines were then crossed in all possible combinations, including reciprocals, to study the mode of inheritance, to test for maternal effects, and to investigate interactions between genes governing leaf and head resistance.

MATERIALS AND METHODS

Parents (P_2, P_3, P_4, and P_6) were chosen based on the area under the disease progress curves (ADPC) for leaves and heads, measured during the summers of 1978 and 1979. Their response to *S. nodorum* infection in 1981 is shown in Figure 1. Even though resistance is quantitative, we have, for easier understanding, assigned a + when the level of resistance was high on the respective plant part and a − when it was low. Parent P_2, designated (−−), is highly susceptible on leaves and heads. P_3 (−+) shows high susceptibility on the leaves and good resistance on the heads, P_4 (+−) shows the reverse of P_3, and P_6 (++) has good resistance on both plant parts. In 1981, average plant heights for the four parents were 109, 120, 103, and 116 cm, and their heading dates were, on the average, 7, 6, 6, and 8 June, respectively.

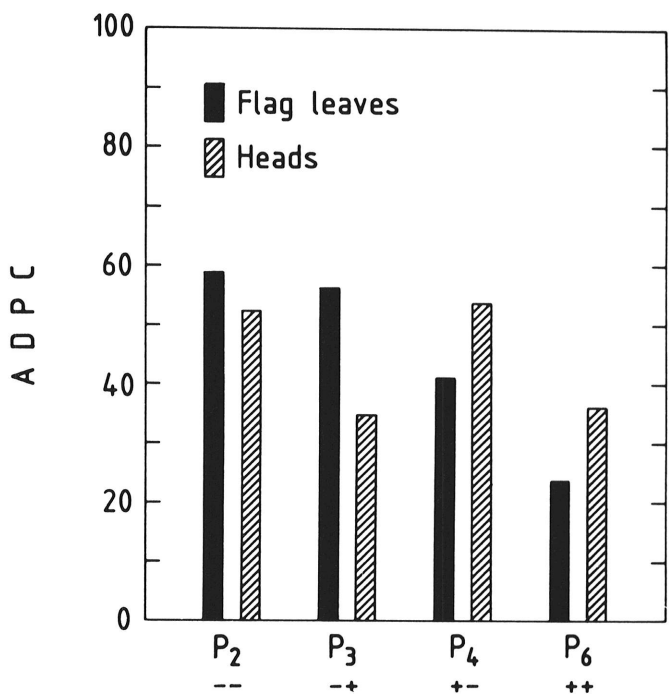

Fig. 1. Areas under the disease progress curves (ADPC) of four winter wheat parents P_2, P_3, P_4, and P_6 and their designation (+ = resistant, − = susceptible) according to the response to infection with *Septoria nodorum* on the respective plant part.

The pedigree of P_2 is Moisson/4*Probus//71803, and 71803 = A273/Weique//Champlein; P_3 and P_4 were selected from a cross between Kawkas and 71803, and P_6 = Danubian Roman//Heine VII/71803. The four parents were crossed in all possible combinations to make up a diallel series.

TABLE 1. Diallel table of the areas under disease progress curves for flag leaves (ADPCFL) and heads (ADPCHD) of winter wheat after inoculation with *Septoria nodorum* for the parents (diagonal), F_1 crosses (above diagonal), and F_1 reciprocal crosses (below diagonal)

Male parent	Female parent				Mean
	P_2 (−−)[a]	P_3 (−+)	P_4 (+−)	P_6 (++)	
ADPCFL					
P_2 (−−)	56.6	71.8	58.7	57.9	61.3
P_3 (−+)	69.3	57.4	61.7	48.1	59.1
P_4 (+−)	62.3	61.6	40.6	37.3	50.4
P_6 (++)	55.7	49.8	34.1	22.5	40.5
Mean	61.0	60.1	48.8	41.4	
ADPCHD					
P_2 (−−)	52.3	52.8	53.3	49.0	51.8
P_3 (−+)	46.6	36.4	47.5	42.0	43.1
P_4 (+−)	48.5	51.3	52.9	39.9	48.3
P_6 (++)	49.9	39.3	41.7	36.0	41.9
Mean	49.3	45.1	48.8	41.7	

[a] + = Resistant, − = susceptible on flag leaves and heads, respectively.

TABLE 2. Analysis of variance of the diallel data for areas under the disease progress curves for flag leaves (ADPCFL) and heads (ADPCHD) of winter wheat inoculated with *Septoria nodorum*

Variable	Source	df	MS	*F* calc.	*P*
ADPCFL	Blocks	4	347.19	3.84	0.0076
	Crosses	3	1748.47	19.33	<0.0010
	Reciprocals	3	1795.87	19.86	<0.0010
	Crosses × reciprocals	9	294.34	3.25	0.0028
	Error	60	90.45		
	Maternal effects	6	17.01	0.18	>0.5
ADPCHD	Blocks	4	887.57	23.03	<0.001
	Crosses	3	254.61	6.61	<0.001
	Reciprocals	3	438.76	11.38	<0.001
	Crosses × reciprocals	9	81.89	2.12	0.041
	Error	60	38.54		
	Maternal effects	6	38.37	0.99	>0.5

F_1 and F_2 seed were produced in the greenhouse after vernalization of parents and F_1 seedlings for 42 days at 4 C. The F_2 seed was derived from randomly selected and selfed F_1 plants. For each of the 28 treatments (four parents and all their combinations, including reciprocals in F_1 and F_2 generations) about 160 single plants were raised in 6-cm Jiffy pots during the winter of 1980–1981. In the spring, 100 of these seedlings were transplanted into the field. The field experiment (Experiment I) was laid out as a randomized complete block design (RCBD) in which every treatment was replicated five times. In each plot, 20 seedlings were planted at a distance of 30 cm within the row. Border rows with cultivar Zenith (medium resistant) were drill-seeded at 23-cm row spacing to simulate a more realistic environment surrounding the test plants and to minimize interplot interactions.

The plants were inoculated twice, at the late-boot stage and after heading of two to four tillers per plant. A spore suspension of 500 L/ha was applied with a tractor with boom and nozzles. Spore concentrations were adjusted to 5×10^6 viable spores per milliliter for the first inoculation date and to 10^6 spores per milliliter for the second. Inoculum was produced from a single-spore isolate on autoclaved wheat kernels (3). The percentage of area covered by *S. nodorum* was estimated three separate times on all leaves, on the flag leaf only, and on the heads, at the following growth stages (22): 61 (6 June), 63 and 65 for all leaves; 65 (1 July), 71 and 72 for flag leaves; 71 (8 July), 72 and 73 (24 July) for the heads. The ADPCs for all leaves (ADPCAL), for flag leaves (ADPCFL), and for the heads (ADPCHD) were calculated and used in the analysis of the data.

Experiment II was carried out during 1981–1982 with the parents and the F_3's from the F_2's of the nonreciprocal crosses in 1981. From each of the 600 F_2 plants (100/cross) about 20 individual F_3 seedlings were raised during the winter in Jiffy pots. In the spring five seedlings per F_3 family were randomly chosen and transplanted next to each other in a field plot. The layout was again a RCBD with five replications and with border rows and spacings as in Experiment I. As an insurance, an identical RCBD, with different randomization, was planted in an adjacent field. Because no irregularities were observed in either field, the two disease ratings of each F_3 family were averaged for the genetic analysis. We inoculated Experiment II at the same growth stages (GS) and with the same isolate and equipment as in 1981. ADPCs were calculated on disease assessments as follows: five for ADPCAL at GS 59, 61, 71, 72, and 73; three for ADPCFL at GS 71, 73, and 81; and four for the heads at GS 72, 73, 81, and 83.

We analyzed the data for the parents and the F_1s according to the diallel method described by Mather and Jinks (13) and for the F_2 and F_3 data by the standard-unit method of Frey and Horner (8). We omit detailed results obtained with the ADPCAL because they were similar to those obtained with the ADPCFL.

TABLE 3. Half diallel tables of the areas under disease progress curves for flag leaves (ADPCFL) and heads (ADPCHD) of winter wheat inoculated with *Septoria nodorum*

Male parent	Female parent				Mean	Covariance Wr	Variance Vr
	P_2 (−−)[a]	P_3 (−+)	P_4 (+−)	P_6 (++)			
ADPCFL							
P_2 (−−)	56.62	70.56	60.54	56.78	61.125	55.099	42.845
P_3 (−+)		57.44	61.61	48.93	59.635	110.507	80.890
P_4 (+−)			40.56	35.71	49.605	209.642	179.526
P_6 (++)				22.52	40.985	240.296	227.129
Mean						153.886	132.598
ADPCHD							
P_2 (−−)	52.34	49.70	50.89	49.46	50.598	11.030	1.740
P_3 (−+)		36.40	49.71	40.65	44.115	60.881	44.675
P_4 (+−)			52.86	40.79	48.563	37.086	28.538
P_6 (++)				35.96	41.715	36.921	31.698
Mean						36.479	26.663

[a] + = Resistant, − = susceptible on flag leaves and heads, respectively.

RESULTS

The inoculation of the different genotypes resulted in the following disease severity levels at the last reading dates: flag leaves 15–100%, heads 5–100%. The ADPCs were normally distributed, and the mean values are presented in Table 1. The large variation among blocks found in the analysis of variance of Experiment I (Table 2) is primarily due to the variation observed in blocks I and IV. These were located adjacently in the front part of the field, where drought caused damage to some of the seedlings after transplantation from the greenhouse. The mean squares for crosses and reciprocals are significant, providing evidence for additive genetic variation among the genotypes tested. The means as well as the mean squares (Tables 1 and 2) are of the same magnitude for both crosses and reciprocals. This suggests the absence of cytoplasmic (maternal) effects, which is further supported by the *F* test shown in Table 2.

The interaction crosses × reciprocals is also statistically significant for both flag leaves and heads. This indicates that the differences among the progenies are not wholly attributable to purely additive genetic variation, but that there must be also some nonadditive variation in the form of dominance or interaction between nonallelic genes.

To test whether dominance is the sole component of the nonadditive variation, analyses of variance (Vr) and covariance (Wr) were performed on the pooled means of crosses and reciprocals, and the Wr's on Vr's were regressed on the data from the half diallel tables (Table 3). Parents with the highest frequency of dominant alleles have the smallest Vr and Wr, thus, their relative position along the Vr/Wr regression line reflects the frequency of dominant alleles. Sorting the parents used in these crosses in order of decreasing dominance gave the following sequences for ADPCFL: P_2, P_3, P_4, P_6; and for ADPCHD: P_2, P_4, P_6, P_3. Thus, susceptibility on both flag leaves and heads is dominant. The ranking is different for the two characteristics measured, but it follows the disease rating for the parents on the respective plant part. The Student's *t* test for deviation from one of the regression lines was also not significant: *t* calc. = 0.015 for ADPCFL and 0.6391 for ADPCHD, the *t* value for $P = 0.05$ being 4.303. This indicates that dominance is present. It does not, however, exclude nonallelic interactions. To further separate these two effects, an analysis of variance on Wr + Vr and one on Wr − Vr were carried out, based on the family means in Table 1. The mean squares were not significant for Wr − Vr, which indicates that nonallelic interactions are small or absent ($P = 0.63$ and $P = 0.66$ for ADPCFL and ADPCHD, respectively). Therefore, dominance seems to account for the major proportion of the nonadditive variation, as indicated by the significant mean squares Wr + Vr for ADPCFL ($P = 0.019$) and to a smaller degree for ADPCHD ($P = 0.409$).

The fixable variation D and the dominance component H were also calculated; estimates of the heritable and nonheritable portions for D and H are given in Table 4. The dominance ratio ($\sqrt{H/D}$), an estimate of the average level of dominance, is 0.86 for ADPCFL and 0.40 for ADPCHD. Heritability values varied between 0.48 and 0.68 and were higher for ADPCFL than for ADPCHD. Heritability estimates by the standard-unit method (8), which are based on F_2 and F_3 family means, were lower (Table 5). The difference may, at least in part, be due to possible genotype × environment interactions because the F_2's and F_3's were grown in two different years.

The correlations between ADPCFL and ADPCHD within F_3 families were low (Table 5), except for the cross $P_2 \times P_3$ (both parents susceptible on the leaves). This suggests independent segregation of the two traits. Correlations would be high especially in the cross P_3 (−+) × P_4 (+−), if genes for leaf and head resistance were linked or if they showed pleiotropic effects.

The means for ADPCFL and ADPCHD of the parents, the F_1's and F_2's (including reciprocals) are graphically presented in Figure 2A and B. Figure 2C and D shows the corresponding means of the F_3's. The pattern fits well the additive-dominance model suggested by the diallel analysis. The dominance of susceptibility, which is particularly evident in the crosses with the overall susceptible parent P_2, is also confirmed.

DISCUSSION

All genetic studies so far have shown that *S. nodorum* resistance on adult plants is polygenically inherited (5,11,14,16,19). The genetic analysis of our wheat crosses shows that the additive-dominance model of Mather and Jinks (13) is adequate to explain the data and confirms earlier findings. Heritability estimates based on the parents and F_1's varied between 0.48 and 0.68 and are of the same magnitude as reported by Brönnimann (5) and by Rosielle and Brown (17). On the other hand, the heritabilities estimated by the standard-units method of Frey and Horner (8) are much lower and averaged only 0.30 and 0.26 for ADPCFL and ADPCHD, respectively. The slow progress made in the past in breeding for resistance to *S. nodorum* and the limited availability of resistant germ plasm in acceptable agronomic background (6) suggest that these low heritability estimates may be closer to reality than those calculated from the diallel analysis using the parents and F_1's.

TABLE 4. Components of variation on genetic parameters in the diallel and heritability estimates for areas under the disease progress curves for flag leaves (ADPCFL) and heads (ADPCHD) of winter wheat inoculated with *Septoria nodorum*

	ADPCFL		ADPCHD		
Component	Nongenetic	Genetic	Nongenetic	Genetic	Expectation
$\underline{V_p}$[a]	18.09	252.84	7.71	82.24	D
$\overline{W_r}$[b]	4.52	149.36	1.93	34.55	1/2 D
$\underline{V_{\bar{r}}}$[c]	2.83	85.78	1.11	15.35	1/4 D
$\overline{V_r}$[d]	11.31	121.29	4.82	21.84	1/4 (D + H)

	ADPCFL	ADPCHD
Additive component of variation $D = 4/7\ (V_p + \overline{W}_r + V_{\bar{r}})$	15.71	75.51
Dominance component of variation $H = 4\overline{V}_r - D$	14.83	11.75
Average level of dominance $\sqrt{H/D}$	0.86	0.40
Narrow sense heritability 1/2 / (1/2D + 1/4H + E)	0.50	0.48
Broad sense heritability (1/2D + 1/4H) / (1/2D + 1/4H + E)	0.68	0.51

[a] $\underline{V_p}$ = Variance of the parent lines.
[b] $\overline{W_r}$ = Mean covariance of arrays.
[c] $\underline{V_{\bar{r}}}$ = Variance of array means.
[d] $\overline{V_r}$ = Mean variance of arrays.

TABLE 5. Correlations between F_2's and F_3's and within F_3's of areas under disease progress curves for flag leaves (ADPCFL) and heads (ADPCHD) of six winter wheat crosses with parents differing in the level of leaf and head resistance to *Septoria nodorum*

	Correlation coefficients between			
	F_2's and F_3's		ADPCFL and ADPCHD	Observations
Cross	ADPCFL	ADPCHD	within F_3's	*n*
$P_2 \times P_3$ (−− × −+)[a]	0.37	0.47	0.51	97
$P_2 \times P_4$ (−− × +−)	0.25	0.15	0.18	96
$P_2 \times P_6$ (−− × ++)	0.15	0.12	0.04	96
$P_3 \times P_4$ (−+ × +−)	0.51	0.30	−0.02	92
$P_3 \times P_6$ (−+ × ++)	0.11	0.15	−0.05	97
$P_4 \times P_6$ (+− × ++)	0.43	0.41	0.02	91
Mean	0.30	0.26	0.14	95

[a] + = Resistant, − = susceptible on flag leaves and heads, respectively.

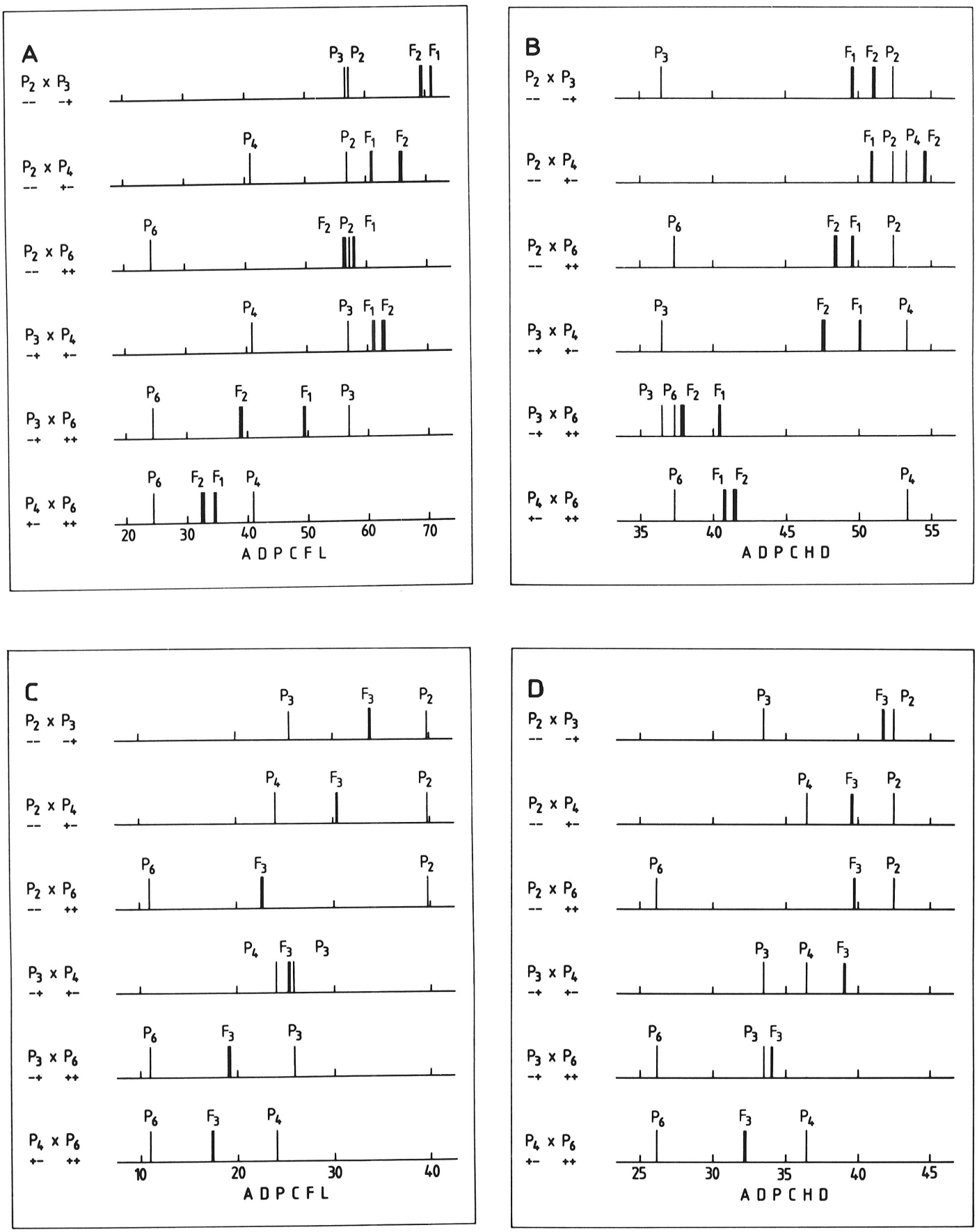

Fig. 2. Mean values of the areas under disease progress curves for flag leaves (ADPCFL) and heads (ADPCHD) of four winter wheat parents, P_2, P_3, P_4, and P_6, the F_1's and F_2's **(A, B)** and the F_3's **(C, D)** for all crosses. The parents differed in their level of resistance to *Septoria nodorum* on flag leaves and on the heads (first subscript = leaf reaction, second subscript = head reaction). **A** and **B** are based on data taken in 1982, **C** and **D** in 1983.

Scott et al (19) reported that the variation in resistance was associated with plant height and lateness. In most of their crosses, the mean disease rating on F_2 plants was intermediate between parents. In some of them, however, deviations toward partial dominance for susceptibility or partial dominance for resistance were also observed. Brönnimann (5) found dominance for susceptibility in the F_1's in three out of four crosses with the cultivar Svenno (most susceptible entry), whereas Nelson and Gates' results (16) indicate dominance for resistance because all F_1 glume blotch ratings were lower than those for the more resistant parent in each cross. In our study, the average level of dominance for susceptibility was estimated at 0.86 and 0.40 for ADPCFL and ADPCHD, respectively. Most of this partial dominance probably originates from P_2, the most susceptible entry (Fig. 2). Because all parents used were similar in ear emergence and plant height, the genetic variation in resistance observed is assumed to be independent of these two factors. This, however, does not exclude the possibility of genetic associations in short straw cultivars or in progenies of short and tall cultivars as shown by Scott et al (19). Departure from single autosomal inheritance of resistance had been suggested by Nelson (15) but could not be confirmed (16). Our results show good evidence that there are no maternal effects.

These crosses have shown that both leaf and head resistance are genetically fixed in the selected cultivars. The inheritance of both types of resistance can be sufficiently explained by the additive-dominance model of Mather and Jinks (13). Evidence for independent segregation of genes controlling the two traits is given by the low correlation between ADPCFL and ADPCHD of the F_2's and F_3's and by the different ranking of parents along the Wr/Vr regression line for the two characteristics. This implies absence of pleiotropic effects and separate sets of genes for leaf and head resistance. Even though heritabilities for both are rather low, there is a reasonable chance of breeding and identifying wheat genotypes with resistance to *S. nodorum* on leaves and heads. However, large single plant populations and strong selection pressure are necessary. To avoid selection of late but susceptible genotypes, grouping in the field as indicated by Fried (9) is helpful.

Because a combination of head and leaf resistance is required for a new variety and both seem to be independently inherited, it appears that screening procedures in the field cannot be replaced by tests on seedlings or detached leaves (2) in the laboratory.

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