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The inheritance of resistance to head blight caused by *Fusarium culmorum* in winter wheat

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Summary

Crosses were made among ten winter wheat genotypes representing different levels of resistance to Fusarium head blight to obtain F_1 and F_2 generations. Parents, F_1 and F_2 were inoculated with one strain of *Fusarium culmorum.* Data on incidence of head blight 21 days after first inoculation were analyzed. Broad-sense heritabilities averaged 0.39 and ranged from 0.05 to 0.89 in the individual F_2 families. The joint-scaling test indicated that the inheritance of Fusarium head blight resistance was adequately described by the additive-dominance model, with additive gene action being the most important factor of resistance. With respect to the non-additive effects, dominance of resistance predominated over recessiveness. The number of segregating genes governing resistance in the studied populations was estimated to vary between one and six. It was demonstrated that resistance genes differed between parents and affected resistance differently.

Introduction

Fusarium head blight of wheat (*Triticum aestivum* L.) is a destructive disease in the humid and semi-humid wheat-growing regions of the world. It induces losses in various ways: by adversely affecting yield and grain quality, by mycotoxins in the grain, and by seedling blight as a result of seed infections. In the Netherlands, head blight is mainly caused by two *Fusarium* spp., viz. *F. culmorum* (W.G. Smith) Sacc. (which is the dominant species) and *F. graminearum* Schwabe. Breeding for resistance to Fusarium head blight is hampered by lack of knowledge of the inheritance of the resistance. Studies of the inheritance of the resistance to Fusarium head blight in wheat are scarce and limited to resistance to *F. graminearum.* In various studies the resistance was inherited from partially dominant to over-dominant, and was controlled by two to five genes (Gu, 1983; Yu, 1982; Zhang & Pan, 1982; Zhou et al., 1987).

Since the aforementioned studies do not unequivocally state how many genes were involved and investigated resistance to *F. graminearum* in spring wheat only, a study was initiated to investigate the inheritance of the resistance to *F. culmorum* in winter wheat. In an earlier paper (Snijders, 1990a), the general and specific combining abilities for head blight resistance to *F. culmorum* of ten winter wheat genotypes were estimated using a set of diallel crosses. The results indicated that the general combining ability effects played a major role, whereas the specific combining ability effects were not statiscally significant. The objective of the present study was to investigate the inheritance of resistance to Fusarium head blight in winter wheat

using a quantitative genetic approach, and to estimate the number of effective factors involved in reducing head blight.

Materials and methods

Host and pathogen

Ten homozygous winter wheat genotypes representing different levels of resistance to Fusarium head blight were crossed in all possible combinations, resulting in 45 crosses (Snijders, 1990a). Each F_1 was subsequently selfed. In 1988 a field trial with a replicated plot design was established, with no more than 10 plants per plot. The trial included 5 plots of each parent, 1–4 plots of each F_1 cross and 1–11 plots of each F_2 family, depending on the amount of seed available. Three marked heads of each plant were inoculated twice with *F. culmorum* strain IPO 39-01. Incidence of head blight, expressed as the percentage of infected spikelets in the three marked heads per plant, was recorded 21 days after first inoculation. For further details on materials and methods see Snijders (1990a).

Statistical analysis

Data on head blight were analyzed on a single plant basis. The variance components for each parental line P_i (with $i = 1..10$) and each F_2 were estimated by equating observed mean squares with expected mean squares based on a one-way analysis of variance with unequal replication (Table 1; Steel & Torrie, 1981).

The variance of the observed within-plot mean square MS_w (Table 1) is estimated by

$$\hat{\sigma}^2_{MSW} = \frac{2 \times (MS_W)^2}{(df_W + 2)}$$

where df_w stands for the degrees of freedom of MS_w (Wricke & Weber, 1986). The weighted average within-plot variance due to environment $\bar{\sigma}^2_{eW}$ was estimated from the σ^2_{eW} of the homozygous parents, taking as weight the reciprocals of the square root of the variance of the within-plot mean square $MS_{W(i)}$ for each parent P_i

$$\hat{\bar{\sigma}}^2_e = \frac{\sum_i \left\{ \frac{1}{\hat{\sigma}_{MSW(i)}} \times MS_{W(i)} \right\}}{\sum_i \left\{ \frac{1}{\hat{\sigma}_{MSW(i)}} \right\}}$$

The variance of the weighted within-plot variance was estimated by:

$$\underset{(\hat{\bar{\sigma}}^2_e)}{\hat{\sigma}^2} = \frac{\sum_i 1}{\left\{ \sum_i \frac{1}{\hat{\sigma}_{MSW(i)}} \right\}^2}, \text{ with in this case } \sum_i 1 = 10.$$

Genetic analysis, heritability

For overall mass selection of individual plants, ignoring classification in plots, the heritability in broad sense for each F_2 would be

$$h^2_{(F2)} = \frac{\sigma^2_g}{\sigma^2_g + \bar{\sigma}^2_{eW} + \sigma^2_{eB}} = \frac{MS_W - \bar{\sigma}^2_{eW}}{MS_W + \frac{MS_B - \bar{\sigma}^2_{eW}}{r'}}$$

where σ^2_g stands for the variance component due to genetic variation, σ^2_{eB} stands for the variance component due to environment between plots, $\bar{\sigma}^2_{eW}$ is the weighted within-plot variance due to environmental variation, r' is the coefficient corresponding to r, the number of plants per plot, for the average

Table 1. Estimates of mean squares for a one-way classification of an F_2 with unequal replication, where σ^2_g stands for the variance component due to genetic variation, σ^2_{eB} stands for the variance components due to environments between plots, $\bar{\sigma}^2_{eW}$ is the weighted within-plot variance due to environmental variation, p is the number of plots and r_j is the number of plants of plot j with $j = 1..p$. SV = source of variation; MS = mean squares; EMS = expected mean squares

SV	MS	EMS
Between plots	MS_B	$\bar{\sigma}^2_{eW} + \sigma^2_g + \frac{1}{p-1}\left\{\sum r_j - \frac{\sum r_j^2}{\sum r_j}\right\}\sigma^2_{eB}$
Within plots	MS_W	$\bar{\sigma}^2_{eW} + \sigma^2_g$

value of the within-plot mean square (Table 1), and MS_B and MS_W stand for the mean squares between and within plots. To obtain an approximate variance of h^2, the 'delta technique' was used (Bulmer, 1985: p. 82):

$$\sigma^2 \left\{\frac{x}{y}\right\} = \frac{\sigma^2_x}{x^2} + \frac{x^2 \times \sigma^2_y}{y^4}$$

The variance of h^2 can be approximated by:

$$\sigma^2_{h^2} = \frac{2(MS_W)^2}{(df_W + 2)} + \frac{\sigma^2_{(\delta^2_e)}}{(\sigma^2_g)^2} + \frac{(\sigma^2_g)^2}{(\sigma^2_g + \sigma^2_{eW} + \sigma^2_{eB})^4} \times \left\{\frac{2(MS_W)^2}{(df_W + 2)} + \frac{1}{(r')^2} \times \left\{\frac{2(MS_W)^2}{(df_W + 2)} + \frac{2(MS_B)^2}{(df_B + 2)}\right\}\right\}$$

where df_B and df_W stand for the degrees of freedom of MS_B and MS_W, respectively.

Genetic analysis, gene action

Generation means were calculated from overall head blight means, irrespective of classification in plots. If differences among generation means are determined only by additive and dominance effects of the genes, i.e. if there is no epistasis and no differential viability or fertility, then the following four relationships should be true:

Table 2. Mean ratings and confidence interval (P = 0.05) for head blight incidence (%) in 10 parents after inoculation with *Fusarium culmorum* strain IPO 39-01 in 1988

Parent	Mean (%)
SVP 75059-28	8.3 ± 1.9
SVP 72017-17-5-10	12.1 ± 4.4
SVP 77078-30	29.9 ± 4.0
SVP 75059-32	36.7 ± 5.1
Saiga	37.7 ± 5.1
SVP 72003-4-2-4	40.8 ± 4.9
SVP 73012-1-2-3	66.6 ± 9.0
SVP 73016-2-4	76.9 ± 6.9
SVP 72005-20-3-1	79.8 ± 6.4
SVP 73030-8-1-1	85.3 ± 5.0

$$\bar{P}_1 = m + [d]$$
$$\bar{P}_2 = m - [d]$$
$$\bar{F}_1 = m + [h]$$
$$\bar{F}_2 = m + {}^1/_2[h]$$

with m = mid-parent value, [d] = pooled additive gene effects and [h] = pooled dominance effects (Mather & Jinks, 1982). The joint-scaling test (Mather & Jinks, 1982) was used to test the predicted relationships between generation means. The parameters m, [d] and [h] were estimated from the four generations by weighted least squares, using the reciprocals of the squared standard errors of each mean as weights. A comparison was made between the observed and expected means under the assumption that the sum of squares minimized in the fitting process was distributed as a χ^2 with three degrees of freedom less than the number of generation means available, in this case 4 − 3 = 1. A high value of χ^2 indicates epistasis.

Genetic analysis, number of genes

The minimum number of effective factors segregating in an F_2 family was estimated by the following equation (Wright, 1968):

$$n = \frac{0.25 \times (0.75 - h - h^2) \times R^2}{\sigma^2_g}$$

with

- n = minimum number of effective factors
- h = $(\bar{F}_1 - \bar{P}_1)/(\bar{P}_2 - \bar{P}_1)$, the degree of dominance
- R = $(\bar{P}_2 - \bar{P}_1)$, the range between extreme genotypes
- $\bar{P}_1$ = mean of the parent with the lowest disease rating
- $\bar{P}_2$ = mean of the parent with the highest disease rating
- $\bar{F}_1$ = mean of the F_1 generation
- σ^2_g = genetic variance among F_2 plants

For convenience the effective factors will be called 'genes'. The underlying assumptions of the equation are: (1) no systematic relation between mean

and variance, (2) no linkage of genes, (3) no epistasis, (4) the relevant genes are of equal effect, (5) one parent supplies only plus alleles of those genes in which the two parents differ, whereas the other parent supplies only minus alleles, and (6) an equal degree of dominance for all plus alleles. The number of genes will be underestimated if the assumptions do not hold.

Results and discussion

Mean ratings and confidence intervals for head blight of the 10 parents are given in Table 2. Mean head blight ratings for F_1 and F_2 generations were given in Snijders (1990a). For various reasons (see Snijders, 1990a), the available data on head blight were restricted to 28 F_1 generations and 39 F_2 generations. On average, there were 13 F_1 plants per cross; the maximum was 28. The corresponding figures for F_2 plants were 50 and 80. Parents, F_1 and F_2 showed continuous distributions. In the F_1 and F_2 generations there was no systematic relation between means and total variances of head blight ($r = 0.07$ and $r = -0.06$, respectively). For the parents however, there was a significant correlation between means and variance of $r = 0.67$ ($df = 8$; $P = 0.05$). No improvement was obtained by transformation of the scale. Therefore the original percentage scale was retained. For five parents, σ^2_{eB} did not differ significantly from 0. From the high quotients of σ^2_{eW} and σ^2_{eB} for parents, F_1 and F_2 it was concluded that the field was homogeneous, which justifies an analysis based on overall means, irrespective of classification in plots. The estimated weighted within-plot variance due to environment $\tilde{\sigma}^2_{eW}$ and its confidence interval ($P = 0.05$) were 103.5 ± 17.6.

Heritability. The broad sense heritabilities and their standard errors for the head blight resistance of the 39 F_2 families are presented in Table 3. As no backcrosses were available, the additive variance could not be separated from the total genetic variance in the numerator of the broad-sense heritability equation. Estimates of broad-sense heritability ranged from 0.05 to 0.89, with an average of 0.39. Table 3 shows that the standard deviations were very large. This is not unexpected, because heritability estimates are based on estimates of components of variance with inherent large standard deviations.

Gene action. The significance of the deviation from zero of the equation $c = 4\bar{F}_2 - 2\bar{F}_1 - \bar{P}_1 - \bar{P}_2$ was tested for the 23 crosses for which both F_1 and F_2 generations were available. For 13 crosses c differed significantly from zero, implying that in these crosses there were epistatic gene effects. However, the various generation means did not have equal variances. Therefore, the predicted relationships between generation means were tested in the joint-

Table 3. Broad-sense heritabilities and standard errors of resistance to head blight caused by *Fusarium culmorum* strain IPO 39-01 for F_2 families of a 10 × 10 diallel of winter wheat genotypes. Parents are listed in descending order of level of resistance

Parent	Parent number	Parent number								
		2	3	4	5	6	7	8	9	10
SVP 75059-28	1	0.15 ± 0.34	0.55 ± 0.31	0.89 ± 0.64	0.27 ± 0.39	0.21 ± 0.34	0.22 ± 1.49	0.27 ± 0.27	0.14 ± 0.34	–[a]
SVP 72017-17-5-10	2	..	0.62 ± 0.32	0.53 ± 0.31	0.27 ± 0.59	0.37 ± 0.31	–	0.33 ± 0.32	0.34 ± 0.30	0.54 ± 0.27
SVP 77078-30	3	..	..	0.35 ± 0.30	0.41 ± 0.35	0.17 ± 0.41	0.76 ± 0.76	0.71 ± 0.53	0.36 ± 0.30	0.20 ± 0.38
SVP 75059-32	4	..	..	..	0.70 ± 0.37	0.35 ± 0.45	0.20 ± 0.53	0.07 ± 0.90	0.49 ± 0.29	0.26 ± 0.27
Saiga	5	..	..	..	..	0.05 ± 1.32	–	0.25 ± 0.30	0.23 ± 0.28	–
SVP 72003-4-2-4	6	..	..	..	..	..	–	0.20 ± 0.45	0.67 ± 0.50	0.81 ± 0.45
SVP 73012-1-2-3	7	..	..	..	..	..	..	0.48 ± 0.33	0.30 ± 0.49	–
SVP 73016-2-4	8	..	..	..	..	..	..	..	0.59 ± 0.40	0.44 ± 0.47
SVP 72005-20-3-1	9	..	..	..	..	..	..	..	..	0.32 ± 0.34
SVP 73030-8-1-1	10	..	..	..	..	..	..	..	..	..

[a] missing combination.

scaling test, using weighted expectations. The estimates for mid-parent values m, pooled additive gene effects [d] and pooled dominance effects [h], and their standard deviations are given in Table 4. The joint-scaling test showed that the additive-dominance model fitted for all crosses except 8719 (Table 4). The additive gene effects were significant (P = 0.05) for 16 crosses, and for 11 crosses they were accompanied by significant dominance effects. In 8 of these 11 crosses the estimated dominance effects were negative, indicating that in these hybrid combinations the levels of resistance were higher than those of the midparent. The degree of dominance varied considerably. The pre-

Table 4. Values[1] and standard deviations for mid-parent values m, pooled additive gene effects [d] and pooled dominance effects [h], and χ^2 values based on the joint scaling test for 23 crosses among 10 winter wheat parents for head blight incidence due to experimental inoculation by *Fusarium culmorum* strain IPO 39-01. The number of independently segregating genes controlling the Fussarium head blight resistance was estimated by Wright's equation. Parents are listed in descending order of resistance

Cross	Cross number	m	[d]	[h]	χ^2 value	Estimated number of genes
SVP 75059-28 ×						
SVP 72017-17-5-10	8718	10.7 ± 3.6	−2.2 ± 3.6	8.8 ± 7.4	0.30	0.1
SVP 77078-30	8735	19.7 ± 3.1	−11.2 ± 3.1*	−6.1 ± 4.5	0.21	0.3
Saiga	8743	23.7 ± 3.5	−15.2 ± 3.5*	−14.3 ± 5.6*	0.26	0.9
SVP 72003-4-2-4	8716	25.1 ± 3.3	−16.7 ± 3.3*	6.6 ± 3.5	0.20	0.8
SVP 73012-1-2-3	8719	49.4 ± 26.9	−40.2 ± 27.2	4.4 ± 45.4	5.7 °	
SVP 73016-2-4	8720	43.5 ± 5.1	−35.1 ± 5.2*	−27.6 ± 6.8*	0.33	3.9
SVP 72005-20-3-1	8717	44.9 ± 5.3	−36.5 ± 5.3*	−16.0 ± 10.6	0.26	3.6
SVP 72017-17-5-10 ×						
SVP 75059-32	8724	24.4 ± 0.1	−12.0 ± 0.1*	4.6 ± 0.1*	0.00	0.5
Saiga	8739	25.7 ± 2.8	−12.8 ± 2.9*	−12.9 ± 4.7*	0.10	1.5
SVP 72003-4-2-4	8702	29.0 ± 11.0	−14.7 ± 11.3	−24.4 ± 11.3*	1.49	1.1
SVP 73016-2-4	8709	44.3 ± 0.9	−32.3 ± 0.9*	−3.6 ± 1.5*	0.00	2.7
SVP 72005-20-3-1	8703	50.7 ± 16.6	−36.0 ± 17.1*	−37.9 ± 20.9	2.13	5.1
SVP 77078-30 ×						
SVP 75059-32	8736	33.4 ± 0.5	−3.4 ± 0.5*	6.7 ± 0.7*	0.00	0.1
SVP 73016-2-4	8733	51.1 ± 8.3	−22.7 ± 8.6*	22.3 ± 8.9*	0.63	1.3
SVP 72005-20-3-1	8730	55.3 ± 2.1	−25.1 ± 2.2*	−14.5 ± 4.1*	0.03	1.3
SVP 73030-8-1-1	8734	56.6 ± 4.4	−27.6 ± 4.5*	−0.9 ± 8.6	0.24	3.4
SVP 75059-32 ×						
SVP 72003-4-2-4	8722	38.0 ± 2.8	−2.1 ± 2.9	−11.0 ± 5.8*	0.09	1.2
SVP 73016-2-4	8726	56.5 ± 1.3	−20.0 ± 1.3*	−14.1 ± 1.8*	0.01	5.8
Saiga ×						
SVP 72003-4-2-4	8737	39.2 ± 0.3	−1.6 ± 0.4*	−9.3 ± 0.5*	0.00	1.0
SVP 73016-2-4	8741	57.4 ± 0.4	−19.6 ± 0.4*	−10.6 ± 0.6*	0.00	1.3
SVP 73012-1-2-3 ×						
SVP 72005-20-3-1	8705	70.1 ± 7.1	−7.2 ± 7.7	2.1 ± 8.9	0.24	0.2
SVP 73016-2-4 ×						
SVP 72005-20-3-1	8708	77.6 ± 2.8	−1.3 ± 2.9	−8.9 ± 4.9	0.05	0.0
SVP 73030-8-1-1	8715	80.8 ± 4.1	−4.5 ± 4.3	−11.2 ± 6.7	0.15	0.1

[1] As for head blight, m, [d] and [h] are expressed on a percentage scale.

* Significant at a probability of 5%.

° Family for which the additive-dominance model did not fit (P = 0.01).

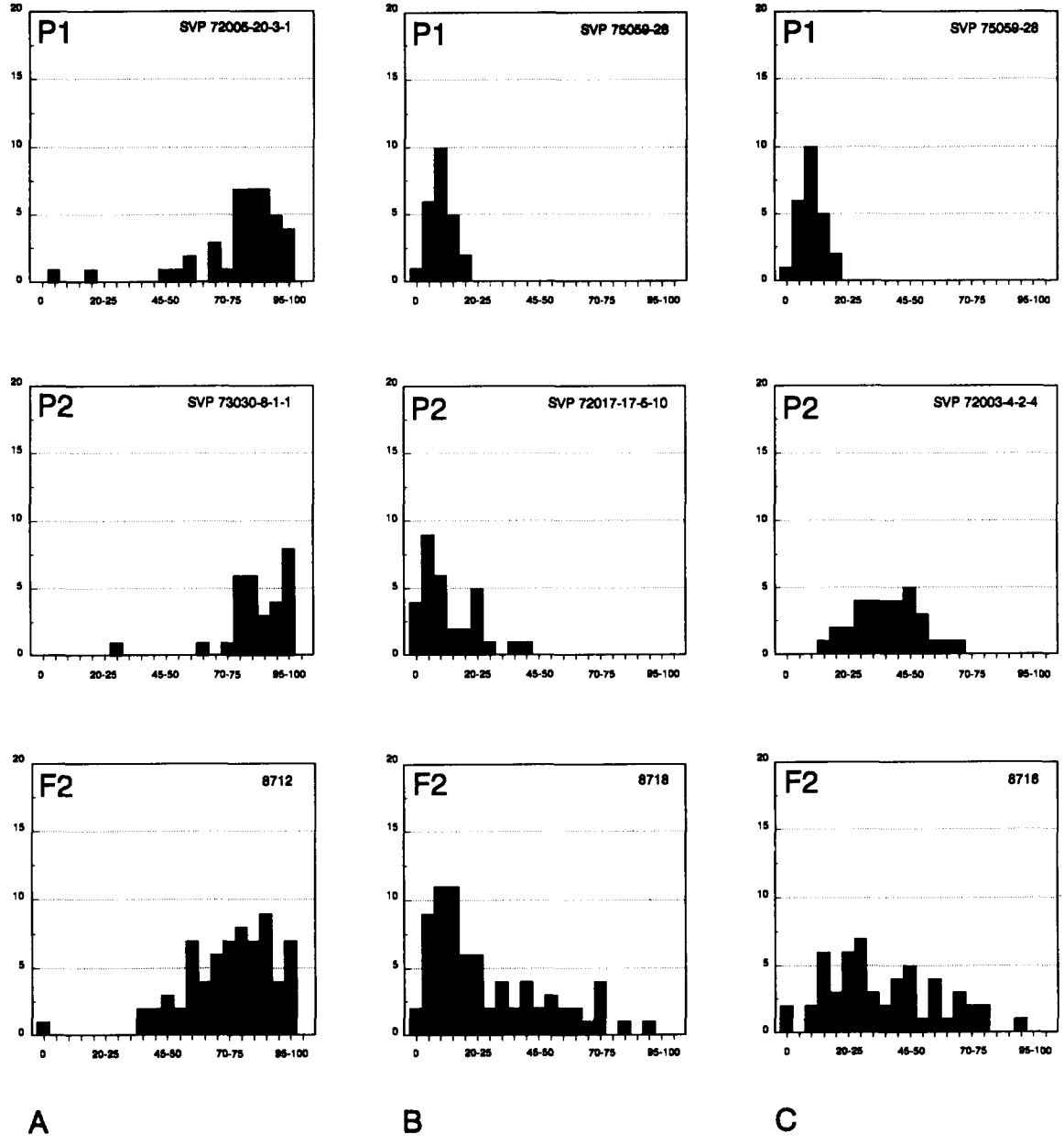

Fig. 1. Distribution of frequencies of plants in 5% classes of Fusarium head blight incidence on a scale of 0–100%, for two parents (P_1 and P_2) and their F_2. Three crosses for which the F_2 distribution suggests transgression are shown. A: cross 8712, B: cross 8718 and C: cross 8716.

dominance of the negative dominance effects in number and degree agrees with results from studies of the combining ability of this data set, where the mean deviation of the F_1s from the mean midparent values indicated heterosis for Fusarium head blight resistance (Snijders, 1990a). Despite the results of the joint-scaling test, the significant dominance effects in crosses 8702, 8722 and 8737 suggest epistasis. These crosses have SVP[1] 72003-4-2-4 as com-

[1] The Foundation for Agricultural Plant Breeding (SVP) is now part of the Centre for Plant Breeding Research (CPO)

mon parent. An earlier paper (Snijders, 1990a) concluded that this genotype had a better general combining ability for resistance than expected from its resistance level. If the increased resistance is the result of an additive × additive epistatic component, it will be fixable in a pure inbred line (Snijders, 1990b).

On the whole, significant additive gene effects occurred more frequently and were larger than dominance effects. This indicates that additive effects predominantly determined the differences in resistance to Fusarium head blight within F_2 populations, and is in agreement with the finding that general combining ability effects were much more important than specific combining ability effects (Snijders, 1990a). Therefore, selecting for resistance on a single-plant basis in a segregating population could be successful if sufficient genetic variation is available.

Number of genes

The number of segregating genes involved in the resistance to Fusarium head blight was estimated for all crosses for which the joint-scaling test had shown that additive and dominance effects could explain the gene action (Table 4). The first three assumptions underlying Wright's equation were met. Of the two resistant parents, SVP 75059-28 differed from the susceptible SVP 72005-20-3-1 in 3.6 genes, and SVP 72017-17-5-10 differed from SVP 72005-20-3-1 in 5.1 genes. In addition to the higher level of resistance of SVP 75059-28, it is concluded that the resistance genes in SVP 72017-17-5-10 had less effect on the increase of resistance than the genes in SVP 75059-28. Crosses 8720, 8709, 8733 and 8726, all with SVP 73016-2-4 as susceptible parent, are even more striking examples of different gene effects. However, these findings do not exclude the possibility that within each partially resistant genotype the resistance genes had equal effects. Assumption 4 states that one parent supplies only plus factors of resistance, whereas the other parent supplies only minus factors. This could not be ascertained for any of the crosses. The F_2 progeny of the cross between the two most susceptible genotypes SVP 72005-20-3-1 and SVP 73030-8-1-1 showed transgression towards resistance (A in Fig. 1). This indicates that even these two susceptible genotypes differed in at least one resistance gene. Apart from the cross between the two susceptible parents, transgressive segregation in the F_2 was only clearly observed for two crosses, 8718 and 8716, and only towards susceptibility (B and C in Fig. 1). The parents of cross 8718 (SVP 75059-28 and SVP 72017-17-5-10) had the highest level of resistance. Although no significant gene effects were found (Table 4), the transgression indicated that some of the resistance genes in both parents must be different. This is not unexpected. The genes of the two parents differed in their effect on resistance and originated from different ancestors (Snijders, 1990a). Additional information on transgression of F_2 derived lines will be presented in a subsequent paper (Snijders, 1990b).

In Table 4 it is shown that the [d] effects increased with the difference in level of resistance between the two parents. No trend was apparent for the [h] effects. Provided that non-allelic genes neither interact nor are linked, the total heritable variance given by k genes in F_2 will be the sum of the k individual contributions, namely $^1/_2\sum d^2 + {}^1/_4\sum h^2$ (Mather & Jinks, 1982). This would mean that the genetic variation has to increase with the difference in resistance levels between the parents, implying an increased heritability. However, heritability estimates did not increase with larger disparity between the parents ($r = -0.07$). This lack of correlation can be attributed to difference in resistance genes between parents as well as to the fact that the estimates for heritability were not accurate.

In Wright's equation R represents the range between the extreme genotypes for head blight resistance. If both parents in a cross show some resistance, but at different levels (as is suggested for several crosses in this study), each may contain plus factors not present in the other. If so, when calculating R it was wrong to assume that the difference in head blight resistance of the two parents was identical to the difference between the two extreme genotypes possible with the genes involved: as a

result, the number of segregating genes was underestimated. Van Ginkel & Scharen (1988) postulated that the assumption of unidirectional gene distribution would be more closely adhered to if the extreme genotypes were selected from the F_2 and used to estimate R. However, in this study the large environmental variances and the continuous distributions of the progenies made it impossible to assign an F_2 plant with extreme resistance to a distinct class.

It may be concluded that the estimated number of segregating genes governing Fusarium head blight resistance in the populations studied varied between one and six. However, the results of the method used to calculate the number of genes should be viewed with caution. Besides the fact that Wright's equation is not a very accurate estimator, possibly also not all assumptions were met. The estimates of number of genes as shown in Table 4 are based on independent assortment. If linkage occurs, the number of genes really present could be higher than the estimated number. Nevertheless, it is clear that Fusarium head blight resistance is governed by several minor genes.

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