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Inheritance of resistance to leaf and glume blotch caused by *Septoria nodorum* Berk. in winter wheat

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Abstract Sixteen crosses between eight winter wheat cultivars were screened for resistance to *Septoria nodorum* leaf and glume blotch in the F_1 and F_4 generations using artificial inoculation in the field. The F_1 of most crosses showed dominance for susceptibility on both ear and leaf. The effects of general combining ability were of similar magnitude as the effects for specific combining ability. On the basis of the phenotypic difference of the parents, no prediction was possible about the amount and the direction of genetic variance in the segregating populations. The variation observed in this study both within and among the segregating populations suggests a quantitative inheritance pattern influencing the expression of the two traits. The components of variance between F_2 families within a population were as high as (for *S. nodorum* blotch on the ear) or higher (for *S. nodorum* blotch on the leaf) than those between populations. Therefore, strong selection within a few populations may be as effective to obtain new resistant genotypes as selection in a large number of populations. In almost all crosses, progenies were found that were more resistant than the better parent. Thus transgression breeding may be a tool to breed for higher levels of resistance to *S. nodorum* blotch. Highly resistant genotypes were found even in combination with two susceptible parents. The genetic source for *Septoria* resistance is probably broader than is generally assumed and could be used to improve *S. nodorum* resistance by combination breeding followed by strong selection in large populations.

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Introduction

Leaf and glume blotch of wheat play a major role as damaging diseases. They are caused by the fungus *Leptosphaeria nodorum* E. Müller [=*Phaeosphaeria nodorum* (E. Müller) Hedjaroude], anamorph *Septoria nodorum* (Berk.) Berk. in Berk.&Broome [=*Stagonospora nodorum* (Berk.) Castellani&Germano]. To simplify matters, in the frame of this report the disease will be called *S. nodorum* blotch (SNB). Among the wheat germplasm, as well as among the related *Triticum* species, no immune reaction to SNB has been identified until now. Nevertheless, some wheat cultivars and lines showed a high level of partial resistance to SNB (Jeger et al. 1983; Tomerlin et al. 1984). Partial resistance of the ear is not, or is only moderately, correlated with the partial resistance of the leaf (Fried and Meister 1987; Bostwick et al. 1993) and both are controlled polygenically (Wilkinson et al. 1990; Bostwick et al. 1993). Resistance to SNB has been determined at the seedling stage (Krupinsky 1972; Krupinsky et al. 1977; Mullaney et al. 1982), in detached leaves (Ecker et al. 1989) and in adult plants (Arseniuk et al. 1991; Bostwick et al. 1993; Loughman et al. 1994; Fried and Meister 1987). However, a screening at the seedling stage gives no indication of resistance reactions on the ear. Information about the genetic basis responsible for the expression of partial resistance in the field is of great importance to wheat breeders. This knowledge might lead to specific germplasm combinations showing a higher level of resistance.

Specific crosses were made between eight winter wheat lines showing different resistance reactions on the ear and the leaves. These lines originate from the actual Swiss or European breeding material. The resistance level of both leaf and ear was evaluated after artificial infection in the field at the adult plant stage for the parental lines and the F_1 and F_4 generation of each cross.

Moreover, the influence of morphological traits on the expression of partial resistance was estimated over all populations and for each population separately. Based on the study of Keller et al. (1994), who were able to distinguish resistant and susceptible wheat cultivars by means of in vitro screening, we wanted to test if this method is useful for resistance screening in early segregating populations. Such a selection would be very useful to wheat breeders. Therefore, one objective of this study was to obtain reliable phenotypic data on the level of resistance against SNB in early segregating generations in the field. Results of the comparison of the field data with the in vitro screening for resistance against *S. nodorum* are discussed in Wicki et al. (1999). The data set obtained by this field assessment over a large number of wheat genotypes allowed us to study the resistance mechanisms responsible for the inheritance of resistance against *S. nodorum* on the leaves and the ears. It was possible to determine the genetic variation between, as well as within, segregating populations and therefore to estimate the probability to detect new sources of resistance. On the basis of 16 different populations, crossing strategies in wheat breeding programs are discussed.

Materials and methods

Parental lines and crosses

Eight winter wheat lines and varieties with contrasting resistance against SNB on ear or leaf, respectively, were selected as parents (Table 1). They were chosen on the basis of the results of the official variety tests of the Swiss Federal Research Station for Agroecology and Agriculture (FAL) Zürich-Reckenholz. The parental lines were divided into two groups (ear and leaf), with two lines showing a high level of resistance and two lines showing a low level of resistance on the corresponding organ in these groups. Within each group, a diallel cross without reciprocal crosses was carried out (Table 2). Four additional crosses were made with entries which at the same time had either a low or a high resistance level on both organs (i.e. SN–×SN+, SN–×Zenith, SN–×Greif, Zenith×Greif).

Field trials

In order to detect quantitative differences in the resistance level to *S. nodorum* leaf and glume blotch, progenies of the 16 crosses in the F_1 and F_4 generations as well as the parental lines were screened in field trials in Zürich-Reckenholz, Switzerland, in 1995 and 1996, respectively, using artificial inoculation as described below.

Field trial F_1 crosses in 1996

F_1 progenies of the 16 crosses were sown in 1995 as a 1-row plot together with the parental lines. In order to avoid interactions with other diseases (powdery mildew, scab, leaf and stripe rust), the trial was sprayed with 1 l/ha of Tiptor (Maag, Switzerland) 6 weeks before inoculation with *S. nodorum*. Oat slugworms were controlled by spraying 1.5 l/ha of Zolone (Maag, Switzerland) at growth stage 50 to 55 (Zadoks et al. 1974). The same protection measures were applied in the field trials with the F_4 populations.

Field trial F_4 populations in 1996

In the meantime, 18–28 individual plants out of each F_2 population were randomly selected (352 in total). From these selected F_2 plants, seeds from one ear were sown as single F_3 head rows (1.2 m long) in autumn 1994. From each F_3 line (head row), 15 ears were harvested and threshed separately. The F_4 seeds were sown in autumn 1995 as 5-row plots, where each row represented a single head row. The design was a rectangular lattice with three replications, including the 352 5-row plots of the progenies of the 16 crosses and the eight parents as six replicated entries per replication (400 plots per replication).

Inoculation procedure

Inoculum for the artificial inoculations was prepared as described by Fried (1989). For the inoculation, a mixture of a broad spectrum of isolates collected in Switzerland was used. The trials were inoculated four times with a spore suspension of 1 million viable spores per ml (400 l/ha, 20 ml per row). The first inoculation was applied at the booting stage (stage 47–49). The second-to-fourth inoculations were applied to the heads. Taking into account the differences in earliness of the different genotypes, the second inoculation was carried out, when the first third of the plants was in stage 59–61, the third when the second third was in this stage (1 week after the first inoculation) and the last when the remaining plants reached this stage (2 weeks after the first inocu-

Table 2 Crossing scheme

Males	Group ear (females)			Group leaf (females)				
	Arina	Iena	Forno	SN–	SN+	Boval	Iena	Zenith
Iena	×							
Forno	×	×						
SN–	×	×	×					
SN+				×				
Boval					×			
Iena					×	×		
Zenith				×	×	×	×	
Greif				×				×

Table 1 Chosen parental lines with their pedigrees, origins and resistance reactions. + High level of resistance, – low level of resistance, 0 Intermediate

Cultivar	Pedigree	Origin	Resistance to ear blotch	Resistance to leaf blotch
Arina	Moisson/(Can3842/Heine VII)	CH	+	0
Iena	Champlein/Courtôt	F	+	–
Forno	72837/Kormoran	CH	–	0
SN–	Major/Hoeser52	CH	–	–
SN+	Arminda/Roazon	CH	+	+
Boval	(Caribo/Hoeser48)/(Can3842/Tano)	CH	–	+
Zenith	Can3842/Heine VII	CH	–	–
Greif	(Maris Hobbit/Carimulti)/Carimulti	D	+	+

lation). In the field trial with the F_4, only two of the three replications were inoculated.

Scoring of data

The following traits were recorded on a single-row basis in the F_1 and F_4 field trials at FAL, Zürich, in 1995 and 1996: days to ear emergence (DEA) and flowering (DFL) from January first, plant height in cm (HCM), percentage of necrosis on each of the top-three leaf layers and percentage of necrosis on the ear. The disease scoring on the leaves was performed five times (1995) and seven times (1996) within 4 weeks during growth stages 50 and 80, and on the ear four times (1995) and seven times (1996) within 4 weeks during growth stages 60 and 85. In order to obtain a normal distribution of the percentage of the diseased area on the ear and leaf, each recording was log (×+1) transformed. For each line, transformed scores of all dates were added up. This resulted in the severity index SNAE for the ear and LNLF for the leaf which was used to determine quantitative differences in the resistance reaction. The colour (wax layer) of the ears (CEA) and the leaves (CLF) was recorded as well. A score of 1 represented a genotype with a strong wax layer, a score of 9 a genotype with no wax layer.

Statistical analysis

Data analysis included simple correlation analysis between the severity indices (SNEA, SNLF), plant height (HCM), wax layer (CEA,CLF) and earliness (DEA, DFL). Heterosis for SNEA and SNLF in the F_1 was determined by comparing the F_1 value to the mid-parent value. On the basis of the crossing scheme, general combining ability (GCA) and specific combining ability (SCA) were calculated for SNEA and SNLF within each group. An analysis of lattice design as well as an analysis of variance (ANOVA) with a complete block design were carried out for the F_4 field trial on the basis of the mean value of the 5-row plots (n=400 per replication) using PLABSTAT (H.F. Utz, Institute of Plant Breeding, University of Hohenheim, Germany). To test the genetic variance between and within populations, the following factors were included in the ANOVA model: blocks (B, the two replications), populations (P) and F_2-derived families within populations (F:P). The following mixed model (Model I) with random factors was used: B+P+F:P+BF:P.

ANOVA was further calculated for each population separately, using the following model (Model II): B+F+BF.

In order to quantify the variance between the F_4 headrows derived from different F_3 plants but the same F_2 plant, a hierarchical model (Model III) was applied by adding the factor headrows (R) nested within family and block: B+P+F:P+BP+BF:P+R:BFP.

The data set for this calculation consisted, therefore, of 4000 values (5 rows×400 families×2 replications). Due to the lack of true replications, the variance components caused by the segregation between the F_4 headrows derived from different F_3 plants, but the same F_2 plant ($\sigma^2_{R:BFP}$), could not be tested for significance. Heritability based on the 400 entries of the lattice design (h^2_o, operational heritability) was calculated according to the following formula:

$$h_o^2 = \frac{\sigma_F^2}{\sigma_F^2 + \frac{\sigma_{ems}^2}{2}}, \text{ where}$$

σ^2_F=variance of different F_2 families over 16 populations,
σ^2_{ems}=effective error of the mean square.

Values for broad-sense heritability (h^2_b) based on the mean values of 5-row-plots (Model II) was calculated for each population according to Hallauer and Miranda (1981):

$$h_b^2 = \frac{\sigma_F^2}{\sigma_F^2 + \frac{\sigma_{FB}^2}{2}}.$$

Results

Parental lines in the F_4 field trial

In the F_4 field trial in 1996, Arina was the most resistant and Boval the most susceptible parent on the ear (15% and 75% necrosis at the last recording date, respectively). The most resistant parent on the leaf was SN+ (35% necrosis on the flag leaf at the last recording date), and the most susceptible parent on the leaf was SN– (90% necrosis on the flag leaf at the last recording date). Table 3 shows the values for SNEA and SNLF of the eight parental lines in the order of increasing SNEA values of the F_4 field trial. Plant height showed a variation between 95 cm for the short-strawed varieties Iena and Greif and 125 cm for the tallest variety Arina (Table 3). Arina, Greif and Zenith, the three most resistant varieties on the ear, had a strong wax layer on the ear (score 2 for colour of ear). Boval with the highest SNEA value of the eight parents had the earliest heading date. Disease severity indices of the parental lines on ear and leaf in the 1995 field trial based on 1-row plots were highly correlated with the disease severities in the 1996 field trial based on 5-row plots (r=0.90 for SNEA and r=0.92 for SNLF, P<0.01). SNEA was not significantly correlated with SNLF for the eight parental lines (r=0.34 in the 1996 field trial , P>0.05). Correlations of SNEA and SNLF with other traits (data from the 1996 field trial) are shown in Table 4. Although moderately high (e.g. r=0.60 between colour of ear and SNEA), these correlations are not significant due to the small number of parental lines.

Field trial F_1

The two most resistant F_1 progenies against ear blotch resulted from the cross Arina×SN– and Arina×Iena (Ta-

Table 3 Parental lines and their resistance reaction on ear and leaf to artificial inoculation with *S. nodorum* from the F_4 field trial in 1996. Days to ear emergence from January 1st (DEA), plant height in cm (HCM), colour of leaf (CLF, 1 strong way layer, 9 no wax layer) and colour of ear (CEA, 1 strong wax layer, 9 no wax layer) are shown

Cultivar	SNEA[a]	SNLF[b]	DEA d	HCM cm	CLF	CEA
ARINA	3.06	23.51	155	125	3	2
GREIF	3.14	18.34	157	95	4	2
ZENITH	3.38	22.3	159	120	3	2
IENA	3.54	21.94	155	95	8	7
SN+	3.64	20.14	158	110	6	4
SN–	4.36	25.23	160	100	6	4
FORNO	4.78	22.31	155	102	7	7
BOVAL	5.59	20.53	151	110	6	6
Mean	3.94	21.79	156.25	107.13	5.38	4.25
s[c]	0.89	2.12	2.87	11.17	1.85	2.19

[a] SNEA: *S. nodorum* severity index on the ear: log (1+ percentage of diseased area of the ear) summed up over seven scorings during growth stages 50–80
[b] SNLF: *S. nodorum* severity index on the leaf: log(1+ average percentage of diseased area of the three top leav layers) summed up over seven scorings during growth stages 60–85
[c] s: standard deviation

Table 4 Correlations of different traits to SNAE and SNLF for the eight parental lines in the F_4 field trial in 1996

Trait 1	Trait 2	*r*	
Days to ear emergence	SNEA	–0.44	n.s.
Days to flowering	SNEA	–0.34	n.s.
Plant height	SNEA	–0.07	n.s.
Colour of leaves	SNEA	0.51	n.s.
Colour of ears	SNEA	0.60	n.s.
Colour of leaves	SNLF	0.28	n.s.
Colour of ears	SNLF	0.24	n.s.
Days to ear emergence	SNLF	0.21	n.s.
Days to flowering	SNLF	0.26	n.s.
Plant height	SNLF	–0.02	n.s.

n.s. $P>0.05$

Table 5 SNEA and SNLF of the 16 F_1 and % heterosis in relation to the mid-parent value in the F_1 field trial in 1996 (positive values: more susceptible than the mean value of the two parents; negative values: more resistant than the mean value of the two parents)

F_1	SNEA	% Heterosis	SNLF	% Heterosis
Arina×SN–	3.52	–4.55	23.89	3.52
Arina×Iena	3.53	14.86	22.79	5.00
Zenith×Greif	3.59	11.94	21.98	7.34
SN–×Greif	3.61	–4.10	22.26	4.98
SN+×Zenith	3.67	3.39	22.52	5.59
SN–×Zenith	3.85	–1.28	23.84	2.21
Iena×Zenith	3.92	19.83	23.20	5.67
Arina×Forno	4.02	9.51	22.72	4.16
Iena×SN–	4.04	5.44	23.12	1.92
SN–×SN+	4.07	–0.76	22.64	2.63
SN+×Iena	4.11	17.99	18.94	–8.44
Iena×Forno	4.14	8.46	20.29	–5.28
Forno×SN–	4.31	–2.82	24.04	5.45
SN+×Boval	4.51	–4.98	19.05	–4.62
Boval×Zenith	4.84	6.64	23.99	12.94
Boval×Iena	4.84	8.17	20.59	–0.04
Mean	4.04	5.48	22.24	2.69

ble 5). Arina×SN– showed a slightly negative heterosis for the severity index on the ear (SNEA), i.e. the F_1 was more resistant than the mean value of the two parents. On the other hand, Arina×Iena showed a high positive heterosis, i.e. the F_1 had about a 15% higher disease severity than the mean of the two parents (Table 5). SN+×Iena and SN+×Boval produced the most resistant progenies on the leaf with negative heterosis (more resistant than the mean of the two parents) for both (Table 5). F_1 progenies from the crosses Boval×Zenith and Forno×SN– showed the highest SNLF values, with a high positive heterosis of 13% (more susceptible than the mean of the two parents) for Boval x Zenith. On average the heterosis was positive for both SNEA and SNLF, with higher values for SNEA. This indicates dominance for susceptibility to SNB, especially on the ear. In the case of SN+×Iena, a high positive heterosis for SNEA was recorded, together with a high negative heterosis for SNLF. General combining ability (GCA) and specific combining ability (SCA) for SNEA and SNLF are summarised in Table 6. Arina, the parent with the highest level of resistance on the ear, had the highest negative GCA for SNEA (–0.24, reduction of necrosis), whereas Forno, with a high level of susceptibility on the ear, was the parent with the highest positive GCA for SNEA (+0.23, increasing necrosis). Iena, with a high level of resistance on the ear, showed only a slightly negative GCA. The highest positive SCA for SNEA (–0.20, reduction of necrosis) was for Arina in the cross with SN– and the highest negative SCA (+0.12, increasing necrosis) was for Forno in the cross with SN–.

Table 6 General combining ability (GCA) and specific combining ability (SCA) for the diallel F_1 crosses within group ear and group leaf in the F_1 field trial in 1996

Group ear		Group leaf	
	GCA ear		GCA leaf
Arina	–0.24	SN+	–1.21
Iena	–0.03	Boval	–0.17
Forno	0.23	Iena	–0.47
SN–	0.03	Zenith	1.86
	SCA ear		SCA leaf
Iena×SN–	0.11	Iena×Zenith	0.43
Iena×Forno	0.01	SN+×Zenith	1.97
Arina×Iena	–0.14	SN+×Iena	–0.76
Arina×SN–	–0.20	SN+×Boval	–0.95
Arina×Forno	0.10	Boval×Zenith	0.93
Forno×SN–	0.12	Boval×Iena	–0.15

F_4 field trial in 1996

At the last recording date on a single-row basis in the 1996 field trial the F_4 lines ranged from 5 to 90% for the percentage of necrosis on the ear and again showed a large variation between and within the 16 populations. The percentage of necrosis on the flag leaf ranged from from 8 to 100% at the last recording date. In the F_4 generation SNEA and SNLF were correlated with r=0.31 ($P>0.01$) across the 16 populations. Plant height in cm (HCM) ranged from 70 cm to 145 cm. This trait was significantly negatively correlated with SNEA (r=–0.32, $P<0.01$) and SNLF (r=–0.25, $P<0.01$). The strongest negative correlation between HCM and SNEA was observed for F_4 lines of the cross SN–×Greif (r=–0.75, $P<0.01$). Days to ear emergence were significantly negatively correlated with SNEA (r=–0.36, $P<0.01$). Colour of the ear and SNEA were correlated with r=0.35 ($P<0.01$).

Heritability based on the 400 entries of the lattice design (h^2_o) was 0.89 ($P<0.01$) for days to ear emergence, 0.91 ($P<0.01$) for plant height, 0.82 ($P<0.01$) for necrosis on leaf, and 0.84 ($P<0.01$) for necrosis on ear. The efficiency of the lattice design (compared to the complete block design) was 102.7 for SNLF, 108.7 for SNEA, 100.9 for plant height and 106.4 for days to ear emergence, indicating little effects of incomplete blocks. This

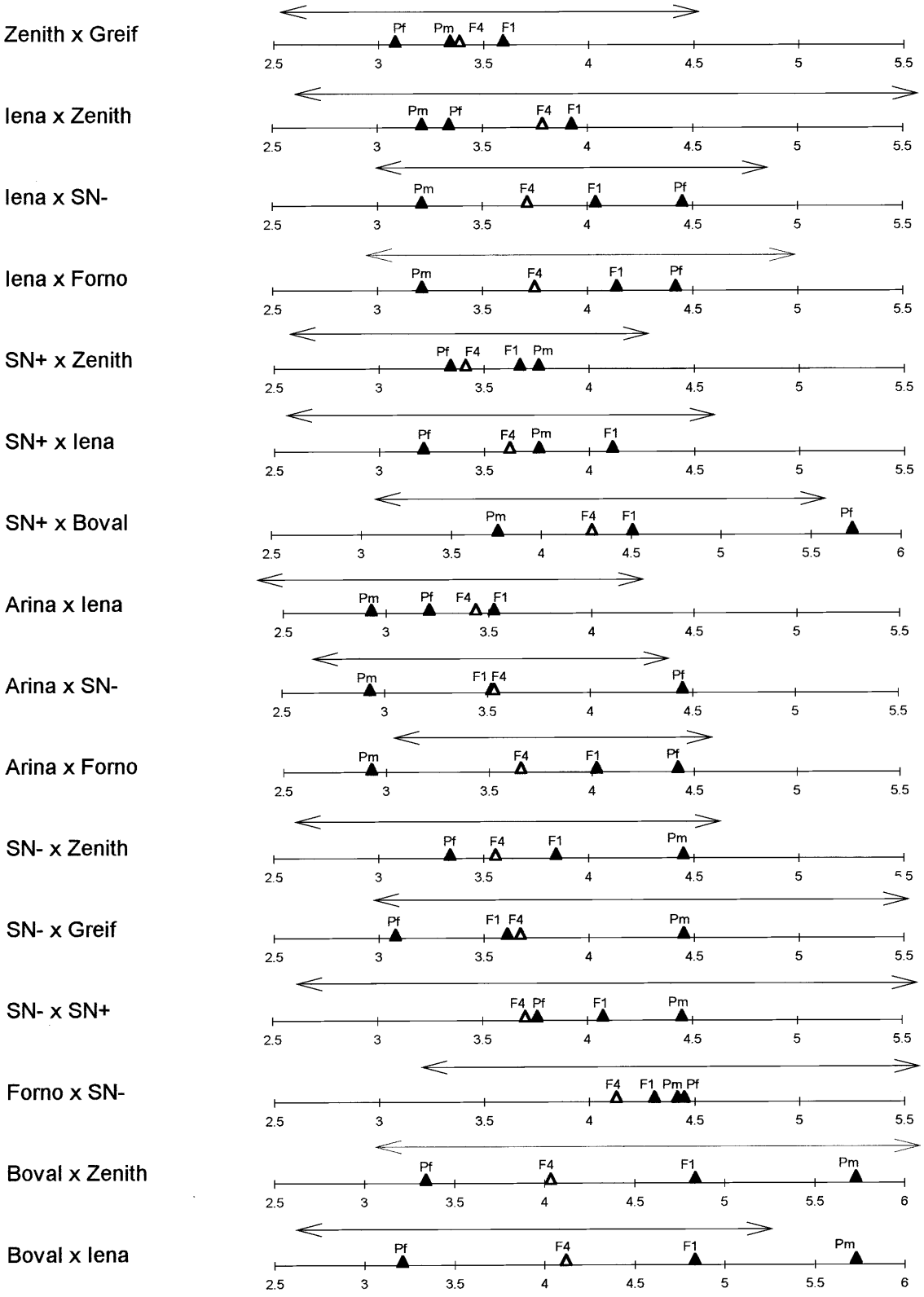

Fig. 1 Mean values of parents, F_1 value and F_4 mean for *S. nodorum* on the ear (SNEA) for all 16 populations, including the range in the F_4. ▲ Pm=mother, ▲ Pf=father, ▲ F_1=F_1 value, △ F_4=F_4 mean, ⟷ range in F_4

allowed the calculation of ANOVA as a randomised complete block design.

Significant differences (P<0.01) between the 16 populations (σ^2_P) as well as between the F_2 families within a population ($\sigma^2_{F:P}$, Model I) were found for SNEA, SNLF, DEA and HCM. For SNEA, the components of

Table 7 Genetic components of variance and heritability for *S. nodorum* on ear and leaf (SNEA and SNLF)

	σ^2_F[a] SNEA	h^2_b SNEA	σ^2_F SNLF	h^2_b SNLF
Zenith×Greif	–0.002	×[b]	0.570*	0.50
Iena×Zenith	0.065*	0.55	–0.228	×
Iena×SN–	0.047**	0.62	1.525**	0.81
Iena×Forno	0.043**	0.70	0.509**	0.62
SN+×Zenith	0.075**	0.66	0.893**	0.75
SN^+×Iena	0.06*	0.57	0.071ns	0.15
SN+×Boval	0.085*	0.53	0.336*	0.52
Arina×Iena	0.081**	0.75	1.630**	0.88
Arina×SN–	0.006ns	0.16	0.591**	0.74
Arina×Forno	0.038*	0.59	0.095ns	0.22
SN–×Zenith	0.084**	0.72	0.455**	0.64
SN–×Greif	0.139**	0.83	1.293**	0.74
SN–×SN+	0.121**	0.79	2.813**	0.95
Forno×SN–	0.025ns	0.30	1.369**	0.82
Boval×Zenith	0.042ns	0.39	0.877**	0.76
Boval×Iena	0.144**	0.77	0.541*	0.56

** $P<0.01$, * $P<0.01$, n.s. $P>0.05$

[a] σ^2_F variance between F_2 families for the 16 crosses (Modell II), h^2_b broad-sense heritability

[b] ×not determined due to negative genetic components of variance

variance between populations compared to the variance within populations had the same magnitude, whereas for SNLF the components of variance between families was higher ($\sigma^2_{F:P}$=0.88) than between populations (σ^2_P=0.56). The variance components between the F_4 headrows derived from different F_3 plants but the same F_2 plant ($\sigma^2_{R:FPB}$, Model III) were two (SNLF)- to four (SNEA)-times lower than between the F_2 families ($\sigma^2_{F:P}$).

Values for broad sense heritability (h^2_b) for the SNEA and SNLF of each population are given in Table 7, together with the genetic variance components of SNEA and SNLF. Genetic variances, and consequently heritabilities, are very low in some cases (e.g. Arina×SN– for SNAE, SN+×Iena for SNLF) and very high in others (e.g. SN–×Greif for SNEA, SN–×SN+ for SNLF). Values for heritability differ strongly between SNEA and SNLF for SN+ Zenith, Arina×SN–, Forno×SN– and Boval×Zenith, which is in accordance with the different directions of heterosis for SNEA and SNLF in Table 5.

Figure 1 shows the mean for SNEA of the parents, the F_1 values, the F_4 means and the range in F_4 for all 16 populations in the 1996 field trial. The F_4 mean deviated in most crosses from the midparent value indicating non-additive gene actions. For SNEA, the F_4 generation of nine crosses showed a higher level of resistance than expected from the parental mean, while the F_4 of two crosses were more susceptible. The population mean was in some crosses even outside the range of the parents (e.g. Forno×SN–). Coefficients of correlation between the genetic variance components and the phenotypic differences between the parental lines of each cross were low, r=0.27 (n.s.) for SNEA and somewhat higher for SNLF (r=0.53, $P<0.05$). This indicates that the amount of variability in segregating populations can not be predicted from the parental difference for SNEA or SNLF-severity indices.

Discussion

High correlations were found between the field trial in 1995 and 1996 for SNEA and SNLF of the parental lines (r=0.95 and 0.88, respectively, $P<0.01$). This indicates that the resistance level can be determined in an accurate way on a 1-row and 5-row plot basis after artificial infection and multiple ratings. Multiple recordings are essential for the assessment of disease development (Walther 1990) and for reducing errors in estimating the percentage of the diseased area. Therefore, the resistance level for the F_4 generation of the 16 crosses provides a reliable basis to compare the resistance under field conditions for the susceptibility to *S. nodorum* metabolites in vitro (Wicki et al. 1999).

Heritability estimates for SNEA and SNLF were higher, lower, or comparable to those reported by Brönnimann (1975) and Rosielle and Brown (1980), who screened only a limited number of genotypes. This identifies the necessity to screen a broad genetic spectrum to study inheritance mechanisms. Heritability estimates differed in some cases strongly between SNEA and SNLF (SN+×Zenith, Arina×SN–, Forno×SN– and Boval×Zenith, Table 7), which is in accordance with the different directions of heterosis for SNEA and SNLF (Table 5). The different magnitudes and the differences for SNEA and SNLF of the heritability estimates found in this study reflect the complex inheritance mechanisms of resistance to SNB.

Effects of heterosis in the F_1 were in most cases positive for SNEA and SNLF, i.e. the F_1 was more susceptible than the midparent value, indicating dominance for susceptibility as found by Brönnimann (1975) and Fried and Meister (1987). Heterosis was more important for SNEA than for SNLF. The production of wheat hybrid seeds would, therefore, not be a tool for improving *Septoria* resistance. However, heterosis effects diminish in later generations due to decreasing heterozygosity. It is therefore possible that breeders discard populations in the F_2 that seem to be susceptible. But, as shown before, even in such populations (for example Forno×SN– for SNEA or Iena×Forno for SNLF, Fig. 1) highly resistant genotypes occurred in later generations. However, large populations and strong selection pressure are necessary to identify such genotypes (Fried and Meister 1987).

Although GCA and SCA effects could not be tested for significance, our data indicate that it is not possible to predict the hybrid performance on the basis of the parental resistance reaction.

The association of resistance to tall, late-maturing plants found in this study for the progenies of the 16 crosses (significant negative correlations for both traits) was in agreement with earlier studies about *Septoria* resistance in wheat (Scott et al. 1982). However, for the eight parental lines, no association was found between SNB and plant height. This was due to the two short-strawed cultivars Iena and Greif (95 cm), which both showed a high level of partial resistance on the ears.

The low coefficients of correlation between SNEA and SNLF found in this study lead to the conclusion that

these traits are inherited independently. In the F_1, the correlation is even negative. In the crosses SN+×Iena and SN+×Boval, the F_1 shows positive heterosis for SNLF, but negative heterosis for SNEA (Table 5), thus representing a contrary reaction. Therefore, different genes and/or gene actions seem to be involved in the expression of glume-blotch and leaf-blotch resistance. This is in agreement with the findings of Fried and Meister (1987) that resistance on the heads is inherited independently from resistance on the leaves and that both are inherited quantitatively.

The variation observed in this study both within and among the segregating populations suggest a quantitative inheritance pattern influencing the expression of the two traits. Continuous variations in SNB response have been reported in numerous studies (Mullaney et al. 1982; Ecker et al. 1989; Bostwick et al. 1993; Loughman et al. 1994). The components of variance between F_2 families within a population were as high as (SNEA), or higher (SNLF) than, those between populations. Therefore, a strong selection within a few populations may be as effective to produce new resistant genotypes as selection in a large number of populations. The lower variation within F_3 families (between F_4 headrows) was due to the higher level of homozygosity in this generation. Assuming an additive-dominance model (Mather and Jinks 1977), the components of variance within the F_4 populations tested in 1996 consist of the variance between F_2 families ($=V_A+1/16\ V_D$), (V_A: additive genetic variance of the F_2 generation, V_D: non-additive genetic (dominance) variance of the F_2 generation), the variance between F_3 progenies within F_2 families ($=1/2\ V_A+1/8\ V_D$) and the variance between individuals within F_3 progenies ($1/4\ V_A+1/4\ V_D+V_E$), (V_E: environmental component of the within-family variance), which is in total $7/4\ V_A+7/16\ V_D+V_E$. Therefore, dominance effects should turn out to be most important in the F_1, and moving from the F_1 to the F_4, the dominance variance V_D diminishes from $1V_D$ (F_1) to $7/16\ V_D$ (F_4). The additive-dominance model is adequate to explain variance in *Septoria* resistance, although some variation in the form of an interaction between non-allelic genes may occur. The graphical presentation of the parental lines, the F_1 values and the F_4 means in Figure 1 underlines this theory. In most cases, the F_1 is more susceptible than the F_4 (mean) and is closer to the susceptible parent, indicating that dominance for susceptibility, as found by Brönnimann (1975) and Fried and Meister (1987), is involved in the inheritance. Ecker et al. (1989) suggested that there are more allelic combinations which increase susceptibility than allelic combinations which increase resistance. Nevertheless, for SNLF in three crosses (Iena×Forno, SN+×Iena and SN+×Boval), and for SNEA in one cross (Forno×SN–), dominance for resistance exists. This indicates that dominant genes for resistance against leaf blotch as found by Frecha (1973) at the seedling stage might also be expressed at the adult stage. On the other hand, such genes may be modified by other genes or certain gene combinations as described by Laubscher et al. (1966) and Kleijer et al. (1977).

When breeders want to improve a trait they usually cross two parents that already express this trait at a high level. On the basis of the results of the present study, this strategy may also be promising for breeding for *Septoria* resistance, as shown in Fig. 1 for Zenith×Greif and SNEA or for SN+×Boval and SNLF. But there are also some peculiarities which might be of interest to breeders. The correlation between the genetic component of variance in the F_4 and the phenotypic difference between the parents is low. This leads to the conclusion that, on the basis of the genetic value of the parents, the genetic variability induced by a specific cross can not be predicted. This is demonstrated in Fig. 1. In the cross Forno×SN–, the parents have almost the same value, but the range in the F_4 is large, and even though the parents represent two susceptible genotypes, a selection for a high level of resistance in the F_4 would be possible. This is also true for the cross SN–×SN+. In the cross SN–×Greif, on the other hand, while there is also a large range, selection for higher levels of resistance than the more-resistant parent seems not to be promising for SNEA but only for SNLF. In some crosses, for example Iena×SN– or Arina×Forno, the range of F_4 lines correspond more or less to the phenotypic difference of the parents.

In almost all crosses some progenies were found that were more resistant than the better parent (range in F_4, Fig. 1). The progenies with the highest level on resistance for SNEA were found in the crosses Arina×Iena, Zenith×Greif, and Boval×Iena. This indicates that Arina and Iena, both with a high level of partial resistance on the ear, carry different resistance genes, and that also parental lines with a low level of resistance (Zenith and Boval for SNEA and SN– and Iena for SNLF) contribute positive alleles increasing resistance. Thus transgression breeding can be a tool to breed for higher levels of resistance to SNB.

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