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Inheritance of resistance to *Fusarium graminearum* in wheat

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Abstract To study the inheritance of resistance in wheat to *Fusarium graminearum*, six resistant cultivars from China were crossed to two susceptible cultivars. The parents and their progenies were evaluated in the greenhouse for resistance to the spread of scab within a spike. A central floret was inoculated by injecting a droplet of inoculum at the time of anthesis. Inoculated plants were kept in a moist chamber for three subsequent nights. The proportion of scabbed spikelets was recorded six-times from 3-days to 21-days after inoculation, and the area under the disease progress curve (AUDPC) was calculated from these proportions. One to three genes, depending on the cultivar, conditioned resistance to scab as reflected by the AUDPC. A simple additive-dominance effect model fitted the segregation data for 8 of the 11 crosses. Dominance and epistatic effects were significant in a few crosses. These effects increased resistance in some crosses but decreased resistance in others. However, relative to additive effects, dominant and epistatic effects accounted for only a small portion of the genetic effects in the populations evaluated. The importance of additive effects means that it should be possible to accumulate different genes to enhance resistance to scab in wheat.

Key words Fusarium head blight · Partial resistance · Quantitative resistance

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Introduction

Scab of wheat, caused mainly by *Fusarium graminearum*, is a destructive disease of wheat (*Triticum aestivum* L.) (Schroeder and Christensen 1963). In recent years, several severe scab epidemics have occurred in North America with losses in excess of $1 billion a year (McMullen et al. 1997). Yield loss results directly from shrivelled grain, which either is blown away during harvest, or has a lighter test weight. Infected grain may also germinate poorly, resulting in seedling blight and a poor stand when it is used as seed (Bai and Shaner 1994). Infected grain may also contain mycotoxins that are toxic to humans and livestock.

The development of disease-resistant cultivars will probably be the most effective strategy to control scab. Wheat scab resistance consists of at least two components: resistance to initial infection and resistance to the spread of the fungus within a spike (Schroeder and Christensen 1963). Resistance to the spread of the fungus within a spike is a relatively stable character and less affected by the environment than is resistance to the initial infection (Bai and Shaner 1994, 1996). In the field, some susceptible cultivars may escape scab because the weather when they are flowering is not conducive to infection, and therefore they appear to be resistant. Uniform inoculation of plants is essential for clear differentiation of resistance to scab spread within a spike among cultivars. To-date, most of the information available about the genetic control of scab resistance is based on field inoculation in which disease severity was probably a function of considerable environmental variance. In terms of these data, several investigators concluded that resistance to scab spread within a spike is controlled by many minor genes (Chen 1983; Liao and Yu 1985; Snijders 1990a, b, d), whereas others concluded that resistance is under the control of a few genes (Bai and Xiao 1989; Bai et al. 1990;Van Ginkle et al. 1996; Yao et al. 1997).

To limit environmental effects, Wang et al. (1982) proposed an in vitro inoculation technique in which de-

tached wheat spikes were inoculated by placing spores in a central spikelet of the spike, and were then cultured in containers with sterile water in a growth chamber with controlled temperature and moisture. With this technique scab symptoms may be confounded by discoloration due to senescence of the detached spike.

Evaluating resistance on plants in the greenhouse can minimize non-genetic variation because the environment can be controlled to a greater extent than in the field, while still allowing the disease to develop in living plants. Since resistance to the spread of *F. graminearum* within a spike has a relatively low environmental variance compared to genetic variance, we evaluated this resistance in our study. The area under the disease progress curve, derived from sequential assessments of the proportion of scabbed spikelets, was used to estimate the genetic parameters associated with resistance. In this paper we report that resistance to the spread of scab symptoms within a spike is mainly controlled by additive gene action, though in a few instances epistatic effects were also noted.

Materials and Methods

Plant materials

Eight wheat cultivars were selected to represent various degrees of resistance to scab (Table 1). The resistant cultivars were crossed to the susceptible cultivars Morocco or Clark in the spring of 1991. Subsequently, F_1s were selfed and backcrossed to their respective parents to produce the F_2 and backcross generations.

The parents, F_1, F_2, BC_1 (backcross of F_1 to resistant parent) and BC_2 (backcross of F_1 to susceptible parent) progenies of 11 crosses were evaluated for scab resistance in the greenhouse in the fall of 1992. Plants of the 11 crosses were randomized on greenhouse benches, and the plants from different generations were randomly arranged within each cross. To evaluate non-heritable effects, eight parents were tested in 1991, 1992 and 1993 (see Table 2), and the F_1s were tested in 1991 and 1992.

In 1992, 175 F_2 plants from the cross Ning 7840/Clark were randomly selected to develop recombinant inbred lines in the greenhouse and growth chamber by single-seed descent. In the spring of 1994 and subsequent greenhouse crop cycles, 9–16 plants per family from the F_5–F_7 and F_{10} progenies were grown and tested for scab resistance in the greenhouse.

In all the trials, seeds were planted in 55 cm×37 cm×7 cm-flats of soil. After seeds germinated, the winter cultivar Clark and its progenies were vernalized at 4°C for 8 weeks, and spring-habit cultivars and those populations derived from crosses with the cultivar Morocco were vernalized for 6 weeks. Following vernalization, seedlings were transplanted to 10-cm-diameter plastic pots filled with a mixture of clay loam soil and peat. Plants were fertilized with urea (46–0-0) 6 and 20 days after transplanting and then grown in the greenhouse with supplemental fluorescent lights (VHO cool white 215 W bulb) set for a 12-h photoperiod at the early stages of growth and a 16-h photoperiod after booting.

Inoculation and disease assessment

The inoculum of *F. graminearum* was a mixture of field isolates that originated from ten randomly selected scabbed seeds of the susceptible cultivar Caldwell grown at the Purdue Agricultural Research Center in 1986. Mung bean liquor medium was used to produce the conidial inoculum. Mung bean liquor was obtained by placing 40 g of mung beans in 1 l of boiling water. The mung beans were steamed for 10 min, and then the broth was filtered through cheesecloth. To prepare the growth medium, 100 ml of the broth was autoclaved in 250-ml Erlenmeyer flasks. The flasks were inoculated with the mycelium of *F. graminearum* and then placed on a shaker for 4 days at 23–25°C to allow conidia to develop. Conidial suspensions were diluted with distilled water to a final concentration of 50000 conidia per ml.

A droplet of conidia (about 1000 spores) was injected with a hypodermic syringe into a central floret of a wheat spike at early anthesis. Inoculations were made from 1400 to 1600 h each day. To insure a high relative humidity after inoculation, we sprayed the inoculated plants with tap water and then placed them in a chamber consisting of a pipe-frame on the greenhouse bench covered with polyethylene sheeting. Temperatures within the moist chamber were 23–25°C and relative humidity was 100%. Plants were kept in this chamber for three consecutive nights after inoculation, from 1600 h in the evening to 0800 h the next morning. During the day, from 0800 h to 1600 h, the chamber was opened. On the 4th day after inoculation, plants were returned to their original positions on the greenhouse bench. The greenhouse temperature averaged 23°C during the day with a range of 19–30°C, and averaged 19°C at night with a range of 17–21°C.

Symptoms on spikes varied from light brown, water-soaked spots on the glumes to bleached spikelets. Spikelets with symptoms were counted 3, 9, 12, 15, 18, and 21 days after inoculation. On the 21st day, all spikelets on each inoculated spike were counted. Disease severity was expressed as the proportion of scabbed spikelets per infected spike. From these proportions we calculated an area under the disease progress curve (AUDPC) for each plant according to Shaner and Finney (1977).

Table 1 Origin, parentage and response of eight wheat cultivars[a] to infection by *F. graminearum*

Cultivar	Origin	Parentage	Reaction[b]
Fu 5114	Fujing, China	Fufan 904/Ning 8017	R
Ning 7840	Nanjing, China	Aurora/Anhui 11//Sumai 3	R
Ning 8331	Nanjing, China	Yangmai 4/Ning 7071	R-MR
Ning 8306	Nanjing, China	263/Fanxiao 5//Ning7302 /Ning 7084/Yangmai 3	MR
Sumai 3	Suzhou, China	Funo/Taiwanmai	R
Sumai 49	Suzhou, China	Su 7922/Ning 7840	R
Morocco	Morocco		MS
Clark[c]	Indiana, USA		S

[a] Resistant cultivars were all provided by Jiangsu Academy of Agricultural Sciences, Nanjing, People's Republic of China, and susceptible cultivars were provided by Purdue University
[b] Cultivar reaction to scab was based on Bai and Shaner (1996). R, MR, MS and S represent resistant, moderately resistant, moderately susceptible and susceptible
[c] See Crop Science (1988) 28:1032

Genetic analysis

Data on scab resistance to fungal spread within a spike were analyzed on a single-plant basis. To compare the resistance among parents, one-way analysis of variance (ANOVA) with an unequal subsample number was conducted (Steel and Torrie 1980). F_5, F_6, F_7 and F_{10} recombinant inbred lines from the cross Ning 7840/Clark were used to calculate heritability based on ANOVA (Steel and Torrie 1980). The minimum number of genes controlling scab resistance in each cross was estimated according to Wright's formulae (Wright 1968).

Joint scaling tests were used to estimate values for genetic effects that best explain differences among generation means based on the means and variances of parental, F_1, F_2, and backcross generations (Mather and Jinks 1982). The additive-dominance model estimates the mid-parent value (m), the additive genetic effect (d),

Table 2 Resistance to the spread of *F. graminearum* within a wheat spike expressed as the area under the disease progress curves[a] of eight wheat cultivars in three experiments

Cultivar	No. of plants	Experiment[b]			Mean[c]
		I	II	III	
Clark	135	9.89±0.59	13.0±0.21	9.99±0.44	10.91[c]
Morocco	83	4.36±0.68	7.84±0.55	8.06±0.70	6.96[b]
Fu 5114	96	1.11±0.16	0.76±0.06	0.82±0.12	0.84[a]
Ning 7840	96	0.85±0.10	0.92±0.11	0.71±0.07	0.80[a]
Ning 8306	65	1.03±0.18	2.33±0.38	3.03±0.48	2.13[a]
Ning 8331	53	0.77±0.11	1.31±0.19	2.83±0.26	1.47[a]
Sumai 3	62	0.71±0.16	1.06±0.09	0.76±0.09	0.92[a]
Sumai 49	87	0.92±0.10	0.75±0.04	0.6±0.00	0.76[a]

[a] Area under the disease progress curve based on the proportion of scabbed spikelets per spike counted at 3-day intervals from 3 to 21 days after inoculation. See text for details. Data within each experiment are the mean and standard error

[b] Means and standard errors for Experiment I (1991), Experiment II (1992) and Experiment III (1993)

[c] Means over three trials followed by a letter in common are not significantly different at P=0.05 according to Fisher's Protected LSD test.

Table 3 Generation-means for reaction to wheat scab caused by *F. graminearum* in 11 crosses of wheat cultivars

Cross	Progeny[a]				
	MP	F_1	F_2	BC_1	BC_2
Fu 5114/Clark	5.88	6.15±0.45 (57)	8.10±0.34 (138)	5.02±0.62 (34)	9.72±0.59 (46)
Fu 5114/Morocco	3.90	4.92±0.90 (19)	4.90±0.35 (118)	2.96±0.50 (23)	6.81±0.68 (30)
Ning 7840/Clark	5.86	5.18±0.68 (101)	5.36±0.27 (222)	2.68±0.20 (165)	9.15±0.33 (162)
Ning 8306/Clark	6.52	6.33±1.21 (13)	9.13±0.38 (123)	6.70±1.12 (17)	10.89±0.55 (54)
Ning 8306/Morocco	4.55	5.61±0.91 (20)	6.40±0.88 (29)	3.76±0.62 (29)	6.89±0.64 (34)
Ning 8331/Clark	6.19	7.16±0.90 (24)	6.55±0.41 (116)	6.22±0.60 (48)	11.48±0.38 (74)
Ning 8331/Morocco	4.22	5.79±0.75 (28)	5.02±0.40 (119)	3.23±0.51 (34)	6.27±0.59 (37)
Sumai 3/Clark	5.92	6.498±0.73 (21)	7.56±0.30 (139)	–[b]	8.68±0.55 (50)
Sumai 3/Morocco	3.94	5.32±0.51 (20)	4.45±0.31 (126)	–[b]	7.33±0.88 (23)
Sumai 49/Clark	5.84	4.65±0.39 (77)	6.14±0.34 (157)	2.66±0.40 (48)	9.22±0.48 (63)
Sumai 49/Morocco	3.86	4.35±0.69 (20)	4.0±0.32 (111)	1.93±0.29 (34)	5.82±0.77 (21)

[a] Mean AUDPC (area under disease progress curve) and standard errors for 11 crosses. Values in parenthesis are the sample sizes for each population. MP=mid-parental value, calculated as $(P_1+P_2)/2$, in which P_1 is the AUDPC of the resistant parent and P_2 is the AUDPC of the susceptible parent. P_1 and P_2 are based on the means of three trials. BC_1 and BC_2 represent backcrosses of the F_1 to resistant and susceptible parents, respectively. F_1 data are the average of two trials

[b] Data for the backcross of the F_1 to the resistant parent were not available

and the dominance effect (h) without considering epistasis. This model describes the allelic interactions within a locus. For more-complicated cases, non-allelic interactions between pairs of loci may play a significant role. In epistatic models, in addition to estimates of (m), (d) and (h), one or more digenic interaction effects of a cross might also be included: the additive × additive effect (i), the additive x dominance effects (j), and the dominance x dominance effects (l). We used all of these effects to fit genetic models to the data. The genetic effects estimated reflected the net effect of all the loci at which the parents differed for scab reaction as measured in our experiments. Since the parents in a cross might differ at several loci, and dominance within these loci and epistasis among these loci might differ, genetic effects can be re-defined as the net directional effects of all relevant loci. These net effects are symbolized as [d], [h], [i], [j], [l]. In the joint scaling tests, the mid-parent values (m) are estimated from the mean values for all homozygous individuals in the parental and segregating generations. Chi-square tests were used to determine how well a particular model fit the data. Effects within a model that fit the data were evaluated for significance with the Z value.

Results

Resistance of parents and F_1 progenies

Variance analysis indicated that parents differed in resistance to scab spread within a spike over three trials (Table 2). Ning 7840, Sumai 3, Sumai 49, and Fu 5114 showed the consistently lowest AUDPCs. In most plants of these cultivars symptoms were restricted to the inoculated spikelet without spread to uninoculated spikelets in the same spike, and variation in AUDPC among the trials was least for those cultivars. Because Ning 8331 and Ning 8306 had relatively higher AUDPC values and larger variation within and among the trials compared to highly resistant cultivars, they are regarded as moderately resistant though the difference was not significant. The AUDPC value in the moderately susceptible cultivar Morocco was at least three-times higher than that for resistant cultivars. Morocco also had greatest variation of AUDPC. Clark had the consistently highest

Table 4 Genetic effects for resistance to *F. graminearum* in 11 crosses involving eight wheat cultivars

Cross	Model fit[a]	Chi-square value	*P*-value[b]	Component fit[c]
Fu 5114/Clark	m[d][h]	12.15	<0.01	
	m[d][h][i]	2.81	>0.10	m[d][h][i]
	m[d][h][j]	9.24	<0.01	
	m[d][h][l]	4.70	>0.05	m[d][h][l]
Fu 5114/Morocco	m[d][h]	0.23	>0.95	m[d]
	m[d][h][i]	0.20	>0.98	m[d]
	m[d][h][j]	0.07	>0.98	m[d]
	m[d][h][l]	0.14	>0.90	m[d]
Ning 7840/Clark	m[d][h]	5.91	>0.10	m[d][h]
	m[d][h][i]	1.24	>0.5	m[d][i]
	m[d][h][j]	4.80	>0.05	m[d][h]
	m[d][h][l]	3.18	>0.10	m[d][h]
Ning 8306/Clark	m[d][h]	2.29	>0.50	m[d][h]
	m[d][h][i]	1.07	>0.50	m[d]
	m[d][h][j]	0.69	>0.50	m[d][h]
	m[d][h][l]	2.04	>0.10	m[d]
Ning 8306/Morocco	m[d][h]	1.52	>0.50	m[d]
	m[d][h][i]	1.44	>0.25	m[d]
	m[d][h][j]	1.33	>0.50	m[d]
	m[d][h][l]	1.35	>0.25	m[d]
Ning 8331/Clark	m[d][h]	18.46	<0.01	
	m[d][h][i]	9.27	<0.01	
	m[d][h][j]	18.41	<0.01	
	m[d][h][l]	17.97	<0.01	
Ning 8331/Morocco	m[d][h]	2.71	>0.25	m[d]
	m[d][h][i]	1.65	>0.25	m[d]
	m[d][h][j]	2.71	>0.25	m[d]
	m[d][h][l]	0.29	>0.75	m[d]
Sumai 3/Clark	m[d][h]	5.92	>0.05	m[d]
	m[d][h][i]	8.97	<0.01	
	m[d][h][j]	0.00	>0.98	m[d][j]
	m[d][h][l]	8.97	<0.01	
Sumai 3/Morocco	m[d][h]	9.49	<0.01	
	m[d][h][i]	0.52	>0.25	m[d][h][i]
	m[d][h][j]	5.16	<0.05	
	m[d][h][l]	1.59	>0.25	m[d][l]
Sumai 49/Clark	m[d][h]	1.49	>0.50	m[d][h]
	m[d][h][i]	0.43	>0.75	m[d][h]
	m[d][h][j]	1.13	>0.50	m[d][h]
	m[d][h][l]	0.69	>0.50	m[d]
Sumai 49/Morocco	m[d][h]	2.30	>0.50	m[d]
	m[d][h][i]	2.07	>0.25	m[d]
	m[d][h][j]	1.29	>0.50	m[d]
	m[d][h][l]	0.50	>0.75	m[d]

[a] m=estimated mean of all homozygous individuals, [d] =additive effect, [h] =dominance effect, [i] =additive x additive epistatic effect, [j] =additive×dominance interaction effect and [l] =dominance×dominance interaction effect

[b] Models having chi-square values with $P>0.05$ are considered to fit the data

[c] The genetic effects listed differed significantly from zero at $P=0.05$ according to the Z-test and thus contributed significantly to the model

AUDPC. The difference in AUDPC among trials was not significant and the correlation of AUDPCs among the trials was high (0.97). The technique used in this study to assess wheat resistance to scab spread within a spike appears to be reliable.

F_1 means were similar to, or higher than, the means of the midparents except for the crosses Ning 7840/Clark and Sumai 49 /Clark, indicating that resistance to scab was partially dominant in Ning 7840/Clark and Sumai 49/Clark, and partially recessive in most other crosses (Table 3). The standard error for the F_1 (Table 3) was higher than that for parents (Table 2) in most crosses. There was a low correlation between mean and variance among F_1s ($r=0.47$, $df=20$) and parents ($r=0.59$, $df=22$), and a log-transformation of AUDPC did not eliminate this weak association. Therefore, untransformed data were analyzed.

Generation-mean analysis

In general, the order of generation-mean AUDPCs from resistant to susceptible were resistant parent, BC_1 (F_1 backcross to resistant parent), F_1 or F_2, BC_2 (F_1 backcross to susceptible parent), and susceptible parent (Tables 2 and 3). For 8 of the 11 crosses, the simple additive-dominance effect model (m [d] [h]) explained the inheritance of scab resistance (Table 4). In this model, the additive gene effect, which increased resistance, was highly significant for all crosses (Table 5). The dominance effect was significant in three of eight crosses. The significant positive dominance effect in the Ning 8306/Clark cross and the negative dominance effect in the crosses Ning 7840/Clark and Sumai 49/Clark indicates the F_1s were more like the susceptible parent than the resistant parent in Ning 8306/Clark, but opposite in the other two crosses.

Table 5 Significance of the genetic effects in the additive×dominance model with a chi-square probability $P>0.05$ for the inheritance of resistance to *F. graminearum* in wheat

Cross	Component[a]	Estimate[b]	Z-value[c]	*P* value[d]
Fu 5114/Morocco	m 4.32±0.26	16.74	<0.01	
	[d]	–3.56±0.26	–13.85	<0.01
	[h]	1.17±0.67	1.75	0.08
Ning 8306/Morocco	m 5.07±0.32	15.76	<0.01	
	[d]	–2.79±0.31	– 9.00	<0.01
	[h]	1.09±0.88	1.24	0.22
Ning 8331/Morocco	m 4.46±0.27	16.44	<0.01	
	[d]	–3.17±0.27	–11.87	<0.01
	[h]	1.29±0.67	1.95	0.051
Ning 8306/Clark	m 7.73±0.21	36.28	<0.01	
	[d]	–5.29±0.21	–25.07	<0.01
	[h]	2.19±0.69	3.17	<0.01
Ning 7840/Clark	m 6.96±0.11	61.58	<0.01	
	[d]	–6.07±0.11	–54.68	<0.01
	[h]	–2.20±0.28	– 7.86	<0.01
Sumai 3/Clark	m 7.02±0.11	64.11	<0.01	
	[d]	–5.95±0.11	–54.34	<0.01
	[h]	0.40±0.51	0.78	0.44
Sumai 49/Clark	m 6.92±0.10	67.83	<0.01	
	[d]	–6.17±0.10	–60.49	<0.01
	[h]	–2.10±0.37	– 5.68	<0.01
Sumai 49/Morocco	m 4.33±0.25	17.24	<0.01	
	[d]	–3.58±0.25	–14.26	<0.01
	[h]	–0.80±0.54	– 1.48	0.14

[a] m=mean, [d]=additive effect, [h]=dominance effect
[b] Estimates of genetic effects and standard errors
[c] Ratio of the estimate to its standard error that measures whether the parameter was significantly different from zero
[d] The genetic effect was considered significant and highly significant if the Z-value had a $P=0.05$ and $P=0.01$, respectively

Table 6 Significance of the genetic effects for resistance in wheat to *F. graminearum* in models with a chi-square $P=0.05$ and an interaction component that was significantly different from zero at $P=0.05$

Cross	Model fit	Component fit	Estimate[a]	Z-value	*P*-value[b]
Fu 5114/Clark	m[d][h][i]	m	9.64±0.90	10.75	<0.01
		[d]	–6.12±0.11	–57.72	<0.01
		[h]	–3.11±1.35	–2.31	0.04
		[i]	–2.76±0.90	–3.06	<0.01
	m[d][h][l]	m	6.88±0.11	64.70	<0.01
		[d]	–6.12±0.11	–57.60	<0.01
		[h]	4.02±1.26	3.19	<0.01
		[l]	–4.35±1.59	–2.73	<0.01
Ning 7840/Clark	m[d][h][i]	m	5.62±0.63	8.94	<0.001
		[d]	–6.09±0.11	–54.60	<0.01
		[h]	–0.38±0.90	–0.42	0.69
		[i]	1.39±0.64	2.16	0.03
Sumai 3/Clark	m[d][h][j]	m	7.05±0.11	62.98	<0.01
		[d]	–5.99±0.11	–53.38	<0.01
		[h]	1.01±0.55	1.85	0.06
		[j]	7.49±2.43	3.08	<0.01
Sumai 3/Morocco	m[d][h][i]	m	2.39±0.86	2.77	<0.01
		[d]	–3.43±0.27	–12.73	<0.01
		[h]	4.19±1.36	3.08	<0.01
		[i]	2.10±0.9	2.32	0.02
	m[d][h][l]	m	4.50±0.27	16.43	<0.01
		[d]	–3.44±0.27	–12.58	<0.01
		[h]	–1.70±1.58	–1.08	0.27
		[1]	3.71±1.78	2.08	0.04

[a] Estimate of genetic effects and standard error
[b] The genetic effect was considered significant and highly significant if the Z-value had a $P\leq0.05$ and $P\leq0.01$, respectively

More complex models with epistatic effects could also explain the data for most crosses (Table 4). All three digenic interaction models were able to explain the inheritance of scab resistance in all crosses with Ning 7840, Ning 8306, and Sumai 49 as the resistant parent, as well as in crosses of Morocco with Ning 8331 and Fu 5114. The crosses Fu 5114/Clark and Sumai 3/ Morocco, for which the simple model was inadequate, were fit by the additive-additive epistatic model (m[d][h][i]) and by the dominance-dominance epistatic model (m[d][h][l]). None of the models fit the data for the cross Ning 8331/ Clark. Four crosses were best fit by the additive×additive epistatic model (m[d][h][i]), two crosses were best fit by the dominance-dominance epistatic model (m[d][h][l]), and four crosses were best fit by the additive×dominance epistatic model (m[d][h][j]).

Of the 26 combinations of the epistatic model and cross with a chi-square probability >0.05 (Table 4), only

Table 7 Estimates of the minimum number of genes for resistance to spread of *F. graminearum* within a wheat spike as measured by the area under the disease progress curve

Cross	n_1[a]	n_2[b]	Mean
Fu 5114/Clark	1.10	1.56	1.33
Fu 5114/Morocco	0.39	0.89	0.64
Ning 7840/Clark	1.1	1.45	1.28
Ning 8306/Clark	0.74	1.58	1.16
Ning 8306/Morocco	0.15	0.23	0.19
Ning 8331/Clark	0.76	1.48	1.12
Ning 8331/Morocco	0.24	0.40	0.33
Sumai 3/Clark	1.60	2.75	2.18
Sumai 3/Morocco	0.47	0.56	0.52
Sumai 49/Clark	0.93	1.25	1.09
Sumai 49/Morocco	0.54	0.92	0.73

[a] Minimum gene number $n_1=(P_1-P_2)^2/8\{V_{F2}-[(V_{P1}+V_{P2})/2]\}$
[b] Minimum gene number $n_2=(P_1-P_2)^2/8\{V_{F2}-[(V_{P1}+V_{P2}+2V_{F1})/4]\}$
In the formulas above, V_i is the variance for the population designated by the subscript, P_1 and P_2 are the AUDPC values for the resistant and susceptible parent, respectively

six had significant epistatic effects (Table 6). A significant additive-additive epistatic effect occurred in three crosses. The dominance-dominance epistatic effect was highly significant in the crosses Fu 5114/Clark and Sumai 3/Morocco. A significant additive-dominance epistatic effect was detected only in the cross Sumai 3 /Clark. Four of the six significant epistatic effects were positive, meaning that the epistatic effects led to a higher AUDPC value. Only two negative epistatic effects were detected, in the cross Fu 5114/Clark. All of these findings indicate that mainly additive genetic effects control wheat scab resistance in these crosses. Dominance and epistasis, mainly additive-additive, could be detected in a few crosses.

Heritability and the number of resistance genes

Heritabilties were high, ranging from 0.91, 0.80, 0.87 and 0.87 for the F_5, F_6, F_7 and F_{10} generations, respectively. When the variances of parents were used to estimate environmental variance, the number of resistance "genes" involved in the crosses was one or two (Table 7). When the weighted variances of parents and the F_1 were used to estimate the environmental variance, similar estimates of gene number were obtained except that three genes were estimated in Sumai3/Clark cross. Estimates of gene number varied for a resistant parent according to which susceptible parent it was crossed. There was an estimate of one gene less in crosses with Morocco compared to those with Clark as a parent. Two genes controlled scab resistance in most crosses, with a range of one to three.

Although segregation in the F_2 population showed a continuous distribution, there were three peaks which usually coincided with the F_1 and each parent (Fig. 1B). The ratio of resistant, or moderately resistant, plants to susceptible plants approximately fit the pattern expected

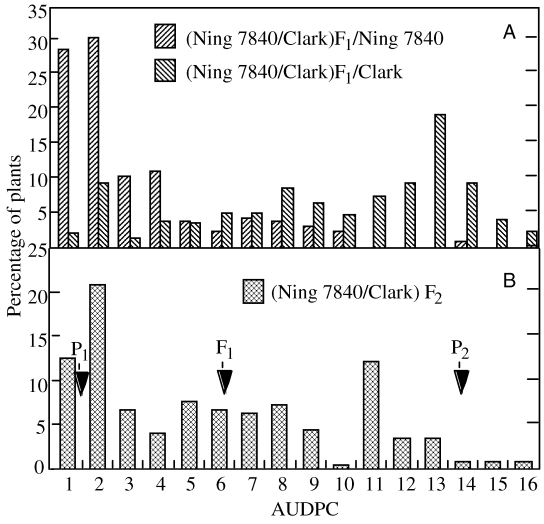

Fig. 1A, B Distribution of the area under the disease progress curve (AUDPC) for cross Ning7840/Clark. **A** (top). Distributions for the backcross to each parent; **B** (bottom). distribution for the F_2. "F_1" indicates the mean AUDPC of wheat scab in the F_1 population. "P_1" and "P_2" indicate the means of Ning7840 and Clark, respectively

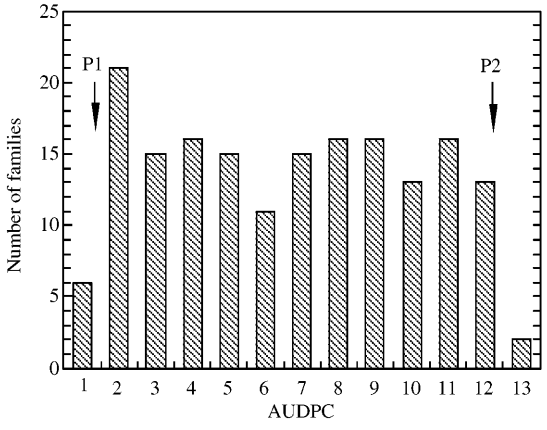

Fig. 2 Distribution of area under the disease progress curve (AUDPC) for F_7 families from cross Ning7840/ Clark. "P_1" and "P_2" indicate the means of Ning7840 and Clark, respectively

if one or two genes for resistance were segregating. In the backcross to the susceptible parent, a similar distribution pattern was observed, but the ratio was different (Fig. 1A). In the BC_1, almost all the plants were resistant or moderately resistant. The interpretation of data from backcross populations was consistent with the hypothesis based on F_2 populations.

The frequency distribution for F_7 families of the cross Ning 7840/Clark was continuous with two major peaks (Fig. 2). The ratio of the frequency of families associated with the two peaks was consistent with 1:1 segregation in a chi-square test (χ^2=0.28, *df*=1, *P*>0.5). In this model, one major gene with some other minor modifier genes can be assumed to control scab resistance in this cross. Two minor peaks can be identified within each major peak. The

segregation ratio for the four peaks is about 1:3:3:1 (χ^2=4.9, *df*=3, *P*>0.1), suggesting the segregation of three genes with one gene having a major effect on scab resistance. The continuous distribution of AUDPC indicates that this trait is quantitative and is affected not only by modifying genes but also by environment. The interpretation of data from the other three generations was consistent with the hypothesis based on F_7 populations.

Discussion

Uniform inoculation of plants under a controlled environment is essential for clear differentiation of resistance to scab-spread within a spike among plant genotypes. In this study, to limit environmental variation, plants were inoculated in the greenhouse by injecting approximately 1000 spores into one floret of a central spikelet in each spike. The area under the disease progress curve (AUDPC) was used to measure the overall performance of a genotype during the disease cycle. Cultivars regarded as resistant, based on performance in the field in China, consistently showed the lowest AUDPCs in the three tests. The symptoms were restricted to the inoculated spikelets for most resistant plants. Susceptible cultivar Clark showed the highest AUDPC value. The rank of resistance level for the eight cultivars was consistent over tests, similar to the results obtained in our previous work (Bai and Shaner 1996). High heritability and highly significant correlations among the trials demonstrate that the technique we used to evaluate resistance to scab spread within a spike is reliable.

The inheritance of resistance to the spread of scab after initial infection was mainly additive because the data from eight crosses were consistent with a simple additive-dominant effect model (m [d][h]), and the additive effect was highly significant and accounted for most of the genetic variation in the model. Our results are consistent with those of Chen (1983) and Snijders (1990 b).

Resistance genes in Ning 7840 and Sumai 49 were partially dominant, whereas resistance genes in the other parents were mainly additive to partially recessive in this study. Among eight crosses for which the simple model was adequate, Ning 7840/Clark and Sumai 49/Clark had a negative dominance effect, meaning that resistance was partially dominant. This agrees with some previous studies (Chen 1983; Liao and Yu 1985; Bai and Xiao 1989; Bai et al. 1990). For most crosses in our study, resistance was partially recessive, but the dominance effect was not significant for those crosses. The degree of dominance for resistance may vary with the genes from different resistance sources, as well as the genetic background in which the genes occur and the environmental conditions that exist when the phenotype is assessed.

Epistasis was detected in some crosses. A simple additive-dominance model did not fit the crosses Fu 5114/Clark and Sumai 3/Morocco. The model assuming additive x additive interaction gave the best fit in these two crosses even though the dominance× dominance epistatic effect was also significant. Dominance and additive×additive epistasis conferred resistance in the cross Fu 5114/Clark but susceptibility in Sumai 3/Morocco. Snijders (1990 b) was aware that epistasis might occur in some crosses, but did not elaborate on the fact. As far as we are aware, the present study is the first to quantify epistasis for scab resistance**.** That epistasis conferred susceptibility suggests that a combination of resistance genes may not increase resistance as expected in some crosses. However, epistasis could only be detected in a few crosses and explained only a small portion of the genetic variation. Therefore, it may not be a serious problem to accumulate resistance from different sources. In the cross Ning 8331/Clark, the lack of fit of any of the four models may be due to a more-complex genetic control of resistance.

Two genes were estimated with Wright's formulas to account for most of the scab resistance in the various cultivars we studied. Three genes were estimated for resistance in the cross Sumai 3/Clark. It must be borne in mind that Wright's equation may underestimate the number of genes that govern a trait. First, the equation only estimates the minimum number of genes controlling a given trait. Second, the assumptions for the equation may not all be met in this study. The evidence for epistasis in some crosses violates Wright's assumption of no epistasis. Clark can be considered an extremely susceptible genotype, so crosses with this cultivar basically meet the assumption of a unidirectional distribution of genes. Severity on Morocco was less than severity on Clark, and crosses of resistant cultivars to Morocco indicated one fewer gene segregating than in crosses with Clark. Thus, Morocco may have one resistance gene and may violate the assumption of a unidirectional distribution of genes. In addition, the assumption of an equal gene effect may not be true in some crosses, as indicated in the Ning 7840/Clark cross.

Some earlier studies provided evidence for the oligogenic control of resistance (Bai and Xiao 1989; Bai, et al. 1990; Yu 1990, Van Ginkle et al. 1996). Snijders (1990b) investigated 23 crosses and found that in 15 of them the estimates of gene number were less than two. Li and Yu (1988) reported that two resistance genes in the cultivar WZHHS were effective through all disease developmental stages. Bai and others (Bai and Xiao 1989; Bai et al. 1990) concluded that resistance to Nanjing isolates of *F. graminearum* in the field at Nanjing, China, might be controlled by two to three major genes with some modifying genes. Yu (1990) also reported that one resistance gene on chromosome 3A was responsible for resistance to scab in the cultivar YGFZ. More recently, Van Ginkle et al. (1996) evaluated recombinant inbred lines from crosses involving Ning 7840 or Frontana as one of the parents in the field. They concluded that two genes controlled scab resistance in each cultivar. In our study, the continuous distribution of progenies made it difficult to find discrete classes in segregating populations, but the segregating populations for most crosses did show a trimodal distribution. The data from different

segregating populations provided evidence to support the hypothesis that one to three genes govern scab resistance. Wright's formulae gave a similar estimate. In the cross Ning 7840/Clark, data from the F5, F6, F_7 and F_{10} families indicated that Ning 7840 has three genes for resistance. One gene may have a major effect and others may have a modifying effect. According to our data and some other studies, we conclude that most probably one to three genes control scab resistance.

From an analysis of all crosses, we conclude that additive effects account for most of the genetic variance for scab resistance, although dominance and epistasis are also important in some crosses. It should be possible to combine resistance genes from different sources into a single cultivar by selecting from transgressive segregants**.** Liu and Wang (1991) reviewed the progress in China toward breeding cultivars for scab resistance. They found that one-tenth of the lines with resistance to scab were selected from transgressive segregants. Sumai 3, a well-known resistant cultivar in China, was derived from a simple cross of two moderately susceptible cultivars, Funo and Taiwanmai. Snijders (1990c) presented evidence for the transgressive segregation of resistance in an F_2 population. Since only a few cultivars have a high degree of resistance, and these materials have many other undesired traits, the use of resistance genes from moderately resistant or moderately susceptible cultivars in a breeding program may permit combining different resistance genes in a genetic background that results in desired agronomic traits.

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