The Relationships Among Plant Stature, Maturity Class, and Susceptibility to Septoria Leaf Blotch of Wheat

Tamar Danon, J. M. Sacks, and Z. Eyal

Former graduate research assistant, Division of Mycology and Plant Pathology, Department of Botany, The George S. Wise Faculty of Sciences, Tel Aviv University; geneticist, Division of Statistics and Experimental Design, Agricultural Research Organization, Volcani Center, Bet Dagan, Israel; and associate professor, Division of Mycology and Plant Pathology, Department of Botany, The George S. Wise Faculty of Life Sciences, Tel Aviv University, Tel Aviv, Israel, respectively.
This research was supported by Grant 1514 from the U.S.-Israel Binational Science Foundation (BSF), Jerusalem, Israel.
Appreciation for technical assistance is expressed to U. Cohen.
Accepted for publication 4 December 1981.

ABSTRACT

Danon, T., Sacks, J. M., and Eyal, Z. 1982. The relationships among plant stature, maturity class, and susceptibility to Septoria leaf blotch of wheat. Phytopathology 72:1037-1042.

The relationships between Septoria leaf blotch severity and plant stature, and maturity class were investigated in parental, F_1, and F_2 populations, and selected lines derived from crosses among 16 parents in an incomplete diallel crossing scheme. Resistant parents were selected among tall (120–145 cm) late maturing wheats, semidwarf early and late maturing wheats, and winter wheat cultivars; susceptible parents included semidwarf (90–110 cm) and dwarf (45–85 cm) cultivars. Based on the analyses of F_1 and F_2 progeny, we concluded that resistance to the virulent *Septoria tritici* isolate IS 398A1 in the winter wheat cultivar Bezostaya 1, the tall late-maturing spring wheats (cultivars Colotana, Fortaleza-1, Polk/Waldron, Sheridan, and Titan), and in the winter wheat cultivar Oasis is controlled by relatively few genes. Cultivars Chris Mutant and Olaf have moderate resistance that appears to be simply inherited. Disease expression in F_1 and F_2 populations of certain crosses may be dictated in part by modifying genes. Small negative correlations were found between plant height and pycnidial coverage in F_2 populations. The low correlations between plant height and severity of Septoria leaf blotch do not support the hypothesis for linkage or pleiotropy between short-stature and susceptibility. The moderate negative correlation ($r = -0.30$) between heading date and resistance in the F_2 population might suggest linkage. Short-statured (50–80 cm), early-maturing (heading <110 days from seedling emergence), resistant plants were recovered in F_2 populations and continued to express resistance to a wide spectrum of virulence in succeeding generations. The implications of these studies on the incorporation of resistance conditioned by different genetic factors in agronomically suitable wheats is discussed.

Additional key words: genetics, *Triticum aestivum*.

Septoria leaf blotch of wheat, a disease caused by *Septoria tritici* Rob. ex Desm. (perfect state: *Mycosphaerella graminicola* (Fuckel) Schroeter), is a major wheat disease in many parts of the world and it causes severe yield reductions (3,12,15,17). The increase in importance of the disease is largely due to the widespread and rapid replacement over large growing areas of tall, susceptible local cultivars by the high-yielding, early maturing, short-strawed wheat cultivars that are susceptible to the pathogen. Severe leaf blotch epidemics in plantings of some vulnerable commercial wheat cultivars cause 30–50% losses in yield. The resulting grain is shriveled and unfit for milling (3).

Breeding for resistance is the most economically feasible disease control measure. However, resistant germ plasm is not abundant, and little is known about the types of resistance and about their inheritance, manipulation, and accumulation. These difficulties, together with the presence of physiological specialization in *S. tritici* (3,4,11), constitute major obstacles in breeding for resistance.

Resistance to Septoria leaf blotch appears to be more widely distributed among wheat (*Triticum aestivum*) cultivars with winter growth habit than among bread wheats with spring growth habits (3,14,18–20). Apparently, genes that control phenotypic traits (eg, plant height) and physiological growth characteristics (long-day photoperiod and vernalization requirements) are intermingled with genetic factors that govern resistance of germ plasm to Septoria leaf blotch (3). Resistance to the pathogen is often associated with late maturity and tall plant stature (3). Tavella (19) studied the relationships between Septoria leaf blotch severity, date of heading, and plant height in cultivar yield trials. He concluded that the taller and later the cultivar, the lower its Septoria leaf blotch score tended to be. Rosielle and Brown (14) reported an association between resistance to Septoria leaf blotch and lateness and tall plant stature in crosses among four wheat cultivars, but they concluded that the correlations were not sufficiently high to be a major obstacle in the selection of resistant early-maturing, short-statured (90–97 cm) wheats in Australia. Scott and Benedikz (16) suggested that the association between height and resistance to *Septoria nodorum* may be due to chance association of shortness and susceptibility in parental lines, to genetic linkage, or to pleiotropy. They concluded that not all of the association of resistance with tallness among F_3 families derived from randomly selected single F_2 plants could be attributed to chance, but may be due to linkage or to pleiotropy.

Resistance to *S. tritici* in different cultivars has been reported to be inherited monogenically (10,13,18,20), digenically (10,14), and trigenically (14). Resistance in the tall winter wheat cultivar Nabob appears to be conditioned by two independent genes, each lacking dominance, but with additive effects (10). In the spring wheat cultivars Lerma 50 and P 14 resistance is conditioned by a single dominant gene (10). The resistance of the tall winter wheat cultivar Bulgaria 88 is governed by a single dominant gene expressed in the adult plant (13), and is incorporated into the soft red winter wheat cultivar Oasis (18). Attempts to recover the resistance of Bulgaria 88 in progenies from crosses to early maturing spring types in Australia (14) and in Israel have failed. Rosielle and Brown (14) reported that the inheritance of resistance in cultivar Seabreeze seems to be determined by recessive genes with at least three loci, while resistance in cultivars Veranopolis and IAS-20 might be due to a single gene. Wilson (20) reported that a single dominant gene governs Septoria resistance in Israel 493 (AUS 16144, Miriam 4/Lakhish 1552-3) and Veranopolis (AUS 1553). Despite all the above efforts, the level of resistance to Septoria leaf blotch in early maturing, short-statured, high-yielding commercial wheat cultivars remains distrubingly low (3,12,15).

The objectives of this study were to examine the relationships

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

0031-949X/82/08103706/$03.00/0

between plant-stature, maturity class, and severity to Septoria leaf blotch of wheat in cultivars of diverse origin, and assess the genetic factors governing those relationships for utilization in breeding programs.

MATERIALS AND METHODS

Wheat cultivars varying in height, maturity, vernalization requirements, and response to Septoria leaf blotch were selected for crosses on the basis of performance in Septoria nurseries over several years. Emphasis was placed when possible on selecting germ plasm with acceptable agronomic characteristics (straw strength, head type, nonshattering and seed size) and resistance to both leaf and yellow rusts of wheat.

Resistant parents were selected among tall late-maturing wheats, semidwarf early and late maturing wheats, and among winter wheat cultivars. The parents were crossed in a 16-parent diallel cross, not including reciprocals (incomplete diallel). The susceptible short-statured parents (dwarfs and semidwarfs) included in the diallel were: Aberdeen (CI 14918), Barkai (V238-8822/Miriam 2), Chris Mutant (CI 17241), Lakhish (Yt//Nrn 10/B21-1C/3/FA), Olesen dwarf (CI 14497), Pacific triple dwarf (CI 14590), and Tordo (Nai 60//Tom Thumb/Son 64/3/LR64/Son 64). The resistant spring wheat parents were: tall late-maturing wheats Colotana (CI 13556) [Colonista/Frontana], Fortaleza-1 (Colonista/Frontana), Polk/Waldron, Sheridan (CI 13586), and Titan (CI 12615); the early maturing, semidwarf cultivar V392-304 (H574/2*5187-23); and the late-maturing semidwarf cultivar Olaf (CI 15930). The resistant winter wheat parents were: Bezostaya 1 (Lutescens 17/Skorospelka 2), and Oasis (CI 15929). The winter wheats were vernalized for 8 wk at 4 C prior to transplanting.

F_1 seeds and, in many cases F_2 seeds, were produced from each cross. During the 1978–1979 season, parents, F_1, and F_2 populations from each cross were sown in spaced rows (60 cm apart), 7 cm separating each plant within a 6-m-long row for each population. Every 10th row was sown with the susceptible cultivar Bet-Dagan 233 to check on the uniformity of inoculation and subsequent spread of Septoria in the trial. The trial was inoculated uniformly with a 10^7 spores per milliliter of suspension of the virulent *S. tritici* isolate IS 398A1 once or twice a week, 15 times during the season on rainy days and/or dewy nights by using an Ulva 8 sprayer. The inoculation terminated when the late-maturing parents reached the end of the milk stage.

During the 1979–1980 season, parents, F_3 bulks, and F_2-derived early maturing, short-statured selected resistant lines of certain crosses were sown in a similar design. This trial was inoculated with a mixture of isolates (including isolate IS 398A1) expressing a broad spectrum of virulence. One hundred plants from each parent, 50 F_1 plants, 200–300 plants of F_2 or F_3 bulk populations, and 50–100 plants of the F_2-derived selected lines were assessed for each cross. Each plant in the trials was evaluated for the following parameters: heading date, plant height, and disease severity (the percentage of the green area of the six uppermost leaves covered with pycnidia of *S. tritici*) all recorded at the end of the milk stage (1,5).

RESULTS

Performance of parents. A summary of phenotypic characteristics and disease expression to a variable population of *S. tritici* in the 1977–1979 growing seasons is presented in Table 1. Disease severities on the parents in the 1978–1979 trial inoculated with the virulent *S. tritici* isolate IS 398A1 are similar to severities observed on these cultivars in the 1977–1979 field nurseries, which were inoculated with a mixture of *S. tritici* isolates, except for the response of Pacific. The vernalized winter wheat cultivars Bezostaya 1 and Oasis had low disease severity, a semidwarf stature, and moderate maturity. The five tall spring wheat cultivars Colotana, Fortaleza-1, Polk/Waldron, Sheridan, and Titan also consistently expressed low severity. In all these resistant cultivars, pycnidia formation remained on the lower leaves despite repeated inoculations over the canopy. The five tall cultivars and Olaf headed 30 days later than the remaining cultivars. The late-maturing semidwarf cultivar Olaf and the early maturing semidwarf wheat V392-304 manifested moderate disease severity. All the dwarf cultivars headed relatively early and had high disease severity.

Severity of disease in F_1 populations. The disease severities of F_1 populations derived from the partial diallel cross of the 16 parents are shown in Table 2. The F_1 populations are grouped in Table 3 into seven cultivar groups based on similarity in disease severity. F_1 populations obtained from crosses with the resistant winter wheat cultivar Bezostaya 1 expressed low mean pycnidial coverages (2–15%) except for the crosses with Aberdeen, Barkai, and V392-304, which resulted in moderately resistant (29–40%) F_1 populations. The means of the F_1 populations derived from crosses between the resistant cultivar Bezostaya 1 and members of groups C_1, D, and E indicate dominance of resistance to isolate IS 398A1. The crosses between Bezostaya 1 and members of groups C_2 and F suggest the possible presence of codominant modifier genes.

TABLE 1. Agronomic characteristics and response to Septoria leaf blotch of wheat of 16 wheat cultivars in the 1977-1979 Septoria nurseries and in the 1978–1979 diallel experimental plot (Expt) at Bet Dagan Experiment Station, Israel

Wheat cultivar	Growth habit	Plant height (cm)			Days to heading			Disease severity (%)[c]		
		77/78	78/79	Expt	77/78	78/79	Expt	77/78	78/79	Expt
Aberdeen	S[a]	45	55	53	101	103	110	65	90	52
Barkai	S	60	65	69	88	95	89	82	90	81
Bezostaya 1	W[b]	90	100	91	111	111	123	2	8	2
Chris Mutant	S	80	85	85	106	115	111	32	40	28
Colotana	S	124	148	136	130	130	128	2	14	14
Fortaleza-1	S	120	125	143	131	135	126	4	1	8
Lakhish	S	80	95	100	94	105	97	85	82	84
Oasis	W	100	100	111	111	102	130	7	8	14
Olaf	S	114	105	105	128	135	129	5	18	24
Olesen	S	40	45	45	99	100	89	80	90	82
Pacific	S	70	75	74	104	106	108	85	50	70
Polk/ Waldron	S	115	140	126	133	131	126	2	8	4
Sheridan	S	120	125	144	133	135	131	2	1	20
Titan	S	120	125	145	129	135	130	4	2	8
Tordo	S	60	60	56	100	112	106	82	90	75
V392-304	S	75	85	92	83	88	86	30	28	18

[a] S = spring growth habit.
[b] W = winter growth habit, vernalized for 8 wk at 4 C prior to transplanting.
[c] Disease severity = mean pycnidial coverage (%) of four uppermost leaves in the 1977–1979 trials and mean coverage of six leaves for the 1978–1979 diallel experimental plot (Expt).

Crosses among the parents of group B (Colotana, Fortaleza-1, Oasis, Polk/Waldron, Sheridan, and Titan) resulted in progeny with low disease severities (mean, 10%) similar to those of the parents. Crosses between cultivars of group B and cultivars in group C (Chris Mutant, Olaf, and V392-304) resulted in progeny with moderate disease severities with a mean of 24%. Progeny from crosses with Pacific and Lakhish had severities between 22 and 57%. Crosses with the susceptible short-statured cultivars Aberdeen, Barkai, Olesen, and Tordo (7) resulted in progenies with high disease severity (mean >50%). The means of the F_1 progenies from crosses between group B and the susceptible groups D, E, and F suggest approximately additive inheritance of resistance to isolate IS 398A1. The cultivars in group C are divided into two subgroups. Those in C_1 have moderate pycnidial coverages (Chris Mutant and Olaf) and produce resistant F_1 progeny in crosses with Bezostaya 1 and moderately resistant F_1 progeny in crosses with group B (mean severity of 18%). Cultivar V392-304 is placed in a separate subgroup (C_2) since it deviates from the other two cultivars in the susceptibility of its F_1 with Bezostaya 1 (mean severity of 29%), and with its F_1 with the cultivars of group B (mean 38%).

The means of the F_1 may imply that the resistance of Chris Mutant is governed by a subset of the recessive factors conditioning resistance in cultivars of group B, while in cultivar V392-304 resistance may be governed by recessive genes other than those of groups B and C_1.

The susceptible parents are divided into three groups. Cultivars in group D (Lakhish and Pacific) produced F_1 progeny with low disease severity (mean of 12%) in crosses with Bezostaya 1, moderate severity (mean of 38%) in crosses with cultivars in group B, and high severity (mean 58–85%) in crosses with cultivars in groups C_1, C_2, D, E, and F. These results suggest the presence of modifying genes for resistance or resistance genes in group D that are not present in cultivars of groups E and F.

The number of genetic factors segregating in the F_2 generation. The number of genetic factors governing resistance to IS 398A1 of *S. tritici* can be estimated by the formula of Burton (2).

$$n = \tfrac{1}{4}(3/4 - h + h^2)\, D^2/(VF_2 - VF_1)$$

in which n is the estimated number of segregating loci in the F_2 generation, VF_2 is the observed variance of the F_2 generation, VF_1 is the observed variance of the F_1 generation, $D = P_2 - P_1$, $h = (F_1 - P_1)/D$, VB is the predicted genetic variance in a segregating F_2 whose genetic resistance is governed by a single locus, and

$$VB = \tfrac{1}{4}(P_1 - \bar{x})^2 + \tfrac{1}{4}(P_2 - \bar{x})^2 + \tfrac{1}{2}(F_1 - \bar{x})^2,\ \bar{x}$$
$$= \tfrac{1}{4}P_1 + \tfrac{1}{4}P_2 + \tfrac{1}{2}F_1.$$

The genetic variance of a quantitative trait is equal to VB/n if the n controlling loci are identical in effect, additive, unlinked, not dominant and if one parental line contributes all the plus factors (6,9). If these conditions are not maintained, the true genetic variance will be greater than VB/n and Burton's formula will underestimate n. The genetic variance in the F_2 can also be estimated by $VF_2 - VF_1$. The relative size of VF_2 to VF_1 is a good indicator of genetic segregation in the F_2 population (6,9). The VF_2 values in Table 4 are typically above 150 whereas the VF_1 values are well under 100. The only three cases in which the VF_2 fall below 100 correspond to virtually zero estimates of 'n'. These two features support an estimate of no segregation for the last three crosses listed in Table 4.

In crosses with Bezostaya 1, one or two segregating loci were manifested. These findings support the previous findings from the F_1 analysis of dominant gene(s) governing resistance in Bezostaya 1 with possible additional modifier gene(s) in this cultivar.

Correlations. The correlation coefficients between disease severity and plant height and disease severity and days to heading for F_1 and F_2 populations in crosses between resistant and short-statured parents are presented in Table 5. The correlations between plant height and pycnidial coverage were small and negative; ie, there was no evidence of linkage or pleiotropic genes which affect

TABLE 2. Mean Septoria pycnidial coverage on the six uppermost leaves of wheat parents and F_1 populations crossed in a half diallel scheme Bet Dagan, Israel, 1978–1979

Wheat cultivars	Parents disease severity (%)	Wheat cultivars													
		Aberdeen	Barkai	Olesen	Tordo	Lakhish	Pacific	V 392-304	Chris Mutant	Olaf	Oasis	Titan	Colotana	Polk/ Waldron	Sheridan	Fortaleza-1
Bezostaya 1	1.6 ± 0.1[a]	38 ± 2.5	40 ± 3.0	10 ± 0.7	2 ± 0.2	8 ± 0.8	15 ± 1.2	29 ± 2.8	2 ± 0.2	6 ± 0.8	0.4 ± 0.02	3 ± 0.4	9 ± 1.3	–	7 ± 0.7	6 ± 0.5
Fortaleza-1	8.1 ± 0.7	49 ± 1.8	36 ± 3.2†[c]	–	57 ± 1.1	37 ± 1.2	39 ± 1.3	48 ± 2.5	22 ± 2.0	10 ± 2.2†	13 ± 0.6†	11 ± 1.2	6 ± 1.5†	–	–	–
Sheridan	20.3 ± 0.7	56 ± 3.9	–	46 ± 1.2	67 ± 1.1	45 ± 1.0	51 ± 0.8	57 ± 2.3	30 ± 1.4	12 ± 0.5	21 ± 1.0	7 ± 2.5†	13 ± 0.5	18 ± 1.6		
Polk/ Waldron	4.0 ±0.2	–[b]	64 ± 3.9	-	66 ± 0.8	57 ± 2.1	37 ± 1.4	–	21 ± 2.0	4 ± 1.9†	12 ± 1.5	16 ± 4.4	11 ± 0.8			
Colotana	14.4 ± 0.2	50 ± 5.2	30 ± 4.4†	72 ± 2.4	67 ± 1.4	37 ± 3.1	22 ± 1.0	13 ± 1.7	19 ± 1.5	15 ± 1.7†	3 ± 0.6†	13 ± 1.3				
Titan	7.6 ± 1.4	59 ± 2.0	47 ± 2.1	60 ± 1.5	79 ± 1.5	37 ± 0.9	32 ± 1.1	–	32 ± 2.1	11 ± 1.2	7 ± 0.7					
Oasis	14.3 ± 1.4	19 ± 1.1	53 ± 1.3	78 ± 1.3	58 ± 2.2	32 ± 0.9	28 ± 1.6	33 ± 2.0	29 ± 1.1	16 ± 3.8†						
Olaf	23.9 ± 0.9	–	–	74 ± 2.5	64 ± 2.4	65 ± 2.0	62 ± 2.7	–	42 ± 5.1							
Chris Mutant	27.7 ± 1.4	76 ± 1.6	79 ± 1.6	77 ± 1.0	72 ± 1.0	50 ± 1.4	82 ± 0.7	20 ± 1.1								
V392-304	18.0 ± 1.2	–	75 ± 1.8	78 ± 2.8	68 ± 0.9	77 ± 1.3	80 ± 1.7									
Pacific	70.4 ± 0.5	–	85 ± 0.8	85 ± 1.3	77 ± 0.6	80 ± 0.6										
Lakhish	83.9 ± 0.4	85 ± 0.9	85 ± 0.8	88 ± 0.6	78 ± 0.9											
Tordo	75.0 ± 0.7	77 ± 0.9	69 ± 0.9	82 ± 1.0												
Olesen	82.4 ± 0.5	85 ± 0.7	87 ± 0.5													
Barkai	81.3 ± 0.9	83 ± 1.6														
Aberdeen	51.8 ± 2.8															

[a] Standard error.
[b] Missing F_1 combination.
[c] † Data from 1979–1980.

TABLE 3. Classification of wheat parents and F_1 populations to severity classes (A–F) based on percent coverage by pycnidia of *Septoria tritici*

Group	Cultivar	P_0[a]	Pycnidial coverage (%)						
			A	B	C_1	C_2	D	E	F
A	Bezostaya 1	2	2	5	4	29	12	6	39
B	Fortaleza-1	8	6	10	16	48	38	57	42
	Sheridan	20	7	15	21	57	48	56	56
	Polk/Waldron	4	···	14	12	···	47	66	64
	Colotana	14	9	9	17	13	30	70	40
	Titan	8	3	11	21	···	35	70	53
	Oasis	14	0.4	11	22	33	30	68	36
C_1	Olaf	24	6	8	42	···	64	74	···
	Chris Mutant	28	2	26	42	20	66	74	78
C_2	V392-304	18	29	38	···	20	78	73	75
D	Pacific	70	15	35	72	80	80	81	85
	Lakhish	84	8	41	58	77	80	83	85
E	Tordo	75	2	66	72	68	77	82	73
	Olesen	82	10	64	76	78	86	82	86
F	Barkai	81	40	46	79	75	85	78	83
	Aberdeen	52	38	47	76	···	85	81	83

[a] Mean pycnidial coverage of parent.

TABLE 4. Means and variances of diseases severity (%) in paretns, F_1, and F_2 and the estimated number of segregating loci in F_2 crosses

Cross	Group	Mean coverage (%)				Variance		Estimated no. of segregating loci[b]
		P_1	P_2	F_1	F_2	F_1	F_2	
Tordo × Bezostaya 1	E × A[a]	75	2	2	47	2	773	1.4
× Colotana	× B	75	14	67	43	51	275	2.6
× Fortaleza-1	× B	75	8	57	64	46	283	2.6
× Sheridan	× B	75	20	67	65	35	299	1.8
× Titan	× B	75	8	79	63	73	412	2.7
× Olaf	× C	75	24	64	57	74	207	2.8
Pacific × Colotana	D × B	73	14	22	23	25	194	3.2
× Fortaleza-1	× B	73	8	39	34	54	196	3.7
× Oasis	× B	73	14	28	41	80	195	4.3
× Polk/ Waldron	× B	73	4	37	54	44	289	2.4
× Sheridan	× B	73	20	51	34	21	153	2.7
× Titan	× B	73	8	32	46	40	439	1.4
Lakhish × Bezostaya 1	D × A	84	2	8	39	28	682	1.7
× Fortaleza-1	× B	84	8	37	36	47	364	2.3
× Oasis	× B	84	14	32	31	36	354	2.1
Bezostaya 1 × V392-304	A × C_2	2	18	29	18	161	318	0.8
× Lakhish	× D	2	84	8	39	28	682	1.7
× Olesen	× E	2	82	10	26	21	512	2.1
× Tordo	× E	2	75	2	47	2	773	1.4
Sheridan × Pacific	B × D	20	73	51	34	21	153	2.7
× Olesen	× E	20	82	46	40	40	345	1.6
× Tordo	× E	20	75	67	65	35	299	1.8
Fortaleza-1 × Lakhish	B × D	8	84	37	36	47	364	2.3
× Pacific	× D	8	73	39	34	54	196	3.4
× Tordo	× E	8	75	57	64	46	283	2.6
× Aberdeen	× F	8	55	49	40	105	442	1.0
Christ Mutant × Oasis	C_1 × B	27	14	29	17	36	167	0.3
× Polk/ Waldron	× B	27	4	21	10	44	140	0.7
V392-304 × Fortaleza-1	C_2 × B	18	8	48	42	168	579	0.8
× Sheridan	× B	18	20	57	70	137	377	1.4
× Bezostaya 1	× A	18	2	29	18	161	318	0.8
Oasis × Polk/Waldron	B × B	14	4	12	12	51	82	0.5
Sheridan × Colotana	B × B	20	14	13	29	82	85	0.1
Lakhish × Tordo	D × E	84	75	78	73	19	39	1.0

[a] Cultivar groups from Table 3.

[b] Minimum numbers calculated from Burton's (2) formula which is based on the assumption that no linkage exists between pertinent genes, one parent supplies only resistant genes and the other only susceptibility genes, all genes are equally important and have the same degree of dominance, and there is no epistasis.

both plant height and resistance to Septoria leaf blotch. The correlation between days to heading and disease severity was low-to-moderate ($r = -0.30$) and negative; ie, late heading was related to low severity. This correlation could imply linkage between the genes controlling heading and those for resistance. The correlation appears to be too low to suggest pleiotropy since the number of genetic loci controlling resistance is small. If the same genes controlled both traits (heading and resistance) the correlations should be very high.

F_2-derived lines. Early-maturing, short-statured resistant plants selected among F_2 populations which were inoculated with isolates IS 398A1 continued to express varying levels of resistance when subjected to a mixture of virulent *S. tritici* isolates of diverse origin. The performance of the best F_2-derived lines combining earliness, short stature, and resistance to a wide spectrum of virulences of *S. tritici* isolates from Israel is presented in Table 6. It was difficult to recover resistant (disease severity <10%) plants among the early maturing, short-strawed lines derived from crosses between early maturing, susceptible dwarf and late-maturing tall, resistant parents. F_2-derived lines expressing high resistance level, nearly as high as the resistant parent, combined with short stature (85–95 cm), and early maturity, were obtained from crosses between Bezostaya 1 and the dwarf cultivar Tordo. Resistant, early, semidwarf lines combined with good head type were recovered from the cross between Bezostaya 1 and V392-304 which possesses moderate resistance to *S. tritici* derived from H574. Rather few plants continued to express high resistance to Septoria leaf blotch in F_2 derived lines from crosses between Bezostaya 1 and the susceptible commercial cultivar Lakhish, which is thought to possess a dominant modifier gene for resistance.

DISCUSSION

The currently grown high yielding wheat cultivars are disturbingly vulnerable to Septoria leaf blotch, and the incorporation of resistance to *S. tritici* remains a high priority breeding objective. Because of the involvement of plant architecture, growth habits, age, maturity, and vernalization in host-parasite interactions, specific genetic factors that contribute to resistance can be difficult to distinguish or assess (3,15,18,19). The difficulties in combining short plant stature (<90 cm) and earliness with resistance to *S. nodorum* led Scott and Benedikz (15) to conclude that pleiotropy or tight linkage or epidemiological factors might have interfered with incorporation efforts. The current analysis of F_1 and F_2 populations derived from an incomplete diallel crossing scheme indicates that resistance to *S. tritici* is governed by genes that are distinct from those controlling short-stature or maturity. Short-statured (50–70 cm), early maturing (heading <110 days from seedling emergence), resistant plants (pycnidial coverage of the four uppermost leaves of less than 15%) could be recovered in F_2 populations (Table 6), and their progeny from selfing continued to express resistance to a wide spectrum of virulences in succeeding generations. The moderate correlations ($r = -0.30$) between days to heading and pycnidia coverage could indicate that high resistance is associated with later maturity. This association may be due to linkage effects, yet, the moderate negative correlation does not seem to support pleiotropy.

Resistance to *S. tritici* isolate IS 398A1 exhibited by the winter wheat cultivar Oasis (CI 15929) is clearly not due to a dominant locus. The resistance to *S. tritici* in this cultivar is incorporated from Bulgaria 88 which is reported to possess a single dominant gene for mature plant resistance (13). The possible discrepancy in the results may be due to differences in physiologic specialization of *S. tritici* populations in Indiana and Israel. The highly virulent *S. tritici* populations in Israel have rendered susceptible some previously reported resistant cultivars. Conversely, Bezostaya 1, which is resistant to a wide *S. tritici* spectrum in Israel, exhibits moderate susceptibility in Australia and Oklahoma (R. E. Wilson and F. J. Gough, *personal communication*).

The utilization of resistant winter wheat germ plasm of diverse sources in spring wheat breeding programs may offer a promising avenue in improving genetic protection to *S. tritici* (3). However, with the relatively slow progress in producing resistant commercial wheat cultivars, breeders may be too eager to resort to rapid improvement by the extensive utilization of a few resistant sources, and thereby reduce diversity and increase genetic vulnerability (8).

Resistance to *S. tritici* manifested by the tall-stature, late-maturing spring wheat cultivars: Colotana, Fortaleza-1, Polk/Waldron, Sheridan, and Titan may be derived from the common parent cultivar Frontana (CI 12470). The failures in incorporating resistance to *S. tritici* from tall, late maturing spring wheat germplasm to short-stature, early maturing wheats are partly due to improper priorities in selecting for resistant plants under weak Septoria epidemics. Strong selection in early generations for short plant stature, earliness, head type, straw stiffness, milling quality, resistance to rust diseases, etc., followed by selection for resistance to *S. tritici* in the selected F_6–F_7 lines, usually resulted in lines deficient in resistance to *Septoria*. The integration of genes for resistance, conditioned by different genetic

TABLE 5. Correlation coefficients between Septoria pycnidial coverage (PCD) and plant height and days to heading for F_1 and F_2 populations in crosses between resistant and short-statured wheat cultivars

Crosses	Correlation coefficients[a] (PCD - plant height)		Correlation coefficients[b] (PCD - heading)	
	F_1	F_2	F_1	F_2
Tordo × Fortaleza-1	0.116	−0.054	−0.187	−0.486
× Sheridan	0.122	−0.329	0.253	−0.385
× Colotana	0.165	−0.564	0.229	0.358
× Titan	0.222	−0.082	−0.174	0.035
Lakhish × Oasis	0.361	0.176	−0.051	−0.339
× Fortaleza-1	0.133	−0.005	−0.379	−0.353
Pacific × Polk/Waldron	−0.277	−0.015	0.210	−0.407
× Sheridan	−0.062	−0.053	−0.44	−0.241
× Colotona	−0.052	−0.061	−0.044	−0.321
× Fortaleza-1	0.328	−0.175	−0.247	−0.245
× Titan	0.356	−0.035	−0.155	−0.391
× Oasis	−0.203	0.067	0.053	−0.366
Aberdeen × Fortaleza-1	0.112	−0.052	−0.444	−0.424
Mean	0.10 ± 0.06[c]	−0.06 ± 0.06	−0.07 ± 0.06	−0.29 ± 0.07
Tordo × Bezostaya 1	0.568	−0.143	−0.679	−0.097
Olesen × Bezostaya 1	0.191	−0.019	−0.106	−0.404
Lakhish × Bezostaya 1	0.108	−0.116	−0.250	−0.113
Mean	0.29 ± 0.15	−0.09 ± 0.03	−0.34 ± 0.19	−0.20 ± 0.10

[a]PCD − plant height = correlation coefficients between pycnidial coverage (PCD) and plant height.
[b]PCD − heading = correlation coefficient between pycnidial coverage (PCD) and days to heading.
[c]± Standard error.

TABLE 6. Plant stature and disease performance of susceptible dwarf, resistant, tall, and semidwarf wheat cultivars and their F_2 derived lines. Populations were inoculated with a mixture of virulent *Septoria tritici* isolates. Israel, 1979–1980

Cultivar	Source	Plant height (cm)	Disease severity[a] (%)
Tordo	Parent	60.3 ± 1.07[b]	73.8 ± 2.56
Colotana	Parent	141.1 ± 1.13	1.8 ± 0.18
Tordo × Colotana	F_2-derived line	92.5 ± 4.20	22.6 ± 2.56
Fortaleza-1	Parent	143.2 ± 0.93	5.4 ± 0.54
Tordo × Fortaleza-1	F_2-derived line	95.2 ± 5.54	28.8 ± 4.48
Sheridan	Parent	145.8 ± 1.11	15.1 ± 0.91
Tordo × Sheridan	F_2-derived line	98.2 ± 2.81	45.0 ± 2.67
Bezostaya 1	Parent	105.2 ± 0.69	5.5 ± 0.45
Tordo × Bezostaya 1	F_2-derived line 1	85.1 ± 5.54	8.7 ± 0.80
	F_2-derived line 2	83.4 ± 4.38	10.7 ± 1.45
	F_2-derived line 3	86.3 ± 5.16	12.3 ± 1.99
	F_2-derived line 4	90.0 ± 3.01	14.3 ± 1.77
	F_2-derived line 5	94.9 ± 4.11	9.7 ± 0.87
Olesen	Parent	70.0 ± 1.19	79.9 ± 1.86
Olesen × Sheridan	F_2-derived line	107.1 ± 3.10	37.9 ± 4.58
Olesen × Bezostaya 1	F_2-derived line 1	97.1 ± 1.96	9.6 ± 0.88
	F_2-derived line 2	103.2 ± 1.85	22.1 ± 2.71
	F_2-derived line 3	101.9 ± 2.28	23.1 ± 2.81
	F_2-derived line 4	69.8 ± 2.74	24.0 ± 2.26
	F_2-derived line 5	99.3 ± 1.98	26.8 ± 2.96
Lakhish	Parent	100.8 ± 1.26	67.7 ± 1.90
Bezostaya 1	Parent	105.2 ± 0.69	5.5 ± 0.45
Lakhish × Bezostaya 1	F_2-derived line 1	85.0 ± 1.58	16.9 ± 1.54
	F_2-derived line 2	107.4 ± 1.96	23.4 ± 2.00
V 392-304	Parent	93.2 ± 0.91	57.9 ± 2.22
V 392-304 × Bezostaya 1	F_2-derived line 1	90.9 ± 1.54	4.9 ± 0.88
	F_2-derived line 2	113.6 ± 1.56	8.8 ± 1.77
	F_2-derived line 3	97.1 ± 1.15	15.1 ± 2.83
	F_2-derived line 4	108.9 ± 2.11	15.4 ± 3.48
	F_2-derived line 5	110.1 ± 1.35	18.2 ± 2.55

[a]The average pycnidial coverage of the four uppermost leaves.
[b]Standard error.

factors into agronomically suitable wheats, by various breeding strategies, may broaden the genetic basis of protection and increase stability against the diverse population of the pathogen.

LITERATURE CITED

1. Bahat, A., Gelernter, I., Brown, M. B., and Eyal, Z. 1980. Factors affecting the vertical progression of Septoria leaf blotch in short-statured wheats. Phytopathology 70:179-184.
2. Burton, G. W. 1951. Quantitative inheritance in Pearl millet (*Pennisetum glaucum*). Agron. J. 43:409-417.
3. Eyal, Z. 1981. Integrated control of Septoria diseases of wheat. Plant Dis. 65:763-768.
4. Eyal, Z., Amiri, Z., and Wahl, I. 1973. Physiologic specialization of *Septoria tritici*. Phytopathology 63:1087-1091.
5. Eyal, Z., and Brown, M. B. 1976. A quantitative method for estimating density of *Septoria tritici* pycnidia on wheat leaves. Phytopathology 66:11-13.
6. Falconer, D. S. 1960. Introduction to Quantitative Genetics. The Ronald Press Company, New York. 365 pp.
7. Fick, G. N., and Qualset, C. O. 1973. Genes for dwarfness in wheat *Triticum aestivum* L. Genetics. 75:531-539.
8. Jain, H. K., and Kubshrestha, U. P. 1976. Dwarfing genes and breeding for yield in bread wheat. Z. Pflanzensucht. 76:102-112.
9. Mather, K., and Jinks, J. L. 1971. Biometrical Genetics. Cornell University Press, Ithaca, NY. 382 pp.
10. Narvaez, I. M. 1957. Studies of Septoria leaf blotch of wheat. Ph.D. thesis, Purdue University, W. Lafayette, IN. 101 pp.
11. Prestes, A. M. 1976. *Septoria tritici* Rob. ex Desm. Host relationships, varietal response, and influence on the development of wheat roots. Diss. Abstr., Series B, 37:2601-2602.
12. Rajaram, S., and Dubin, H. J. 1977. Avoiding genetic vulnerability in semidwarf wheat. Ann. N. Y. Acad. Sci. 287:243-254.
13. Rillo, A. O., and Caldwell, R. M. 1966. Inheritance of resistance to *Septoria tritici* in *Triticum aestivum* subsp. *vulgare* 'Bulgaria 88' (Abstr.) Phytopathology 56:597.
14. Rosielle, A. A., and Brown, A. G. P. 1979. Inheritance heritability and breeding behaviours of resistance to *Septoria tritici* in wheat. Euphytica. 28:385-392.
15. Saari, E. E., and Wilcoxson, R. D. 1974. Plant disease situation of high yielding dwarf wheats in Asia and Africa. Annu. Rev. Phytopathol. 12:49-68.
16. Scott, P. R., and Benedikz, P. W. 1978. Genetics of the association between height and resistance to *Septoria nodorum* in wheat. (Abstr.) Page 284 in: Proc. 3rd Int. Congress Plant Pathology, Munich, W. Germany.
17. Shaner, G. 1981. Effect of environment on fungal leaf blights of small grains. Annu. Rev. Phytopathol. 19:273-296.
18. Shaner, G., Finney, R. E., and F. L. Patterson. 1975. Expression of effectiveness of resistance to Septoria leaf blotch. Phytopathology 65:761-766.
19. Tavella, C. M. 1978. Date of heading and plant height of wheat varieties as related to Septoria leaf blotch damage. Euphytica 27:577-580.
20. Wilson, R. E. 1979. Resistance to *Septoria tritici* in two wheat cultivars, determined by independent, single dominant genes. Austr. Plant Pathol. 8:16-18.