Genetic Analysis of Adult-Plant Resistance to Leaf Rust in Five Spring Wheat Genotypes

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ABSTRACT

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Inheritance of adult-plant resistance to leaf rust, caused by *Puccinia triticina*, was studied in the progeny of a one-way diallel cross involving five CIMMYT-derived adult-plant resistant wheat (*Triticum aestivum*) genotypes and a susceptible wheat 'Avocet-YrA'. F₁ progenies, F₂ populations, F₂-derived F₃, and F₄-derived F₅ lines were field evaluated under artificial epidemics with leaf rust race MCJ/SP. Adult-plant resistance to leaf rust was incompletely dominant in crosses with the susceptible parent and was found to be controlled by additive interactions of *Lr34* with at least two to three additional genes. Transgressive segregation giving rise to plants or lines with higher and lower levels of resistance than the parents was observed in all F₂ and F₅ derivatives of the resistant-parent intercrosses and suggested that, apart from *Lr34*, some of the other additive genes were nonallelic. Although specific combining ability was significant in some generations, general combining ability was found to be the major component of variation. Among generations, the estimates of the narrow-sense heritability of adult-plant resistance to leaf rust ranged from 0.67 to 0.97.

Additional keywords: diallel analysis

Leaf rust, caused by *Puccinia triticina* Eriks., is the most widely distributed and most common of all three wheat rusts (24). On a worldwide basis, it probably causes more yield losses than the other wheat rusts (25). Khan et al. (13) determined that, for leaf rust severities of up to approximately 20%, every 1% of the flag leaf area covered by rust pustules caused a reduction of 1% in grain yield in winter wheat cultivars grown in Mississippi. However, losses to leaf rust are highly influenced by the growth stage at disease onset (24). Chemical control of leaf rust often is practically unmanageable and uneconomical. Genetic resistance is the most economical method of reducing yield losses due to leaf rust (14). Most of the catalogued leaf rust resistance genes (19)

confer race-specific hypersensitive responses that remain effective for a short period of time, but may then become ineffective as races with virulence to the resistance genes develop in the pathogen population.

Substantial economic benefits are likely to be associated with the development of race-nonspecific resistance to leaf rust in many wheat-producing areas, especially in areas where farmers change cultivars slowly (37). Race-nonspecific adult-plant resistance to leaf rust, often referred to as slow rusting (6) or partial resistance (21), is characterized as a type of interaction between the host plant and pathogen in which plants show less disease severity compared with the susceptible check, despite compatibility or susceptible infection types. Several studies report the inheritance of such resistance to leaf rust (3,4,8,16,20,35). Genetic studies based on either latent period or the area under the leaf rust progress curve often have shown that race-nonspecific adult-plant resistance to leaf rust is under oligogenic or polygenic control of genes having additive effects with relatively high heritability (8,16,27).

Durable resistance to leaf rust often involves race-nonspecific, additive adult-plant resistance genes (18). Attempts have been made in several wheat breeding programs to accumulate additive resistance genes into high-yielding wheat back-

grounds to achieve higher levels of race-nonspecific resistance. Resistant parental genotypes studied in this research were developed in a breeding project at the International Wheat and Maize Improvement Center (CIMMYT), aimed at combining as many additive resistance genes as possible through intercrossing adult-plant resistant genotypes. Genotypes were selected for the high seedling infection type accompanied by low leaf rust severities in the adult stage (32).

The objectives of the present research were to (i) determine the number of genes that confer a high level of adult-plant resistance to leaf rust and (ii) study the combining abilities and gene effects involved in such resistance in five wheat genotypes.

MATERIALS AND METHODS

Plant material. Five adult-plant resistant genotypes and a susceptible genotype, 'Avocet-YrA' (Table 1), were intercrossed in a one-way diallel mating design in a greenhouse at the University of Alberta, Edmonton, Canada. Avocet-YrA is a reselection from the Australian cv. Avocet that shows high susceptibility to current leaf rust races in Mexico, where field experiments were carried out. Crosses were made by hand-emasculatation and artificial pollination using the approach method (1). Parental genotypes were planted in 15-cm-diameter pots filled with a Metro-mix (W. R. Grace & Co., Ajax, Ontario, Canada) soil mixture and grown in a greenhouse maintained at 20 and 15°C (day and night, respectively) and a 16-h photoperiod. From each cross, 90 to 150 F₁ seed were harvested. Ten F₁ seed from each cross were grown in a greenhouse with the same growing conditions as mentioned for crossing, and self-fertilized to produce F₂ seed that were harvested from individual plants in each cross. Consequently, F₂-derived F₃ lines of the susceptible × resistant crosses were harvested from F₂ single plants that were planted in the greenhouse. The F₅ single seed-descent lines of the diallel cross were developed by advancing generations using the single seed-descent method (23). In each generation, single plants of the crosses were grown in root containers (25 mm in diameter and 140 mm deep) containing Metro-mix. One seed was harvested from each single plant in each generation and replanted to achieve the next

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generation. The F₄ seed were space planted at the University of Alberta Edmonton Research Station in the summer of 2000 and F₅ single seed-descent lines were harvested from single F₄ plants.

Test sites. Field evaluations were carried out at two CIMMYT Research Stations. The first was at El-Batan (Mexico State) in the highlands of central Mexico (19°N, 2,249 *masl*) during the 2000 crop season, starting in mid-May and concluding in early October. The second was at Ciudad Obregon (Sonora State) in north-western Mexico (22°N, 7 *masl*) during the winter 2000–01 crop season, starting in late November and concluding in early April. Each site has a favorable environment for leaf rust development.

Experimental procedure. Parental lines, F₁ progenies, F₂ populations derived from the diallel crosses, and F₃ lines from the susceptible × resistant crosses were evaluated at El-Batan during the summer 2000 crop season. F₁ and F₂ diallel experiments, comprising six parental lines and 15 F₁ crosses or F₂ populations, were planted separately, each in a three-replication randomized complete block design. Fifteen seed were planted in each F₁ experimental unit on a double-row bed, 1 m long and 70 cm wide. In each F₂ experimental unit, 120 seed were space-sown, about 10 cm apart, on two double-row beds, 3 m long and 75 cm wide. The F₃ lines of the susceptible × resistant crosses (52 to 80 lines from each cross) were planted in one replication with plot sizes similar to the F₁ experiment, with parental lines repeated after every 10 lines as checks. Each F₃ line was grown in an individual plot with 3 g of seed planted to obtain at least 60 plants per plot.

The experimental design used for planting F₃ and F₅ lines at Ciudad Obregon during 2000–01 was an augmented randomized complete block design where 10 check genotypes (Table 2) were planted in 3 and 10 replications of F₃ and F₅, respectively. Each of these replications formed a complete block. Unassigned plots then were formed within each block and were filled with unreplicated experimental lines (27). The six parental lines, a total of 564 F₃ lines from the susceptible × resistant crosses (66 to 155 lines from each cross), and 1,802 F₅ lines from the diallel cross

(76 to 142 lines from each cross) were included in this test. Each block, comprising 200 experimental units, included 190 lines and 10 checks. Plot size and seed density were the same as in the F₃ experiment at El-Batan. The F₃ lines included in the Ciudad Obregon study were different from those studied at El Batan.

Artificial epidemics and LR race. In all field experiments, one row of the highly susceptible cv. Morocco was planted as spreader on one side of the plots in the middle of the 0.5-m-wide pathways and around the field. Artificial inoculation was carried out with the Mexican *P. tritici* race MCJ/SP (28) by spraying the spreader rows with urediniospores suspended in a light-weight mineral oil Soltrol-170 (Bartlesville, OK) with 300 mg of spores per liter of oil, about 4 and 6 weeks after planting at El-Batan and Ciudad Obregon, respectively. Fresh inoculum was obtained by multiplying urediniospores on the susceptible cv. Morocco in a greenhouse. According to Singh (28), race MCJ/SP has the following avirulence/virulence formula: *Lr2a,2b,2c,3ka,9,16,19,21,24,25,28,29,30,32,33/1,3,3bg,10,11,12,13,14a,14b,15,17,18,20,22b,23,26,27+31*.

The race MCJ/SP was considered suitable for the study of adult-plant resistance to leaf rust because all parents included in this study show a high (susceptible) infection type in the seedling tests and reduced leaf rust severity as adults in field tests (Table 1) with it.

Leaf rust assessment. Leaf rust severities were assessed visually twice following the modified Cobb's scale (22). Based on the practice used in CIMMYT (36), the first rating was done when the leaf rust severity of the susceptible parent, Avocet-YrA, reached 80 to 100% and the second rating was done 10 to 15 days later, when the leaves of the susceptible parent had dried out due to leaf rust infection. F₁ progenies and F₃ and F₅ lines were assessed based on the average rust severity on the flag and penultimate leaves in a plot. For this purpose, several leaves were examined and an overall visual estimate of the disease severity was recorded for each plot. In the F₂ diallel experiment, however, single plants in each plot were rated separately for disease severity. Flag and penultimate leaves of the two most advanced

tillers of each plant were observed, to determine the average severity estimate of each F₂ plant. Average leaf rust severity for each F₂ plot then was computed.

Genetic analysis. For the segregation analysis, F₃ lines of the susceptible × resistant crosses were classified into four phenotypic groups: homozygous for the parental type resistance (HPTR), homozygous for the parental type of higher susceptibility (HPTS), either segregating or homozygous for disease levels higher than the resistant parent but less than the susceptible parent (SegI), and segregating for disease levels equal to the susceptible parent (SegS). The F₅ lines were classified into three groups: HPTR, HPTS, and other segregants. HPTR lines were assumed to be derivatives of the F₂ or F₄ single plants homozygous for all resistance alleles; HPTS lines were assumed to be the derivatives of the F₂ or F₄ single plants homozygous for all susceptibility alleles; SegI lines were assumed to be the derivatives of the F₂ single plants homozygous for resistance allele for at least one locus; and SegS lines were assumed to be the derivatives of the F₂ or F₄ single plants heterozygous for resistance allele for at least one locus and homozygous for susceptibility alleles at all other loci. To test the distribution of the observed phenotypic frequencies against those expected for segregation models, χ^2 analyses were carried out.

Quantitative genetic analyses were performed on the mean values of the terminal leaf rust severities of experimental units, separately for the F₁, F₂, and F₅ diallel experiments, and for the susceptible × resistant crosses in the F₃ generation, tested in Ciudad Obregon. The residuals were not normally distributed as determined by the Proc UNIVARIATE procedure (SAS Institute, Cary, NC). Therefore, mean disease severity of each plot was transformed by the arcsine of the square root to normalize the scale. Analyses of variance (38) were performed for the terminal leaf rust severities separately for the F₁, F₂, F₃, and F₅ experiments, with treatments being parents and crosses in F₁ and F₂ and parents, crosses, and checks in F₃ and F₅. For the unbalanced F₃- and F₅-line experiments, analyses of variance were performed following the model for the augmented design proposed by Scott and

Table 1. Wheat genotypes included in genetic analysis with their pedigree, and adult-plant and seedling responses to *Puccinia tritici* race MCJ/SP

Genotype	Cross and selection history	Leaf rust response	
		Adult plant	Seedling ^z
Avocet-YrA	WW191/WW15/Egt	Susceptible	4
Saar	Sni/Trap#1//Bav92 CG25-099Y-099M-4Y-2M-3Y-0B	Resistant	3–4
Simorgh	Sni/Yaco//Bav92 CG29-099Y-099M-10Y-2M-3Y-0B	Resistant	3–4
Homa	HD2281/Yaco/3/Kauz*2/Trap//Kauz CG54-099Y-099M-10Y-3M-5Y-0B	Resistant	3–4
Parastoo	Pvn//Car422/Ana/3/Kauz*2/Trap//Kauz CG62-099Y-099M-3Y-3M-3Y-0B	Resistant	3–4
Cocnoos	Trap#1/PBW65//Star CG99-099Y-099M-3Y-7M-1Y-0B	Resistant	3–4

^z Seedling responses are infection types based on a 0-to-4 scale (24) where infection types 3 and 4 are considered compatible or susceptible.

Milliken (26). Minor modifications were made in order to estimate the effects of treatment and line-treatment; to break the sum of squares for treatment into parents, crosses, and checks; and to estimate the effect of parents versus crosses using a single-degree of freedom contrast (SAS Institute). Combining ability analyses were performed following the model I, method IV of Griffing (12) with treatment as fixed effect. Parents were not included in the combining ability analyses, to obtain unbiased estimates of general combining ability (GCA) and specific combining ability (SCA) parameters (7,30). Diallel Analysis and Simulation Software (5) was used to estimate the GCA and SCA effects and the components of variance. Narrow-sense heritability of adult-plant resistance to leaf rust was estimated separately for each generation as the ratio of additive variance to phenotypic variance.

Mean terminal leaf rust severities of genotypes were computed for the F_1 and F_2 diallels using the MEANS statement in the Proc GLM procedure (SAS Institute). However, because the F_3 and F_5 experiments were unbalanced, least square means were computed for each genotype, using LSMEANS statement in Proc GLM procedure (SAS Institute).

Minimum numbers of additive genes in the resistant parents were estimated based on the formula proposed by Bjarko and Line (3), using data from the F_5 experiment: $N = (GR)^2/4.27(V_{F_5} - [V_{P_1} + V_{P_2}]/2)$, where N is the minimum number of effective additive genes, GR is the genotypic range, V_{F_5} is the variance of the F_5 lines, and V_{P_1} and V_{P_2} are the variances of the resistant and susceptible parents, respectively. The formula proposed by Wright (40) also was used to estimate the minimum number of genes: $N = (P_s - P_r)^2 \times$

$[1.5 - 2h(1 - h)]/8(V_{F_2} - V_E)$, where P_r and P_s are the mean parental values of the resistant and susceptible parents, respectively, $h = (F_1 - P_s)/(P_s - P_r)$, V_{F_2} is the variance of the F_2 , and V_E is the environmental variance. The formulae are based on the assumptions that resistance genes that segregate in a cross occur in only one parent, segregating resistance genes are not linked, all resistance genes have equal effects, additive-dominance and genotype-environment effects do not exist, and dominance is equal at all loci.

RESULTS

Terminal leaf rust severities of parental genotypes, F_1 progenies, F_2 populations, and least-square means of the leaf rust severities of F_3 and F_5 lines are presented in Table 2. Least square means are the treatment means adjusted for other effects in the experimental design and were used in F_3 and F_5 experiments because they were unbalanced for the number of lines in each cross. All resistant parents displayed low levels of terminal leaf rust severities, whereas the susceptible parent exhibited a high severity. Susceptible $\times$ resistant crosses had intermediate average leaf rust severities in all generations. Although the leaf rust severities of the parents varied slightly in generation trials, the correlations (r) between generations were high and ranged between 0.93 ($P < 0.01$) and 0.97 ($P < 0.01$).

Susceptible $\times$ resistant crosses. All F_1 susceptible $\times$ resistant progenies displayed intermediate levels of leaf rust severity (Table 2). The frequency distributions of leaf rust severity of the F_2 , F_3 , and F_5 lines derived from the susceptible $\times$ resistant crosses showed continuous variation, with only a few lines having the leaf rust severities of the resistant and susceptible parents (Table 3). The frequency distributions were significantly skewed, with population means shifting from the midparental values toward the resistant parent (Table 2).

The frequency of the four or three phenotypic groups in the F_3 and F_5 lines of the susceptible $\times$ resistant crosses are presented in Table 4. Because F_3 lines tested at two locations were not identical, frequencies of the four phenotypic groups in F_3 also were pooled and tested against the hypotheses of three and four gene ratios. The expected four gene frequencies for testing the segregation ratio in crosses of 'Avocet S' with 'Homa' and 'Parastoo' were modified, assuming that lines that were homozygous for four and three genes could not be distinguished based on the severity phenotype and were grouped into HPTR class. The ratios at both testing sites individually and when pooled were in agreement with the hypothesis of the control of resistance by at least three independent additive genes in cvs. Saar, Simorgh, and Cocnoos, and four independent genes in Homa and Parastoo. The distribu-

Table 2. Mean leaf rust severities of parents, F_1 plants, and F_2 populations tested at El-Batan, and least square population means of F_3 and F_5 lines tested at Ciudad Obregon, derived from a one-way diallel cross involving five resistant and one susceptible wheat genotypes along with checks^y

Tested	El Batan		Ciudad Obregon	
	F ₁	F ₂	F ₃	F ₅
Susceptible $\times$ resistant crosses				
Avocet-YrA $\times$ Saar	19.3 cd	30.8 b	40.4	46.0
Avocet-YrA $\times$ Simorgh	26.5 bc	28.4 b	44.9	45.3
Avocet-YrA $\times$ Homa	38.3 b	18.5 c	35.8	36.7
Avocet-YrA $\times$ Parastoo	6.4 efg	12.2 c	34.0	24.1
Avocet-YrA $\times$ Cocnoos	38.1 b	33.3 b	44.2	40.3
Standard error	...	...	2.1	3.9
Resistant $\times$ resistant crosses				
Saar $\times$ Simorgh	10.0 def	6.5 d	...	11.4
Saar $\times$ Homa	3.3 fg	2.6 efg	...	10.7
Saar $\times$ Parastoo	6.4 efg	3.8 def	...	8.7
Saar $\times$ Cocnoos	6.4 efg	5.9 de	...	10.7
Simorgh $\times$ Homa	10.0 efg	6.5 d	...	7.8
Simorgh $\times$ Parastoo	5.0 efg	2.5 efg	...	5.9
Simorgh $\times$ Cocnoos	6.4 efg	6.0 de	...	9.2
Homa $\times$ Parastoo	1.0 g	1.1 g	...	2.3
Homa $\times$ Cocnoos	13.2 de	4.8 def	...	5.9
Parastoo $\times$ Cocnoos	5.0 efg	3.5 defg	...	2.7
Standard error	...	...	...	1.1
Parents				
Avocet-YrA	100.0 ab ^z	100.0 a	81.2	99.8
Saar	5.0 efg	1.9 fg	6.4	12.9
Simorgh	10.0 def	4.4 def	6.4	10.9
Homa	1.0 g	3.1 defg	6.9	6.5
Parastoo	1.0 g	1.0 g	1.9	5.4
Cocnoos	3.3 fg	1.0 g	1.5	0.8
Standard error	...	...	12.7	15.5
Checks				
Avocet-YrA	...	...	83.2	98.9
Avocet+Lr34	...	...	10.7	10.5
Thatcher	...	...	75.6	79.4
Thatcher+Lr34	...	...	1.1	1.0
Jupateco S	...	...	99.4	99.9
Jupateco+Lr34	...	...	26.4	30.2
AC-Nanda	...	...	38.0	32.4
Cook	...	...	18.8	13.9
Saar	...	...	11.5	10.8
Cocnoos	...	...	1.8	2.0
Standard error	...	...	11.4	12.5

^y Means followed by letters in common within generations do not differ significantly based on Tukey's standardized range test on arcsine transformed data ($P < 0.05$). Range test is not valid for least square means.

^z Leaf rust severity recorded when the susceptible check Avocet-YrA reached 80 to 100%, based on the modified Cobb's scale (22).

tion of F₅ lines in susceptible × resistant crosses further verified the F₃ results.

Resistant-parent intercrosses. The F₅ lines of the resistant-parent intercrosses were tested at Ciudad Obregon to study the similarity or diversity of resistance genes in the parents. Although none of the F₅ lines resembled the leaf rust severity of the susceptible parent Avocet-YrA, few lines with leaf rust severities higher than those of the parents could be found (Table 3). In crosses of Saar with Homa, Parastoo, and Cocnoos, and in the cross Homa × Cocnoos, few lines were found with disease

severities as high as 50 to 70%. This indicated that, although parental lines have at least one gene in common, the other additive genes were different. Crosses of Simorgh and Homa with Parastoo displayed less segregation for disease severity in both F₂ and F₅ generations, indicating that perhaps two additive genes were common in the parents of the respective crosses.

Quantitative genetics analysis. Analyses of variance for the terminal leaf rust severities in all experiments are given in Table 5. In the F₃ experiment, only the susceptible × resistant crosses were in-

cluded. In other experiments, all crosses in the diallel mating were tested. The differences among the parental lines, F₁ crosses, F₂ populations, and F₃ and F₅ lines were highly significant. The parents versus F₁ crosses, F₂ populations, and F₃ and F₅ lines were highly significant, which was an indication of a deviation of F₁ crosses and F₂, F₃, and F₅ population means from mid-parental values. The differences among the check genotypes tested in the F₃ and F₅ experiments at Ciudad Obregon also were highly significant. GCA mean squares were highly significant in F₁, F₃, and F₅

Table 3. Frequency distribution of leaf rust severity of F₂ plants, and F₃ and F₅ lines derived from a one-way diallel cross involving five resistant and one susceptible wheat genotypes, when inoculated with *Puccinia triticina* race MCJ/SP

	No. of plants or lines with disease severity ^z												
Cross, generation	1	5	10	20	30	40	50	60	70	80	90	100	Total
Susceptible × resistant													
Avocet-YrA × Saar													
F ₂	28	35	44	43	38	22	8	23	2	31	4	2	280
F ₃	1	1	2	20	32	26	23	17	18	3	8	4	155
F ₅	1	4	8	32	6	14	13	11	14	6	8	6	123
Avocet-YrA × Simorgh													
F ₂	57	41	49	26	21	31	4	23	2	29	4	10	297
F ₃	1	1	1	16	28	15	14	17	10	6	2	1	112
F ₅	3	7	8	20	15	20	8	13	4	9	7	6	120
Avocet-YrA × Homa													
F ₂	85	51	23	22	15	14	2	17	0	12	2	3	246
F ₃	0	6	4	18	11	8	8	8	4	1	0	0	68
F ₅	3	21	7	20	15	16	14	6	12	3	3	2	122
Avocet-YrA × Parastoo													
F ₂	72	63	50	26	13	8	1	5	0	2	2	2	244
F ₃	3	2	9	28	31	11	9	10	6	3	0	0	112
F ₅	14	8	15	34	11	9	7	10	7	3	3	2	123
Avocet-YrA × Cocnoos													
F ₂	52	23	23	17	14	29	2	29	1	21	6	11	228
F ₃	1	1	1	18	19	22	24	18	7	3	1	0	115
F ₅	4	0	2	24	20	20	11	15	4	9	5	2	116
Resistant × resistant													
Saar × Simorgh													
F ₂	67	147	63	33	1	0	0	0	0	0	0	0	311
F ₅	3	38	41	51	9	0	0	0	0	0	0	0	142
Saar × Homa													
F ₂	229	50	13	6	3	0	0	0	0	0	0	0	301
F ₅	27	22	16	42	15	7	2	3	0	0	0	0	134
Saar × Parastoo													
F ₂	167	65	23	15	1	0	0	0	0	0	0	0	271
F ₅	49	11	14	29	16	7	2	1	0	0	0	0	129
Saar × Cocnoos													
F ₂	169	60	29	24	15	4	0	0	0	0	0	0	301
F ₅	21	25	20	39	11	1	1	0	1	0	0	0	119
Simorgh × Homa													
F ₂	126	86	19	16	9	3	4	1	0	0	0	0	264
F ₅	28	45	23	20	1	0	0	0	0	0	0	0	117
Simorgh × Parastoo													
F ₂	190	67	16	0	0	0	0	0	0	0	0	0	273
F ₅	45	54	29	10	0	0	0	0	0	0	0	0	138
Simorgh × Cocnoos													
F ₂	181	64	31	29	13	5	0	0	0	0	0	0	323
F ₅	18	24	10	19	4	1	0	0	0	0	0	0	76
Homa × Parastoo													
F ₂	283	6	1	0	0	0	0	0	0	0	0	0	290
F ₅	83	44	6	0	0	0	0	0	0	0	0	0	133
Homa × Cocnoos													
F ₂	214	42	23	26	14	0	0	0	0	0	0	0	319
F ₅	31	35	14	14	2	2	1	0	0	0	0	0	99
Parastoo × Cocnoos													
F ₂	228	46	16	18	5	0	0	0	0	0	0	0	313
F ₅	52	31	18	7	1	0	0	0	0	0	0	0	111

^z Leaf rust severity data based on the modified Cobb's scale (22) when the susceptible check 'Avocet-YrA' reached 80 to 100% in susceptible × resistant crosses, and 12 to 13 days later when it had necrotic leaves in resistant × resistant crosses (Jupateco+Lr34 check displayed between 50 and 60% severity at that time).

diallel experiments, whereas SCA mean squares were significant only in F₁ and F₂ diallel experiments. Components of variance ratios were 0.69, 0.94, and 0.98 for the F₁, F₂, and F₅ diallel experiments, respectively. The components of variance ratio estimated for the F₁ diallel was relatively smaller than those estimated for the F₂ and F₅ diallel experiments. This probably was due to greater SCA estimates in

the F₁ experiment. The relatively high estimates for the components of variance ratios indicated that GCA was of major importance in predicting the progeny performance. The greater importance of GCA effects also was evident based on the relatively high correlations between parental means and GCA effects ($r = 0.89$, $P < 0.05$ for F₁; $r = 0.94$, $P < 0.01$ for F₂; and $r = 0.97$, $P < 0.01$ for F₅). Narrow-sense

heritability estimates were 0.67, 0.92, and 0.96 for F₁, F₂, and F₅ diallel experiments, respectively. Similar to the component of variance ratios, the heritability estimate for the F₁ diallel was relatively smaller than those for the F₂ and F₅, probably due to the greater dominance effects estimated in the F₁ experiment.

Estimates of GCA effects for parents and SCA for the crosses are given in Table

Table 4. Frequency distribution of leaf rust severity of F₃ and F₅ lines derived from crosses of the susceptible genotype ‘Avocet-YrA’ with five resistant wheat genotypes, when inoculated with *Puccinia triticina* race MCJ/SP

Generation/tested in, cross ^u	No. of lines with reaction					No. of genes ^z	χ^2	P value
	HPTR ^v	HPTS ^w	SegI ^x	SegS ^y	Others			
F ₃ /El-Batan								
Saar	1	1	39	37	...	3	1.51	>0.50
Simorgh	1	2	33	34	...	3	2.87	>0.25
Homa	3	1	51	25	...	4	1.79	>0.50
Parastoo	3	1	44	20	...	4	2.18	>0.50
Cocnoos	0	0	26	26	...	3	3.11	>0.25
F ₃ /Ciudad Obregon								
Saar	3	3	93	56	...	3	1.43	>0.50
Simorgh	3	1	60	48	...	3	1.49	>0.50
Homa	4	1	43	18	...	4	2.63	>0.25
Parastoo	5	1	71	35	...	4	0.80	>0.99
Cocnoos	4	1	58	52	...	3	4.34	>0.10
F ₃ /pooled								
Saar	4	4	132	93	...	3	0.08	>0.99
Simorgh	4	3	93	82	...	3	1.34	>0.50
Homa	7	2	94	43	...	4	3.78	>0.25
Parastoo	8	2	115	55	...	4	2.57	>0.25
Cocnoos	4	1	84	78	...	3	2.78	>0.25
F ₅ /Ciudad Obregon								
Saar	6	10	...	...	107	3	2.00	>0.25
Simorgh	14	11	...	...	95	3	1.89	>0.25
Homa	20	5	...	...	97	4	0.52	>0.75
Parastoo	15	5	...	...	103	4	3.56	>0.10
Cocnoos	4	8	...	...	104	3	4.22	>0.10

^u Avocet-YrA crossed with genotypes listed.

^v Homozygous for the parental type resistance (homozygous for all resistance alleles).

^w Homozygous for the parental type or higher susceptibility (homozygous lacking all resistance alleles).

^x Either segregating or homozygous for disease levels higher than that of the resistant parent but less than that of the susceptible parent (homozygous for at least one resistance allele).

^y Segregating with disease levels reaching the susceptible parent response (heterozygous for at least one locus and homozygous for susceptibility alleles at other loci).

^z Tested for segregation of the indicated number of genes. The ratio used in F₃ was 0.0156 HPTR:0.0156 HPTS:0.5625 SegI:0.4062 SegS expected for the segregation of three additive genes and 0.0507 HPTR:0.0039 HPTS:0.6328 SegI:0.3125 SegS for the segregation of four genes. The ratio used in F₅ was 0.0837 HPTR:0.0837 HPTS:0.8327 others for the segregation of three genes and 0.1884 HPTR:0.0366 HPTS:0.775 others for the segregation of four genes. Four gene ratios are adjusted based on the combined frequency of lines homozygous for three and four genes into the HPTR category, assuming that it was not possible to distinguish such lines based on rust severity response.

Table 5. Mean squares (MS) for the arcsine-transformed terminal leaf rust severity of parents, F₁ crosses, F₂ populations, and F₅ lines derived from a one-way diallel cross, involving susceptible genotype, ‘Avocet-YrA’ and five resistant wheat genotypes, and F₃ of susceptible × resistant crosses, when inoculated with *Puccinia triticina* race MCJ/SP

SV ^z	Generation ^y						
	df	F ₁	F ₂	F ₃		F ₅	
		MS	MS	df	MS	df	MS
Block	2	< 0.001	< 0.001	2	0.036	9	0.008
Treatment (T)	20	0.309**	0.317**	20	0.545**	30	1.850**
Parents	5	0.979**	1.020**	5	0.479**	5	1.450**
Parents vs. crosses	1	0.074**	0.073**	1	0.693**	1	0.132**
Crosses	14	0.086**	0.083**	4	0.382**	14	0.535**
GCA	5	0.187**	0.221**	...	...	5	0.116**
SCA	9	0.031**	0.007**	...	...	9	0.001
Crosses × block	28	0.004	0.001	...	...	...	...
Checks	...	...	...	9	0.854**	9	2.554**
Line/T	...	...	...	557	0.053	1765	0.048**
Error	40	0.002	< 0.001	31	0.066	105	0.005

^y * and ** = Significant at $P < 0.05$ and 0.01 , respectively.

^z SV = source of variation, GCA = general combining ability, and SCA = specific combining ability.

6. The F_1 crosses, F_2 populations, and F_5 lines involving parents with negative GCA effects were more resistant than the average of the respective generations, whereas significant positive GCA indicated greater susceptibility. Parastoo had the greatest negative GCA in all experiments that was significant in F_1 and F_5 , and highly significant in F_2 . This indicated that the most resistant F_1 progenies, F_2 populations, or F_5 lines on average had Parastoo as one of the parents. The relative values of GCA effects were slightly inconsistent across generations.

The SCA of the F_1 progenies, F_2 populations, and F_5 lines are presented in Table 6. In crosses involving the susceptible parent, Parastoo had the greatest negative SCA effect in all generations (SCA = -12.11, $P < 0.01$ for F_1 ; SCA = -7.74, $P < 0.01$ for F_2 ; and SCA = -4.30, $P < 0.01$ for F_5). This indicated that Parastoo had the greatest reduction in average terminal leaf rust severity when crossed with the susceptible parent. In the resistant parent intercrosses, the cross Saar × Cocnoos had significant and highly significant negative SCA effect in F_2 and F_5 , respectively (SCA = -3.24, $P < 0.05$ for F_2 and SCA = -2.52, $P < 0.01$ for F_5). As for the GCA effects, slight inconsistency in SCA effects of the crosses also was observed across generations.

Estimates of the minimum numbers of additive genes in the resistant parental lines based on the F_5 data from the susceptible × resistant crosses are presented in Table 7. The estimates based on the two formulae were between 2.17 and 4.02.

DISCUSSION

The resistant parents included in this study were selected at CIMMYT, Mexico for seedling and adult plant compatibility, characterized by a high infection type, accompanied by low adult plant leaf rust severity to common Mexican races of *P.*

tritricina (32). They all displayed moderately susceptible to susceptible infection types in field evaluations, confirming the compatible nature of the host–pathogen interaction that is a known feature of partial (21) or slow-rusting resistance (6).

F_1 progenies showed intermediate levels of terminal leaf rust severity, significantly higher than those of the resistant parents. This implied that adult-plant resistance to leaf rust in the evaluated genotypes was incompletely dominant. Segregation analyses indicated the presence of at least three to four genes having additive effects in each of the resistant parents. When quantitative methods of analysis were used, the estimates of the number of additive genes ranged between 2.17 and 4.02. These numbers are different from those estimated by the Mendelian method of segregation analysis. However, they are in agreement with the involvement of more than a single locus in the inheritance of adult-plant resistance in the genotypes. The difference in the estimated numbers may have been due to the fact that estimates based on the quantitative methods of genetic analysis may be biased and influenced by the visual estimates of disease severity (39).

All resistant parents displayed leaf tip necrosis phenotype, which is known to be either pleiotropic or tightly linked to the slow-rusting adult-plant resistance gene

Lr34 (29). Moreover, the resistant parents were derived from intercrosses of *Lr34*-carrying genotypes. Therefore, we conclude that *Lr34* was one of the genes present in all parents. The frequency distribution of the leaf rust severity in F_3 and F_5 lines derived from the susceptible × resistant crosses were skewed, with population means shifting toward the resistant parent. This may have resulted from unequal effects of *Lr34* and additive genes.

The adult-plant resistance gene *Lr46* is likely to be present in Saar and Simorgh. They both are three-way cross derivatives having Sonoita 81 as one of the parents. Sonoita 81 shares common parentage with the Mexican cv. Pavon 76 (coefficient of parentage = 0.48; estimated by the International Wheat Information System; 10), which has *Lr46*. However, this hypothesis needs to be further investigated.

Although we assume that gene *Lr34* is common in all resistant parents, some of the F_5 lines derived from intercrosses of resistant parents had disease severities considerably higher than their parental responses. This indicated that some of the additive genes in the parents were nonallelic; therefore, they can be accumulated into new breeding lines to further enhance the level of resistance. The transgression for the maximum infection level varied among the resistant × resistant crosses. The high-

Table 7. Minimum number of effective additive genes for resistance to *Puccinia tritricina* race MCJ/SP in five wheat genotypes

Avocet-YrA crossed with	Number of genes estimated based on ^z	
	Formula 1	Formula 2
Saar	2.18	2.19
Simorgh	2.42	2.50
Homa	3.20	2.17
Parastoo	3.21	3.09
Cocnoos	4.02	2.50

^z Formulae 1 and 2 are based on Bjarko and Line (3) and Wright (40), respectively.

Table 6. Estimates of general (on diagonal) and specific combining ability for the terminal leaf rust severity in F_1 (top), F_2 (middle), and F_5 (bottom) generations of a one-way diallel cross, involving susceptible genotype 'Avocet-YrA' and five resistant wheat genotypes^z

Parent	Avocet-YrA	Saar	Simorgh	Homa	Parastoo	Cocnoos
Avocet-YrA	16.00**	-4.45	-0.61	9.05**	-12.11**	8.13*
	16.94**	4.25*	1.79	-4.05*	-7.74**	5.75**
	24.88*	0.04	3.33**	-2.20*	-4.30**	3.12**
Saar	...	-4.75*	3.46	-4.86	8.63**	-2.78
	...	-1.47	-1.78	-1.54	2.31	-3.24*
	...	0.68	-3.03**	2.41*	3.10**	-2.52**
Simorgh	...	...	-1.91	-1.36	4.13	-5.61*
	...	...	-1.36	2.38	0.83	-3.22*
	...	...	-4.38	-0.45	0.80	-0.64
Homa	...	...	...	0.08	-1.86	-0.95
	...	...	...	-5.46**	3.54*	-0.33
	...	...	...	-6.89	0.30	-0.05
Parastoo	...	...	...	...	-10.41*	1.21
	...	...	...	...	-8.14**	1.05
	...	...	...	...	-9.91*	0.09
Cocnoos	...	...	...	...	...	1.00
	...	...	...	...	...	-0.50
	...	...	...	...	...	-4.37

^z * and ** = Significantly different from zero at $P < 0.05$ and 0.01 , respectively.

est levels of leaf rust severities (50 to 70%) in the F_5 generation was found for the cross Saar $\times$ Cocnoos, whereas the cross Homa $\times$ Parastoo gave rise to lines with no more than 10% leaf rust severities. Saar and Cocnoos also share less common parentage based on the pedigree information and seem to carry different genes apart from *Lr34*, which is common to both. In other words, between Saar and Cocnoos, other than gene *Lr34*, two additional genes could be present in each parent. They share Kauz*2/Trap//Kauz as a common parent. The F_5 lines in the resistant-parent intercrosses with terminal leaf rust severities as high as 60% probably are those with *Lr34* alone. This further indicated that a single additive leaf rust adult-plant resistance gene does not provide acceptable level of resistance. This was in accordance with the previously published reports (18,31) of leaf rust severities of up to 50 to 60% when *Lr34* was deployed alone. A further example involves gene *Lr46*. Singh et al. (34) reported leaf rust severities of up to 60% when the *Lr46* gene was present alone. The leaf rust severities of the three *Lr34* isogenic lines in Thatcher, Jupateco, and Avocet backgrounds, in F_3 and F_5 experiments, were not higher than 30%. The data presented in Table 2 are from an evaluation made when Avocet-YrA had just reached 80 to 100% rust severity. The data on resistant $\times$ resistant crosses (Table 3) were recorded about 12 to 13 days later to allow a higher variation in severity of the lines. Thatcher+*Lr34* is a very late line in Ciudad Obregon and the expression of *Lr34* may be different. Moreover, previous studies on this line (Thatcher+*Lr34* line RL6058) have determined involvement of factors other than just *Lr34* (31).

Additive gene effects appeared to be more important than nonadditive gene effects in the inheritance of adult-plant resistance to leaf rust, as indicated by the quantitative analysis in our study. The GCA, which represents the average performance of a genotype and mainly is reflective of the additive gene effects, was significant in all generations. The SCA also was found to be significant in F_1 and F_2 , but the mean squares for SCA were much smaller than the mean squares of GCA. Moreover, the components of variance ratio, proposed by Baker (2), were 0.67 for F_1 and close to unity for F_2 and F_5 . Our conclusion that additive gene effects contributed the most in the inheritance of adult-plant resistance to leaf rust is consistent with results from previous studies on race-nonspecific adult-plant resistance to leaf rust in other wheat genotypes (8,20,27).

In addition to the additive gene effects, there also was evidence for the presence of significant nonadditive gene effects in our study. The model that is used in the combining ability analysis assumes that epistasis and genotype-environment effects are

negligible. Therefore, any nonadditive effect is referred to as dominance. The significant parent-crosses effect in analysis of variance for all generations indicated the presence of a significant deviation of F_1 crosses, and F_2 and F_5 population mean values from midparental values. The frequency distributions of the leaf rust severities of the susceptible $\times$ resistant crosses also were skewed. Moreover, significant SCA effects in F_1 and F_2 and smaller components of variance ratio estimated for F_1 pointed to the presence of nonadditive gene effects. Messmer et al. (20) also reported the presence of significant dominance effects in the inheritance of adult-plant resistance to leaf rust in wheat.

It also should be noted that, because leaf rust severity assessment was based on a visual scale, there could be the possibility of overestimation of nonadditive effects. The application of a quantitative genetic approach in the study of gene effects has been argued (39) for having underestimates of additive variance and overestimates of nonadditive variance. Therefore, we relate the significant nonadditive variance estimated in this analysis, at least in part, to the overestimation of such effects due to the visual scale of disease assessment that was used. Gene *Lr34* is known to interact strongly with other genes (11). This interaction in lines with *Lr34* in association with one or more additional genes could produce a level of resistance greater than expected from a simple additive effect. The nonadditive effects in this study may have been caused by this enhancement effect of *Lr34*.

A high level of adult-plant resistance to leaf rust in the resistant parents included in our study was found to be inherited by a few genes, with additive gene effects being the major contributor. Our results were in agreement with the previously published reports (3,4,35,36) of the inheritance of adult-plant resistance to leaf rust to be the result of additive interactions of a few genes. Resistance of some widely grown cultivars carrying *Lr34* and additional genes having additive effects has remained effective for a long period of time (36). A high level of resistance in the resistant parents included in our study, therefore, is expected to be of a durable nature.

Transgressive segregation giving rise to lines with higher levels of resistance than those of parents was observed in the intercrosses of resistant parents. Other researchers (4,8,17) also reported the presence of transgressive segregation for latent period and receptivity of leaf rust in wheat. Incorporating the additive genes from different sources, therefore, will be possible through intercrossing adult-plant resistant genotypes carrying different additive genes. It has been demonstrated (32,33) that incorporating these additive genes in high-yielding cultivars by means of top-crossing or limited back-crossing may

result in the achievement of resistance levels reaching near-immunity while maintaining or even increasing the actual yield potential.

The estimates of the narrow-sense heritability ranged from 0.67 to 0.97 among generations. This indicated that the terminal leaf rust severity in the field was moderately to highly heritable. Other researchers (7,9,16,27) reported moderate to high heritability for the latent period and the area under the leaf rust progress curve. Kuhn et al. (16), based on the higher heritability estimates of latent period over pustule size, suggested that selection could be done for a longer latent period. Kolmer and Liu (15), however, recommended the use of direct genetic analysis, because latent period may not always be predictive of adult-plant resistance to leaf rust. It can be concluded from the results of our study that breeding for adult-plant resistance would be possible by selection for disease severity at the critical stages of growth. Selection must be done under an epidemic of *P. tritici-na* with a high spectrum of virulence for any known seedling resistance gene that may act epistatically and mask the effect of adult-plant resistance genes. It also should be noted that selection pressure for plants with very low leaf rust severities in the early segregating generations may result in deleting the heterozygous plants that would be capable of giving progenies with higher levels of resistance.

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