

Heritability and Number of Genes Governing Adult-Plant Resistance to Powdery Mildew in Houser and Redcoat Winter Wheats

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ABSTRACT

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Heritability and number of genes controlling adult-plant resistance to powdery mildew (*Blumeria graminis* f. sp. *tritici*) was studied in three winter wheat (*Triticum aestivum* L.) crosses. Parents, F₁, F₂, F₃, and backcross populations were evaluated in the field under naturally occurring inoculum of powdery mildew. Number of genes controlling adult-plant resistance in Houser and Redcoat was determined by both qualitative and quantitative methods. Mildew severity was used for qualitative esti-

mates, and area under the disease progress curve values were used for quantitative estimates. Adult-plant resistance to powdery mildew in Redcoat and Houser is controlled by two to three genes. Disease reactions of the parents and progenies of resistant × susceptible crosses indicated that resistance in both cultivars is partially dominant and additive. Broad-sense heritability, estimated by the variance components method, ranged from 0.57 to 0.94, while heritability estimated by the standard units method ranged from 0.19 to 0.35. Selection for adult-plant resistance among progeny derived from crosses with Redcoat and Houser would likely be most effective in advanced generations.

Powdery mildew caused by *Blumeria graminis* (DC.) E.O. Speer f. sp. *tritici* Ém. Marchal (syn. *Erysiphe graminis* f. sp. *tritici*), is an important disease of wheat (*Triticum aestivum* L.) in areas with a maritime or semicontinental climate (1). The most economical and environmentally safe control is achieved with the utilization of resistant cultivars. In most cases, genes conferring hypersensitive types of resistance have been used in wheat (1), but this resistance has been ephemeral (20,22).

Resistance to powdery mildew that retards infection, growth, and reproduction of the pathogen in adult plants, but not in seedlings, has been termed "slow mildewing," "adult-plant resistance" (7), or "partial resistance" (8). Henceforth, this type of resistance will be referred to as adult-plant resistance (APR). APR to powdery mildew is more durable than hypersensitive resistance (22) and has been identified in wheat (1), barley (*Hordeum vulgare* L.) (13), and oats (*Avena sativa* L.) (14). To effectively breed for this type of resistance, information on the genetics and mode of inheritance is essential.

Unfortunately, little is known about the genetics and inheritance of APR to powdery mildew in wheat (1,8). APR to powdery mildew has been detected in cultivars that either have no identified major-gene resistance or in which major-gene resistance has been overcome (1). Bennett (1) speculated that several modern European cultivars possess APR in addition to the major gene *Pm2*. Shaner and Finney (23) reported that APR to powdery mildew in wheat behaves genetically as a quantitative trait. Using monosomic analyses, Chae and Fischbeck (3) reported that genes on 14 chromosomes are involved in the expression of APR to powdery mildew in the cultivar Diplomat. Based upon transgressive segregation, Hautea et al (8) suggested that APR to powdery mildew in wheat is polygenic in nature. Using generation mean analyses, they found that additive gene effects were most important for mildew resistance in four spring wheat crosses. They reported heritability estimates as high as 32% using parent-offspring regression and standard units methods.

Jones et al (15) attributed APR to barley mildew (*B. graminis* f. sp. *hordei*) to additive and dominance effects of as many as

five independent genes with no evidence of nonallelic interaction or epistasis. Heun (9) reported narrow-sense and broad-sense heritability estimates of 88 and 94%, respectively, for quantitative resistance to powdery mildew in barley. In oats, Jones (12) postulated that four to nine genes, depending on growth stage, governed APR to mildew (*B. graminis* f. sp. *avenae*).

The objectives of our study were to determine the number of genes and heritability of APR to powdery mildew in Houser and Redcoat winter wheats using both qualitative and quantitative genetics methods.

MATERIALS AND METHODS

Parents and crosses. The cultivar Becker (PI 494524) is susceptible to powdery mildew (16), and Houser (CI 17736) and Redcoat (CI 13179) have APR (11,21). Crosses among these cultivars, which included Houser × Becker, Redcoat × Becker, and Houser × Redcoat were made in the greenhouse. The parents, F₁, BC₁P₁ (F₁ × Becker), BC₁P₂ (F₁ × resistant parent), F₂, and F₃ generations were evaluated in field nurseries at Warsaw, Virginia, under natural inoculum of powdery mildew. The nurseries were grown on Suffolk soils (fine-loamy, siliceous, thermic Typic Hapludult). Seeds of parents, F₁, BC₁P₁, BC₁P₂, and F₂ were space-planted in 7.5-m long rows in mid October 1991. One row of Becker was planted in alternate rows to facilitate mildew increase and spread. The distance between rows was 30 cm. Depending on the cross, population sizes ranged from 21 to 31 plants per parent, 25 to 45 plants per F₁, 41 to 66 plants per BC₁P₁, 46 to 52 plants per BC₁P₂, and 129 to 217 plants per F₂.

Twenty seeds of F₃ families derived from 97 to 100 randomly selected F₂ plants were space-planted in 3-m long rows in the field on 20 October 1992. A row of each parent was planted for every 20 rows of F₃s. The experimental plot was surrounded by a border of two rows of Becker.

Cultural practices. In 1991, fertilizer was applied in the fall at the time of land preparation at a rate of 33.6 kg/ha of nitrogen, 56 kg/ha of phosphorous, and 90 kg/ha of potassium. Nitrogen fertilizer was top-dressed in early March 1992 at a rate of 101 kg/ha. In 1992, fall fertilizer was applied at a rate of 33.6 kg/ha of nitrogen, 30 kg/ha of phosphorous, 74 kg/ha of potassium,

and 11 kg/ha of sulfur. Nitrogen fertilizer was top-dressed in the spring at a rate of 45 and 56 kg/ha when the plants were at Zadoks (31) growth stages 25 and 30, respectively.

Disease assessment. Powdery mildew severity (0–50% leaf area covered) on penultimate leaves was assessed using the James disease assessment key (10). Several penultimate leaves of each plant were evaluated visually, and an average mildew severity per plant was determined. The two APR cultivars were similar in maturity (growth stage), and Becker was slightly earlier. Flag leaves of the parents and progenies had emerged when the first disease assessment was made. In the first year, mildew severities of all plants for parents, F₁, BC₁P₁, BC₁P₂, and F₂ populations were recorded at weekly intervals for four weeks. Becker, Houser, and Redcoat were at growth stages 59, 45, and 47, respectively, when the first reading was taken. In the second year, mildew severities of five randomly selected plants for each parent and F₃ family were recorded at weekly intervals for 3 wk. In that year, Becker, Houser, and Redcoat were at growth stages 55, 45, and 47, respectively, at the time of first reading. In addition, mildew severity of all 20 plants per F₃ family were recorded when Becker, Houser, and Redcoat were at growth stages 77, 73, and 75, respectively. Area under the disease progress curve (AUDPC) was calculated for each plant from mildew severity ratings following the formula used by Bjarko and Line (2).

Gene number and heritability estimates. Number of genes controlling adult-plant resistance to powdery mildew in Houser and Redcoat were estimated by both qualitative and quantitative methods. For qualitative analyses, mildew severities from the third assessment of parents, F₁, BC₁P₁, BC₁P₂, and F₂ populations were used. In the 1992–1993 experiments, mildew severity values obtained from the final assessment of all 20 plants of each F₃ family and parents were used to estimate gene number. Individual plants of a cross or family were classified for reaction type as resistant, susceptible, or intermediate. A plant was considered resistant if its mildew severity was less than or equal to the mean mildew severity plus one standard deviation of the resistant parent; whereas, a plant was considered susceptible if its mildew severity was more than or equal to the mean mildew severity minus one standard deviation of the susceptible parent. Plants having mildew severities in between these two groups were classified as intermediate.

The observed number of resistant plus intermediate and susceptible plants of F₂ and backcross generations were tested for various genetic ratios by chi-square analysis. F₃ families were grouped into three classes: homozygous resistant, homozygous susceptible, and segregating. Reaction types of homozygous families were compared with those of the F₂ plants from which they were derived to verify their proposed genotypes. The F₃ families in the three groups also were tested for genetic ratios using chi-square analysis. Based on BC₁P₁, F₂, and F₃ genetic ratios, the number of genes present in the resistant parents was determined.

AUDPC values were used to estimate gene numbers by quantitative methods. For F₃ populations, the mean AUDPC value for each family was used. AUDPC values ranged from 6 to 497 within F₂ populations and from 17 to 335 within F₃ populations, and a systematic relationship was observed between the mean and standard deviation for AUDPC values of different popula-

tions. Subsequently, AUDPC values were log transformed using Wright's (30) method of scale transformation to stabilize the variances of populations with different mean values. This transformation stabilized the variances of parents and F₁ and F₂ populations grown in 1991–1992, and the distribution of each of these populations was normal as tested by the Shapiro-Wilk statistic (24) and the Kolmogorov-Smirnov test (28). The F₃ populations did not show normal distribution when AUDPC values were transformed using Wright's log transformation method; however, simple log transformation stabilized the variances of parents and F₃ populations and normalized the population distribution. Quantitative estimates of minimum number of effective factors (will be called genes hereafter for convenience) controlling APR were obtained for F₂ and F₃ generations using the formulas of Wright (30), with necessary modifications for F₃ generation analyses using the formula of Cockerham (4) as follows: for F₂ generation,

$$n = (GR)^2/8[V_{F_2} - (V_{PS} + V_{PR} + 2V_{F_1})/4]$$

for F₃ generation,

$$n = (GR)^2/5.33[V_{F_3} - (V_{PS} + V_{PR})/2]$$

where GR is genotypic range, estimated as the difference between the mean response of two parents; V_{PS}, V_{PR}, V_{F₁}, V_{F₂}, and V_{F₃} are variances of susceptible parent, resistant parent, F₁, F₂, and F₃, respectively; and *n* is the estimated number of genes. Both formulas assume that no linkage exists between the loci involved, the effects of all loci involved are equal, dominance and epistasis are absent, and all genes for resistance are in a single parent of the cross. The presence of linkage, dominance, or unequal effects at different loci will result in an underestimation of the actual number of segregating genes present, while the presence of epistasis may cause either an overestimation or an underestimation of the actual number of segregating genes. In most cases, these formulas give a conservative estimate of the number of genes involved (2). Standard error of gene number estimates was obtained following the formula of Lande (17).

Log transformed values of AUDPC were used to estimate heritabilities. Broad-sense heritability was estimated in the F₂ and F₃ generations by the variance components method after Nyquist (19). Heritability in standard units was estimated using the method of Frey and Horner (5).

RESULTS

Powdery mildew was severe in both years of this study, and conditions were favorable for mildew development from the first to last assessment date.

Houser × Becker cross. Powdery mildew severity for Houser, Becker, and their F₁ was 2.9, 29.8, and 8.6%, respectively, which indicated that resistance is partially dominant. An F₂ population of 205 plants segregated for 200 plants with resistant (R) or intermediate (I) reaction types and five with susceptible (S) type (Table 1). This segregation fit a 63(R+I) to 1(S) ratio, indicating that Houser has three partially dominant genes for resistance. However, the observed F₂ segregation pattern also fit (0.20 > *P* >

TABLE 1. Segregation ratios for adult-plant resistance to powdery mildew and chi-square tests of populations for crosses between the mildew-susceptible wheat cultivar Becker and mildew-resistant Houser

Population	No. of plants		No. of F ₃ lines			Ratio tested	χ ²	<i>P</i>
	R ^a + I	S	Hom ^b R	Seg ^c	Hom S			
BC ₁ P ₁ ^d	36	7	7:1	0.91	0.25–0.50
BC ₁ P ₂	52	0
F ₂	200	5	63:1	0.66	0.25–0.50
F ₃	2	94	1	1:62:1	0.38	0.75–0.90

^a R = resistant, I = intermediate, and S = susceptible reaction type.

^b Hom = homozygous.

^c Seg = segregating.

^d BC₁P₁ = F₁ × Becker and BC₁P₂ = F₁ × Houser.

0.10) a 61(R+I) to 3(S) ratio that would be expected for two partially dominant and one recessive genes. The segregation pattern of the BC₁P₁ (F₁ × Becker) population fit a 7(R+I) to 1(S) ratio, which was expected for the segregation of three partially dominant genes controlling resistance in Houser. As expected, all 52 plants of BC₁P₂ (F₁ × Houser) produced only resistant or intermediate plants. In the F₃, two homozygous parental-type resistant, 94 segregating, and one homozygous parental-type susceptible lines were obtained. This segregation fit a 1:62:1 ratio and further supported the hypothesis that resistance in Houser is controlled by three partially dominant genes, which were additive when present together.

The number of genes estimated using quantitative methods was 2.92 ± 0.43 and 2.75 ± 0.70 in the F₂ and F₃ generations, respectively (Table 2). This is in agreement with the three-gene estimate obtained by the qualitative method. Broad-sense heritability, estimated by the variance components method, was 0.76 and 0.79 in F₂ and F₃ generations, respectively (Table 2). Heritability estimated in standard units was 0.35 and was significantly different from zero.

Redcoat × Becker cross. Redcoat, Becker, and their F₁ had mean mildew severities of 1.1, 24.4, and 4.9%, respectively, suggesting that resistance is partially dominant. Segregation in the F₂ generation produced 205 plants with resistant or intermediate disease levels and 12 susceptible plants (Table 3). This segregation fit a 15(R+I) to 1(S) ratio, and indicated that Redcoat has two partially dominant genes for resistance to powdery mildew. However, the observed F₂ segregation also fit (0.70 > P > 0.50) a 61(R+I) to 3(S) ratio, expected for two partially dominant and one recessive genes for resistance. Segregation of the BC₁P₁ (F₁ × Becker) population fit a 3(R+I) to 1(S) ratio, which was expected based on the hypothesis of two partially dominant or two partially dominant and one recessive genes controlling resistance in Redcoat. As expected, none of the 46 plants of BC₁P₂ (F₁ × Redcoat) were susceptible. Classification of the F₃ families fit a ratio of one homozygous parental-type resistant to 14 segregating to one homozygous parental-type susceptible families, which was expected for segregation of two dominant and additive genes.

Quantitative estimates of gene number for APR in Redcoat based on AUDPC values were 2.03 ± 0.23 and 2.88 ± 0.90 in F₂ and F₃ generations, respectively (Table 2). Thus quantitative estimates of gene number are in close agreement with the estimate of two to three genes obtained by qualitative methods. Estimates of heritability for APR in the broad sense were 0.94 and 0.76

in F₂ and F₃ generations (Table 2). Heritability estimated by the standard units method was 0.31 and was significantly different from zero.

Houser × Redcoat cross. Houser, Redcoat, and their F₁ had mean mildew severities of 2.3, 1.4, and 2.4%, respectively. We evaluated an F₂ population of 129 plants and did not observe any segregants with a disease level similar to that of Becker. Mildew severities of F₂ plants ranged from 0.3 to 8%, whereas Becker had average mildew severities ranging from 24 to 30%. In an F₃ population of 100 families derived from randomly selected F₂ plants, none of the families had plants with mildew severities as high as those observed for Becker. However, based on postulated gene numbers in the APR parents, F₂ and F₃ population sizes were not large enough to conclude if Houser and Redcoat share common genes for resistance. Broad-sense heritability, estimated by the variance components method, was 0.57 and 0.78 in F₂ and F₃ generations, respectively (Table 2). Heritability estimate in standard units was 0.19 and was significantly different from zero.

DISCUSSION

Both qualitative and quantitative inheritance of APR or slow-disease development have been reported for different cereal host-pathogen interactions (2,6,12,15,25-27). Therefore, we used both qualitative and quantitative models to determine the number of genes segregating for APR to powdery mildew in the wheat cultivars Houser and Redcoat.

The parents and progenies differed in growth stages; however, flag leaves of all plants had emerged when the first mildew reading was taken. Gustafson and Shaner (7) found that powdery mildew severity on penultimate leaves of susceptible and slow-mildewing cultivars was statistically indistinguishable when plants were inoculated in the greenhouse at growth stages 37 or 40. However, they also observed that disease severity means were clearly distinguishable when plants were inoculated between growth stages 43 and 84. In a separate study, involving seven parents and their F₁s derived from a diallel cross, we studied the correlation between AUDPC on penultimate leaves and heading date (*unpublished data*). Among the parents and F₁s, Julian heading date ranged from 117 to 141 days, and AUDPC ranged from 2.3 to 316. The correlation ($r = -0.34$, $n = 28$) between heading date and AUDPC was not significant ($P > 0.05$). As in the current study, the first disease assessment was made when flag leaves of parental and F₁ plants had emerged. Therefore, the effect of differences

TABLE 2. Quantitative estimates of gene number and heritability for adult-plant resistance to powdery mildew for three winter wheat crosses

Cross	Gene number estimates		Heritability estimates ^a		
	F ₂	F ₃	F ₂ variance components	F ₃ variance components	Standard units
Houser × Becker	2.92 ± 0.43	2.75 ± 0.70	0.76	0.79	0.35 (0.53**)
Redcoat × Becker	2.03 ± 0.23	2.88 ± 0.90	0.94	0.76	0.31 (0.46**)
Houser × Redcoat	0.57	0.78	0.19 (0.29**)

^a Values in parentheses are the computed coefficients before multiplication by a factor of 2/3 to adjust for inbreeding in F₂ plants. ** = Significant at the 0.01 level of probability.

TABLE 3. Segregation ratios for adult-plant resistance to powdery mildew and chi-square tests of populations for crosses between the mildew-susceptible wheat cultivar Becker and mildew-resistant Redcoat

Population	No. of plants		No. of F ₃ lines			Ratio tested	χ ²	P
	R ^a + I	S	Hom ^b R	Seg ^c	Hom S			
BC ₁ P ₁ ^d	56	10				3:1	3.41	0.05-0.10
BC ₁ P ₂	46	0			
F ₂	205	12				15:1	0.31	0.50-0.75
F ₃			4	90	4	1:14:1	2.50	0.25-0.50

^a R = resistant, I = intermediate, and S = susceptible reaction type.

^b Hom = homozygous.

^c Seg = segregating.

^d BC₁P₁ = F₁ × Becker and BC₁P₂ = F₁ × Redcoat.

in plant growth stage on mildew severity among the parents and progenies was reduced to a great extent by taking mildew notes after flag leaf emergence. In the current study, we obtained similar estimates of gene number by both qualitative and quantitative methods for populations evaluated in two years, which also indicates that growth stage and environment had little effect on estimates of gene number.

Mildew reactions of the resistant and susceptible parents and progeny derived from resistant \times susceptible crosses indicated that APR to powdery mildew is partially dominant and additive in these crosses. Qualitative estimates of gene number based on F_2 , F_3 , and backcross generation data indicated that three partially dominant genes control APR to powdery mildew in Houser. Quantitative estimates also indicated a three-gene segregation pattern in both F_2 and F_3 generations for the Houser \times Becker cross.

Qualitative estimates indicated that either two partially dominant genes or two partially dominant and one recessive gene govern APR in Redcoat. Quantitative estimates support two- and three-gene segregation patterns in F_2 and F_3 generations, respectively, for the Redcoat \times Becker cross. On the basis of pedigree information and analysis of an F_2 population from the cross between the cultivars Redcoat and Jufy I, Wolfe (29) reported that Redcoat has a single recessive gene for powdery mildew resistance, which gave a differential reaction identical to that of *Pm5* in the cultivar Hope. However, Leath and Heun (18) found that Redcoat was susceptible to 11 isolates of powdery mildew and does not possess *Pm5* or other major genes for powdery mildew resistance. From our results, it is likely that two to three genes control APR to powdery mildew in Redcoat.

There was limited segregation in the Houser \times Redcoat cross. Plants with higher disease severities than Houser and Redcoat were observed in both F_2 and F_3 generations of this cross; yet, plants with mildew severities as high as Becker were not observed. However, a definite conclusion as to whether the two APR parents share common genes for resistance could not be drawn, because sample size was inadequate to observe segregation of more than three genes.

Estimates of broad-sense heritability in the three crosses were moderate to high. However, heritability estimates in standard units were low in all three crosses. This difference between heritability estimates in the broad sense and in standard units in the present study indicates that nonadditive genetic variance is involved in the inheritance of APR to powdery mildew in these cultivars. Heritability estimates in the cross between the two resistant cultivars were lower than those of the crosses between resistant and susceptible cultivars. This may have been due to the segregation of more genes in resistant \times resistant than in resistant \times susceptible crosses. Estimates of heritability in standard units in this study are in agreement with those obtained by Hautea et al (8) for APR to powdery mildew.

This study indicated that two to three genes govern APR to powdery mildew in Houser and Redcoat wheats. Because heritability estimates in standard units for APR were low, selection for APR should be more effective in advanced generations of populations derived from crosses with these cultivars.

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