Inheritance of Slow Rusting and the Relationship of *Sr* Genes to Slow Rusting in the Wheat Line FKN

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ABSTRACT

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Inheritance of resistance (slow rusting) to stem rust (*Puccinia graminis* f. sp. *tritici*) was studied in two crosses involving the slow-rusting spring wheat (*Triticum aestivum*) line FKN and two fast-rusting lines, W3498 and 3-106. The area under the disease progress curve (AUDPC) was used to measure the rate of rusting. Progenies in the F_6 generation of the crosses with FKN were distributed symetrically about the mean. Heritability estimates were 66 and 52% for FKN/W3498 and FKN/3-106 crosses, respectively. The heritability estimate for the cross between the fast-rusting parents was 20%. No relationship was found between the number of *Sr* genes in individual F_6 progenies and the AUDPC. Although a significantly smaller AUDPC was associated with some *Sr* genes and combinations of *Sr* genes, other single *Sr* genes or combinations of them were associated with higher AUDPCs.

Additional key words: vertical resistance, horizontal resistance, gene pyramiding.

Resistance (slow rusting) to stem rust caused by *Puccinia graminis* Pers. f. sp. *tritici* has been identified in several wheat (*Triticum aestivum* (L.)) cultivars by Wilcoxson et al (16). The inheritance of slow rusting in seven spring wheat cultivars was determined by Skovmand et al (13), who concluded that additive gene action was predominant in the genetic control of slow rusting and that narrow-sense heritability was 82%. Quantitative inheritance of rate-limiting resistance (slow rusting, horizontal resistance [15], general resistance [9]) has been reported in various host-pathogen systems (2,3,5,7).

The spring wheat line FKN, CI 13145, was developed from crosses among the cultivars Frontana, Kenya 58, and Newthatch (4). Original sources of FKN contained the stem rust resistance genes *Sr*5, *Sr*6, *Sr*7, *Sr*8, and *Sr*9b. In addition, FKN has been long recognized as a line with a high degree of slow rusting because of its ability to retard epidemics in plants inoculated with races of *P. graminis* f. sp. *tritici* virulent to all five *Sr* genes. This line has never been grown commercially but has been used in several breeding programs as a source of resistance.

The objective of this study was to determine the nature of inheritance of slow rusting in FKN and the effect of the presence or absence of *Sr* genes on slow rusting.

MATERIALS AND METHODS

Two lines of spring wheat susceptible to stem rust were used as fast-rusting parents in crosses with FKN. Line W3498 was obtained originally from the University of Sydney, Australia, and is derived from crosses among the cultivars Little Club, Gabo, and Charter. Line 3-106 was developed at the University of Minnesota from a cross of cultivars Baart and Prelude (11), which are both fast rusting (16). Families from reciprocal crosses between each of these lines and FKN were advanced to the F_5 generation by single-seed descent. Reciprocal crosses between W3498 and 3-106 were made, and the resulting families were advanced by single-seed descent to the F_4 generation. Seeds from single F_5 plants of the FKN crosses and F_4 plants for the W3498/3-106 crosses were bulked for field plantings. The numbers of families available for study were 156 from W3498 × FKN, 36 from FKN × W3498, 53 from 3-106 × FKN, 18 from FKN × 3-106, 43 from W3498 × 3-106, and 26 from 3-106 × W3498. Problems with seed germination during generation advancement reduced the number of progenies available for use in the experiment.

Parents and families from each pair of crosses were planted in hill plots at Rosemount, MN, in separate randomized complete blocks with three replicates. Families from reciprocal crosses were coded independently to allow individual evaluation of each cross. By randomizing reciprocal crosses together, families could be pooled if reciprocal cross differences were not significant. Hills were planted 30.5 cm apart with 10–15 seeds per hill. The entire plot was surrounded with Era, a semidwarf spring wheat cultivar resistant to races of the stem rust pathogen prevalent in Minnesota.

The plot was sprayed with butrizol (4-*N*-butyl-1,2,4 triazole, Indar LC-70, Rohm and Haas Co., Spring House, PA 19477) to control leaf rust (*P. recondita* Rob. ex Desm. f. sp. *tritici*) when plants were at growth stage 3–4 (tillers formed, leaves often twisted spirally; beginning of pseudostem erection, leaf sheaths beginning to lengthen) of the Romig scale (1). Only traces of leaf rust developed.

Plants in the entire plot were inoculated with urediospores of *P. graminis* f. sp. *tritici* race 151-QSH suspended in soltrol 170 (a

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paraffinic oil from Phillips Petroleum Co., Bartlesville, OK 74003) at the rate of 0.1 g of urediospores per 300 ml of oil per 180 m of row when the plants were in growth stage 8–9 (last leaf visible but still rolled up, head beginning to swell; ligule of last leaf just visible) (1). Race 151-QSH has the necessary virulence genes to produce fully susceptible-type lesions (type 4) (14) on lines that carry *Sr*5, *Sr*6, *Sr*7a, *Sr*8, and *Sr*9b (10) reported to be in FKN (4). Rust severity was estimated with the modified Cobb's scale (8) five times at weekly intervals, beginning at growth stage 10–12 (boot stage to heading) and continuing until most families were at growth stage 24 (early milk) or higher. The area under the disease progress curve (AUDPC) for each hill was calculated with the computer program described previously (2,3,13).

The presence of *Sr*5, *Sr*6, *Sr*7a, *Sr*8, and *Sr*9b in each family was determined with appropriate races of *P. graminis* f. sp. *tritici*. Remnant seed or seed harvested from the field plots of each family was used. All plants were maintained before and after inoculation in a growth chamber at 21 ± 2 C and a 12-hr day.

TABLE 1. Mean area under the disease progress curve (AUDPC) for the parental wheat lines and progenies of combined reciprocal crosses inoculated with *Puccinia graminis* f. sp. *tritici* race 151-QSH

Line	AUDPC[a]
Parents[b]	
FKN	59.0 ± 7.4
3-106	343.2 ± 20.6
W3498	377.6 ± 39.1
Crosses	
FKN/3-106	220.2
FKN/W3498	206.9
3-106/W3498	374.8

[a] Mean of three replications, with the standard error of the mean for parents.
[b] FKN = a Frontana, Kenya 58, Newthatch derivative; 3-106 = a Baart, Prelude derivative; W3498 = a Little Club, Gabo, Charter derivative.

TABLE 2. Number of progenies with *Sr* genes (*Sr*5, *Sr*6, *Sr*8, *Sr*9b) from crosses between slow-rusting (FKN) and fast-rusting (3-106 and W3498) wheat lines

Number of *Sr* genes	Number of progenies per cross[a]	
	FKN/3-106	FKN/W3498
0	4	10
1	13	37
2	25	53
3	20	28
4	3	9
Total	65	137

[a] FKN = a Frontana, Kenya 58, Newthatch derivative; 3-106 = a Baart, Prelude derivative; W3498 = a Little Club, Gabo, Charter derivative.

TABLE 3. Mean squares (MS) for area under the disease progress curves among progenies of wheat crosses with and without each *Sr* gene and the resulting F ratio

Cross	Lines with *Sr* gene		Lines without *Sr* gene		
Sr gene	No.	MS	No.	MS	F ratio[a]
FKN/W3498[b]					
5	53	13,504.2	84	15,550.4	1.15
6	93	14,859.6	44	14,542.7	1.02
8	64	12,461.9	73	16,763.6	1.34
9b	53	12,188.8	84	14,358.1	1.18
FKN/3-106[c]					
5	21	5,056.3	44	7,186.7	1.42
6	44	4,348.0	21	10,492.7	2.41*
8	33	6,174.6	32	6,845.5	1.11
9b	37	7,735.6	28	4,355.5	1.78

[a] F ratio obtained by using the larger value as the numerator and the smaller as the denominator; * = significant at $P = 0.05$.
[b] FKN = a Frontana, Kenya 58, Newthatch derivative; W3498 = a Little Club, Gabo, Charter derivative.
[c] 3-106 = a Baart, Prelude derivative.

RESULTS

Initial stem rust severities were less than 1% in all hills. Many resistant (slow-rusting) lines had initial disease severities 0–0.1%. Terminal severities ranged from 5 to 80%.

Mean AUDPC values were not significantly different for reciprocal crosses in any of the three combinations; therefore, all subsequent analyses were performed on combined data. Mean AUDPC values for parental cultivars or lines and crosses are shown in Table 1. Progenies from all crosses were symmetrically distributed around the mean (Figs. 1 and 2). Progenies more resistant than FKN occurred only in the FKN/W3498 crosses. Transgressive segregation occurred in both directions in the crosses between the fast-rusting parents, 3-106 and W3498.

Broad-sense heritability estimates, calculated from the ratio of genetic variance to total variance, were 66 and 52% for the FKN/W3498 and FKN/3-106 crosses, respectively. The estimate of heritabilty for the 3-106/W3498 crosses was 20%.

The gene *Sr*7a was not present in the F_6 progenies of either cross with FKN. Subsequent checking revealed that *Sr*7a occurred at a low frequency in progeny of FKN plants used in these crosses. Other sources of FKN had *Sr*7a plus the other four genes (*Sr*5, *Sr*6, *Sr*8, and *Sr*9b) reported to be in FKN (4). When plants of FKN without *Sr*7a were grown in the field or greenhouse, their appearance and reaction to stem rust were similar to those of FKN plants with *Sr*7a.

Progenies that were segregating for one or more *Sr* genes were removed from the population before the relationship of *Sr* genes to slow rusting was analyzed. Consequently, 137 progenies homozygous for the *Sr* genes in question were available from the FKN/W3498 crosses and 65 were available from the FKN/3-106 crosses (Table 2). As expected, none of the progenies of the 3-105/W3498 crosses contained the *Sr* genes in question. Heritability estimates from these reduced populations were within 1% of the estimates obtained from the original data.

The relationship between the number of *Sr* genes in the F_6 progenies of FKN crosses and the area under the disease progress curve is shown in Fig. 3. The points on the graph represent the mean AUDPC for progenies with the indicated number of genes. The regression equation was obtained by regressing the number of genes identified in each progeny against the AUDPC for that progeny. The number of *Sr* genes present did not affect the range of AUDPC values obtained. The R^2 values were extremely low, and regression coefficients were not significant, suggesting that slow rusting is not related to the number of *Sr* genes in individual progenies.

Comparisons of mean squares of progenies with and without each *Sr* gene (Table 3) were significant in only one case, suggesting that the genetic backgrounds in progenies were distributed randomly.

Regression analysis was used to study the relationship between slow rusting as measured by AUDPC and individual *Sr* genes or combinations of genes. For the FKN/W3498 crosses, significant negative regression coefficients were obtained for *Sr*9b and the combinations *Sr*5 and *Sr*9b and *Sr*5 and *Sr*8. Significant positive regression coefficients were obtained for the combinations *Sr*6 and *Sr*8 and *Sr*5, *Sr*6, and *Sr*8. The amount of variation (R^2) explained by the most complicated equation (all possible combinations of genes) was less than 10%. Data were separated into two sets, lines with and lines without the gene or genes that regression analysis indicated were contributing significantly to the sum of squares for regression. Separate analyses of variance were made on each set, and the means and mean squares for lines were compared (Table 4). Progenies with *Sr*9b, and the combinations *Sr*5 and *Sr*9b and *Sr*8 and *Sr*9b, had a significantly lower AUDPC than progenies without these genes. Mean squares for lines were not significantly different. Environmental variances (σ_e^2) were similar in lines with or without the genes (Table 4). Genotypic variances (σ_g^2) were lower for lines with these genes. Progeny with *Sr*6 and *Sr*8 and *Sr*5, *Sr*6,

and *Sr*8 had a greater AUDPC than progeny without these combinations. Mean squares for lines σ_e^2 and σ_g^2 were similar in these comparisons.

The coefficient of determination (R^2) was less than 1% for the FKN/3-106 crosses. Significant negative regression coefficients were obtained only for *Sr*9b. Significant positive regression coefficients resulted with *Sr*6 and the combinations *Sr*5 and *Sr*8; *Sr*6 and *Sr*9b; and *Sr*6, *Sr*8, and *Sr*9b. When the data were separated into sets as described above, lines with *Sr*9b had a significantly lower AUDPC (Table 4) than lines without *Sr*9b. Mean squares for lines were not different in the comparison (Table 3). Comparison of lines with *Sr*6 or the combination *Sr*5 and *Sr*8 showed that the mean AUDPC was significantly greater when the genes were present than when the genes were absent. Mean squares for lines were significantly lower for sets with *Sr*6 and the combination *Sr*5 and *Sr*8, suggesting reduced variation in these combinations. Environmental variances (σ_e^2) were similar in all comparisons. Except for the *Sr*9b comparison, progenies with the genes in question had reduced genotypic variances (σ_g^2).

Correlations among *Sr* genes were not significant in either cross, suggesting that the genes were distributed in the population at random.

DISCUSSION

Our results agree with earlier evidence that slow rusting in wheat stem rust (13), barley leaf rust (3), wheat leaf rust (2), and oats crown rust (5) is inherited as a quantitative character. Estimated broad-sense heritabilities for the two FKN crosses were reasonably high and suggest that genetic advance could be made for slow rusting by selection criteria based on the AUDPC in crosses between slow- and fast-rusting parents.

Regression analysis allowed the examination of the relationship between *Sr* genes and slow rusting (Fig. 1) when plants were inoculated with a race of *P. graminis* f. sp. *tritici* capable of overcoming all the *Sr* genes present in the population. For these crosses, the number of genes present did not signficiantly affect rust development as measured by AUDPC. In fact, in FKN/3-106 crosses, AUDPC increased slightly as the number of *Sr* genes in the progeny increased. However, the number of lines available in this cross was low. These data suggest that for this model, pyramiding (6) of the identified *Sr* genes would not result in increased levels of slow rusting.

Ideally, studies of gene pyramiding should be made with isogenic material to minimize background effects. In this study, we demonstrated that in most cases, the variation, as measured by mean squares, was similar in lines with or without the genes, suggesting that genetic backgrounds were similar. One of us, Southern (*unpublished*), attempted to combine the *Sr* genes known to be in FKN (*Sr*5, *Sr*6, *Sr*7, *Sr*8, and *Sr*9b) in a homogeneous background. Although not all possible gene combinations were obtained, the degree of slow rusting was not associated with higher numbers of *Sr* genes.

In the FKN/W3498 crosses, a smaller AUDPC was measured in those progenies containing *Sr*9b, alone or in combination with *Sr*5 and *Sr*8, than in progenies not containing those genes (Table 4); however, other combinations such as *Sr*6 and *Sr*8 and *Sr*5, *Sr*6, and

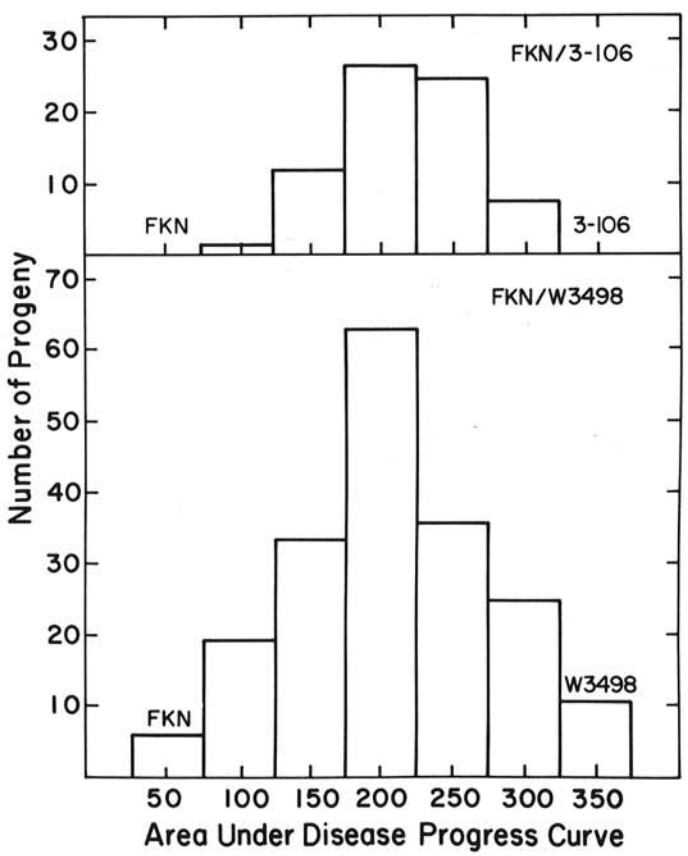

Fig. 1. Frequency distribution for area under the disease progress curve of F_6 lines of two spring wheat crosses infected with *Puccinia graminis* f. sp. *tritici* race 151-QSH. Parents are indicated above the class in which they fall.

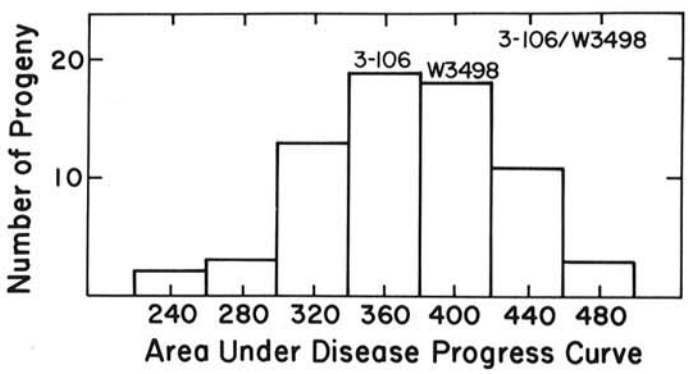

Fig. 2. Frequency distribution for area under the disease progress curve of F_5 lines of a spring wheat cross infected with *Puccinia graminis* f. sp. *tritici* race 151-QSH. Parents are indicated above the class in which they fall.

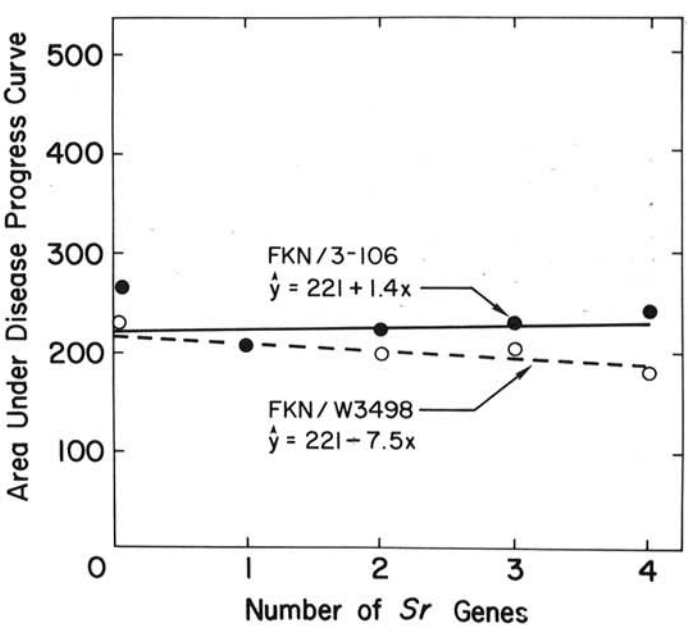

Fig. 3. Relationship of the area under the disease progress curve (AUDPC) to the number of *Sr* genes in F_6 lines from two spring wheat crosses infected with *Puccinia graminis* f. sp. *tritici* race 151-QSH. Data points represent the mean AUDPC for lines with the indicated number of genes.

TABLE 4. Number of wheat lines, mean area under the disease progress curve ($\bar{x}$) of lines, mean squares (MS) for lines, and environmental (σ_e^2) and genotypic (σ_g^2) variance associated with lines in the F_6 generation of crosses sorted into populations with and without the indicated *Sr* gene

Cross gene(s) *Sr*	Lines with *Sr* gene(s)					Lines without *Sr* gene(s)				
	No.	$\bar{x}$[a]	MS[b]	σ_e^2	σ_g^2	No.	$\bar{x}$[a]	MS[b]	σ_e^2	σ_g^2
FKN/W3498[c]										
9b	53	181.3	12,188.8	2,359.0	3,276.6	84	222.8**	14,358.1	2,139.8	4,072.7
5,9b	25	169.9	7,666.2	2,484.0	1,725.4	112	215.0**	15,179.4	2,170.5	4,336.3
6,8	42	220.7**	11,115.9	2,315.8	2,933.4	95	200.6	15,984.7	2,154.7	4,610.0
8,9	23	171.0	7,498.5	1,646.6	1,950.6	113	214.0**	15,241.4	2,339.8	4,300.5
5,6,8	20	225.7**	10,335.2	2,319.1	2,672.0	116	203.5	15,274.0	2,163.8	4,370.1
FKN/3-106[d]										
6	44	229.5**	4,348.0	1,688.0	886.7	21	211.6	10,492.7*	1,289.2	3,067.8
9b	37	216.2	7,735.6	1,622.7	2,037.6	27	233.6**	4,355.5	1,539.6	938.6
5,8	7	244.0**	1,300.7	1,127.7	57.7	58	221.3	6,895.0*	1,654.6	1,746.8
6,9b	23	230.0	4,243.8	1,694.7	849.7	42	220.3	7,633.8	1,513.4	2,040.2
6,8,9b	14	230.0	3,181.9	1,954.0	576.0	51	222.0	7,211.6	1,481.5	1,910.0

[a]** Indicates that means for lines with or without the gene(s) indicated are significantly different at $P = 0.01$ (t test).
[b]* Indicates that mean squares for lines with or without the gene(s) indicated are significantly different at $P = 0.05$ (F test).
[c]FKN = a Frontana, Kenya 58, Newthatch derivative; W3498 = a Little Club, Gabo, Charter derivative.
[d]3-106 = a Baart, Prelude derivative.

*Sr*8 had increased AUDPC values. Variation, as measured by mean squares for lines, was not greatly reduced in lines with either of these genes or combinations. One would expect smaller AUDPC means and reduced variation in lines with higher numbers of genes if gene pyramiding increased the degree of slow rusting. This was not obtained.

Progenies with *Sr*9b in the FKN/3-106 crosses also had increased slow rusting compared with progenies without *Sr*9b; however, the mean squares for lines with *Sr*9b were slightly higher than for lines without *Sr*9b, although not significant. Progenies with *Sr*6 and the combination *Sr*5 and *Sr*8 had increased levels of disease. Only seven progenies contained the *Sr*5 and *Sr*8 combination, so these results should be interpreted with caution.

Skovmand et al (12) demonstrated slow rusting in lines possessing the dominant *Sr*6 allele but concluded that *Sr*6 per se was not responsible for the slow rusting because fast-rusting individuals occurred in the groups of lines containing *Sr*6. Our results suggest that *Sr*9b might contribute to slow rusting, but fast-rusting progenies containing *Sr*9b were identified. Our conclusion is similar to that of Skovmand et al (12) in suggesting that factors other than *Sr*9b contribute to slow rusting. In addition, in this study, lines possessing *Sr*6 from the FKN/3-106 crosses had higher average AUDPC than lines without *Sr*6.

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