The Relationship Between Slow-Rusting and Some Genes Specific for Stem Rust Resistance in Wheat

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ABSTRACT

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The relationship between slow-rusting and genes for specific resistance against stem rust was studied in F_5 lines derived from diallel crosses among the wheat cultivars Idaed 59, Kenya 58, Thatcher, Lee, Marquis, Prelude, and Baart. Slow-rusting in the F_5 lines was measured in natural epidemics of stem rust and by calculating the area under the rust progress curve derived from weekly estimates of stem rust severity. Lines that possessed either the dominant or recessive allele of genes for specific resistance, *Sr*5, *Sr*6, *Sr*7b, *Sr*11, and *Sr*Tt1, were identified in a seedling test in the greenhouse using appropriate cultures of *Puccinia graminis* f. sp. *tritici*. Then the slow-rusting ability of lines possessing the dominant allele of an *Sr* gene was compared to the slow-rusting ability of half-sib lines possessing the recessive allele of the same *Sr* gene. There was no effect on the development of stem rust attributable to the recessive or to the dominant alleles of *Sr*5, *Sr*7b, and *Sr*11. There was an association between the development of stem rust and the dominant allele of the *Sr*Tt1 gene, but slow-rusting was not due to this allele. The genes conditioning slow-rusting may be located on the same chromosome as the *Sr*Tt1 locus and linked to *Sr*Tt1. There also was an interaction between slow-rusting and the dominant allele of gene *Sr*6. The F_5 lines possessing the dominant allele of *Sr*6 rust more slowly than the lines possessing the recessive allele. However, in the group of lines with the dominant allele of *Sr*6, there were some fast-rusting lines. This indicated that slow-rusting was not due to the *Sr*6 gene, per se, but due to associated genes for slow-rusting.

Additional key words: *Puccinia graminis*, horizontal resistance, generalized resistance, adult plant resistance.

Recent reports (20, 25) indicate that the slow-rusting type of resistance against wheat stem rust is a heritable trait; in the crosses studied, it was controlled by two to 12 pairs of genes (20). The objective of this research was to study whether there is a relationship between the genes that control slow-rusting and other genes that are known to control specific races of *Puccinia graminis* Pers. f. sp. *tritici* Eriks. & E. Henn.

MATERIALS AND METHODS

The spring wheat (*Triticum aestivum* L.) cultivars Baart (C.I. 1697), Idaed 59 (C.I. 13631), Kenya 58 (C.I. 12471), Lee (C.I. 12488), Marquis (C.I. 3641), Prelude (C.I. 4323), and Thatcher (C.I. 10003) were studied. Idaed 59 rusted slowest, Baart and Prelude rusted rapidly, and the other cultivars were intermediate between Idaed 59 and the fast rusters (20, 25). These cultivars were crossed to form a complete diallel series. The progenies of the 21 crosses were advanced to the F_4 by single-seed descent. Seed from a single F_4 plant was harvested to form an F_5 line and 80 F_5 lines from each of the 21 crosses were developed.

The slow-rusting characteristic of the F_5 lines was evaluated in 1974 and 1975 in a "sets-in replicates" design with eight sets in each of two replicates. Each set consisted of 10 randomly chosen F_5 lines per cross and the seven parents, making a total of 217 entries. The entries were planted 30 cm apart with 10 seeds per hill in mid-May at Rosemount, Minnesota.

The severity of stem rust on the F_5 lines was evaluated in epidemics of *Puccinia graminis* f. sp. *tritici*, on 26 June 1974, just after tillering, and on 14 July 1975, just after anthesis, and thereafter at 1-wk intervals for 6 wk in 1974 and for 4 wk in 1975, using a modified Cobb scale (14). When rust severities were less than 1% [10 uredia/culm (7)] the uredia were counted and converted to percentages by equating one uredium per hill to .01%, 10 uredia per hill or one uredium per plant to .1%, etc.

The slow-rusting characteristic of the F_5 lines was indicated by calculating the area under the stem rust

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progress curve from the weekly stem rust severity ratings made on each hill. The area under the curve was calculated with the Fortran IV subroutine AREA and the associated subroutine INTEG of Bevington (3). The matrices were inverted using the subroutine INVERT of Davies (5).

In 1974, the epidemic resulted from natural infection. In 1975, the plants were inoculated with race 15-TLM, a common race in the area, to ensure that an epidemic would develop. In both years the predominant races in the plots and surrounding fields were similar. The races present and the infection types they produced on the parent cultivars are shown in Table 1.

Leaf rust (caused by *Puccinia recondita* Rob. ex Desm. f. sp. *tritici*) was controlled with Indar LC-70® (Rohm and Haas Co., Spring House, PA 19477) (4-butyl-4H-1,2,4,-triazole) applied at the rate of 566 ml/ha. No leaf rust developed in 1974, but there was a trace in 1975.

The presence of the race-specific genes, *Sr* genes 5, 6, 7b, 11, and Tt1, in the progenies of the 21 crosses was determined in F_6 generation seedlings. Some lines could not be tested for the presence of *Sr* genes because viable seed was not produced in the field. The *Sr* genes were identified using cultures of *P. graminis* f. sp. *tritici* that possessed appropriate virulence and avirulence (Table 2). The *Sr* genes reported to occur in the parent cultivars are: Baart, none; Prelude, none; Kenya 58, *Sr*6, and 7b (9); Lee, *Sr*9g and 11 [(9) and *personal communication*, R. A. McIntosh, University of Sydney, Sydney, Australia]; Marquis, *Sr*7b, 18, 19, and 20 (2, 10); Thatcher, *Sr*5, 9g, 12, and 16 [(10, 11, 19) and *personal communication*, R. A. McIntosh, University of Sydney, Sydney, Australia]; and Idaed 59, *Sr*Tt1 (*personal communication*, D. V. McVey, University of Minnesota, St. Paul, Minnesota). The *Sr* genes that we identified in the parent cultivars were: Baart, none; Prelude, none; Kenya 58, *Sr*6; Lee, *Sr*11; Marquis, *Sr*7b; Thatcher, *Sr*5; and Idaed 59, *Sr*Tt1. Gene *Sr*7b, which has been reported to occur in Kenya 58 (9), was not present in our line of Kenya 58. The other *Sr* genes reported to occur in our parent cultivars were not studied because appropriate cultures of the pathogen were not available. The infection types produced on the parent cultivars by cultures of *P. graminis* f. sp. *tritici* capable of detecting genes *Sr*5, 6, 7b, 11 and Tt1 are shown in Table 3.

One-wk-old seedlings were inoculated with uredospores suspended in oil (Soltrol 170®, Phillips Petroleum Co., Special Products Div., Borger, TX 79007) and incubated at 100% relative humidity for approximately 20 hr. The seedlings then were transferred to a greenhouse maintained at about 18 C. The infection types were ascertained 2 wk after inoculation.

On the basis of the seedling tests, the F_5 lines were assigned a genotype for specific resistance and grouped according to genotype as follows: (i) groups of full-sib F_5 lines (lines with the same parents) with two homozygous dominant alleles of the *Sr* genes in all combinations taken two at a time, and (ii) groups of half-sib F_5 lines (lines with one parent in common) in which assignment to a group was dependent on the *Sr* gene in the common parent. The individual F_5 lines in each half-sib group were either homozygous dominant or homozygous recessive for the *Sr* gene in the common parent.

The normality of the population distribution, according to the area under the progress curve, for F_5 lines within the full-sib and half-sib groups, was tested using the Kolmogorov-Smirnov test (21). The areas under the stem rust progress curve of the groups were compared using a one-way analysis of variance.

RESULTS

The stem rust epidemics in 1974 and 1975 were considered to be natural epidemics, even though the plots were inoculated in 1975. In both years, the race-15 group constituted 70 percent of the collections from the hill plots and about 90% of the collections from the station; race

TABLE 1. Races of *Puccinia graminis* f. sp. *tritici* identified in collections made within F_5 lines and elsewhere at the Agricultural Experiment Station, Rosemount, MN in 1974 and 1975, their virulence/avirulence formulas for specific *Sr* genes, and the infection types produced in the parental cultivars

Wheat stem rust race[a]	Virulence/ avirulence formula	No. isolates/race on: F_5 lines 1974	F_5 lines 1975	Station 1974	Station 1975	Infection types on parent cultivars:[b] Baart	Kenya Prelude	Kenya 58	Kenya Lee	Marquis	Thatcher	Idaed 59
56 MBC	5,7b/6,11,Tt1	0	2	0	0	S[c]	S	0;	;2⁻	S	0;1	0
11-32-113 RKQ	5,6,7b,Tt1/11	0	3	0	1	S	S	S	2⁻	S	S	S
11-32-113 RPQ	5,7b,11,Tt1/6	0	0	0	1	S	S	0;	S	S	S	S
11-32-113 RTQ	5,6,7b,11,Tt1/	0	2	0	1	S	S	S	S	S	S	S
11-32-113 RCC	5,7b/6,11,Tt1	1	0	0	0	S	S	0;	;2⁻	S	S	0;1
15 TBM	5,7b,Tt1/6,11	1	0	0	0	S	S	0;	2⁻	S	S	S
15 TDM	5,7b,Tt1/6,11	2	0	1	2	S	S	0;	2⁻	S	S	S
15 TLM	5,7b,11,Tt1/6	10	9	8	2	S	S	0;	S	S	S	S
15 TNM	5,7b,11,Tt1/6	13	13	79	56	S	S	0;	S	S	S	S
151 QSH	5,6,11/7b,Tt1	5	3	3	4	S	S	S	S	2	S	0
151 QFB	5/6,7b,11,Tt1	2	0	2	1	S	S	0;	2⁻	2	S	0;1

[a]The number refers to the race as identified on the standard differential cultivars [(22) E. C. Stakman et al. 1962. U.S. Dep. Agric. Bull. E-617 (R-v)]. The letter refers to the race as identified on single-gene differential lines [(16) A. P. Roelfs and D. V. McVey. 1972. Plant Dis. Rep. 56:1038-1039].

[b]Host response was similar to known checks within the test conditions used.

[c]The susceptible reaction (S) was indicated by infection types 3 and 4.

151-QSH was present in about 10% of the hill-plot collections and in less than 7% of the station collections. The other races were identified in one of the two years, in less than 10% of the collections (Table 1). The predominant races, 15-TLM and 15-TNM, were virulent on Baart, Prelude, Thatcher, Marquis, Lee, Idaed 59, and on Kenya 58 at high temperatures; they were avirulent on Kenya 58 at low temperatures. Race 151-QSH was virulent on Baart, Prelude, Thatcher, Kenya 58, and Lee, and avirulent on Marquis and Idaed 59 (Table 1).

The races in the epidemics occurred randomly throughout the field plots in both years. Races with particular virulence or avirulence were not found consistently in collections from lines or parents that contained certain genes for specific resistance. Thus, the isolates of the race-15 group were identified from plants with the *Sr*6 gene as frequently as from plants with other genotypes.

TABLE 2. Cultures of *Puccinia graminis* f. sp. *tritici* with the respective virulence and avirulence formulas which are used to identify genes for specific resistance to wheat stem rust (*Sr* genes)

Wheat stem rust race[a]	Culture number[b]	Virulence/avirulence for selected *Sr* genes
29-HJC	70-44-64-A	7b,6/5,11,Tt1
17-HNL	68-41-73-A	7b,11,Tt1/5,6
33-LCL	70 B451	5,Tt1/7b,11,6
56-MBC	74-46-570-A	5,7b/11,6,Tt1
32-RKQ	72-25-639-C	5,7b,6,Tt1/11
15-TLM	65-39-2	5,7b,11,Tt1/6
151-QFB	70-00-1370-C	5/7b,11,6,Tt1
151-QSH	69-21-399	5,11,6/7b,Tt1

[a]The number refers to the race as identified on standard differential cultivars [(22) E. C. Stakman et al. 1962. U.S. Dep. Agric. Bull. E-617 (R-v.)]; the letters refer to the race as identified on single-gene differential lines [(16) A. P. Roelfs and D. V McVey. 1972. Plant Dis. Rep. 56:1038-1039].

[b]These cultures are stored under these numbers in the Cereal Rust Laboratory, University of Minnesota, St. Paul.

In 1974, the infection types observed in the F_5 lines in the field plots indicated that the lines were susceptible. This also was true in 1975 for most F_5 lines, but in 1975 some F_5 lines were moderately susceptible as follows: 10 lines in Marquis × Idaed 59, 18 in Kenya 58 × Idaed 59, 15 in Thatcher × Idaed 59, 25 in Lee × Idaed 59, 5 in Prelude × Idaed 59, 9 in Baart × Idaed 59, 5 in Kenya 58 × Marquis, 28 in Thatcher × Kenya 58, 5 in Lee × Kenya 58, 2 in Prelude × Kenya 58, 1 in Baart × Kenya 58, 2 in Lee × Thatcher, and 2 in Prelude × Thatcher.

The mean and range for area under the stem rust progress curve varied among the 21 crosses (Table 4). In each cross, lines were observed that rusted more slowly or more rapidly than the parents. The normality of the progeny distribution in each of the crosses is shown in Table 4 and Fig. 1. The distributions of the progenies in the crosses with Idaed 59 and Kenya 58 were skewed toward slow-rusting. The distribution of the progenies in the other crosses was normal.

Analysis of variance indicated that the differences among the crosses for slow-rusting were significant. The interactions of crosses × year, crosses × replicates in years, and crosses × sets × replicates in years also were significant, but the interactions of crosses × sets and crosses × sets × year were not. The year, replicates in years, and set effects were nonsignificant. The significant interaction between crosses and years probably resulted from the fact that, except for Prelude × Kenya 58, the crosses with Kenya 58 had a lower mean area under the stem rust progress curve in 1975 than in 1974; all other crosses had higher mean areas under the curve in 1975 than in 1974.

Details on the inheritance of slow-rusting with *P. graminis* f. sp. *tritici* in the crosses mentioned in this paper may be seen elsewhere (20).

The mean areas under the stem rust progress curve for the half-sib F_5 lines, homozygous for the dominant or the recessive alleles of the *Sr* genes, are listed in Table 5. The areas under the curve for the genotypes *Sr*Tt1*Sr*Tt1 and *Sr*6*Sr*6 differed significantly from each other and from the means of all other genotypes. The means of the lines that were homozygous dominant or homozygous

TABLE 3. Infection types produced on parent wheat cultivars inoculated with cultures of *Puccinia graminis* f. sp. *tritici* with virulence/avirulence appropriate to detect stem rust resistance genes *Sr*5, 6, 7b, 11 and Tt1

Wheat stem rust race[a]	Parent cultivar[b]						
	Baart	Prelude	Kenya 58	Lee	Marquis	Thatcher	Idaed 59
29-HJC	S	S	S	;2⁻	S	S	;1
17-HNL	S	S	0;	S	S	S	S
33-LCL	S	S	0;	;2⁻	2	S	S
56-MBC	S	S	0;	;2⁻	S	0;1	0
32-RKQ	S	S	S	;2⁻	S	S	S
15-TLM	S	S	0;	S	S	S	S
151-QFB	S	S	0;	;2⁻	2	S	0;1
151-QSH	S	S	S	S	2	S	0

[a]The number refers to the race as identified on the standard differential cultivars [(22) E. C. Stakman et al. 1962. U.S. Dep. Agric. Bull. E-617 (R-v.)]; the letter refers to the race as identified on single-gene differential lines [(16) A. P. Roelfs and D. V. McVey. 1972. Plant Dis. Rep. 56:1038-1039].

[b]The susceptible reaction (S) was indicated by infection types 3 or 4; host response was similar to known checks within the test conditions.

recessive for the genes *Sr*5, 7b, and 11, and the means of the lines that were homozygous recessive for the genes *Sr*Tt1 and *Sr*6 did not differ significantly from each other except that the mean of the lines with the *Sr*11*Sr*11 genotype differed from the values for the *sr*6*sr*6 genotype.

The range in area under the stem rust progress curve for each of the groups of half-sib F_5 lines is given in Table 5. The maximum area under the curve was greater than 1,000 for all the genotypic groups, while the minimum area under the curve ranged from less than 10 for the genotypes *Sr*Tt1*Sr*Tt1, *Sr*11*Sr*11, *Sr*6*Sr*6, and *sr*Tt1*sr*Tt1 to 435 for the genotype *sr*7*bsr*7b.

TABLE 4. Mean area and range for area under the stem rust progress curve for each cross infected with *Puccinia graminis* f. sp. *tritici*

Cross	Area (Mean ± s.e.)	Range
Marquis × Idaed 59	264 ± 38	7.6 - 1,111.9[a]
Kenya 58 × Idaed 59	187 ± 36	2.9 - 1,215.2[a]
Thatcher × Idaed 59	306 ± 41	0.9 - 1,146.8[a]
Lee × Idaed 59	270 ± 39	1.0 - 1,028.5[a]
Prelude × Idaed 59	390 ± 44	2.6 - 1,340.6[a]
Baart × Idaed 59	461 ± 45	3.6 - 1,164.1[a]
Kenya 58 × Marquis	516 ± 37	32.5 - 1,170.9
Thatcher × Marquis	698 ± 15	109.0 - 948.0
Lee × Marquis	732 ± 13	422.8 - 955.2
Prelude × Marquis	718 ± 17	435.2 - 1,065.5
Baart × Marquis	798 ± 13	487.7 - 1,055.0
Thatcher × Kenya 58	206 ± 34	0.2 - 937.2[a]
Lee × Kenya 58	480 ± 36	10.3 - 1,150.5[a]
Prelude × Kenya 58	590 ± 44	21.7 - 1,239.3[a]
Baart × Kenya 58	538 ± 34	13.4 - 1,108.2
Lee × Thatcher	629 ± 27	36.4 - 1,068.9
Prelude × Thatcher	713 ± 25	72.4 - 1,079.8
Baart × Thatcher	731 ± 18	364.3 - 1,059.4
Prelude × Lee	720 ± 22	192.6 - 1,132.9
Baart × Lee	734 ± 13	376.5 - 1,033.5
Baart × Prelude	869 ± 17	513.1 - 1,242.5

[a]Distributions that varied significantly from a normal distribution [(21) R. R. Sokal and J. F. Rohlf. 1969. Biometry: the principles and practice in biological research. W. H. Freeman and Co., San Francisco. 776 p.].

The frequency distributions for areas under the stem rust progress curves for half-sib lines, grouped according to their specific resistance, are shown in Fig. 2. The distributions of the lines with the dominant allele of *Sr*Tt1 and of *Sr*6 were significantly different from a normal distribution. The distribution of lines in the other genotypic groups did not differ from a normal distribution (Fig. 2 and Table 5).

There was no association between area under the stem rust progress curve and the dominant or the recessive alleles of the genes *Sr*5, *Sr*7b, and *Sr*11. The distribution of lines in each genotypic group was normal (Table 5 and Fig. 2), and there were lines in each group that were of the slow- or fast-rusting types.

There was an association between area under the stem-rust progress curve and the allelic form of the genes *Sr*Tt1 and *Sr*6. The mean area under the curve for the genotypes *Sr*Tt1*Sr*Tt1 and *Sr*6*Sr*6 was significantly different from that of genotypes *sr*Tt1*sr*Tt1 and *sr*6*sr*6 (Table 5). The distributions of the lines with genotypes *sr*Tt1*sr*Tt1 and *sr*6*sr*6 were normal while those of the lines with genotypes *Sr*Tt1*Sr*Tt1 and *Sr*6*Sr*6 were not (Table 5). The area under the stem rust progress curve tended to be high for the genotypes *sr*Tt1*sr*Tt1 and *sr*6*sr*6 and low for *Sr*Tt1*Sr*Tt1 and *Sr*6*Sr*6. However, in the genotypic groups with the dominant and the recessive allele of *Sr*Tt1, there were lines with equally low and high areas under the progress curve (Fig. 2). In the groups with the dominant or recessive allele of *Sr*6, there were lines with equally high areas under the curve but the low area under the curve was much lower in lines of the *Sr*6*Sr*6 genotype than they were in the lines of the *sr*6*sr*6 genotype (Fig. 2).

Information concerning the area under the stem rust progress curve for the full-sib lines (those lines with the same parents with two homozygous dominant alleles) is shown in Table 6 and Fig. 3. The populations of the full-sib lines were somewhat small, but the data tend to agree with those obtained from the populations of the half-sib lines. The populations in which *Sr*Tt1 was associated with *Sr*6, 5, 7b, or 11 tended to have low mean areas under the curve; populations in which gene *Sr*6 was associated with *Sr*5, 7b, and 11 tended to have intermediate mean areas under the curve; and populations in which the genes *Sr*5,

TABLE 5. Mean and range of area under the stem rust progress curve for groups of half-sib F_5 lines that were homozygous for the dominant or recessive alleles of genes for specific resistance to wheat stem rust caused by *Puccinia graminis* f. sp. *tritici*

Specific resistance genotype	Common parent	Area[y] (mean ± s.e.)	Number of F_5 lines	Area range
*Sr*Tt1*Sr*Tt1	Idaed 59	143 ± 17 a	144	3 - 1,164[z]
*Sr*6*Sr*6	Kenya 58	244 ± 21 b	127	1 - 1,010[z]
*Sr*11*Sr*11	Lee	701 ± 13 c	167	4 - 1,151
*sr*Tt1*sr*Tt1	Idaed 59	732 ± 35 cd	92	8 - 1,257
*sr*5*sr*5	Thatcher	738 ± 19 cd	113	69 - 1,079
*Sr*5*Sr*5	Thatcher	743 ± 19 cd	83	275 - 1,148
*Sr*7b*Sr*7b	Marquis	753 ± 12 cd	143	275 - 1,112
*sr*11*sr*11	Lee	765 ± 17 cd	80	242 - 1,133
*sr*7b*sr*7b	Marquis	792 ± 18 cd	54	435 - 1,066
*sr*6*sr*6	Kenya 58	818 ± 20 d	81	147 - 1,178

[y]Means followed by a different letter are different according to Duncan's multiple range test, $P = 0.01$.

[z]The population distribution varied significantly from a normal distribution [(21) R. R. Sokal and J. F. Rohlf. 1969. Biometry: the principles and practice in biological research. W. H. Freeman and Co., San Francisco. 776 p.].

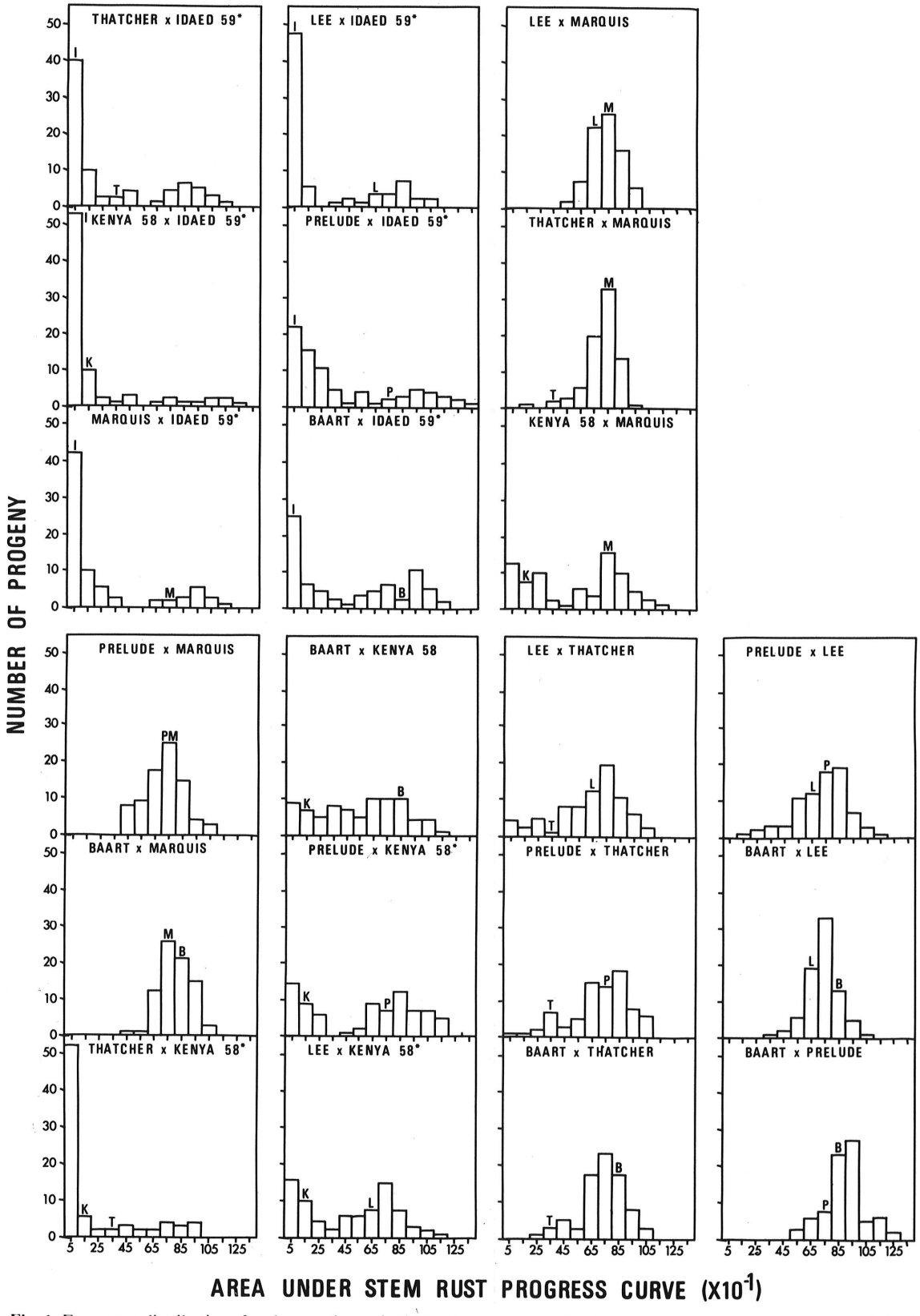

Fig. 1. Frequency distributions for slow-rusting as indicated by area under the stem rust progress curve of progenies of 21 wheat crosses infected with *Puccinia graminis* f. sp. *tritici*. The area under the curve for the parents is indicated by the letter above certain bars. The asterisk indicates distributions that were not normal.

7b, or 11 were associated together in any combination tended to have the highest mean areas under the curve. From the frequency distributions (Fig. 3) it can be seen that the association of *Sr*Tt1 and *Sr*6 with each other or with the other *Sr* genes resulted in progeny with low areas under the curve and the distributions were not normal.

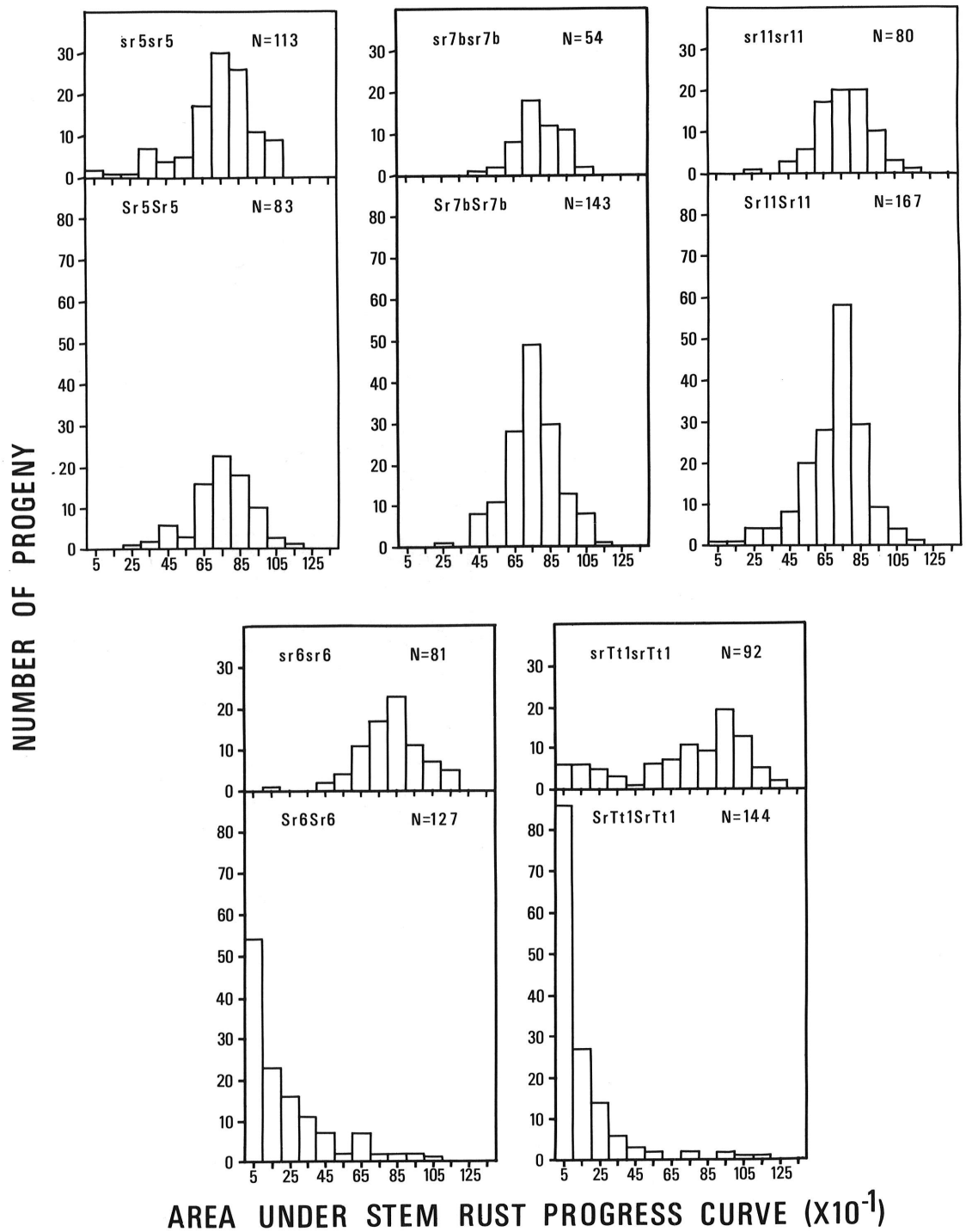

Fig. 2. Frequency distributions for area under the stem rust progress curve for half-sib F_5 lines, homozygous for the dominant or recessive allele of genes *Sr*5, 6, 7b, 11, and Tt1 that control specific resistance against wheat stem rust (*Puccinia graminis* f. sp. *tritici*). Class intervals were 100 units. N = number of progenies of a given genotype.

The association of the genes *Sr*5, 7b, and 11 with each other in any combination resulted in progeny with relatively wide ranges for area under the curve and the distributions were similar to normal distributions.

DISCUSSION

It has been suggested that the same genes control both specific and general resistance (1, 13, 26); when the genes function singly, the plants indicate specific resistance but when they function together, the plants indicate general resistance. This view has been opposed by van der Plank (24), who believes that the two types of resistance are genetically distinct. The opinion of van der Plank also appears to be supported by others. Pope (15) and Sharp and Volin (18) have shown that minor additive genes that control general resistance of wheat to stripe rust may modify the action of genes for specific resistance. Black (4) demonstrated that the resistance of potato to late blight that was due to a gene for specific resistance was enhanced by the presence of genes for general resistance. Knight (8) found a single gene in cotton that controlled specific resistance to bacterial blight, but it was ineffective in the absence of genes for general resistance.

Our data support the concept that race-specific resistance to wheat stem rust is genetically different from the slow-rusting type of resistance. The presence of the genes *Sr*5, 7b, and 11 had no effect on the development of the disease. There was a relationship between slow-rusting and *Sr*Tt1 and 6, but these genes by themselves did not control the slow development of the disease. Lines were identified that were extremely fast-rusting even though they possessed *Sr*Tt1 or *Sr*6. In recent work not yet published, we evaluated some of our slow-rusting lines that did not possess any of the *Sr* genes, *Sr*5, 6, 7b, 11, or Tt1, with several hundred cultures of *Puccinia graminis* f. sp. *tritici* and found that these lines did not possess any designated *Sr* genes.

Among the progenies from the crosses with Idaed 59, there was an association between the slow development of stem rust and the dominant allele of the *Sr*Tt1 gene. The lines with this dominant allele tended to rust slowly. However, there were a few lines with the dominant allele that rusted rapidly and there were a few lines with the recessive allele that rusted slowly. Apparently, the dominant allele of *Sr*Tt1 does not control the slow development of stem rust. The genes responsible for slow-rusting are possibly located on the same chromosome as the *Sr*Tt1 locus and they may be linked.

Among the progenies from the crosses with Kenya 58, there was an association between slow development of stem rust and the dominant allele of the *Sr*6 gene. However, the association between slow-rusting and *Sr*6 appeared to be different from that with *Sr*Tt1. The *Sr*6 gene controls resistnace to the races 15-TLM and 15-TNM at 18.3 C (65 F) but not at temperatures above 23.9 C (75 F) (6). Apparently the temperatures in the field plots during both years in this study were high enough at critical periods to allow uredia to form on lines with the dominant allele of *Sr*6. The result was that most of the lines that possessed *Sr*6 rusted slowly, but some rusted rapidly.

Most of the lines with the recessive allele of *Sr*6 tended to rust rapidly, but a few rusted moderately slowly. These facts indicate that there is an interaction between the specific resistance conditioned by *Sr*6 and genes that result in slow development of stem rust, but that the slow-rusting is not caused by *Sr*6. We concluded that the effectiveness of the resistance controlled by the *Sr*6 allele was apparently enhanced by the genes that controlled slow development of stem rust.

The results from comparing the slow development of stem rust in lines with two homozygous dominant alleles for specific resistance parallel the results from the comparison of lines with one homozygous dominant allele. Slow-rusting was associated with the dominant allele of *Sr*Tt1 and there was an apparent interaction between the dominant allele of *Sr*6 and slow-rusting. Lines with the dominant alleles of both *Sr*Tt1 and *Sr*6 all rusted slowly. There was no apparent effect on slow-rusting from any other combination of *Sr* genes with the exception of the combination of the dominant alleles of *Sr*Tt1 and *Sr*11. Lines with this combination had low means and low maximum area under the stem rust progress curve. Only a few lines were available for these comparisons and the possibility that the combination of *Sr*Tt1 and *Sr*11 will affect slow-rusting should be investigated further.

The slow-rusting character has been stable in a number of tests with various races and in several locations (25), but the testing of this character is still somewhat limited, both in terms of races and geographical area. Unknown and/or undetected genes for specific resistance in lines or cultivars may be involved in making the lines appear to be slow-rusting, especially if the unknown genes were of the same type as *Sr*6.

In the cultivars Thatcher, Lee, Marquis, Prelude, and Baart, the resistance due to the slow-rusting character is apparently under polygenic control (20). In the progenies from the crosses which had Thatcher, Lee, or Marquis as one of the common parents, there was no association between slow-rusting and either allelic form of the *Sr*5, 7b, and 11 genes. However, other genes for specific resistance have been reported in these cultivars (2, 9, 10, 11, 19). The relationship between slow rusting and these *Sr* genes should be investigated.

The gene(s) that condition(s) slow-rusting in Idaed 59 is

TABLE 6. Mean and range of area under the stem rust progress curve for groups of full-sib F_5 lines homozygous for two dominant alleles of genes for specific resistance to wheat stem rust caused by *Puccinia graminis* f. sp. *tritici*

Homozygous dominant for *Sr* genes	Area[1] (mean ± s.e.)	Number of F_5 lines	Area range
Tt1 and 6	20 ± 4 a	21	1 - 85
Tt1 and 11	27 ± 7 a	29	1 - 173
Tt1 and 5	86 ± 24 ab	31	1 - 725
5 and 6	107 ± 34 ab	40	1 - 937
Tt1 and 7b	124 ± 30 ab	35	8 - 910
6 and 11	239 ± 65 bc	13	10 - 658
6 and 7b	317 ± 60 c	22	33 - 936
5 and 11	593 ± 63 d	18	36 - 1068
5 and 7b	655 ± 34 d	30	109 - 948
7b and 11	718 ± 24 d	28	422 - 908

[1] Means followed by a different letter are different according to Duncan's multiple range test, $P = 0.01$.

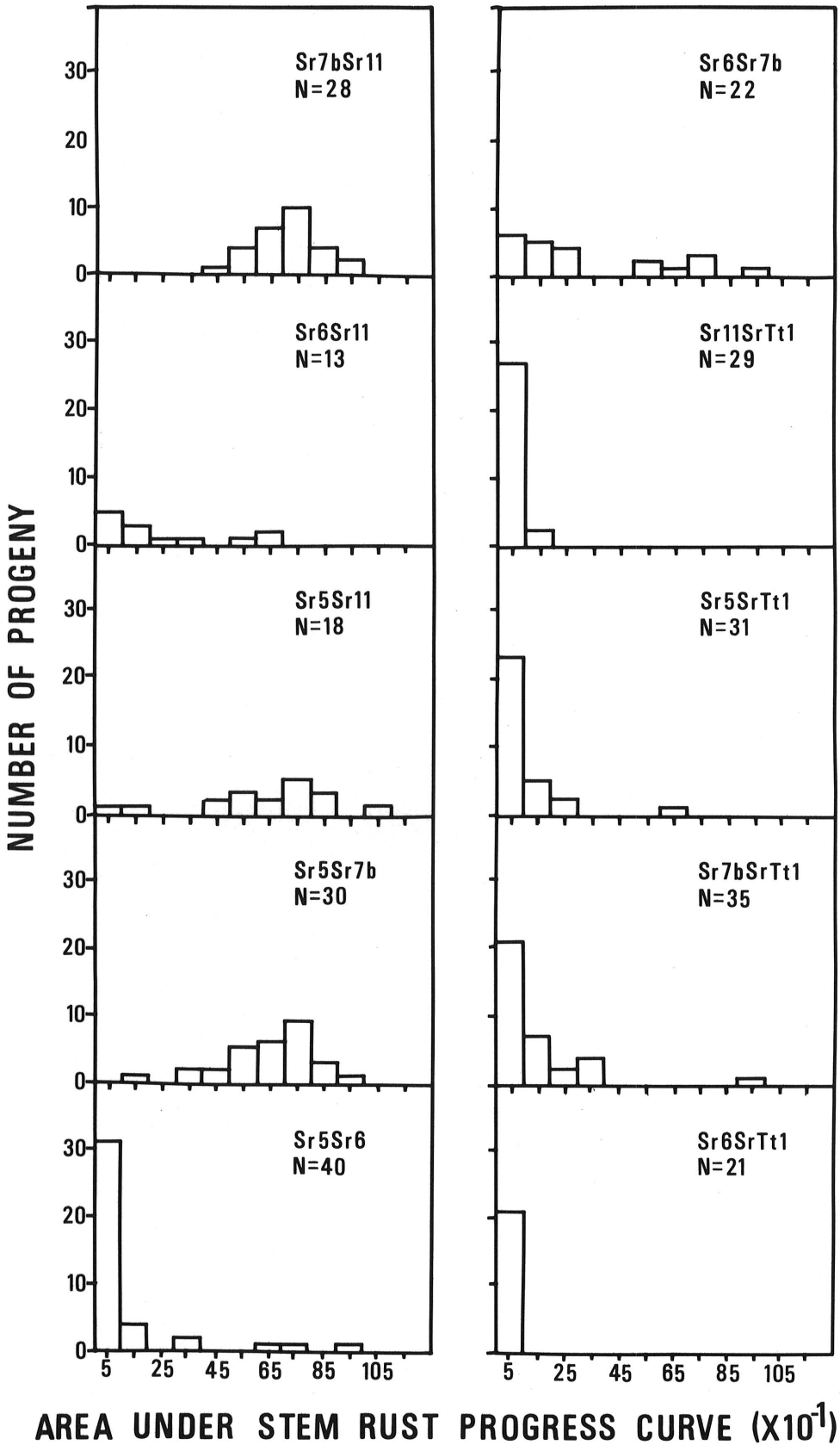
Sr7bSr11
N=28
Sr6Sr7b
N=22
Sr6Sr11
N=13
Sr11SrTt1
N=29
Sr5Sr11
N=18
Sr5SrTt1
N=31
Sr5Sr7b
N=30
Sr7bSrTt1
N=35
Sr5Sr6
N=40
Sr6SrTt1
N=21
NUMBER OF PROGENY
0
10
20
30
5
25
45
65
85
105
AREA UNDER STEM RUST PROGRESS CURVE (X10-1)

Fig. 3. Frequency distribution for area under the stem rust progress curve for full-sib F_5 lines homozygous for two dominant alleles of genes *Sr*5, 6, 7b, 11, and Tt1 that control specific resistance against wheat stem rust (*Puccinia graminis* f. sp. *tritici*). Class intervals were 100 units. N = number of progenies of a given genotype.

(are) apparently linked to the dominant allele *Sr*Tt1. It is not clear whether the slow-rusting possessed by Idaed 59 is controlled by one or several genes. The number of genes in the crosses with Idaed 59 was estimated to be less than three (20), and since all the other cultivars have some genes that condition slow-rusting, it may be that only one gene conditioning slow-rusting is found in Idaed 59.

Rowell and McVey (17) found that low receptivity in Idaed 59 was controlled by one dominant gene, and it may be that it is this character that causes this cultivar to rust slowly. On the other hand, slow-rusting in Idaed 59 could also be due to a number of linked genes. The *Sr*Tt1 gene for specific resistance found in Idaed 59 (D. V. McVey, Cereal Rust Laboratory, St. Paul, MN, *personal communication*) is from *Triticum timopheevi* (12), and since the gene(s) conditioning slow-rusting appears to be linked to the dominant allele of *Sr*Tt1, the gene(s) for slow-rusting may also have originated from *T. timopheevi*. If so, it is possible that a number of linked genes resulting in slow development of stem rust was responsible for the slow-rusting found in Idaed 59.

The interaction between the specific resistance due to the dominant allele of *Sr*6 and slow-rusting should be studied in other cultivars that possess the *Sr*6 gene. If this interaction is common, it may indicate that *Sr*6 is a "strong" gene, as defined by van der Plank (23), due to its association with general resistance, and not because of a loss of fitness of stem rust races with virulence on *Sr*6.

One problem in our work was the lack of a wheat cultivar that lacked genes for specific resistance or slow-rusting. We used the cultivars Prelude and Baart because they rusted rapidly and the uredia were large. We assumed that they had few, if any, genes for resistance. It is now apparent that they possess genes that control the rate of rust development.

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