Inheritance of Stem Rust Resistance in Triticum aestivum, 'C. I. 14115', a Powdery Mildew Differential

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ABSTRACT

Inheritance of seedling resistance to stem rust was studied in the F_3 and F_4 of *Triticum aestivum* 'C. I. 14115' × susceptible 'Little Club'. Resistance to culture 111-SS2 (physiologic race 111) of *Puccinia graminis* f. sp. *tritici* was conditioned by two independent dominant genes and to culture 69-21-399 (physiologic race 151) by a single dominant gene. The gene for resistance to culture 69-21-399 was associated with one of the genes for resistance to culture 111-SS2. Neither of the three genes was associated with the gene for mildew resistance, *Pm1.* The presence of the gene, *Sr15*, for resistance to stem rust was demonstrated by I. A. Watson in several F_3 and F_4 families by testing them with certain cultures indigenous to Australia. Tests conducted by A. P. Roelfs with cultures of physiologic races 17A, 32, and 177 (which collectively had genes for avirulence corresponding to genes for resistance, *Sr5, Sr6, Sr7b, Sr8, Sr9a, Sr9b, Sr9d, Sr10, Sr11,* and *Sr14*) failed to indicate additional genes for resistance in 40 F_5 families (from 16 F_3 families) homozygous for susceptibility to cultures 111-SS2 and 69-21-399.

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Additional key words: Erysiphe graminis f. sp. *tritici,* wheat, genetics.

Triticum aestivum L. 'C. I. 14115' ('C. I. 13836'/8 'Chancellor') was developed by Briggle (2) to differentiate cultures of *Erysiphe graminis* DC. ex Merat f. sp. *tritici* Em. Marchal. The line has a single known gene for mildew resistance (designated *Pm1*) from C. I. 13836 (1), a common wheat from Mexico (7). A gene for mildew resistance at the same locus occurs in cultivars "AsII', 'Axminster', 'Converse', 'Farrertrou', 'Fram I', 'Kenya 744', 'Norka', 'Pika', 'Thew', and 'TU 4' (1,3,5). In Axminster, the *Pm1* locus was located on the long arm of chromosome 7A (8).

Watson and Luig (10) reported a dominant gene (designated *Sr15*) for stem rust resistance in Thew and Norka that was closely linked with the genes on chromosome 7A for resistance to powdery mildew and leaf rust. Pathologic tests indicated that *Sr15*, effective against certain Australian cultures of physiologic race 21 of *Puccinia graminis* Pers. f. sp. *tritici* Eriks. & E. Henn., occurred also in Axminster, 'Birdproof', 'Bonus W33', Converse, 'Festival', 'Huron' (C. I. 3315), 'Kenora', Kenya 744, 'Normandie', and 'Sweden W1230', Cultures capable of detecting *Sr15* are unknown in the United States.

The purpose of this investigation was to estimate the number of genes in C. I. 14115 for seedling resistance to stem rust, and to determine if our test culture of *P. graminis* f. sp. *tritici* has the gene for avirulence that corresponds to *Sr15*. A preliminary report has been published (6).

MATERIALS AND METHODS.—Single-spore culture 111-SS2 of *P. graminis* f. sp. *tritici* was our primary test culture in studies of seedling resistance in the F_3 and F_4 of 'Little Club' × C. I. 14115. Usually, 30 to 40 plants were tested in each F_3 family, but in some the number was as few as 15, and in others as many as 60. Culture 111-SS2, used extensively in studies of stem rust resistance (3), is known to have genes for avirulence corresponding to genes for resistance, *Sr6, Sr7b, Sr8, Sr9d, Sr10, Sr11, Sr12, Sr13, Sr16, Sr18, Sr19,* and *Sr20.* Our method of inoculation has been described (3). After inoculation, the plants were incubated in a greenhouse at 25±5 C under 16,140 lx (1,500 ft-c) or more of natural and supplemental fluorescent light for 12 h or more per day.

When scored for reaction, the F_3 families were divided into three classes: (i) those homozygous for susceptibility; (ii) those segregating resistant and susceptible plants in numbers fitting 3:1 ratios when tested by X^2; and (iii) those homozygous for resistance at one or more loci, or segregating for resistance at more than one locus. The class that appeared to segregate for monogenic resistance was subdivided into additional classes, based on infection types in resistant plants.

In F_3 families of less than 48 individuals, some segregating families will fit the calculated expectancies of both 3:1 and 15:1 ratios. As a result, the number of families classed as segregating for a single factor will be exaggerated if two genes condition nearly similar phenotypes. To avoid this, we tested 10 F_4 families derived from each F_3 family classed as segregating in a 3:1 ratio. We used the data to affirm the number of genes segregating in each parental F_3 family and the infection types conditioned by them singly.

In addition to greenhouse tests, growth-chamber

TABLE 1. Segregation among F_3 families from Little Club × C. I. 14115 for seedling reaction to culture 111-SS2 of *Puccinia graminis* f. sp. *tritici*

Reaction of F_3 families	Number of families[a]	
	Observed	Expected
Inseparable segregating and homozygous resistant	321	300.3
Seg. 3:1 (0;1 and 4)	55	54.6
Seg. 3:1 (3- and 4)	39	54.6
Homozygous susceptible	22	27.3

[a]Fitted to a 11:2:2:1 ratio, $0.05 < P < 0.10$.

tests of randomly selected F_3 families that had segregated three resistant seedlings to one susceptible were made. These plants were simultaneously inoculated with culture 111-SS2 and culture No. 6 of *E. graminis* f. sp. *tritici* (obtained from A. L. Scharen), which was avirulent on stocks having *Pm1*. They were incubated in a growth chamber programmed for 12-h days at a constant 17±2 C. Selected F_3 families were tested also with culture 69-21-399 (physiologic race 151) of *P. graminis* f. sp. *tritici* (obtained from D. V. McVey).

Infection types were described by the symbols proposed by Stakman et al. (9). All plants classed susceptible had infection types as high as those in Little Club.

RESULTS.—C. I. 14115, Chancellor, and Little Club developed infection types 0;, 0; and 1, and 4, respectively, when inoculated with culture 111-SS2. A X^2 test for heterogeneity indicated that segregation patterns among 437 F_3 families derived from four F_1 plants of Little Club × C. I. 14115 were homogeneous ($0.05 < P < 0.10$). Twenty-two families were homozygous for susceptibility, 94 segregated in 3:1 ratios of resistant to susceptible plants, and 321 were either homozygous resistant or segregated for resistant and susceptible reactions in other than monogenic ratios. Among the segregating 94 families, 55 produced low infection types of 0; to 1, and 39 produced a low infection type of 3-. From these data, we concluded that resistance of C. I. 14115 to culture 111-SS2 was conditioned by two dominant independent genes (Table 1).

Sixteen F_3 families that had segregated 0; to 1 and 4 infection types and eight that had segregated 3– and 4 infection types were inoculated simultaneously with cultures 111-SS2 and No. 6. The results (Table 2) indicated that neither of the genes for resistance to culture 111-SS2 was linked with *Pm1*, and that either C. I. 14115 did not have *Sr15*, or culture 111-SS2 lacked the corresponding gene for avirulence necessary for its detection. To facilitate discussion, we assigned temporary symbols *SrCI1* and *SrCI2* to the genes that conditioned 0; to 1 and 3- infection types, respectively.

Culture 69-21-399 (physiologic race 151) of *P. graminis* f. sp. *tritici*, which produced an infection type 2 in Norka (D. V. McVey, *personal communication*), was obtained from the Cereal Rust Laboratory, USDA, University of Minnesota, and used to inoculate Chancellor, C. I. 14115, Little Club, 14 F_3 and F_4 families homozygous for *SrCI1*, and 42 selected F_3 families that had segregated three resistant plants (*SrCI2*) to one susceptible to culture 111-SS2. Chancellor developed a slightly higher infection type (3- to 3c) than C. I. 14115 (3-). Little Club and families having *SrCI1* singly were fully susceptible. Among the 42 families having *SrCI2*, four reacted differently to culture 69-21-399 than they had to culture 111-SS2 (Table 3), indicating that resistance to the two cultures was conditioned by different but associated genes. Infection types in resistant plants of the 42 families ranged from 3- (similar to those in C. I. 14115) to 3-3c, (similar to those in Chancellor). However, gradual intergradations from 3- to 3c precluded consistent distinction of the two types, and they were treated as the range of a single phenotype.

Additional tests indicated that resistance to cultures 111-SS2 and 69-21-399 was conditioned by different genes. Firstly, 12 of 80 F_4 families (from 11 F_3 families that had segregated monogenically to culture 111-SS2) reacted differently to cultures 111-SS2 and 69-21-399. Of seven families homozygous for resistance to culture 111-SS2, five segregated and two were homozygous for susceptibility to culture 69-21-399. Two families that segregated to culture 111-SS2 were homozygous for susceptibility to culture 69-21-399, and of three families homozygous for susceptibility to culture 111-SS2, one was homozygous for resistance and two segregated to culture 69-21-399. Secondly, among 40 F_5 families (derived from 16 F_3 families), fully susceptible to culture 111-SS2 in tests in our laboratory and to single cultures of physiologic races 17A, 32, and 177 in tests conducted in the Cereal Rust Laboratory, eight segregated resistant (3- infection type) and susceptible plants to culture 69-21-399 in approximate 3:1 ratios. Consequently, the gene for resistance to culture 69-21-399 was provisionally designated *SrCI3*.

Twenty-four F_3 and 43 F_4 families that had reacted variously to cultures 111-SS2, 69-21-399, and No. 6 were tested by I. A. Watson with cultures of *P. graminis* f. sp. *tritici* known to have the gene for avirulence corresponding to *Sr15* and with two strains of *E. graminis* f. sp. *tritici*. Watson stated (*personal communication*), "There was complete correlation between the behavior to powdery mildew and to stem rust when the strains used could not attack plants with *Sr15*. The expression of resistance resembled that normally expected when *Sr15* is present".

DISCUSSION.—The foregoing results indicate that C. I. 14115 has at least four genes for resistance to stem rust: two (*SrCI1* and *SrCI2*) that condition resistance to culture 111-SS2, one (*SrCI3*) that conditions resistance to culture 69-21-399, and the previously designated *Sr15*. The apparent independence of *SrCI1*, *SrCI2*, and *SrCI3*, from *Pm1*

TABLE 2. Joint segregation of two genes considered separately for resistance to culture 111-SS2 of *Puccinia graminis* f. sp. *tritici* and the gene, *Pm1*, for resistance to culture No. 6 of *Erysiphe graminis* f. sp. *tritici* among plants in combined F_3 families of Little Club × C. I. 14115

Reaction to Culture No. 6	Reaction to culture 111-SS2			
	No. of Plants[a,c]		No. of plants[b,c]	
	Resistant (Infection types 0; to 1)	Susceptible (Infection type 4)	Resistant (Infection type 3-)	Susceptible (Infection type 4)
Resistant	251	67	150	43
Susceptible	87	23	57	13

[a]From 16 F_3 families indicated to be homogeneous by the X^2 test for heterogeneity, $0.05 < P < 0.10$.
[b]From eight F_3 families indicated to be homogeneous by the X^2 test for heterogeneity, $0.05 < P < 0.10$.
[c]The X^2 test for independence indicated that *Pm1* was not associated with either gene which conditioned resistance to culture 111-SS2 (respectively, $0.05 < P < 0.10$ and $0.10 < P < 0.20$ for genes conditioning 0; to 1 and 3- infection types).

TABLE 3. Joint segregation for reaction to cultures 111-SS2 and 69-21-399 of *Puccinia graminis* f. sp. *tritici* among 42 F_3 families from Little Club × C. I. 14115

Reaction to culture 111-SS2	No. of families[a]		
	Reaction to culture 69-21-399		
	Homozygous resistant	Segregating	Homozygous susceptible
Homozygous resistant	7	3	0
Segregating	0	20	0
Homozygous susceptible	0	1	11

[a]The X^2 test for independence indicated that genes for resistance to cultures 111-SS2 and 69-21-399 were associated, $P < 0.01$.

indicates that their presence in C.I. 14115 probably derives from Chancellor.

SrCI3 conditioned a 3– infection type in C. I. 14115 and a 3- to 3c in Chancellor. Since, relative to stem rust reaction, C. I. 14115 is known to differ from Chancellor only by the presence of *Sr15*, the lowered infection type probably resulted because *Sr15* modified the interaction of *SrCI3* and the corresponding gene for avirulence. Modifying effects of a presumably ineffective gene in a specific host-gene/parasite-gene interaction is not unprecedented (11).

Recent work by Luig and Rajaram (4) showed that infection types conditioned by interactions of specific genes for resistance and those for avirulence in *P. graminis* f. sp. *tritici* are modified by the genetic background of the stocks in which the genes for resistance are assessed. Usually, a specific gene for resistance conditioned lower infection types when combined with other genes, than when in stocks otherwise devoid of genes for resistance.

The genes for stem rust resistance in C. I. 14115, whether singly or in combination, do not provide adequate protection against several cultures in the natural population of *P. graminis* f. sp. *tritici*. However, we believe that in future programs of breeding stem rust-resistant wheats, much greater emphasis will be placed on the cumulative effects of gene interactions than in the past. Consequently, the genes from C. I. 14115 may be useful in building broad-spectrum resistance conditioned by several interacting gene combinations.

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