# Genetics of Resistance to Stem Rust in Thirteen Wheats of Diverse Origins

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## ABSTRACT

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Inheritance of seedling resistance to culture 111-SS2 of *Puccinia graminis* f. sp. *tritici* was studied in crosses of 13 resistant wheat (*Triticum aestivum* and *T. turgidum*) cultivars and selections with susceptible *T. aestivum* 'Little Club'. The resistant wheats included: Gai Printemps, Docteur Mazet, Primepi, Opal, Cleo, Kenya Page, Kenya Hunter, Webster, Ninguta 157, D7314, N4, a *T. turgidum* selection, and a *Triticum* sp. selection. Resistance was conditioned by one gene in the *T. turgidum* selection, by two genes in Docteur Mazet and D7314, by four genes in Primepi; and by three genes in each of the remaining wheats. Genes for resistance were dominant and independent in all crosses. Lines derived from each cross also were tested with 24 additional cultures. A comparison of the reaction of each line with those of 66 tester stocks having known genes for resistance indicated that Opal, Kenya Page, Kenya Hunter, and *Triticum* sp. have *Sr7b;* Docteur Mazet and *Triticum* sp. have *Sr10;* D7314 has *Sr15;* Kenya Page and Kenya Hunter have *Sr17;* Gai Printemps, Docteur Mazet, Primepi, Opal, Cleo, Kenya Page, Kenya Hunter, and Ninguta 157 have *Sr18;* and Webster has *Sr30.* The unidentified genes conditioned intermediate levels of resistance to a narrow array of biotypes of *P. graminis* F. sp. *tritici.* Consequently, their value in current U.S. breeding programs appears to be limited.

Use of resistant cultivars is the most economical method currently available for controlling stem rust of wheat which is incited by *Puccinia graminis* Pers. f. sp. *tritici* Eriks. & E. Henn. Resistance of newly developed cultivars to prevalent biotypes of *P. graminis* f. sp. *tritici* often is conditioned by a single gene. When confronted by a large population of wind-borne urediospores with a capacity to widen its virulence by several methods (3), the resistance gene has on the average been rendered ineffective after about 5 yr (6).

Reducing the vulnerability of wheat to rust epiphytotics and extending the productive life of cultivars is a major goal of plant disease research. The use of genetic diversity, locally with varied gene combinations in pure and multiline cultivars, and regionally by managed deployment of specific genotypes, seemingly offers the most effective method of attaining that goal (2,7). Gene management strategy requires knowledge of the effectiveness of many single resistance genes and their interactions in specific combinations.

The objectives of work reported in this paper were to identify as many resistance genes as practicable in wheats of diverse origin, develop wheat lines having the resistance genes separately, and evaluate them for usefulness in breeding programs.

## MATERIALS AND METHODS

Thirteen cultivars and selections of wheat (*Triticum aestivum* L., *T. turgidum* L., and an unidentified *Triticum* species) resistant to our primary test culture of *P. graminis* f. sp. *tritici* were used as male parents in crosses with near-universally susceptible *T. aestivum* 'Little Club' (Table 1). Except for Webster, seed of the resistant wheats were obtained from the USDA Germplasm Resources Laboratory, Agricultural Research Center-West, Beltsville, MD 20705. Seed of Webster was taken from stocks maintained by the second author. Segregating populations used to estimate the numbers of genes conditioning resistance in each cross were derived from four $F_1$ plants, with three exceptions. Populations from crosses of *T. aestivum* 'N4', the *T. turgidum* selection, and *Triticum* sp. No. 3902 were obtained from one, two and three $F_1$ plants, respectively.

Single-spore culture 111-SS2 of *P. graminis* f. sp. *tritici* served as the primary test culture. Culture 111-SS2, which is used extensively in prior studies of stem rust resistance (4,5,15), is known to be virulent to plants carrying resistance genes *Sr5, Sr9a, Sr14,* and *Sr15.* Our methods of inoculating and scoring for seedling reactions to rust infection have been described (5), however, descriptions of some aspects of the procedures will be repeated here. First, $F_3$ families were divided into three classes: (i) those homozygous for resistance at one or more loci, or segregating for resistance at more than one locus; (ii) those segregating resistant and susceptible plants in numbers fitting 3:1 ratios when tested by $\chi^2$; and (iii) those homozygous for susceptibility. When

TABLE 1. Identity and source of resistant wheats investigated for inheritance of resistance to stem rust in crosses with susceptible *Triticum aestivum* 'Little Club'

| Name or designation | PI or CI number[a] | Source |
|---|---|---|
| *T. aestivum* | | |
| Gai Printemps | 315986 | France |
| Docteur Mazet | 315979 | France |
| Primepi | 316002 | France |
| Opal | 315837 | Netherlands |
| Cleo | 315841 | Netherlands |
| Kenya Page | 290747 | Kenya |
| Kenya Hunter | 299423 | Kenya |
| Webster | 3780 | Russia |
| Ninguta 157 | 7170 | China |
| D7314 | 8925 | China |
| N4 | 11053 | China |
| *T. turgidum* | 8116 | China |
| *Triticum* sp. No. 3902 | 8326 | China |

[a] PI and CI are abbreviations for Plant Introduction and Cereal Investigations, respectively. In this table, PI numbers have six digits and CI numbers have either four or five.

appropriate, or possible, the second class was subdivided into additional classes based on infection types in resistant plants. Ten $F_4$ families descended from reserve seed of each $F_3$ family classed as homozygous for susceptibility, or as segregating in a 3:1 ratio, were tested for seedling reaction. The results were used to confirm and adjust the classification of $F_3$ families before subjecting the data from the $F_3$ to statistical analysis.

The most difficult aspect of classifying both $F_3$ and $F_4$ families within a cross was distinguishing among families with similar infection types that were conditioned by different single genes for resistance. When the infection types (phenotypes) conditioned by different single genes could not be separated with confidence, the data from families with similar infection types were combined and tested for goodness of fit to genetic ratios that treated them as a single class.

Chi-square tests were used to determine the probability of homogeneity among $F_3$ populations within each cross involving more than one $F_1$ plant, and to determine the probability of observed class distributions fitting ratios assumed from segregation patterns.

The $F_4$ families classed as homozygous for monogenic resistance were advanced to the $F_5$ generation. Selected $F_5$ families were tested for reactions to culture 111-SS2 and, with a few exceptions, to 24 additional cultures maintained by the USDA Cereal Rust Laboratory. The cultures used in addition to 111-SS2 had the following Cereal Rust Laboratory identification numbers and race codes (in parentheses) (11,12): 59-51-A (BCB), 73-45-399C (BFC), 75-45-1622-A (DKC), 74-45-1331-B (GDC), 72-21-1157-C (GLC), 71-08-184-C (HJC), 68-41-73-A (HNL), 70-B458-A (LCB), 76-34-513-A (MBC), 74-36-923-B (NFB), 76-38-54-C (QCB), 75-45-1694-A (QCC), 72-00-1370-C (QFB), 69-21-399 (QSH), 71-21-550-C (RHR), 76-32-744-C (RFQ), 72-00-1369-C (RTQ), 73-47-293-B (SBC), 72-21-1184-B (TMB-1, *Sr15* avirulent), 74-32-A21 (TMB-2, *Sr15* virulent), 73-21-848 (TDM), 76-14-280-C (TLM), 76-24-268-B (TNM-1, *Sr17* avirulent), and 76-32-744-8 (TNM-2, *Sr17* virulent). Hereafter, for brevity, race codes are used in this paper to indicate all cultures except 111-SS2.

Culture QCC was unique in that it caused a low infection type (0;to 2=) in the near-universally susceptible Little Club. We assumed that the resistance of Little Club was conditioned by a single gene, which we provisionally designated *SrLC*.

To verify the virulence-avirulence genotypes of the cultures, each (except QCC) was used to inoculate 66 wheat stocks having the following resistance genes either singly, in known combinations, or in combinations with unidentified ones; permanently named genes in the *Sr* series, *5, 6, 7a, 7b, 8, 9a, 9b, 9e, 9f, 9g,* and *10* through *30*; and genes tentatively designated as Mg-X, *Tt-1, Tt-2, Tt-3, Tmp, Kota-2, dp-2, Gt,* and *SrLC*. Stocks having *Sr13, 22, 24, 25, 26, 27, 29, Tt-2,* and *Gt* were resistant to all cultures. Conversely, those having singly *Sr9f, 18, 19,* and *20* were susceptible to all cultures except 111-SS2. The various patterns of reactions to the cultures across the remaining stocks are omitted from this paper for brevity.

Reactions of each $F_5$ family to the 25 cultures were compared with reactions of the 66 stocks for similarities that would indicate those families and stocks most probably having common genes for resistance.

## RESULTS AND DISCUSSION

**Inheritance of resistance.** Data from testing $F_3$ and selected $F_4$ families indicated that seedling resistance to culture 111-SS2 was controlled by dominant and independent genes in each cross. Four genes conditioned resistance in one cross, three in eight crosses, two in three crosses, and one in one cross (Table 2). Little Club × Ninguta 157 was the only cross among the 13 in which the distribution of reaction classes was a poor fit ($P < 0.05$) to the proposed ratio. However, the distribution among classes of $F_3$ families derived from each of four F1 plants of Little Club × Ninguta 157 were good fits to the proposed ratios when analyzed separately, and the $\chi^2$ test for heterogeneity indicated that the combined data were homogeneous ($P > 0.10$).

Adult plant resistance was evident in the field at College Station, TX, in 1975 among Kenya Hunter and Kenya Page derived lines ($F_5$) susceptible as seedlings to culture 111-SS5. No attempt was made to identify the genes for adult plant resistance.

**Pathogenicity tests.** Forty-eight $F_5$ families (hereafter termed lines) classed as monogenic and homozygous for resistance to culture 111-SS2 were selected from the 13 crosses and inoculated in the seedling stage with 24 additional cultures. By comparing the reactions of each line with those of the 66 stocks with known genes for resistance, we tentatively identified previously named genes *Sr7b, 10, 15, 17, 18,* and *30* (Table 2) as being responsible for resistance of 24 of the lines. Both *Sr7b* and *10* were present in one line from Little Club × *Triticum* sp. No. 3902. Gene *Sr15*, not detectable with culture 111-SS2 (4), was indicated in one line from Little Club × D7314 by cultures TBM-1, TLM, TNM-1, and TNM-2. Resistance of the remaining 24 lines could not be related to any genes known to be in the tester stocks.

Segregation among or within lines for plants susceptible to culture QCC indicated that of the resistant parents only Opal had the gene *Sr*LC. We tested 12 lines from Little Club × Opal (including the two listed in Table 3) with culture QCC and all were homozygous for low infection types indistinguishable from those in Little Club. Gene *SrLC* was absent from 12 of the wheats used in this study, we have data from other crosses involving Little Club which indicate that another 13 selections and cultivars lack *SrLC*. They are: CI 8458, CI 8925, CI 9275, CI 10487, CI 10613, CI 11050, and CI 12612 from China; CI 14115 and Lee from the United States; Lucero (CI 14047) from Argentina; Ngesi (PI 314911) from Rhodesia; Skorospelka 3b (PI 316439) from Russia; and Vila Velha

TABLE 2. Inheritance of seedling reaction to culture 111-SS2 of *Puccinia graminia* f. sp. *tritici* in the $F_3$ from crosses of 13 resistant wheats with susceptible cultivar Little Club and identified *Sr* genes for resistance

| Resistant parent | Number of $F_3$ families | Ratio of classes[a] i | ii | iii | No. of genes | $P(\chi^2)$ | *Sr* genes identified |
|---|---|---|---|---|---|---|---|
| Gai Printemps | 372 | 57: | 2:4 | 1 | 3 | >0.30 | *Sr18* |
| Docteur Mazet | 106 | 11: | 2:2: | 1 | 2 | >0.50 | *Sr10, Sr18* |
| Primepi | 389 | 247: | 2:2:2:2: | 1 | 4 | >0.50 | *Sr18* |
| Opal | 93 | 57: | 2:2:2: | 1 | 3 | >0.80 | *Sr7b, Sr18* |
| Cleo | 329 | 57: | 2:2:2: | 1 | 3 | >0.10 | *Sr18* |
| Kenya Page | 283 | 57: | 2:2:2: | 1 | 3 | >0.30 | *Sr7b, Sr17, Sr18* |
| Kenya Hunter | 289 | 57: | 2:2:2: | 1 | 3 | >0.05 | *Sr7b, Sr17, Sr18* |
| Webster | 397 | 11: | 4: | 1 | 2 | >0.50 | *Sr30* |
| Ninguta 157 | 364 | 57: | 2:4: | 1 | 3 | >0.02 | *Sr18* |
| D7314 | 107 | 11: | 2:2: | 1 | 2 | >0.50 | *Sr15* |
| N4 | 274 | 57: | 6: | 1 | 3 | >0.50 | None |
| *T. turgidum* | 137 | 1: | 2: | 1 | 1 | >0.05 | None |
| *Triticum* sp. No. 3902 | 153 | 57: | 6: | 1 | 3 | >0.50 | *Sr7b, Sr10* |

[a] Classes i, ii, and iii were, respectively, those families either homozygous or segregating for resistance at more than one locus, those segregating resistant and susceptible plants in numbers fitting 3:1 ratios, and those homozygous susceptibility.

TABLE 3. Lines from 12 wheat crosses monogenic for resistance to culture 111-SS2 of *Puccinia graminis* f. sp. *tritici* and additional cultures (designated by race codes[a]) to which they were resistant

| Resistant cultivars and derived lines[b] | Common infection types[c] | Cultures in addition to 111-SS2 causing low infection types |
|---|---|---|
| Gai Printemps | | |
| 75-113L | 3=3− | QCC |
| 75-133L | 3− | BCB, HNL, QCC |
| 75-129L | 3−3 | HNL, QCC |
| Primepi | | |
| 75-145 | 3− | BCB, HNL, QCC |
| 75-159 | 3− | BCB, HNL, TBM-1 |
| 75-156L[d] | 12n | BCB, HNL, QCC, TBM-1 |
| Opal | | |
| 75-70L | 12− | BCB, HNL, QCC |
| 75-72L | 13− | BCB, HNL, QCC, TBM-1 |
| Cleo | | |
| 75-87L[e] | 3− | HNL, QCC, TBM-1 |
| 75-93L | 3− | BCB, HNL, QQC |
| Kenya Page | | |
| 75-13L[e] | 3− | QCC |
| Kenya Hunter | | |
| 75-36L[f] | 3− | BCB, HNL, QCC, TBM-1 |
| Webster | | |
| 75-353,-361, and -326 | 2− | BCB, QCB, QFB, RKO, RTQ |
| Ninguta 157 | | |
| 75-328[e] | 3= | HNL |
| 75-325 | 3− | BCB, HNL |
| D7314 | | |
| 75-308L | 13− | QCC |
| 75-306 | 3− | BCB, HNL |
| N4 | | |
| 75-332L | 2− | HNL, QCC |
| 75-337 | X+ to 3 | None |
| *T. turgidum* | | |
| 75-316 | 23= | HNL |
| *T.* sp. No. 3902 | | |
| 75-349 | 12−2 | HNL |
| 75-350 | 13− | None |

[a] Cereal Rust Laboratory race codes.
[b] Line designations ending with the letter "L" indicates that the line had a gene from Little Club tentatively designated *SrLC* which conditioned resistance to culture QCC.
[c] Infection types on some lines varied slightly with culture. Infection types 0; and 2= always developed when lines having *SrLC* were inoculated with culture QCC.
[d] Tested only with cultures 111-SS2, BCB, HNL, QCC, and TBM-1.
[e] Not tested with culture BCB.
[f] Has a gene for resistance not detected in tests of the $F_3$ and $F_4$.

(PI 313095) from Brazil.

Gene *Sr18* was the most common of the six resistance genes, tentatively identified as belonging to the *Sr* series. It was present in eight of the 13 resistant parent cultivars. Loegering and Harmon (8) reported that *Sr18* was almost universally present in bread wheats, it was known to be absent only from Little Club, Chinese, and Prelude. Later, Baker et al (1) reported that *Sr18* was absent from Brevit, Eureka, Federation, Gular, Kenya C6204, Koala, Morroco, Norka, and Yalta. Our data indicated that three additional cultivars of *T. aestivum* (Webster, D7314, and N4), the selection of *T. turgidum,* and the unidentified *Triticum* sp. lacked *Sr18.*

Disregarding resistance conditioned by *SrLC,* every cultivar except Docteur Mazet had one or more genes for resistance which could not be identified with known *Sr* genes by the pathogenicity tests. Resistance conferred by these newly recognized unnamed genes was expressed by infection types ranging from 1 to 3$^-$ (usually 2$^-$ and 3$^-$) and was effective against only a few cultures.

Similarities of infection types resulting from infection with 111-SS2 and other cultures precluded recognition of different exact genotypes among the derived lines having unnamed resistance genes. However, the lines could be separated into five groups based on their reaction patterns to the cultures (excluding QCC); (i) five lines derived from Gai Printemps, Kenya Page, D7314, N4, and *Triticum* sp. No. 3902 were resistant only to culture 111-SS2; (ii) five lines derived from Gai Printemps, Ninguta 157, N4, *T. turgideum,* and *Triticum* sp. No. 3902 were resistant to cultures 111-SS2 and HNL; (iii) six lines derived from Gai Printemps, Primepi, Ninguta 157, D7314, Opal, and Cleo were resistant to cultures 111-SS2, BCB, and HNL; (iv) four lines derived from Primepi, Opal, and Kenya Hunter were resistant to cultures 111-SS2, BCB, HNL, and TBM-1; and (v) three lines derived from Webster, and having the same gene, were resistant to cultures 111-SS2, BCB, QCB, QFB, RKQ, and RTQ.

The unnamed gene from Webster conditioned resistance to more cultures than did unnamed genes in the other cultivars, and the resistance was directed against biotypes that have been more prevalent in the United States during recent years than biotypes suppressed by unnamed genes from the other cultivars (9–14).

The narrow spectrum of resistance conditioned by the unnamed genes, compared to that conditioned by many named ones, against the prevalent races and biotypes limits the value of the unnamed genes in breeding programs. Among the tentatively identified *Sr* genes in the derived lines, *Sr7b* conferred resistance to 12 cultures, *Sr10,* to seven, *Sr15* to four, *Sr17* to seven, and *Sr30* to 24. Furthermore, as mentioned earlier, the tester stocks with permanently named *Sr* genes *12, 22, 24, 25, 26, 27, 29,* and those temporarily designated *Tt-2* and *Gt* conferred resistance to all cultures. Thus, the greatest value of the newly recognized unnamed genes appears to be their potential for defense against races and biotypes which may arise with a genetic capacity to overcome the named genes.

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