

## Close Association of Two Factors for Avirulence in *Puccinia graminis tritici*

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### ABSTRACT

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The corresponding genes for avirulence on Webster and *Agropyron intermedium* derivative W3592 in *P. graminis tritici*, are distinct. A change in one gene, however, is likely to affect the other.

*Additional key words:* double mutants, gene interactions.

In 1963, a rust-resistant wheat cultivar, Festiguay, was made available to farmers in Australia. Festiguay was derived from a cross supposedly involving Festival (*Sr9b*, *Sr15*; *Lr20*) and Uruguay C10837, but subsequent studies (1) showed that the cultivar carries the Webster gene *Lr2a* for resistance to *Puccinia recondita* Rob. ex Desm. (wheat leaf rust), and one major gene for resistance to *Puccinia graminis* Pers. f.sp. *tritici* Eriks. & E. Henn. (wheat stem rust). The latter gene also was found in Webster (1), but because Festiguay also has a minor gene for resistance to *P. graminis tritici* it is the more resistant of the two cultivars (Luig, *unpublished*). Recently, the gene for resistance to *P. graminis tritici* in Webster and Festiguay was designated as *Sr30* (4).

A year after the release of Festiguay, a single isolate of *P. graminis tritici* was found which proved virulent on both cultivars. It was first assumed that the virulent strain, then identified as 21-2,8 [for classification of Australian strains see (2, 3, 5, 6)] had its origin as a simple mutation from the common strain 21-2 (2). Later work, however, has shown that it also differed from its putative parent by being able to attack plants of the *Agropyron intermedium* (Host) Beauv. derivative W3592, number 9 of the Australian supplementary differentials.

Gene *Sr30* conditions "2+" infection types in seedlings, but allows considerable rust development on adult plants at high temperatures. The pustules, although restricted in size, would provide for strong competition against any mutants virulent on plants with *Sr30*, and this competitive effect may have been a major factor in maintaining the resistance of Festiguay for 5 yr after its release. In 1968 two new strains, 21-2,3,7,8,9 and 194-2,3,7,8,9 appeared which were fully virulent on Festiguay and they increased rapidly. Surprisingly, both strains proved virulent on W3592 which at that time was resistant to nearly all Australian strains. The main difference between the two strains is their behavior on plants with *Sr9g* [Acme, Kubanka (4)]. Luig and Watson (2) have

considered the possibility that 194-2,3,7,8,9 and 21-2,3,7,8,9 could have originated as mutants for virulence on Festiguay from the common strain 21-2,3,7, but noted slight differences between the resistant infection types produced by 21-2,3,7,8,9 and 21-2,3,7 on plants with *Sr8*. Including this difference, both strains specific for Festiguay differ from their putative parent 21-2,3,7 by at least three characters, and consequently it seems unlikely that they have arisen by mutation. Therefore, the same authors (2) advanced an alternative explanation, viz., that the two strains are introductions to Australia.

Although the popularity of Festiguay declined immediately after it became susceptible, the two strains virulent on this cultivar increased in frequency. In the following years, mutational changes for increased virulence at other loci in the two strains gave rise to several strains which now are predominant in eastern Australia. All these strains are virulent on both Festiguay and *A. intermedium* derivative W3592. To study the possible association of virulence on these two cultivars, a mutation experiment using the mutagen ethyl methane sulfanate (EMS) was carried out.

### MATERIALS AND METHODS

The strain of *P. graminis tritici* used for the mutation experiment was 34-1,2,3,4,5,6,7,11 (culture 75-L-9). It was derived from the common field strain 21-2,3,4,5,7 after six EMS treatments resulting in seven pathogenic changes (Luig and Watson, *unpublished*).

Uredospores were treated with a 1.2% aqueous EMS solution in a flask shaker for 2 hr. After filtering and washing, the spores were dispersed in Odorless Mineral Spirit and sprayed on approximately 1,200 seedlings each of Festiguay and W3592. Seedlings of other wheats also were included. After remaining in the misting chamber for about 18 hr, the pots containing the seedlings were spaced out in the glasshouse. After 2 wk, all primary leaves were screened for mutants with increased virulence. The mutants detected along with parent culture 75-L-9 were tested on wheat sets consisting of: (i) the

twelve international differentials, (ii) the eleven Australian supplementals, and (iii) several additional genotypes (3).

### RESULTS AND DISCUSSION

No pustules that exhibited increased virulence on Festiguay (*Sr30*) were detected. Two fully virulent mutants were found on W3592. Both mutants produced identical infection types and each differed from 75-L-9 by two mutations for increased virulence. On W3592 they conditioned infection type "3+" and on Festiguay a semi-resistant "2+3="; the respective infection types of 75-L-9 were "3" and "2=". On Webster (*Sr30*) both mutants produced "3 3+", in contrast to the infection type by 75-L-9 which was "2+". Further comparative pathogenic tests with the two mutants have demonstrated that the increase of virulence on plants with *Sr30* is an intermediate step, similar to those reported for other host-pathogen relationships (7).

The same mutation experiment also yielded three mutants for virulence on Einkorn (*Sr21*), one mutant for virulence on Vernal Emmer (*Sr9e*), and four color mutants (orange-yellow).

The recovery of two double mutants for increased virulence on Festiguay and on W3592 after EMS treatment, suggested that the corresponding genes for pathogenicity on these two genotypes are very closely linked. The existence of three field strains, 21-9, 21-6,9, and 343-1,2,3,5,6,9 all virulent on W3592 but avirulent on Festiguay, nevertheless, indicates that the fungal genes

are distinct. On the other hand, not a single Australian strain avirulent on W3592, can attack Festiguay. It is possible that the gene for avirulence on W3592 is located adjacent to the one controlling avirulence on plants with *Sr30* so that it becomes sensitive to changes taking place in the latter gene.

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