POWDERY MILDEW ON BARLEY

Pathogen variability in South Australia. Resistance genes in cv. Galleon.

by

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ABSTRACT

Powdery mildew disease of barley occurs every year in South Australia. It is occasionally severe when moderately cool and humid conditions prevail during the tillering phase.

Two techniques (mobile nursery and detached leaf) were used to study the pathogen variability of *Erysiphe graminis* DC. ex. Merat f.sp. *hordei* Em. Marchal. The mobile nursery surveys were conducted over five years (1981-1985) to monitor changes in the spectrum of virulence and individual gene frequencies. The same virulence gene spectrum was present in the pathogen populations in several areas of South Australia throughout the period 1981 - 1984. During 1985 one new virulence gene matching the resistance in the commercial cultivar Forrest was detected. The resistance in another cultivar, Galleon, did not 'break down' within the five years although by 1985 its commercial cultivation rose to 40% of the barley area of South Australia.

The race specific resistance genes Ml-a6, Ml-k and Ml-41/145 were found to be susceptible to the pathogen populations in all surveyed areas. The relative frequencies of the matching virulence genes varied only slightly over time and location. The relative frequency for V-k was always almost 100%.

In the mobile nursery tests the resistance gene Ml-v showed a variable reaction (1-3 infection types), in most cases was moderately susceptible. In the controlled conditions of the detached leaf test it gave a type 3 infection indicating that the virulence gene for Ml-v was present in the pathogen populations. Reasons for this variable reaction and the status of the Ml-v gene are discussed. Since only the Ml-k gene has previously been exploited in commercial cultivars grown in South Australia the virulence genes V-a6, V-41/145 and V-v are "unnecessary" to the local pathogen populations; the implications of this are discussed.

The Ml-a, Ml-7, Ml-a9, Ml-a12, Ml-g , Ml-h, Ml-(CP) and ml-o3 mildew resistance genes were found to be resistant in all surveyed areas. Either the matching virulence genes for these were absent, or present, at a very low frequency in the pathogen population which could not be detected by the sampling technique used.

A study was undertaken into the nature of the resistance in the cultivar Galleon to elucidate its apparent durability. The aim was to compare the resistance gene(s) in Galleon with available known genes, and to locate the Galleon gene(s) to a particular chromosome and to determine the linkage relationship with the two structural genes on chromosome 5 that control hordein polypeptides of the seed storage proteins in barley.

Resistance in the cultivar CI-3576, the parental source of Galleon's resistance, was also studied in progenies of cross to a susceptible cultivar. Three independent dominant genes for resistance were indicated.

The F1s, F2s, F3s, and three-way-cross populations from crosses of Galleon with susceptible cultivars showed the resistance to be conditioned by one dominant gene in some plants and by two independent dominant genes in other plants. Thus commercial Galleon is a mixture of genotypes for reaction to E. *graminis* f. sp. *hordei*.

In F2s and three-way-cross populations from a single plant selection of Galleon crossed with other resistant cultivars the gene in Galleon segregated independently from Ml-a, Ml-a7, Ml-a9, Ml-a12, Ml-g and Ml-h. The infection reaction type of the Galleon gene also showed differences from Ml-k, Ml-a6, Ml-41/145, Ml-p, Ml-at, Ml-(CP) and ml-o3.

The resistance gene in Galleon is, tentatively designated as Ml-(ga) and was found to be loosely linked with the Hor-1 locus (C Hordein) at a recombination percentage 35.7 ± 4.46. Nearly independent segregation exists between Hor 2 (B Hordein) and this locus. It was deduced that Ml-(ga) is on chromosome 5 closer to the centromere than the Hor-l locus and close to Ml-p.