Inheritance of Resistance to Race T-1 of *Tilletia caries* in Minturki and Cooperatorka Wheats

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The most satisfactory method of control of bunt (*Tilletia caries* (DC.) Tul. and *T. foetida* (Wallr.) Liro) of wheat is the growing of resistant varieties combined with seed treatment. In breeding resistant varieties a knowledge of the mode of inheritance of resistance to the 31 races which have been identified (16) is desirable. At the present time, no gene has been identified which will control all races. However, it has been shown that a combination of two genes will give protection against at least 25 races (11). As new races are discovered, it may be necessary to find new resistance genes in the host. It is therefore advisable to investigate the genetics of resistance in the presence of individual races of bunt. In this field of study, Briggs and his students have determined the inheritance of the resistance of 16 wheat varieties to race 1 of *T. caries*. Seven different genes for resistance have been identified (1, 2, 3, 4, 5, 6, 7, 8, 13, 17). In this paper the genetics of resistance of two varieties, Minturki, C.I. 6155, and Cooperatorka, C.I. 8861, to race T-1 is reported.

MATERIALS AND METHODS

Minturki was selected from Odessa × Turkey and Cooperatorka is of Crimean origin (12). At the California Station Minturki had shown an average of 0.6% of bunt infection over a 14-year period. Cooperatorka was bunt-free during the 7 years it had been grown. Both are known to be susceptible to some races in the Pacific Northwest (15).

Minturki and Cooperatorka were crossed with the completely susceptible variety Baart and with the resistant varieties Martin, Turkey 3055, Rio, and Selection 1403, which are testers for the *MM, TT, RR, and HH* genes, respectively.

F2 progenies of the crosses with Baart were grown in duplicate rod-rows. The 80 seeds sown in each row had been thoroughly blackened with chlamydospores of race T-1 of *T. caries*. This collection has been perpetuated and used at this station since 1919. F2 progenies of the test-crosses were handled similarly except that they were grown in single rod-rows. Parent checks and small F2 populations were included.

The plants were pulled when nearly mature and classified as diseased or not diseased.

RESULTS FROM CROSSES INVOLVING MINTURKI

The distributions of parental plots and F2 progenies by 5% infection classes for the 1949 trials are given in table 1. The range of infection in Baart, the susceptible variety, was from 53.2% to 74.9% with an average of 64.4%, a considerably lower value than the 16-year average of 73.9%.

The average infection of Minturki in 1949 was 20.7% with a range of 3.8% to 38.9%. Minturki had been highly resistant at Davis with a previous high of 3.2% infection. Thus, there was an apparent sudden break in resistance. There are three possible explanations: eliminating races, mutation in Minturki, or environmental interaction in the 1948-49 season. The first two possibilities were ruled out by return to normal Minturki in 1950. The third possibility relates to the physiology of the plants was disturbed by the conditions occurring in 1948-49 in such a manner that there was a breakdown in the resistance mechanism. Considerable frost injury was noted in the nursery; this was not particularly favorable for bunt, as the low level of infection in Baart; therefore, it appeared to be operating against the Minturki type of resistance. Environmental effects on the pathogen-host relationships have been reviewed by Tapke (1).

Segregation of susceptible or heterozygous plants from crosses with the testers showed that the *HH* genes were definitely not present in Minturki (table 1). Previous studies (Briggs, unpublished) conclusively that *MM* was not present.

There were no definite minima or maxima on the distribution curve of Minturki × Baart (fig. 1) which could be interpreted as distinguishing genotypes. Since there appeared to be a very small proportion of progenies in the class representing the most resistant Minturki, it was concluded that resistance is probably due to the action of two or more genes with recombination. El Khishen and Briggs (13) and Baker (5) have shown that the cumulative effect of two weak genes can produce a high level of resistance.

The type of distribution curve observed was similar to that of a small sample of *Tilletia* spores in the population of susceptible plants. Expected mean infection percentages from F3 genotypes were assigned as shown, i.e., the center of the modal class of Minturki plots was 17.5% was designated for the Minturki genotypes, and the value 67.5%, based on infection in Baart, was assigned to the susceptible genotype, *mmvv*. The gene *H* homozygous condition was given the value 4.3%.