Inheritance of Black Point Resistance in Durum Wheat

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ABSTRACT

The green-kernel test was developed and used to test segregating populations of reciprocal Leeds × Golden Ball (durum wheat) crosses for reaction to *Helminthosporium sativum*. Progeny of the crosses did not fit the expected Mendelian ratios. Analyses of variance of differences in disease ratings within cultivars, between cultivars, and within segregating progeny of reciprocal crosses were calculated. The genetic variances of F_2 plants, F_3 families, backcross-F_1 plants, and backcross-F_2 families were significantly greater than the genetic variance within the parents. Disease ratings equal to those of the resistant parent, Leeds, were recovered in all segregating populations, indicating that selections for resistance to black point could be made in segregating populations.

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Additional key words: heritability, genetic variance, *Helminthosporium sativum*.

The importance of the black point disease of cereals is reflected in the degradation of grain quality due to kernel discoloration (1). Infected kernels are typified by a dark discoloration of the embryo region and the crease or suture of the kernel. Heavily infected seeds are discolored, shrivelled and of inferior quality (2). The disease also results in decreased kernel weight (6). Although species of *Alternaria* and *Fusarium* have been associated with the disease, *Helminthosporium sativum,* Pammel, King & Bakke (Imperfect stage of *Cochliobolus sativus* Ito & Kurib.) is usually the most prevalent organism associated with the disease (2, 6).

Environmental factors such as humidity and temperature are important in disease development (1, 4, 6). However, the inheritance of resistance has not been well elucidated. Differences in host susceptibility have been reported; the durums are generally more susceptible than hard red spring wheats (7). Difference in response to black point have also been noted among durum varieties (7). Small kernels have been related to resistance but are undesirable in durum wheat (3). In 1925, Griffee (5) reported that at least three genes were involved in resistance to head blight of barley caused by *H. sativum*.

In prior experiments (1, 6) plants were inoculated by either spraying inoculum onto the heads or dipping the heads in an inoculum suspension and incubating at high humidity. In dipping and bagging, spike maturity, inoculum density, and exposure time to high humidity must be controlled accurately for reliable results. In this study, the green-kernel test was developed as a rapid and reliable method of measuring resistance to *H. sativum* in studies of the inheritance of resistance to black point in Leeds durum wheat.

MATERIALS AND METHODS.—Green kernels (berry, caryopsis) of durum cultivars, *Triticum durum* Desf., were excised in the early soft dough stage of development. The stage of maturity of the kernels is critical since kernels inoculated prior to the early soft dough stage incorrectly may be rated susceptible, and those inoculated after the green color has disappeared may be rated incorrectly as resistant. Kernels were placed crease up on moistened filter paper in petri dishes. One drop of the spore suspension, consisting of 10,000 spores/ml in a 0.1% aqueous solution of Tween 20, was placed on the crease of each kernel. Eight drops of 25% lactic acid were added to 1,000 ml of the spore suspension to retard bacterial growth. Kernels were incubated in petri dishes enclosed in aluminum foil for 3 to 5 days at room temperature (21 C).

Kernels were evaluated for damage and discoloration on a scale of 1-5. Resistant: 1 = No visible symptoms, 2 = Embryo tip of kernel slightly discolored; Susceptible: 3 = Embryo tip of kernel severely discolored; crease may show slight discoloration, 4 = Embryo tip of kernel severely discolored; crease with moderate discoloration, 5 = Kernel discolored and damaged along the entire crease.

This method of evaluating durum for reaction to *H. sativum* is hereafter referred to as the "green-kernel" test.

Leeds (C.I. 13768) and Golden Ball (C.I. 6227) were selected as resistant and susceptible parents, respectively. Seed from single heads of each variety were planted, reciprocal crosses were made, and F_1 progeny of these crosses were backcrossed to Leeds. Green kernels of the parents, F_1, F_2, F_3, Backcross-F_1 (BC-F_1) and Backcross-F_2 (BC-F_2) progeny were analyzed for their reaction to *H. sativum*.

A chi-square (χ^2) test for goodness of fit was used to analyze segregating populations. Kernels were classified as resistant (1-2) or susceptible (3-5) for the χ^2 analysis. An analysis of variance with unequal subclass numbers was used to analyze disease ratings of individual kernels rated on a 1-5 scale. These analyses were used to determine if significant differences existed in disease ratings within varieties, between varieties, and within segregating progeny of reciprocal crosses of Leeds × Golden Ball. Variance components were calculated from the analysis of variance to compare genetic variation of the parents to that of segregating populations.

Coefficient of variability values were computed to compare the precision of estimates of the green-kernel test in evaluating genetic variation.

RESULTS AND DISCUSSION.—The cultivar, Leeds, was resistant (average rating 1.8) while Golden Ball was susceptible (average rating 2.9) to *H. sativum*.

F_1 kernels from reciprocal crosses were mostly susceptible (average rating 3.2). Therefore, it was assumed that recessive gene(s) conditioned resistance to *H. sativum*.

TABLE 1. Variance components of black point evaluations of parents and progeny of reciprocal Leeds × Golden Ball durum wheat crosses

Cultivar or line	$\hat{\sigma}g^2$ [a]	$\hat{\sigma}sg^2/\hat{\sigma}p$ [b]	F-test[c]
Leeds (L)	.0709		
Golden Ball (GB)	.1724		
L and GB combined	.1296		**[e]
F_1 (L × GB and GB × L)	.1459		
F_2 L × GB	.7496		
F_2 GB × L	.2067		
F_2 combined	.5035	**	
F_3 L × GB	.2778		
F_3 GB × L	.3243		
F_3 combined	.3488	**	
BC-F_1 L × (L × GB)	.3417		
BC-F_1 L × (GB × L)	.1877		
BC-F_1 combined	.3871	*[d]	
BC-F_2 L × (L × GB)	.3081		
BC-F_2 L × (GB × L)	.0819		
BC-F_2 combined	.2119	**	

[a]Genetic variance component estimate within parental and segregating population.
[b]Genetic variance of segregating population tested by the combined parental genetic variance.
[c]Testing differences between parent and reciprocal populations.
[d]*Significant $P = 0.05$.
[e]**Significant $P = 0.01$.

One hundred sixty nine F_2 kernels from the Leeds × Golden Ball cross and 295 kernels from the reciprocal were tested for resistance to *H. sativum* using the green-kernel test. Kernels from the Leeds × Golden Ball cross segregated 40 resistant to 129 susceptible and the reciprocal segregated 44 resistant to 251 susceptible. Although several families fit a monogenic recessive gene hypothesis, χ^2 tests for heterogeneity indicated that the families were not homogeneous and should not be combined for the genetic analysis. If three families from the Leeds × Golden Ball cross and six from the reciprocal were omitted from the analysis the families were homogeneous, but the combined data did not fit a monogenic recessive gene hypothesis. Families segregating heterogeneously for reaction to *H. sativum* indicate that the parental lines may have been heterozygous. An occasional susceptible kernel in Leeds and resistant kernels in Golden Ball gave further evidence of heterozygosity.

One hundred seventy seven F_3 families consisting of 15-25 kernels each were inoculated with *H. sativum*. F_3 families of the Leeds × Golden Ball cross segregated into three classes; 29 resistant, 40 segregating to 19 susceptible. The families fit a 1:2:1 ratio for a monogenic recessive gene pair but approximately half the families in the segregating class did not fit a one resistant to three susceptible ratio. F_3 families from the Golden Ball × Leeds cross segregated 15 resistant, 63 segregating to 11 susceptible and did not fit the 1:2:1 ratio for a monogenic recessive gene pair. The indication of a monogenic gene pair but the failure of segregrating populations to fit hypothesized ratios could be also explained by heterozygous parents.

BC-F_1 kernels from the cross Leeds × (Leeds × Golden Ball) segregated 34 resistant to 36 susceptible and satisfactorily fit a monogenic recessive gene ratio ($P > .75$). BC-F_2 families from this cross segregated 26 resistant to 40 segregating, but approximately half the families in the segregating class did not segregate one resistant to three susceptible. BC-F_1 kernels from the Leeds × (Golden Ball × Leeds) cross segregated 11 resistant to 45 susceptible and did not fit a monogenic recessive ratio. BC-F_2 families segregated 20 resistant to 43 segregating and did not fit monogenic recessive ratios.

Since progeny from reciprocal crosses did not fit expected Mendelian ratios, an analysis of variance was used to determine if there were significant differences in disease ratings within parents, between parents and within segregating populations. Variance components were calculated to estimate the genetic variability of parents compared to variability of segregating progeny from the crosses, and to determine if resistance was heritable and selectable.

Differences in disease ratings were detected between the two parental cultivars Leeds and Golden Ball. No significant differences were detected between reciprocal F_2, F_3, BC-F_1, or BC-F_2 populations, thus no evidence for maternal influence was detected (Table 1).

Significant genetic variation within each of the parental cultivars and segregating populations was detected from their analyses of variance. These genetic differences within the parents were a probable cause of our inability to fit the progeny to qualitative genetic ratios.

The genetic variance of the F_2, F_3, BC-F_1 and BC-F_2 generations significantly exceeded the genetic variance within the parental cultivars. Thus, genetic segregation for black point resistance and susceptibility in the segregating generations was apparently sufficient to allow effective selection of desired types since plants with disease ratings equal to the resistant parent, Leeds, were recovered.

The green-kernel test allowed adequate precision and accuracy in testing for black point. The coefficient of variability of the test ranged from 14% for the F_2 combined data, to a high of 26% for the F_3 of the Golden Ball × Leeds cross.

Statistical significance of differences among plants was readily obtained which indicated less variability among seeds of a plant (a direct influence of the test) than among plants. As in much biological material, an association between the mean disease rating and the error mean square values was observed.

Testing segregating progeny of a Leeds × Golden Ball cross with *H. sativum* using the green berry test, indicated that a single recessive gene conditioned resistance to black point. Since the segregating progenies did not fit hypothesized Mendelian ratios, the segregating populations were analyzed using variance components. Adequate genetic variance was present in the segregating populations to select resistant types. The statistical analyses indicated that Leeds and Golden Ball were heterogeneous for disease reaction to black point which could explain the failure of segregating populations to fit hypothesized ratios.

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