

Although heterosis in yield and other traits is evident from the data, we cannot be certain whether the mutant gene causing the necrotic leaf spot is solely responsible or whether the thermal neutron seed treatment caused other mutations that contribute to heterosis.

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1. Role of β -glucosidase in monogenic resistance to *Helminthosporium turcicum* in maize.

β -glucosidase catalyzes the hydrolysis of phenolic glucosides to their corresponding aglycones. These free phenolics, which are produced after cellular disruption, are highly toxic and are believed to play a role in disease resistance. In maize, the production of the cyclic hydroxamic acid DIMBOA (2,4-dihydroxy-7-methoxy-1,4-benzoxazine-3-one) from its glucoside upon cellular disruption has been recently implicated in the resistant mechanism.

We have developed a rapid fluorometric procedure for the assay of β -glucosidase. Preliminary data indicate that the susceptible genotype (htht) has a significantly higher level of β -glucosidase than the resistant genotype (HtHt). The enzyme (which has been shown to be localized primarily in the cell wall) causes the formation of the fungitoxic aglycone as a result of its mixing with DIMBOA-glucoside during cellular disruption. The toxic DIMBOA may contain the fungus until phytoalexin is produced at which point the differential genotypic reaction would begin.

It has been previously described, and observed in our study, that the infection flecks on the resistant genotype may occur as much as 15 hours earlier than in the susceptible genotype. Since apparently no phytoalexin is being produced at this early stage, it is our contention that the lower β -glucosidase levels in the resistant genotype may account for the earlier disease reaction.

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