2. New evidence of heterosis in crosses between an induced mutant "Necrotic leaf spot" N25 and normal N25 inbred lines.

In an earlier report, Hornbrook and Gardner (Radiation Botany 10: 113-117, 1970) found evidence that when an induced mutant of inbred N25, which caused necrotic blotches to appear on the leaves and reduced yield, was crossed to normal N25, the F_1 hybrid outyielded the normal parent. Reviewers of our original manuscript did not believe that our data were sufficiently conclusive to justify our conclusion so more extensive tests were planned. In 1970, the mutant line, the normal parent and the F_1 hybrid were grown in 20 replications. Ten replications had a plant population density of 51,666 plants per hectare and the other 10 had 68,888 plants per hectare.

Results indicated that maximum yields were obtained at the lower rate of planting, that there is no evidence of genotype--plant population density interaction and that the F_1 hybrid does indeed show significant heterosis above the normal parent. Means are presented in Table 1.

Table 1

Mean values of agronomic traits measured on mutant and normal lines of N25 and their F_1 hybrid

Genotype	Means of traits measured							
	Grain yield	Ear length	Ear diameter	Ears/plant	Barren plants %	Plant ht.	Ear ht.	Days to flower
	(q/ha)	(cm)	(cm)			(cm)	(cm)	
Normal N25	25.6	15.7	3.70	0.80	20.2	186	46	74.9
Mutant N25	19.0	13.8	3.51	0.80	19.8	190	54	76.0
F_1 hybrid	36.2	16.5	3.84	0.95	4.9	187	50	74.6

The hybrid outyielded the better parent by over 40%, had larger ears, more ears, and fewer barren plants. The hybrid is intermediate in plant and ear height and equal to the earlier parent in earliness of flowering.

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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