the resistant inbred Pa 32 crossed with the susceptible parents W153R and Pa 33 also clearly segregated in a ratio of 3 resistant to 1 susceptible and 1:1, respectively. These data strongly support the hypothesis for a monogenic dominant control of resistance to strain A in the maize inbred lines Pa 11, Pa 405, and Pa 32. Additional studies are being conducted with the resistant inbred lines Oh 7B, Pa 422P and Pa 884P. At present the exact nature of gene control for these is not clear.

Recent data support the hypothesis of 3 genes with complementary action controlling resistance to strain B in the resistant inbreds Pa 32 and Pa 422P. The data (Table 1) from F₂ population of Pa 32 with susceptible lines Pa 881P, WF9 and Pa 54 indicate a good fit for a ratio of 27 resistant to 37 susceptible. Another inbred, Pa 422P, also appears to have 3 genes with complementary action for resistance to strain B.

Continued tests are underway to determine: 1. the complexity of inheritance to the control of resistance to maize dwarf mosaic virus strain A and strain B and 2. the relationship, if any, between these two genetic systems controlling resistance to MDMV in a number of resistant maize inbreds.

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1. Genetic control of phytoglycogen accumulation.

Investigations on the control of phytoglycogen accumulation in maize endosperm by the genes <u>ae</u> and <u>su</u>₁ were accomplished by quantitating the phytoglycogen from the 16 genotypes resulting from all possible crosses between <u>normal</u>, <u>ae</u>, <u>su</u>₁ and <u>ae</u> <u>su</u>₁. Background effects were minimized by having <u>normal</u> and the mutants in the BC-2 generation of the inbred W64A. The phytoglycogen was extracted in HgCl₂ from 20 day old kernels and was quantitated by using glucoamylase from <u>Aspergillus niger</u>

to hydrolyze the polysaccharide to glucose. The glucose was then measured as a reducing sugar. The results of different dosage levels of \underline{ae} with homozygous \underline{su}_1 are given below:

Genot	type	Phytoglycogen in mg/g dry wt. + std. dev.	Absorbancy Maxima (mu)	β-amylolysis limit (%)
+ + <u>ae</u>	su1 su1 su1 su1 su1 su1 su1 su1 su1 su1	$\frac{\mathbf{su}_{1}}{\mathbf{su}_{1}} \qquad \frac{343.4 \pm 7.1}{232.9 \pm 13.6}$	475 475 475 475	40.9 40.2 41.9 45.7

Only the endosperms homozygous for \underline{su}_1 contained phytoglycogen. Increasing doses of \underline{ae} decreased the amounts of phytoglycogen. The double mutant (\underline{ae} \underline{ae} \underline{su}_1 \underline{su}_1 \underline{su}_1) contained phytoglycogen in contrast to an earlier report from this laboratory (Black \underline{et} \underline{al} . 1966, Genetics 53:661-668); however, they were using a different and more heterogeneous genetic background.

Absorbancy maxima in an iodine-potassium iodide and saturated calcium chloride solution indicated the phytoglycogens from each of the genotypes were identical. However, the β -amylolysis limit of the double mutant was higher than the others, suggesting that it may be a more loosely branched phytoglycogen.

Studies are in progress to analyze the starches from these genotypes with regard to the ratio of amylose and amylopectin and the structure of the amylopectin. Studies are planned to survey the genotypes for branching and debranching enzymes.

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2. Phenotypic dosage effects exhibited by Ae in combination with wx.

It has been observed in this laboratory that Ae exhibits a dosage effect which reflects the genotype of the endosperm. Ae Ae Ae wx wx wx and Ae Ae ae wx wx wx endosperms are full and waxy in phenotype, the two genotypes being indistinguishable. However, Ae ae ae wx wx wx endosperms are tarnished waxy and appear to be smaller in size. The phenotype of