in neutral CsCl density gradients. A $Cot_{\frac{1}{2}}$ of 4×10^{-2} $M \cdot s \cdot l^{-1}$ was measured from renaturation kinetics. This value indicated the presence of repeated sequences in the GC-rich fraction. However, this rate is too slow for a single repeating unit of 200 bases. Thus, the GC-rich fraction represents either a family of repeated sequences or fragments of a larger, less-repeated sequence. Studies are continuing to distinguish these possibilities and to determine the length and distribution of these sequences.

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Glucosidase in maize seedlings

(a) Inheritance of electrophoretic variants. Glucosidase bands in starch gels (pH7.8 Tris-citrate) were developed using a 6 bromo-2 naphthyl β-D glucoside and Fast Garnet GBC salt. Most inbred lines contain a single isozyme band, but in 2 of the 40 lines assayed (P10 and P11) a faster band was observed. Hybrids between variants have 3 isozyme bands, the additional band being a hybrid isozyme of intermediate migration. The segregation of the isozyme variants in F_2 and BC progeny is consistent with the hypothesis that the isozymes are determined by alleles (Table 1). We propose to call this gene glucosidase and the alleles Glu-A and Glu-C. A third variant, Glu-B, has recently been observed.

Table 1. Inheritance of glucosidase isozyme variants. C and A represent lines breeding true for the isozyme variants. χ^2 was calculated on the Null hypothesis that C and A are determined by alleles.

	Number of Progeny					
Cross	C	C/A	A	Σ	χ^2	P
C/A self	5	10	6	21	.04	>.05
C/A x C	9	10	-	19	.05	>.05
C/A x A	-	9	8	17	.05	>.05

(b) Null mutants at the Glu locus. Glucosidase activity has been implicated in the mechanism of plant disease resistance by mediation of the release of pathogen-toxic materials from inactive glycosidic precursors (Pridham, J. B., 1960, Phenolics in Plants in Health and Disease, Pergamon Press, New York). For the glucosidase determined by the gene Glu this supposed role can be tested by determining whether resistance is still expressed in a plant homozygous for a null mutation of the Glu gene.

Mutants were obtained by EMS treatment of seed homozygous for Glu-A, Gdh-F and Adh-S. During the latter half of the study seedling roots were immersed in water for 12 hours prior to electrophoresis to induce the Adh2 gene and allow scoring at this locus. By using electrophoretically recognizable pollen (Glu-C Gdh-N Adh-F) we should have been able to recognize a variety of mutant types at the 4 loci. However, only one class of apparent mutation was observed (Table 2). These ears carried sectors of seed containing only the pollen parent isozyme for glucosidase C. Plants from 17 of the 33 apparent mutants were selfed. In all cases the F_2 progeny segregated, having either the pollen parent isozyme or no glucosidase activity (Table 3), and in most cases where numbers were sufficient the segregation ratio did not differ significantly from a 3:1 ratio. Thus, absence of the female parent isozyme segregates as an allele of the glucosidase gene. Similarly in crosses Glu-A/Glu-C x Glu-C/mutant, progeny occurred in the ratio 1A:1AC:2C. The homozygous nulls obtained from the F_2 progeny breed true.

Table 2. Summary of ears screened for EMS mutants.

Expt. 1*	Glu	Gdh	Adh	Adh-2
Total No. Ears	38	34	33	16
Ears Mutant	5	0	0	0
%	13			
Expt. 2*				
Total No. Ears	149	143	155	83
Ears Mutant	28	0	0	0
%	19			

*EMS Concentration: Experiment 1, 0.16 M
Experiment 2, 0.08 M

Table 3. Segregation of glucosidase isozymes in selfed progeny of plants from 'mutant' sectors.

Mutant No.	C	Null	Σ
G1	12	5	17
G2	18	4	22
G3	12	4	16
G5	11	4	15
G6	16	6	22
G8	12	5	17
G9	10	6	16
G10	15	7	22
G12	11	3	14
G13	14	7	21
G15	9	6	15
G18	4	2	6
G24	9	5	14
G25	14	5	19
G28	9	8	17
G31	3	5	8
G33	8	5	13

Complementation studies between the different nulls are in progress, and in several F_1s so far examined there was no evidence of activity. Thus, by all criteria the null mutants behave as true alleles of the glucosidase gene. However, the high frequency with which they occurred (18% of tested ears) and the absence of other mutant types at this or the other three loci tested leave open some important questions. Further work is aimed at determining the true nature and origin of these nulls.

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