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1. Cytoplasmic male sterility research: M_2 generation from streptomycin treatments.

Streptomycin was used in an attempt to induce cytoplasmic male sterility in maize (Briggs, 1973). In this research streptomycin was used in concentrations of .001, .005, .01, .05, .10, .150% and a control. Seeds of an inbred line of corn were germinated for 30 hours at 27°C; at the end of this time some radicles had emerged. Subsets of experiments were performed; in one set the germinated seeds were placed embryo down in Petri dishes on Kimpak that was saturated with the streptomycin solution. In the second set germinated seeds were completely submerged in flasks of the streptomycin solution. In another experiment dry seeds (ungerminated) were placed embryo down in Petri dishes on Kimpak that was saturated with the streptomycin solution. All these experiments were conducted for 24 hours at 25°C. Briggs (1973) can be consulted for further details on this research.

Plants from the streptomycin treated seeds were self-pollinated in the M_1 generation and good seed set was obtained from most plants in the treatments. The material was self-pollinated in order to eliminate any sterile plants that may have been in the population which could have arisen spontaneously or by seed mixtures. Seed from the

self-pollinated M_1 ears was planted out ear-to-row in the M_2 generation.

If a cell were mutated to cytoplasmic male sterility in the M_1 generation and if this cell along with nonmutated cells made up the ear (female inflorescence), a chimeric situation would exist and if this material were planted in the next generation, (M_2), male sterile and male fertile plants would be detected. These two types of plants would be found in a ratio which would be dependent on the ratio that the mutated and nonmutated cells made up the ear. If the cell that was mutated to cytoplasmic male sterility went to make up the tassel (male inflorescence), this male sterile sector would not produce pollen; hence it would not be transmissible and would be lost.

Data from these experiments are in Table 1. The sterile plants found in the M_2 generation were sib-pollinated by fertile plants from the same row.

Table 1

Data from M_2 generation of streptomycin treated seeds

Type of streptomycin seed treatment (see text)

	Germinated				Germinated submerged				Dry			
	1	2	3	4	1	2	3	4	1	2	3	4
Control	0				0				0			
.001%	1	0	.0005	1.5	0				14	9	.50-.60	21.9
.005%	0				0				0			
.01%	0				18	10	.50-.60	28.1	0			
.05%	16	3	.999	25.0	0				0			
.10%	11	11	.10-.20	17.2	13	13	.30-.40	20.3	0			
.150%	0				0				0			

1 = No. sterile plants out of 64.

2 = No. of ears with seed.

3 = P value from X^2 (3:1 ratio)

4 = Percentage of sterile plants.

Stein and Steffensen (1959) and Steffensen (1968) indicated that there were 5 to 6 cells in the apical meristem of maize seeds. In the paper by Stein and Steffensen (1959) this was determined by irradiation of seeds where they found a sector that included about 18% of the leaf. More extensive sector data and cell number counts were presented by Steffensen (1968).

The ear is borne at the axil of a leaf; hence it might be assumed that, ontogenetically speaking, the ear and leaf arise similarly from the meristem of the seed. Therefore, if these 5 to 6 cells give rise to the leaves in the upper part of the plant, then a similar situation could also exist for the ear and 16.6 to 20.0% (for 6 and 5 cells, respectively) of the seeds on an ear would carry mutations for cytoplasmic male sterility. This assumes that one cell is mutated in the seed and the seeds from the M_1 ear are properly sampled and planted in the M_2 generation. If seed were heterozygous for a recessive gene for male sterility that arose spontaneously and the plant were self-pollinated or if the streptomycin induced a recessive gene for male sterility, in both instances the male sterile plants would occur in 25% (3:1) of the plants in the M_2 generation. In this research as reported no male sterile plants or plants with male sterile sectors were noted in the M_1 generation in any of the treatments, but the seed that was treated with streptomycin was from a lot that had been sib-pollinated and could carry genes for male sterility in the heterozygous condition.

The data in Table 1 give the probability values from the X^2 test. The cases that have P values of .999 and the two that have P values of .50-.60 are the best fits to a 3:1 ratio. However, the P values of .10-.20 and .30-.40 with 17.2% and 20.3%, respectively, do not give as good a fit to a 3:1 ratio as those previously discussed, but do fit rather closely to the 16.6% and 20.0% that might be obtained if one cell in the M_1 generation had mutated to cytoplasmic male sterility. Sixty-four seeds were planted and this is taken as the number of plants. Even though the field stands were good, a reduction in stand could modify these results. The treatment with one sterile plant (P = .0005) does not fit the above hypothesis, but could be easiest explained by poor sampling. However, since no seed set was obtained it will not be

possible to determine whether it was a genetic or cytoplasmic male sterile or a sterile caused by something not readily explained. These explanations of the data do not take into account any restorer genes that might be present in the material.

However, to determine if these sterile plants are genetic male steriles or cytoplasmic male steriles they will be planted out in this next generation ear-to-row.

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1. Nine maize peroxidase loci and their tissue specificities.

We reported previously (1972 MNL 46:29-33) on seven loci governing peroxidase polymorphisms in corn. Two additional loci have been identified and will be reported here, together with observations on the tissue specificities of these and other peroxidases in maize.

Two new peroxidases have also been localized on vertical and horizontal gels since our 1972 report, bringing to a total of 13 the number of peroxidases we have identified. The approximate order of these enzymes is indicated below as they appear on horizontal 7% acrylamide gels at pH 8.1 (O = origin):

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 -                     O                               +
 1    5     4    7     8    B 3  E    6 D       2 9 A
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Enzymes are currently given numbers in the order in which loci controlling their genetic polymorphisms are recognized, while enzymes designated by alphabetic symbols have not yet shown genetic polymorphisms in our