



MAIZE GENETICS COOPERATION

NEWS LETTER

56

March 15, 1982

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and
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Some sources of general information on maize genetics and cytogenetics:

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- The Mutants of Maize. M. G. Neuffer, L. M. Jones and M. S. Zuber, Crop Sci. Soc. Am., Madison, Wisc., 1968.
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- Evolution of Crop Plants, Chap. 37, pp. 128-136. N. W. Simmonds, ed., Longman, N.Y., 1976.
- Corn and Corn Improvement, 2d edition, G. F. Sprague, ed., Amer. Soc. Agron., 1977.
- Maize Breeding and Genetics. D. B. Walden, ed., Wiley, N.Y., 1978.
- Maize Research and Breeders Manual No. IX. C. B. Henderson, Illinois Foundation Seeds, Inc., Box 722, Champaign, Illinois 61820, 1980.
- Quantitative Genetics in Maize Breeding. A. Hallauer and J. B. Miranda, Iowa State Univ. Press, Ames, 1981.
- Maize. E. Häfliger, ed., CIBA-GEIGY Monograph, Basle, Switzerland, 1979.

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I. FOREWORD

Fifty years ago a communication dated October 5, 1932, signed by "M. M. Rhoades, Sec'y," transmitted his "Report of a meeting held during the Genetics Congress on August 26th by those interested in corn genetics." The meeting was attended by 45 persons, and a committee was formed (Brink, Jones, Mangelsdorf, Stadler, and Emerson, chairman) to establish a "central seed repository." Rhoades was designated custodian of the stocks, which were to be maintained by 10 cooperators (Emerson, Beadle, Brink, Jones, Burnham, Stadler, Jenkins, Sprague, Eyster, Lindstrom), who would grow the stocks and supply fresh seeds for the repository. Of course this was the formalization of an already enthusiastic cooperation among scientists in maize genetics that is still intact today. It established the base for a stock center and the impetus for the News Letter, but it was the openness, mutual respect and cordiality that made progress so rapid (one wag has called it a husky, eerie zeal).

How can we honor that spirit and further the exploration of the genetic systems of *Zea* and our ability to manipulate them? In addition to continuing to openly exchange and share information and materials, we can take account of the research of our colleagues in publications and cite their pertinent work; we can acknowledge seed stocks and information supplied by others or by the Stock Center; we can tell other scientists of our colleagues' findings; we can carry out the linkage experiments, the allelism tests and the tests of alternative hypotheses that are essential to further progress. In short, we can continue with the same enthusiasm.

Is this News Letter a Primary Reference? It seems at times to be misunderstood as such. Citations from the News Letter in publications of course should only be by permission of the writer of the item, but it might also be a good idea to cite the information as a "personal communication" (along with the issue and pages). Misunderstanding of the function of the News Letter is also indicated, according to experiences of some authors, by comments of referees to the effect that the paper under review belongs in the News Letter. In refereed publications, primary publication of new genetic variations and techniques and theories is regular for most species, and maize ought not to be different.

We note with enthusiasm the birth of related newsletters. The Plant Molecular Biology Newsletter is entering its third year, with a burst of maize enthusiasm (see information near the back of this issue). The Plant Genetics Newsletter, issuing from the new Genetics Section of the Botanical Society of America, will contain meeting notes, book reviews, topical articles, employment news, etc. (Dr. Dennis M. Travis, University of Maine, Presque Isle, Maine 04769, is the editor). A corn breeding newsletter has been proposed and is under consideration.

Some new publications of interest:

- Hallauer, A., and J. B. Miranda, 1981. Quantitative Genetics in Maize Breeding. Iowa State Univ. Press, Ames. 468 pp.
- Häfliger, E., editor, 1979. Maize. CIBA-GEIGY Monograph, Basle, Switzerland.
- Micu, V. E., 1981. Genetical Studies of Maize. Shtiintsa, Kishinev, Mold. S.S.R. 231 pp. (in Russian).

If you teach a laboratory, a number of interesting genetics laboratory experiments with corn, soybeans and peas (including exercises with the anthocyanin system of corn) are given in the catalog and manual of Williams Laboratories (P.O. Box 43, Williams, Indiana 47470).

About 900 copies of this issue will be sent to research workers, laboratories and libraries around the world. The costs of preparation, reproduction and mailing as well as office support are borne by the U. S. Department of Agriculture in facilities provided by the University of Missouri. We are all grateful for this indispensable support and encouragement.

A few copies are still available of the wall-size reproduction of the linkage map, with locus names, made available through the efforts and generosity of William F. Sheridan. Copies will be sent on request to Coe at the University of Missouri.

Back issues of News Letter No. 30 (1956) to date will be sent upon request; a microfilm of volumes 1-29 and 33 is available for \$9.50 U.S.; orders should be sent to Coe at the University of Missouri and checks should be made out to Maize Genetics.

Airmail service to addresses outside the U.S. will be provided for \$3.00 if received by January 1st.

The deadline for the next issue (number 57, 1983) is January 1, 1983. Reports submitted normally should consist of information bearing on genetic understanding or genetic manipulation of maize. Brief items containing specific data, specific observations, and specific methods are of most value. Communications are received and assembled with minimum editing.

Because the text will be retyped, it is more important that it be double-spaced and follow the simple format (to shorten preparation and typing time), and that it be accurate, than that it be spotless copy.

Because tables and figures are usually reproduced "as is," it is most important that they be compact and single spaced, accurate and ready for the camera.

References should be used sparingly; when needed, they should be identified in abbreviated form within the text, including authors' initials to facilitate indexing.

The encouragement and support of my colleagues in planning and preparing this News Letter is most appreciated. Ming-Tang Chang, Christine Curtis, Rodney Higgins, David Hoisington, Bryan Kindiger, Stephen Modena, and Scott Poethig helped with proofing and editing of parts; Kathryn Kind and Christopher Browne with mockup and various other tasks. Shirley Kowaleski has again contributed to the year-round office load and the compilation of the publications list. Mary Nelson again has composed, guarded accuracy and clarity, and has produced final copy with earnestness and spirit.

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II. REPORTS FROM COOPERATORS

AMES, IOWA
Iowa State University

The effects of temperature on striping in Mu plants

In last year's News Letter (MGCNL 55:2-3, 1981) I presented a rather lengthy account of the striping phenomenon observed in Mu lines on some occasions. This phenomenon was found only rarely in plants grown in Iowa, Hawaii and Davis, California but was observed frequently in Mike Freeling's nursery in Berkeley, California. In last year's report, I suggested that the Berkeley climatic conditions might account for the striping. On returning to Iowa State University last spring, I looked up the average minimum temperature and average maximum temperature for Berkeley during the growing period. These turned out to be about 55 F and 70 F respectively.

In an attempt to duplicate these conditions, I grew both mutator and standard lines in a growth chamber on a 12-hour dark 12-hour light cycle with a 55 F dark temperature and a 70 F light temperature. These conditions, of course, fall considerably short of duplicating Berkeley's growing conditions. For one thing the light-70 F period should have been considerably longer and the dark-55 F period shorter. In Berkeley, of course, the temperature does not drop down to a constant 55 F as soon as the sun sets or go up to a constant 70 F as soon as the sun rises. Also, it is not possible to program in the periods of time when the temperatures in Berkeley fail to reach or exceed both the average maximum and minimum temperatures. All things considered, I would guess the growing conditions in the growth chamber are more adverse than those experienced in Berkeley. Plants grow very slowly under these conditions and frequently are observed to have pale yellow leaf tips.

We did indeed get striping in mutator plants under these conditions. However, the same striping pattern is found in standard lines as well. Several different plant virologists, shown these plants (both Mu and standard), have indicated that the striping is similar to that found in plants infected with viruses.

The cool nighttime temperatures seem to be necessary for the striping response. Plants grown at 70 F night and day do not show this striping.

Donald S. Robertson

The effects of ultraviolet light (U.V.) or ethylmethanesulfonate (EMS) mutagenesis on Mu

To determine if synergistic interaction occurred between Mu and ultraviolet light the pollen of both Mu plants and control plants was irradiated for 0', 30', 35', 40', and 45' and then applied to the silks of standard plants. The frequency of plants in these outcross progeny that segregated for seedling mutants equals the mutation frequency.

To determine if U.V. had a synergistic effect at each U.V. dose, the mutation frequency for standard plants at each U.V. dose was calculated. The number of plants tested in the 0' Mu population was multiplied by the mutation frequency determined for irradiated standards for each U.V. dose to determine how many U.V. induced mutant bearing plants would be expected in the 0' Mu population if it had been irradiated by that particular dosage of U.V. Thus, a calculated mutation frequency was determined for the combined effect of Mu and U.V. for each dosage based on an additive (non-synergistic) model. If a synergistic effect is occurring for any particular dose of U.V. then the observed mutation frequency in irradiated Mu outcrosses should be greater than the calculated Mu value for a given U.V. dose.

Thus, for each dose, the ratio of observed to calculated mutation frequency was used to calculate synergism. The results of these calculations are plotted in Figure 1.

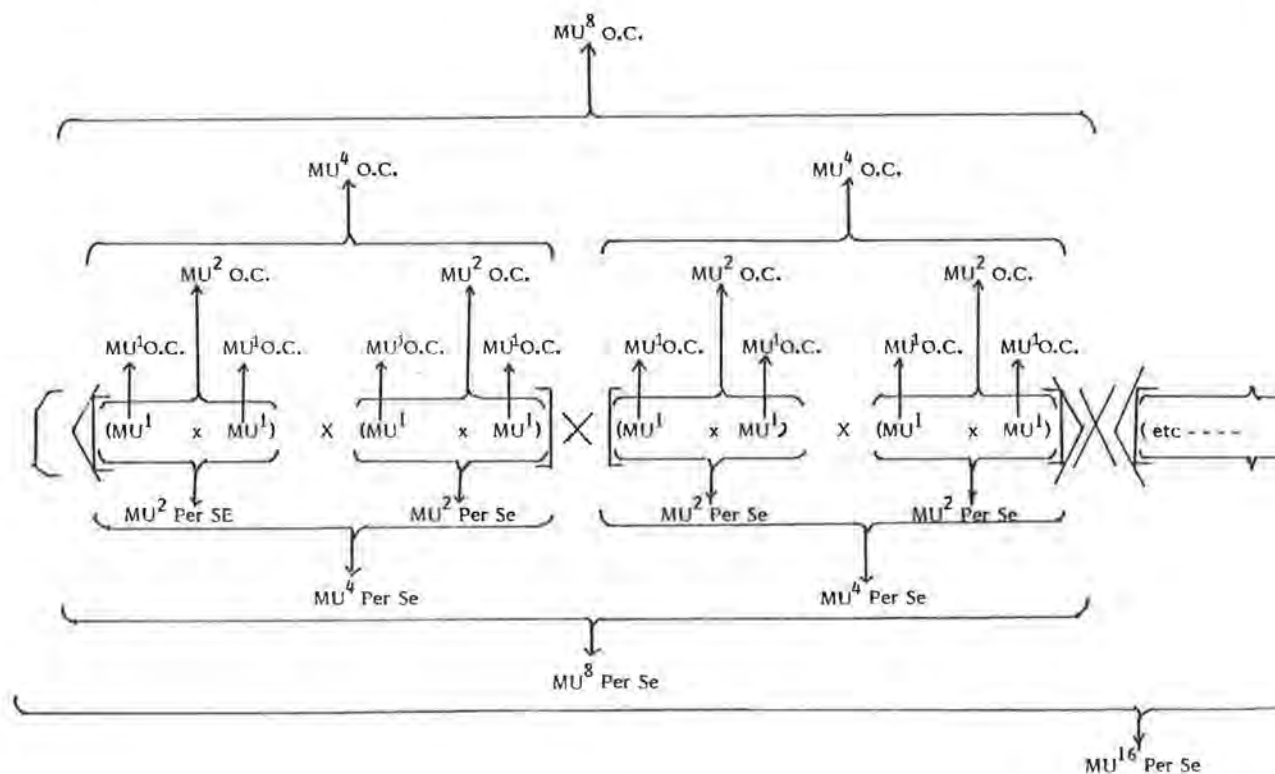


Figure 1

Synergistic effects were observed for 30', 40', and 45' of irradiation. The effect at 40' was significant (at 5% level).

These results suggest a synergistic effect is occurring. Most of the populations so far tested are small, which probably accounts for the up and down nature of the curve. The 30' point had the largest test populations (i.e., 526 for \underline{Mu} crosses and 213 for control crosses). The numbers for the other irradiation times were: 35' \underline{Mu} 155, control 131; 40' \underline{Mu} 139, control 130; 45' \underline{Mu} 161, control 138. The 0' \underline{Mu} population was 525 for 30' and 178 for 35', 40' and 45'.

U.V. irradiation of lysogenic bacteria is known to induce the replication of the temperate virus. If \underline{Mu} is viral in nature and is inserted into the chromosome in a lysogenic state, it also may be activated by U.V. and thus there may be more viruses available for insertion (resulting in mutations) in irradiated pollen than in nonirradiated \underline{Mu} pollen, thus producing a synergistic effect. I am not aware of studies on the effect of U.V. on the mutagenic effect of the various known transposable DNA systems. It would be interesting to know if they respond in a manner similar to \underline{Mu} in this regard.

If \underline{Mu} interferes with the DNA repair systems involved in correcting the thymine dimers induced by U.V., such a synergistic response might be expected.

Next summer larger populations of the U.V. experiments will be grown and the dosage curve will be extended.

Mutator and standard pollen were treated with EMS following the techniques developed by Neuffer. This pollen was used in crosses to standard lines to test for a synergistic effect of this mutagen in Mu plants. There does not appear to be any such effect although, again, the populations are small: Mu controls = 189, Mu-EMS = 128 and Standard EMS = 256. No mutants were found in 139 0' U.V. standard plants or in 280 no-EMS standard plants.

Donald S. Robertson

The effect of inbreeding mutator stocks on Mu activity

The exact nature of the mutator system (Mu) is not known at the present time. Its transmission does not follow traditional Mendelian patterns. When mutator plants are outcrossed generation after generation most of the outcross progeny have mutator ability. If mutator is confined to the chromosomes at one or more defined loci there must be some mechanism for selecting Mu bearing chromosomes for transmission. Evidence bearing on such a system of selective transmission has been negative, to date. In any case, this model would predict that by intercrossing Mu plants, the mutation frequency might be increased by twofold, but an increase above twofold would not be expected. Mutator could be chromosomal and increases in mutation frequencies above twofold could be found if Mu were transposable in nature and thus capable of inserting copies of itself into new chromosomal locations. The normal outcrossing procedure for propagation of Mu would tend to hold the number of transposable elements more or less constant because with each outcross the haploid mutator genome is being combined with a virgin genome into which Mu elements can be inserted. On intercrossing (i.e., inbreeding) Mu lines, however, the number of Mu elements would begin to accumulate. After several generations of inbreeding there could indeed be many Mu elements distributed over the genome, resulting in a mutation frequency considerably higher than twofold.

If Mu is transmitted extrachromosomally (it is transmitted with equal facility through the male and female) it might well build up in numbers upon inbreeding, resulting in mutation frequencies greater than two times those observed in Mu outcrosses.

To determine if inbreeding resulted in a twofold or greater increase in mutation frequency, a series of crosses was made.

The simplest and most efficient method of inbreeding is to self-pollinate for several generations. This is not practical with Mu stocks because progeny of selfs consist of many weak, runty, deformed plants (mutants?). Things do not improve with additional generations of selfing. To avoid these difficulties crosses were made between two different Mu outcrosses. Lines were chosen that produced two ears, one for selfing and one for crossing. The selfs permit the elimination of any plants that carry a mutation induced in one or more of its parental gametes. The protocol followed in this experiment is outlined in Figure 1. In the first generation, four different families of Mu outcrosses were planted and crosses made between individual plants of these four families. The male parent in each cross was self-pollinated, crossed to the female Mu plant and outcrossed to a standard line. The second ear of the female Mu parent was selfed and the plant was also outcrossed to a standard line. The parent plants are termed Mu¹ plants and their outcrosses are Mu¹ outcrosses. The F1 of the cross between the two Mu lines was called Mu² per se. Fifty or more seeds of each outcross were planted, the resulting plants self-pollinated and a sample of seeds from each ear was tested for the presence of seedling mutants. The same procedure was followed for the Mu² per se cross. The next generation two Mu² plants were crossed and at the same time outcrosses to standards were made. The

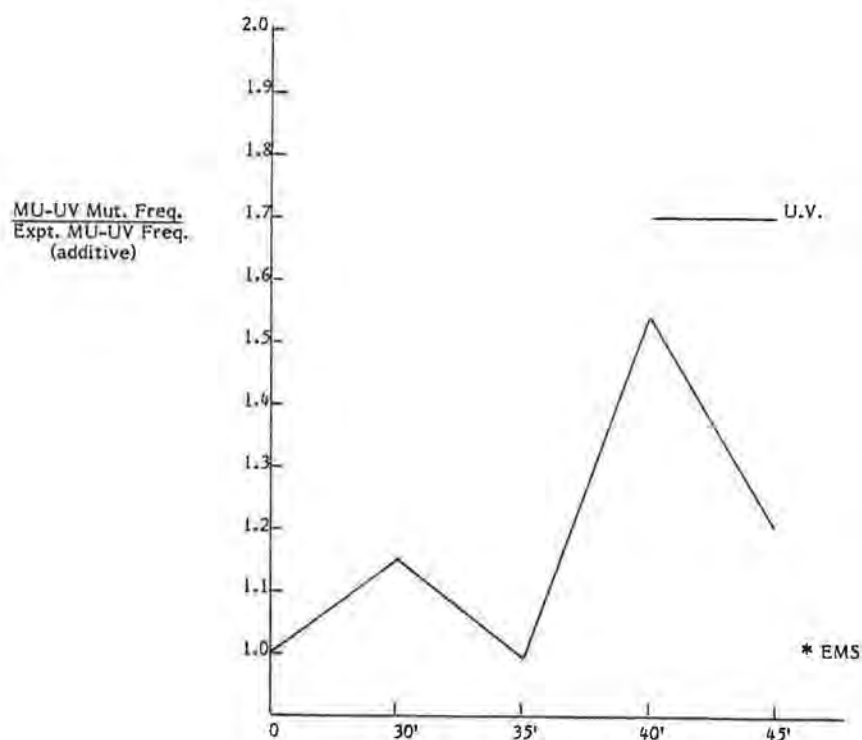


Figure 1

product of $\underline{Mu}^2 \times \underline{Mu}^2$ was termed \underline{Mu}^4 per se and the outcrosses \underline{Mu}^2 outcrosses. Next, \underline{Mu}^4 plants were crossed and outcrossed to give \underline{Mu}^8 per se and \underline{Mu}^4 outcrosses, and lastly, \underline{Mu}^8 plants were crossed to give \underline{Mu}^{16} per se, and outcrossed to produce \underline{Mu}^8 outcrosses.

The results are plotted in Figure 2.

When \underline{Mu}^2 (= $\underline{Mu} \times \underline{Mu}$) plants are produced, the male \underline{Mu} parents contribute to the \underline{Mu}^2 plants their mutant bearing pollen grains in a frequency expected of single \underline{Mu} plants. The female parent also contributes to the \underline{Mu}^2 plants, through the ovules, a frequency of mutants expected of single \underline{Mu} plants. Thus, the \underline{Mu}^2 per se plants should have twice the frequency of mutation that the \underline{Mu}^1 parents have. The \underline{Mu}^2 mutant frequency, however, is greater than expected. Instead of two times the \underline{Mu}^1 frequency it is 2.26 and 2.84 times. For the latter value, the difference between the calculated two times \underline{Mu}^1 rate and \underline{Mu}^2 is close to being significant at the 5% level ($\chi^2 = 3.0936$; $p = 5\%$, $\chi^2 = 3.841$). Since both the \underline{Mu}^1 parents were tested as males the greater than expected increase in mutation frequency observed in \underline{Mu}^2 plants would be expected if more mutants were transmitted through the female \underline{Mu}^1 parent. However, previous tests of transmission of mutants in reciprocal crosses of \underline{Mu} plants revealed significantly fewer mutants transmitted through the female. There are at least three possible explanations for the greater than twofold increase: 1) there is something about \underline{Mu} sperm nuclei traversing \underline{Mu} female tissue that results in additional mutants being produced in the sperm; 2) the \underline{Mu} sperm and \underline{Mu} egg nuclei in a common embryo sac may result in additional mutants being produced in either or both of them; or 3) in the zygote the presence of two \underline{Mu} genomes may induce some additional mutations.

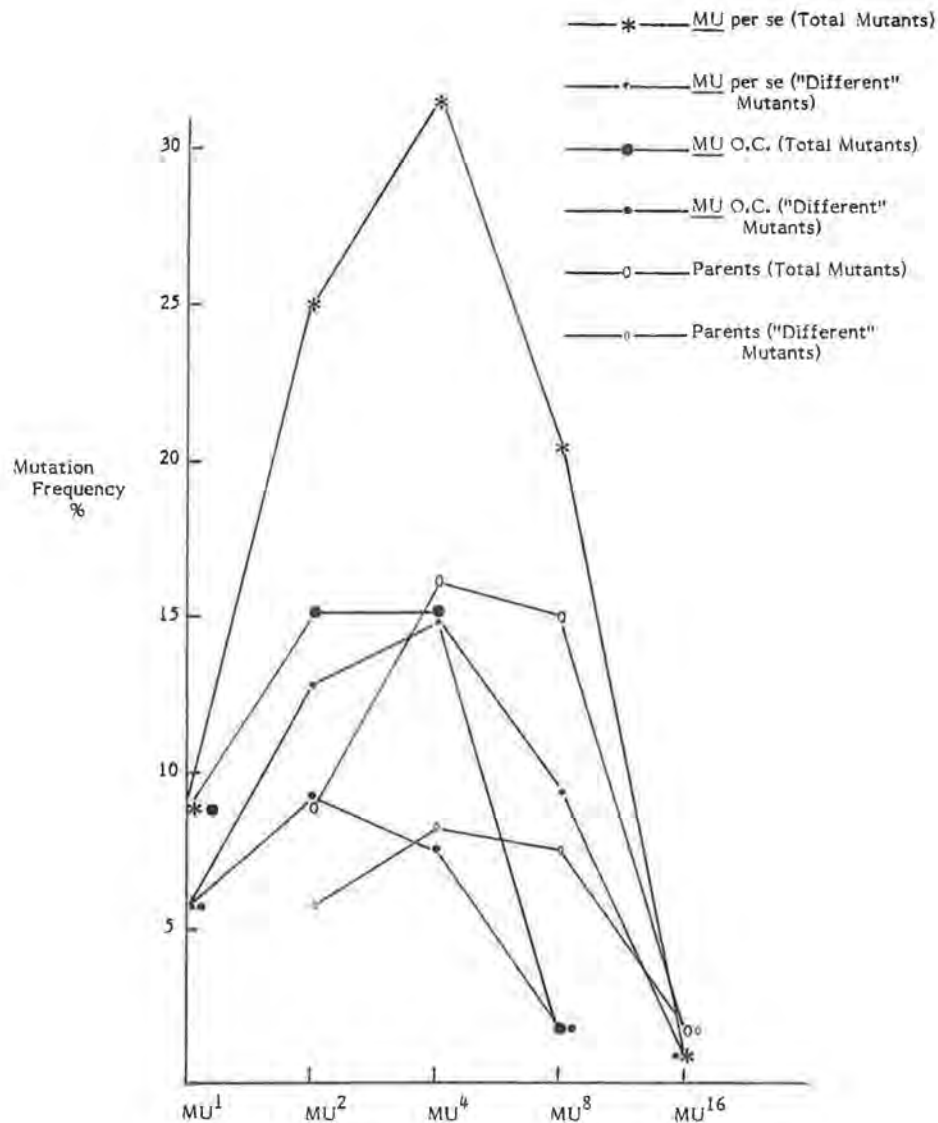


Figure 2

When $\underline{\text{Mu}}^2$ plants are used in outcrosses, they do not have two times the mutator activity as might be expected for either of the models dealing with the nature of the mutator system suggested earlier in this report. The mutation frequency of $\underline{\text{Mu}}^2$ outcrosses does not approach that of the $\underline{\text{Mu}}^2$ per se progeny. For the total mutant values the difference is very significant. Thus, combining two $\underline{\text{Mu}}$'s in a single individual produces less of an effect than combining the products of two separate $\underline{\text{Mu}}$ individuals. The effect of two $\underline{\text{Mu}}$'s combined in a single individual is not additive. Each $\underline{\text{Mu}}$ under these conditions seems to be less effective than when present in separate plants. In other words, the two $\underline{\text{Mu}}$'s together are more effective than a single $\underline{\text{Mu}}$ but not twice as effective as a single $\underline{\text{Mu}}$. Thus it is reasonable to assume that $\underline{\text{Mu}}^4$ per se would show another increase in mutation frequency, and indeed it does.

$\underline{\text{Mu}}^4$ outcrosses, on the other hand, show virtually no change in mutation frequency when compared to $\underline{\text{Mu}}^2$ outcrosses. Thus it appears that four doses of $\underline{\text{Mu}}$ is either not adding anything more to the system or the increases in number of $\underline{\text{Mu}}$'s may be

resulting in their partial inactivation. Inactivation is suggested because Mu^8 per se is observed to have a statistically very significantly lower rate than Mu^4 per se. When Mu^8 is outcrossed, there is observed very low mutation activity, in spite of the fact that Mu^8 per se had a mutation frequency over twice that of Mu^1 . Eight doses of Mu seems to be too much for the normal functioning of Mu 's. Almost all Mu activity seems to be lost. This loss of activity is reflected in Mu^{16} per se, which has a mutation frequency slightly less than the Mu^8 outcrosses.

Inactivation of Mu was suggested above as an explanation for the loss of mutator activity in these plants with putative high doses of Mu . If it is indeed an inactivation phenomenon due, perhaps, to high concentrations of Mu , then outcrossing for several generations of the Mu^{16} per se plants to non- Mu lines or continued outcrossing of Mu^8 outcrosses, again for several generations, may restore high mutability to these lines. Restoration of mutator activity under these conditions would only occur if the Mu inactivation is reversible. The loss of activity may be due to permanent inactivation of Mu or perhaps an actual destruction of Mu . In either case, it would not be possible to restore Mu activity by outcrossing.

There are other possible explanations. Perhaps cells with high doses of Mu can not compete. If there is some mitotic sorting out mechanism for Mu such as occurs for plastids in variegated plants, then the cells with few or no Mu 's would outcompete those loaded with Mu and give rise to an essentially non- Mu plant. Yet it is difficult to envision why such sorting out would take the Mu activity down to such a low level (to about 1%) when Mu plants with 5-9 percent mutation frequency do not seem to have their vigor affected. Such a precipitous drop in only one generation would not seem likely on the basis of this model.

In discussions of these results with Dr. Hugo Dooner, he suggested that sterility may be involved. Higher doses of Mu may be causing abortive pollen and/or ovules, leaving only low dose Mu or zero Mu ovules and/or pollen to function. I did not follow pollen sterility in these experiments. We have had occasion, however, to screen large numbers (several hundred) of Mu^1 plants for pollen sterility and a lesser number (about 50) of Mu^2 per se plants and in neither instance were many plants with abortive pollen found. An occasional plant with lower sterility (35% or less abortive pollen) did occur and a very rare semi-sterile plant was found.

Table 1. Ear sterility for the Mu crosses.

Mu <u>per se</u> progeny	% Normal seed set	% Semi-sterile seed set	Mu outcrosses	% Normal seed set	% Semi-sterile seed set
			Mu^1	52	48
Mu^2	24	76	Mu^2	71	29
Mu^4	14	86	Mu^4	58	42
Mu^8	51	49	Mu^8	74	26
Mu^{16}	30	70			

I did classify the ears for semi-sterile seed set throughout this experiment (Table 1). Seed set is not a very good gauge of actual sterility because it is so easily influenced by environmental factors, particularly in more inbred stocks such as the Mu per se lines. These lines did indeed have a high frequency of ears with poor seed set. But note that Mu¹ outcross lines also have high incidences of ear sterility although pollen examination has established that Mu¹ outcrosses show very little pollen abortion. There does not seem to be any consistent pattern of increasing sterility at higher Mu doses in either the per se crosses or the outcrosses expected if high doses of Mu were inducing sterility.

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A purification scheme for the bronze-encoded UDPglucose:flavonoid glucosyl transferase

The enzyme UDPglucose:flavonoid 3-O-glucosyl transferase (UFGT) catalyzes the 3-O-glucosylation of flavonols and anthocyanidins (Larson, R. L., *Phytochemistry* 10:3073, 1971; Larson, R. L. and Lonergan, C. M., *Planta* 103:361, 1972). Studies on stable and unstable recessive mutants of the bronze locus have provided evidence that UFGT is encoded at this locus (Larson, R. L., and Coe, E. H., *Biochem. Genet.* 15:153, 1977; Dooner, H. K., and Nelson, O. E., *Biochem. Genet.* 15:509, 1977, and *PNAS* 74:5623, 1977). A partial purification scheme for UFGT has been described (Dooner, H. K., and Nelson, O. E., *PNAS* 74:5623, 1977). We have prepared highly purified UFGT by DEAE-cellulose, CM-cellulose, Affi-Gel Blue and hydroxylapatite chromatography. A simple assay and the purification scheme are briefly described here.

The assay is carried out in a buffer containing 25 mM Tris (pH 8.0), 10 mM CaCl₂, 1mM dithiothreitol (DTT), 2 mM UDPG, 1.25 μ Ci/ml C¹⁴ UDPG (240 mCi/mmoles, Amersham), 25 μ g/ml human or bovine serum albumin, and 2 mM quercetin (dissolved at 40 mM in EGME). The enzyme is assayed in a volume of 20 μ l, adding 1-5 μ l of the enzyme to a suitably concentrated reaction mixture. After incubation at 37 C for 30 min, the reaction mixture is extracted with 100 μ l of *n*-amyl alcohol by vortexing; the phases are separated by a brief centrifugation in a Beckman Microfuge and the alcohol phase is removed to a double GF/A glass fiber filter (Whatman). The filter is dried and counted in a liquid scintillation spectrometer. When the labeled products appearing in the alcohol phase are analyzed by paper chromatography, the major labeled component is quercetin 3-O-glucoside; small amounts of labeled material having a different R_f are observed when crude extracts of mature kernels are assayed, but these are no longer detected after chromatography of the enzyme on DEAE-cellulose.

Large-scale preparations are made from mature Bz kernels. Dry kernels are pulverized to a fine powder using a high speed flour mill (Magic Mill II, available from Magic Mill, 235 West 200 South, Salt Lake City, Utah 84101). The powder is extracted in the cold with 3 ml/g powder of 0.1 M NaCl, 0.05 M Tris-HCl (pH 7.5), 1 mM DTT containing 20 mg/ml AG 1-X2 anion exchange resin (Bio-Rad) to adsorb pigments. Extraction is carried out for 1 hr with gentle stirring. The extract is centrifuged at 10,000 rpm for 20 min at 2-4 C in a Sorvall GSA rotor. The supernatant is filtered through siliconized glass wool. The pellet is resuspended in 1/3 of the original volume of buffer by brief homogenization in a Waring blender, respun and the supernatants are combined and dialyzed overnight in the cold against 0.01 M NaCl, 0.01 M Tris HCl (pH 7.5), 1 mM DTT, 25% glycerol, then centrifuged at 10,000 rpm for 20 min at 2-4 C. The supernatant is again filtered through glass wool and loaded on a short, large-diameter DEAE-cellulose column (2 ml bed volume/g

starting material), equilibrated with 0.01 M NaCl, 0.01 M Tris-HCl (pH 7.5), 1 mM DTT, 10% glycerol. The column is washed with 3 column volumes of 0.1 M NaCl, 0.01 M Tris HCl (pH 7.5), 1 mM DTT, 10% glycerol and then eluted with an 0.1-0.2 M NaCl gradient in the same buffer. The enzyme begins to elute at about 0.15 M NaCl.

Active fractions from the DEAE-cellulose column are pooled and dialyzed overnight against 0.08 M NaCl, 0.02 M MES (pH 5.0), 1 mM DTT, 50% glycerol and applied to a CM-cellulose column equilibrated with the same buffer containing 10% glycerol. The column contains 0.5-1.0 ml packed resin/g starting material. The column is washed with equilibration buffer and the material flowing through the column is assayed for enzymatic activity. More than 90% of the total protein eluting from DEAE-cellulose with UFGT adsorbs to the CM-cellulose column at this pH, but the UFGT enzyme does not. The pooled active fractions are dialyzed against 0.01 M NaCl, 0.01 M Tris-HCl (pH 7.5), 1 mM DTT, 50% glycerol and applied to an Affi-Gel Blue (Bio-Rad) column, using 1-2 ml packed bed volume/100 g starting material. The column is equilibrated with the same buffer containing 10% glycerol. After loading, the column is washed with 3 column volumes of 0.1 M NaCl, 0.01 M Tris-HCl (pH 7.5), 1 mM DTT, 10% glycerol and eluted with an 0.1-0.5 M NaCl gradient in the same buffer. The pooled active fractions from this column are dialyzed against 4 mM Na₂HPO₄ (pH 7.0), 1 mM DTT, 50% glycerol and applied to a hydroxylapatite column (Bio-Rad HTP) equilibrated with 4 mM Na₂HPO₄ (pH 7.0), 1 mM DTT, 10% glycerol, using 1 g packed bed volume/200 g starting material. The column is eluted with a 4-10 mM phosphate gradient containing 1 mM DTT and 10% glycerol. The pooled active fractions are dialyzed against 0.01 M Tris HCl (pH 7.5), 0.01 M NaCl, 1 mM DTT, 50% glycerol and stored at -20 C.

All procedures are carried out in the cold. The order of the CM-cellulose and Affi-Gel Blue columns can be reversed. The amount of protein contained in the active fractions becomes difficult to measure directly by UV absorbance beyond the CM-cellulose chromatography step and active fractions are generally examined by polyacrylamide gel electrophoresis to monitor purity. The proteins co-eluting with the enzymatic activity from DEAE-cellulose, CM-cellulose, Affi-Gel Blue and hydroxylapatite columns are displayed on a Coomassie Blue-stained SDS polyacrylamide gel in Figure 1. The arrow indicates the 50 kD protein identified as UFGT. The



Fig. 1. SDS polyacrylamide gel electrophoretic analysis of proteins co-purifying with EFGT activity. The protein co-eluting with UFGT activity during (a) DEAE-cellulose chromatography, (b) CM-cellulose chromatography, (c) Affi-Gel Blue chromatography, and (d) hydroxylapatite chromatography. The arrow indicates the protein identified as UFGT.

enzyme becomes increasingly unstable with purification and the assays should be done in the presence of serum albumin after the DEAE-cellulose step. Enzyme purified through DEAE-cellulose chromatography is stable for many months in 0.01 M Tris HCl (pH 7.5), 0.01 M NaCl, 1 mM DTT, 50% glycerol at -20 C. Highly purified enzyme can be stored frozen in liquid nitrogen in the same buffer. However, the effect of repeated freezing and thawing has not been determined. The purified protein from mature tissue migrates as a closely-spaced doublet of the same apparent molecular weight on 2-dimensional isoelectric focusing and SDS-polyacrylamide gels. Only one of the two spots is present in immature aleurone tissue. We do not know whether the charge heterogeneity is introduced during tissue dehydration and storage or during purification.

N. Fedoroff and J. Mauvais

Peptide mapping of the *Sh1* encoded sucrose synthetase and the residual sucrose synthetase

Sucrose synthetase is coded by the *Sh1* locus on chromosome 9. Chourey and Nelson (Biochem. Genet. 14:1041, 1976) demonstrated that a residual sucrose synthetase activity in *sh1* strains could be attributed to a protein with a slightly faster mobility on native gels. The two proteins are similar by many criteria (immunochemical, catalytic, subunit molecular weight) but show different developmental profiles (Chourey, Molec. Gen. Genet., in press, 1981). This report presents further evidence for the hypothesis (Chourey and Nelson, 1976) that the two sucrose synthetases are coded by two separate genes.

There are 3 maize strains available that are believed to be partial or complete deletions of the *Sh1* gene on chromosome 9. These are the two x-ray induced mutants *sh-bz-x2* and *sh-bz-x3* (Mottinger, Theor. Appl. Genet. 43:190, 1973) and *sh-bz-m4* (McClintock, Carnegie Yearbook 55:323, 1956). Antibody raised to sucrose synthetase purified from a *Sh1* strain is able to precipitate a protein from 20 days after pollination (DAP) endosperm extracts of the three deletion strains. This protein has a molecular weight (88K) identical to sucrose synthetase from *Sh1* endosperms on SDS gels. By titrating immuno-precipitates of *Sh1* endosperm extracts the level of the protein in the deletion endosperms was determined to be approximately 1-2% of that found in *Sh1* extracts.

In order to obtain enough of the residual protein for peptide mapping, the sucrose synthetases were partially purified. 20 DAP endosperms of each strain were ground in 2 volumes of 10 mM Tris-HCl (pH 7.5), 10 mM MgCl₂, and centrifuged at 23,000 g for 20 minutes. The supernatants were applied to Affigel-Blue columns equilibrated in the same buffer in the cold. The columns were washed with loading buffer and the sucrose synthetase fractions eluted at room temperature with 2 mM UMP, 10 mM NaCl and 10 mM Tris-HCl (pH 7.5). These concentrated fractions were immuno-precipitated in 0.15 M NaCl, 20 mM Tris-HCl (pH 7.5), 10% glycerol, 0.5% NP-40 and 0.5 mg/ml BSA. The antigen-antibody complexes were bound to protein A-Sepharose using 4 mg protein A-Sepharose/microliter of antiserum. The precipitates were washed twice with 0.5 M NaCl, 20 mM Tris-HCl (pH 7.5), 0.5% NP-40, and then once with the same buffer lacking NaCl. The antigen-antibody-protein A-Sepharose complexes were taken up in 20 mM DTT and 2% SDS, boiled for 2 minutes and applied to a 10% SDS acrylamide gel. This gel was stained for 30 minutes with Coomassie, destained one hour, and the 88K bands cut out. The gel pieces were inserted into the slots of a slightly wider SDS acrylamide gel and digested with chymotrypsin (7.5 micrograms/lane) as described by Cleveland et al. (J. Biol. Chem. 252:1102, 1977). This gel (Figure 1) was silver stained (Oakley et al., Anal. Biochem. 105:361, 1980).

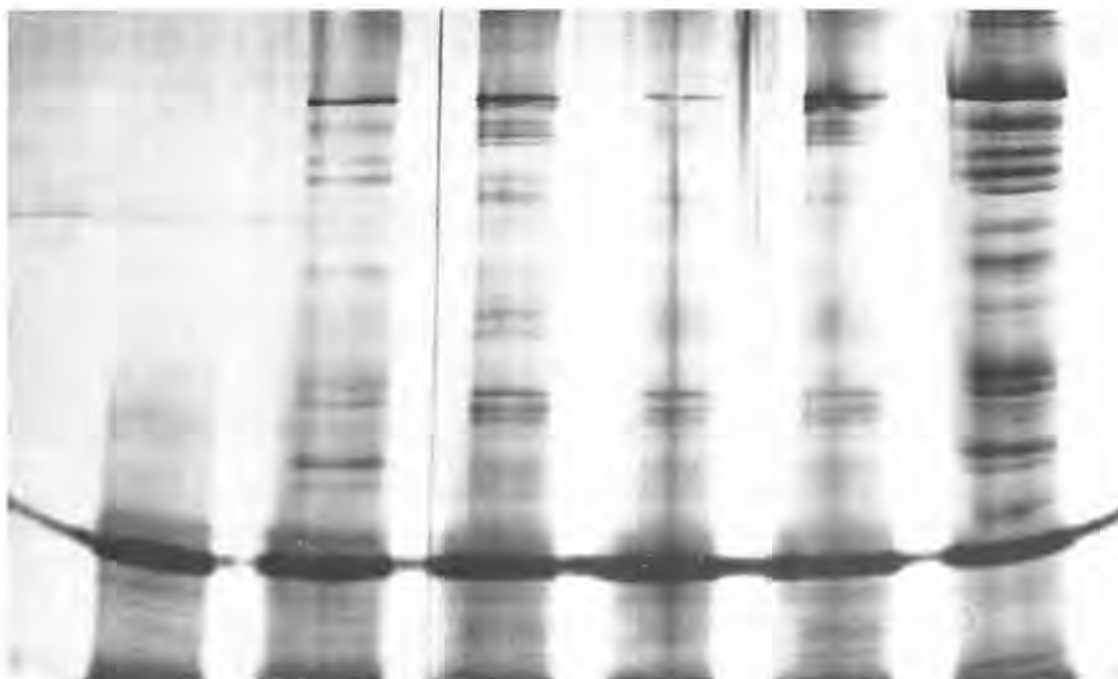


Fig. 1. Partial proteolytic maps of proteins immunoprecipitated with sucrose synthetase antibody: (a) chymotrypsin alone; (b) purified sucrose synthetase; (c) sh-bz-m4; (d) sh-bz-x3; (e) sh-bz-x2; (f) Sh1.

It is evident that the 3 deletion strains (lanes c, d and e) have identical peptide map patterns that are different from the pattern in the Sh1 lanes (b and f). Chourey (1981) was able to detect the residual protein in a Sh1 extract by greatly overloading a native gel. There are probably peptide fragments from the residual sucrose synthetase that are beyond the level of detection in lanes b and f.

In summary, the peptide maps offer fairly conclusive evidence that the two sucrose synthetase proteins are coded by two different genes.

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Does T-toxin bind specifically to Texas male-sterile cytoplasm mitochondria?

To test whether we could detect a specific binding of T-toxin to mitochondria isolated from Texas material, we first prepared some ^3H -labeled T-toxin. Cultures of Drechslera (= Helminthosporium) maydis race T were raised on solid medium containing 0.08 M sodium acetate (250 mCi ^3H -acetate/litre medium). T-toxin was extracted according to the method of Y. Kono and J. M. Daly (1979, Bioorganic Chemistry 8:391)

and had an estimated specific radioactivity of 400 mCi/mole. Two separate experiments were then made with mitochondria from N and T cytoplasm material. In the first experiment mitochondria were isolated from five-day-old dark-grown shoots of inbred A188 (N and T). Using an assay of malate oxidation (DCPIP reduction at 600 nm, P. A. Peterson et al., 1974, Plant Disease Reporter 58:777) the concentration of cold or radioactive T-toxin necessary to cause total inhibition of mitochondrial activity was determined. With the Texas cytoplasm mitochondria a concentration of 90 ng/ml T-toxin was sufficient to cause complete inhibition within one minute. With the Normal cytoplasm mitochondria a hundred-fold increase in toxin concentration caused less than 10% inhibition over the same time period. For the binding studies, mitochondrial suspensions were prepared just as in the enzyme assay experiments and suspended in the same buffer solution (total volume 2.5 ml) at a concentration of 0.20 mitochondrial mg protein/ml. Samples were incubated with toxin for five minutes before they were diluted to 10 ml with ice-cold PBS and filtered through Whatman GF/C glass fibre filters. The filters were rinsed with an additional 2 x 8 ml PBS. Centrifugation of the filtrate indicated that 95% of the mitochondria remained on the filters, which were then put into scintillation vials with 10 ml of a colloidal scintillant (Instagel, Packard Inst. Co.) for determination of radioactivity.

The results showed a relatively high level of unspecific attachment of T-toxin to the mitochondrial preparations but no evidence for specific binding to the T cytoplasm mitochondria. This was confirmed in a second experiment using mitochondria from WF9 (N and T cytoplasm). The results are open to different interpretations. We favor the explanation that T-toxin does not cause the observed inhibition of mitochondrial processes through a direct association with a specific protein species, but rather acts by, for example, destabilizing an already weakened membrane (cf., B. G. Forde and C. J. Leaver, 1980, in *The Plant Genome*, eds. D. R. Davies and D. A. Hopwood). The presence of receptor sites cannot be ruled out, however, since some specific binding of T-toxin to Texas cytoplasm mitochondria might have been masked by the high unspecific attachment of the lipophilic T-toxin molecule to the mitochondrial membrane.

Peter Simons and Richard Brettell

An anomalous line derived from a Texas male-sterile cytoplasm tissue culture:
A case of nuclear reversion?

Instability in the expression of Texas cytoplasmic male-sterility has been observed, particularly when material is passed through tissue culture (B. G. Gengenbach et al., 1977, PNAS 74:5113 and R. I. S. Brettell et al., 1980, TAG 58: 55). Analysis of the newly male-fertile, T-toxin resistant lines has up to now indicated that the reversion is invariably cytoplasmic in origin. Fertility and T-toxin resistance show maternal inheritance and the change in phenotype is correlated with alterations in mitochondrial DNA. However, we have recently found one male-fertile, T-toxin resistant line (32D) derived from an embryo culture of the cross (WF9T/W22 x A188Nrf) x A188Nrf which has shown an exceptional 'restorer' effect when used as pollinator. Preliminary data, yet to be extended or repeated, are as follows:

	Fertile (4-5)	Intermediate (2-3)	Sterile (1)	Total	Toxin Reaction
A188Trf x 32D	0	2	14	16	all sensitive
(A188Trf x 32D) x 32D ^a	5	6	0	11	all sensitive
32D x 32D (selfed)	18	0	0	18	all resistant
A188Trf x A188Nrf	0	0	17	17	all sensitive

^aIn this cross one of the 14 sterile plants from the first cross was used as female parent.

Male-fertility was classified according to the Duvick scale as used by J. B. Beckett (1971). Resistance to T-toxin was assessed by applying T-toxin directly to a young, rapidly-growing leaf.

Nuclear as well as cytoplasmic revertants have been described and well characterized for S cytoplasm (J. R. Laughnan and S. J. Gabay, 1978, in *Maize Breeding and Genetics*, ed. D. B. Walden). Further experiments will be necessary to confirm that the restorer effect of line 32D may similarly be ascribed to changes in the nuclear genome. Meanwhile I should be interested to hear of any other observations by cooperators pointing to nuclear reversion in Texas cytoplasm, and will supply small amounts of seed from the line 32D on request.

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Analysis of mitochondrial DNA from four fertile maize lines obtained from a tissue culture carrying Texas cytoplasm

This contribution describes a further study of changes occurring in the mitochondrial DNA (mtDNA) of maize derived from Texas cytoplasm tissue cultures. Changes in the male-sterile, T-toxin sensitive phenotype have been observed in two independent laboratories as the result of a passage through tissue culture (B. G. Gengenbach et al., 1977, PNAS 74:5113 and R. I. S. Brettell et al., 1980, TAG 58:55). Similar phenotypic changes were also described in Texas cytoplasm material subjected to a mutagenic treatment by A. Cornu et al. (1980, MGCNL 54:42). Restriction enzyme analysis led B. G. Gengenbach et al. (1981, TAG 59:161) to remark that increased variation in mtDNA was probably the consequence of passage through tissue culture, and that the change in phenotype was not simply the result of a selection of N-type mitochondrial genomes already present at low frequency in Texas cytoplasm.

Here we have examined four lines obtained from four plants regenerated from a culture carrying Texas cytoplasm. The culture was initiated from an embryo which derived from crossing (WF9T/W22) x A188Nrf with pollen from W22rf. The culture was maintained according to Brettell et al. with and without a selection for resistance to T-toxin imposed by incorporating T-toxin in the culture medium. Four resistant, fertile plants were regenerated to give four lines as follows:

<u>Number of months cultured before plant regenerated</u>	<u>Selection for resistance to T-toxin</u>	<u>Pollination</u>	<u>Line obtained</u>
10	Yes	selfed	V6
15	No	selfed	V18
15	Yes	selfed	V23
16	Yes	not-controlled	V24

When tested under field conditions these lines were uniformly male-fertile and scored as resistant to T-toxin.

Mitochondrial DNA was extracted from each of the four lines and subjected to a BamHI digest. The fragments were separated on a gel and suggested that a series of rearrangements had occurred, when compared to Texas material that had not passed through tissue culture. Such rearrangements were also observed by Gengenbach et al. in tissue culture-derived lines which continued to express the male-sterile, T-toxin sensitive phenotype, and so are not necessarily related to the appearance of fertile, T-toxin resistant plants. A XhoI digestion similarly revealed a number of apparent rearrangements. However, when taken with the data of Gengenbach et al. it would seem that one of the XhoI fragments present in the Texas cytoplasm controls is altered in all lines so far examined which express the fertile, T-toxin resistant phenotype. This fragment, which may thus relate to the change in phenotype, is about 6.6 kb in length and has now been isolated from a parental T-cytoplasm line, nick-translated and hybridized to the mtDNA of the plants obtained from tissue culture. In three of the lines (V6, V23 & V24) homology appeared at about 6 kb. In V18 on the other hand there was homology at about 20 kb.

HindIII and BamHI digests were also made from the chloroplast DNA of the lines obtained from culture, but in contrast to the digests of mtDNA, they showed a pattern constancy.

A mystery remains as to how and when these rearrangements are occurring. Interestingly, there were similarities between the three lines (V6, V23 & V24) which were derived from the culture maintained in the presence of T-toxin and kept separate from that which gave V18. This might indicate that the rearrangement events are occurring early in the culture sequence (the unselected and selected culture lines were separated four months after the embryo culture was initiated) rather than during regeneration.

Finally, we note that restriction enzyme analysis of mtDNA from plants derived from N cytoplasm cultures also indicates sequence changes (R. J. Kemble and R. B. Flavell, personal communication). This suggests that the apparent 'instability' of mtDNA in tissue culture is not a specific property of Texas cytoplasm.

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In vivo labeling of embryo and endosperm proteins in intact kernels with ^{35}S -methionine

Proteins of the embryo and endosperm are readily labeled with ^{35}S -methionine following the introduction of the label directly into kernels on developing ears. For the labeling experiments, immature ears (20-24 days post-pollination) were brought into the lab from the field, and a portion (about the distal third) of the

ear broken off. A ring of kernels near the point of breakage were removed, and the label was introduced into selected kernels on the ear segment by injection of the solution directly into the base of the kernel, using a Hamilton syringe. The amount of ^{35}S -methionine introduced into each kernel was about 80 uci (1.76×10^8 dpm), in a volume of 7.5 - 10 μl . Injected kernels were labeled with a marking pen for later identification. The ear segment was incubated at 30 C for 2-4 days in a water-saturated atmosphere. After the appropriate incubation period, injected kernels were removed from the ear, and each embryo and endosperm individually macerated in 1 ml of SDS sample buffer (62.5 mM Tris-HCl, pH 6.8, 2.3% sodium dodecyl sulfate, 5% 2-mercaptoethanol, 10% glycerol). These samples were boiled for 5 minutes and centrifuged in a Brinkmann tabletop centrifuge for 2-5 minutes. Aliquots (1-5 μl) of the supernatant were spotted onto filter paper disks, and placed in cold 10% trichloroacetic acid (TCA) to precipitate macromolecules. The filters were boiled in the same solution for ten minutes and washed with 5% TCA, followed by a wash with 95% ethanol. The filters were dried and counted in a scintillation counter. Incorporation of label was on the order of 10^4 dpm/ μl sample, for both embryo and endosperm, for an incorporation efficiency of 5-10%. The samples were electrophoresed in polyacrylamide gels, stained with Coomassie Brilliant Blue, and dried down. The dried gels were used to expose Kodak X-OMAT AR film. The bands on the exposed autoradiograms corresponded to the stained bands in the gel.

The efficiency of labeling in this manner is variable, and the embryo and endosperm of the same kernel may show a significant difference in the amount of label incorporated. It should be noted that the site of injection of the label is critical: attempts to introduce the label by injection into the crown were not successful, as most of the label solution was extruded. Kernels injected at the crown show very little, if any, incorporation of label by the TCA precipitation assay, and no bands were detectable on autoradiograms in lanes in which such kernels were run. Studies involving the introduction of labelled amino acids into kernels with the ear remaining on the plant are in progress at the present time.

The ^{35}S -methionine used in these preliminary studies was of the same quality as that used routinely in our laboratory as a tracer in an in vitro protein synthesizing system (Amersham SJ.204, 1385 ci/mmol).

Alan L. Kriz

An Ac dosage effect on the protein products of the wx-m-9 gene

The size of the Wx protein produced by the wx-m-9 gene (McClintock, Carnegie Inst. Ann. Rep. #62) depends on the dosage of this controlling element mutant, in which Ac is inserted in the gene locus. In 20-day wx-m-9/wx/wx endosperm only a single Wx protein is produced as revealed by silver stain SDS acrylamide electrophoresis. The amount of protein produced is variable but it is of uniform size, slightly larger than the protein specified by the Wx allele. This protein is also produced by germinal revertants of wx-m-9 resulting from prezygotic transposition of Ac. In both wx-m-9/wx-m-9/wx and wx-m-9/wx-m-9/wx-m-9 20-day endosperm, a protein of this size is not detected and instead, one finds a double Wx protein band--one protein slightly larger and the other slightly smaller than the one-dose protein. The two protein bands are of about the same low intensity.

The wx-m-9 allele is not fully suppressed by the presence of the Ac element and some amylose is synthesized. As transpositions occur very late in two-dose wx-m-9 endosperm and practically not at all in three-dose wx-m-9 endosperm, it is proposed that the two Wx proteins in the double band are the products of the Ac "suppressed" gene and that the single intermediate sized Wx protein of wx-m-9/wx/wx endosperm is the product of the reverted gene in which Ac was transposed away from

the locus. Analysis of our data further suggests that we are dealing with included genes, the Ac "gene" being inserted within the limits of the wx structural gene. As shown by McClintock (Carnegie Inst. Ann. Rep. #64) to be the case for wx-m-7, we propose that functioning of the Ac gene and the Wx gene are mutually exclusive. In one-dose wx-m-9, Ac is active early and the Wx gene is inactive early except in those cells in which Ac had been transposed away from the locus. In two- and three-dose endosperm where Ac is not active early in endosperm development, the Wx gene functions early and the other two double band Wx proteins are produced from mRNA in which the Ac segment was excised. Similar results are obtained when the dosage of wx-m-9 is kept constant and Ac dosage varied by the use of other non-linked Ac elements.

Drew Schwartz and Craig S. Echt

Analysis of the Mp mutant wx-B3-M

In last year's newsletter (MGNL 55:8) we presented evidence that the wx controlling element mutants m-1, m-6, B3 and possibly m-8 and B4 lie within the limits of the Wx structural gene. The evidence given for the Mp mutation in B3, originally obtained from Oliver Nelson, was based on the observation that this mutant produces an altered, inactive Wx protein. However, additional B3 material, subsequently obtained from the Coop., gave completely different results. It turns out that B3 had been established in two different backgrounds, W22 and M14, and that the mutant maintained in each background is different. The B3 we originally reported on, which produces the normal amount of an altered Wx protein, is in the W22 background and recent analysis shows that it does not have any Mp transposing activity. In contrast, the B3 allele that is in the M14 background does have Mp activity but produces very little Wx protein. These alleles will be referred to as B3-W (W22) and B3-M (M14). B3-M was the allele that Nelson used in his mapping studies (MGNL 50:109). The B3 pedigree, provided by Nelson, shows that the B3-M allele had been backcrossed into M14 through four generations. The B3-W allele came from a cross of B3-M to W22 and two subsequent backcrosses to W22 followed by two generations of self-pollination. We have shown that the differences in Wx protein level and Mp activity between B3-W and B3-M are not due to the differences in background. The evidence indicates that B3-W is a stable germinal derivative of B3-M. In addition, the mutable B3-M allele seems to undergo autonomous changes in the endosperm to produce both an active and inactive Wx protein.

Since Nelson used the B3-M and not the B3-W allele in his mapping studies, it is important to establish that the B3-M mutant, like B3-W, involves an alteration within the structural gene limits. A pollen recombinational analysis was performed on the two B3 alleles according to the methods of Nelson (Genetics 60:507). The data are given in Tables 1 and 2. It is concluded that there is no detectable recombination between the two B3 alleles. Hence, the Mp element of B3-M is at or very close to the site of the B3-W mutation and most probably lies within the structural gene limits. The evidence presented below that the B3-M mutant can give rise to an altered Wx protein also indicates that Mp lies within the structural gene.

The Mp activity of B3-M is shown by the somatic reversion to Wx in endosperm heterozygous for B3-M and the wx Ds alleles m-1, m-6 or B4. Some autonomous somatic reversions to Wx are also observed in B3-M/wx/wx endosperm. Whereas the Mp element of B3-M is as active as Ac in inducing endosperm reversions in the wx Ds alleles, B3-M is only very slightly active in inducing its own reversions. Each B3-M/wx/wx endosperm has only a few small Wx sectors containing only one or a few cells, as seen with IKI staining.

TABLE 1
Pollen recombinational analysis of B3-M/B3-W heteroallele

	avg. # <u>Wx</u> grains $\times 10^{-5}$	# plants	total # pollen grains
<u>B3-W/B3-W</u>	2.4 \pm .33	3	1,539,000
<u>B3-M/B3-M</u>	6.8 \pm 3.1	3	1,239,000
<u>B3-M/B3-W</u>	4.1 \pm 2.3	4	2,229,000

TABLE 2
Pollen recombinational analysis corrected for rare high Wx counts

	avg. # <u>Wx</u> grains $\times 10^{-5}$	# pollen grains	# slides with aberrantly high # <u>Wx</u> grains / total slides
<u>B3-W/B3-W</u>	2.4 \pm .33	1,539,000	0/36
<u>B3-M/B3-M</u>	3.8 \pm .75	1,090,000	4/36
<u>B3-M/B3-W</u>	1.8 \pm .29	1,975,000	6/55

about 40,000 grains /
slide

The higher numbers of Wx grains and larger standard deviations in the B3-M parent and in B3-M/B3-W (Table 1) seem to be due to the presence of the mutable B3-M allele. The probable cause is occasional premeiotic reversion events since only a few slides had an aberrantly high number of dark staining Wx grains. When these few slides are discounted (Table 2) a more representative estimate of recombination is obtained. However, whether this correction is made or not, it can be seen that the frequency in the heteroallele is lower than the mean of the parental frequencies, so no recombination is evident.

The Wx protein can be extracted from endosperm starch granules and visualized in SDS polyacrylamide gels following electrophoresis and staining (MGNL 55:9). As previously reported, the Wx protein from B3-W is produced in the normal amount. The Wx protein from B3-M, however, is usually present in a greatly reduced amount (roughly 5% the one-dose B3-W level) and decreases with increasing dosage of the B3-M gene or with the presence of additional Ac's. When individual kernels are screened for their Wx protein, about 10% are exceptional and show an increased amount of Wx protein above that seen in the rest of the population. The degree of the increase varies, and although it is usually small, it can approach the one-dose level seen for B3-W or Wx. Because mature kernels from these same crosses do not show an increase in blue staining starch with IKI, most of the Wx protein produced by these exceptional B3-M kernels must be inactive.

It also appears that, even in the non-exceptional kernels, most of the Wx protein present is enzymatically inactive. This is based on a comparison of the amount of Wx protein and blue staining starch produced in the endosperm by B3-M to that produced by the wx Ds mutant m-6 in response to B3-M. In the absence of Mp or Ac, m-6 is stable and produces neither reversions nor Wx protein. When one Ac is present, numerous reversions are seen in the endosperm and a Wx protein is produced that appears about 1,000d larger than the standard 60,000d Wx protein in migration comparisons in Laemmli SDS gels. In m-6/m-6/B3-M heterozygotes two Wx proteins are made, the standard sized B3-M protein and the apparently larger m-6

protein. In silver stained gels the amount of B3-M Wx protein is only slightly less than the amount of m-6 Wx protein. Based solely on the protein ratios, one would predict that B3-M should give rise to the same degree of Wx reversion as m-6 does in response to B3-M or Ac. However, the amount of blue staining starch in B3-M/wx/wx endosperm is at least 10-fold less than that seen in m-6/m-6/B3-M or m-6/m-6/wx; Ac endosperm. Such disparity between the amount of Wx protein and blue staining starch produced by B3-M suggests that nearly all of the Wx protein produced is enzymatically inactive. Though some active protein must be produced, because a few small Wx sectors occur in B3-M kernels, this small amount of protein would hardly be detectable.

It is proposed that B3-M is undergoing the normal amount of autonomous transposition expected of an Mp mutant but that an inactive, altered Wx protein is the result. It is only rarely that a "mistake" is made and an active Wx protein is produced. Preliminary isoelectric focusing analysis suggests that the proteins produced by B3-M and B3-W have the same isoelectric point.

The production of an altered Wx protein by m-6 reversions provides further evidence that the Ds element in m-6 lies within the structural gene limits.

Craig Echt and Drew Schwartz

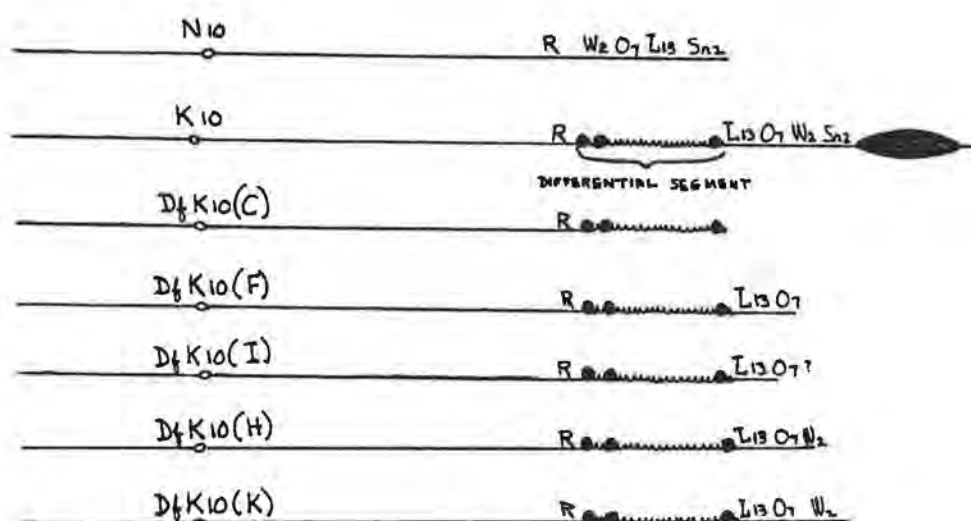
The architecture of abnormal chromosome 10

In our 1981 Maize News Letter report we stated that an abnormal chromosome 10 (K10) terminally deficient for W2 and Sr2 possessed the 07 locus. This gene had been placed in normal chromosome 10 (N10) between the W2 and Sr2 loci and the linear order in the distal end of 10L was believed to be R - W2 - 07 - Sr2. Our data suggest that the linear order in the distal tip of abnormal 10 is R - 07 - W2 - Sr2, i.e., the 07 through W2 segment is inverted in abnormal 10 relative to its orientation in normal 10. However, we questioned if the putative order in N10 is correct, since it was largely based on F2 data from plants with different genetic backgrounds and recombination values are known to be influenced by genetic modifiers. We now present evidence that the tentative linear order in the distal tip of 10L is indeed correct and that the segment including W2, 07, and L13 genes, which lies between R and Sr2 in N10, is inverted in abnormal 10.

The deficient abnormal chromosome 10 mentioned above was one of 11 deficient abnormal chromosomes 10 that we isolated in our high-loss studies. They all arose following rupture of a dicentric bridge arising at the second microspore mitosis as a consequence of the delayed replication of knob heterochromatin. Delayed (incomplete) knob replication occurs in those microspores with two or more B chromosomes. Five of the deficient abnormal chromosomes 10 had terminal deficiencies for the distal end of the long arm of 10. They were not involved in any structural rearrangement. The remaining six deficient abnormal chromosomes 10 possessed a translocated segment from a heterologous chromosome. Most of our attention has been given to the five simple terminal deficiencies, which were designated as DfK10(C), DfK10(F), DfK10(H), DfK10(I), and DfK10(K). All five were tested for possession of specific loci by ascertaining the phenotype of DfK10/N10 compounds where N10 carried a recessive mutant allele. If the hemizygous individuals expressed the mutant trait, the tested DfK10 chromosome lacked the wild type allele present in the parental K10 chromosome. Our findings are summarized in the following table where the + sign indicates that the tested Df chromosome had the wild type allele, a - sign denotes a lack of it and an * signifies that tests have not been completed. The listing of the tested loci at the top of the table is their linear order in N10.

Linear order in	N10	→	R	W2	07	L13	Sr2	
Linear order in	K10	→	R	L13	07	W2	Sr2	Knob
Genic content of	DfK10(C)	→	+	-	-	-	-	-
Genic content of	DfK10(F)	→	+	+	+	-	-	-
Genic content of	DfK10(I)	→	+	+	*	-	-	-
Genic content of	DfK10(H)	→	+	+	*	+	-	-
Genic content of	DfK10(K)	→	+	+	*	+	-	-

All five of the DfK10 chromosomes lack the K10 knob and the Sr2 locus, since the loss of the dominant Sr2 allele present in K10 was used in screening for DfK10 chromosomes. The relative lengths at pachynema of N10, K10, and the five K10 chromosomes with simple terminal deficiencies are given below:



- DfK10(C) Deficient for L13 07 W2 Sr2 and the K10 knob. Arose from a break in K10 to the left of L13.
- DfK10(F) Deficient for W2 Sr2 and the K10 knob. Arose from a break in K10 between 07 and W2.
- DfK10(I) Deficient for W2 Sr2 and the K10 knob. Arose from a break in K10 between 07 and W2.
- DfK10(H) Deficient for Sr2 and the K10 knob. Arose from a break in K10 between W2 and Sr2.
- DfK10(K) Deficient for Sr2 and the K10 knob. Arose from a break in K10 between W2 and Sr2.

~ In DfK10(C)/N10 heterozygotes the two chromosomes 10 are of the same length at pachynema. The differential segment with its three small knobs allows recognition of the DfK10 chromosome. Despite having a length equal to that of N10, it is deficient for all tested loci distal to R, indicating that these missing genes are situated in the euchromatic region of K10 located between the differential segment

and the K10 knob. Cytogenetic studies of all the DfK10 isolates are in agreement with the conclusion that this segment of euchromatin distal to the differential segment is homologous in gene content to the terminal 0.3 of N10 where the linear order is W2 07 L13 Sr2. However, the linear order of the W2 07 L13 region is inverted in K10 where the linear sequence is R L13 07 W2 Sr2 knob (the inverted stretch of chromatin is underlined). This conclusion, which we reported in 1981 based on the uncertain location of 07, has now been confirmed by employing the L13 mutant which was shown by Peter Mascia to lie five crossover units proximal to Sr2.

DfK10(C) and DfK10(F) are the most instructive since their constitution for all extant loci in distal 10L has been determined. DfK10(F), cytologically a shorter deficiency than DfK10(C), is deficient for the K10 knob, the Sr2 and W2 loci, but not for 07 or L13. DfK10(C) lacks all of these marker genes. It should be, and cytologically is, a longer deficiency than DfK10(F). It should be recalled that the DfK10 isolates all arose in high-loss plants in which faulty knob replication at the second microspore mitosis, induced by B chromosomes, results in dicentric bridges at anaphase. They are ruptured. The telophase nuclei become transformed into sperm cells. A sperm with a broken end produced by bridge breakage may unite with the egg pronucleus to form the zygote. It is known from McClintock's earlier work that healing of freshly broken ends occurs in the zygote and they subsequently behave as stable ends. There is no breakage-fusion-bridge cycle, which can lead to structural changes such as duplications. That is, we recover unchanged in the zygote the immediate products of bridge breakage in the ultimate division of the male gametophyte.

We first considered the possibility that even though the portion of K10 carrying the W2 07 L13 segment was transposed distally relative to its position in N10, the linear order with respect to R and Sr2 remained the same. However, if this were true, all breaks in the dicentric bridge between 07 and W2 would yield DfK10 chromosomes deficient for Sr2, L13, and 07 but not for W2. This was not the case since Df(F) has the L13 and 07 genes but not W2 or Sr2. Such a chromosome could not arise if the order were normal. With an uninverted order, all breaks between R and W2 result in chromosomes lacking W2 07 and L13, but DfK10(F) is not deficient for 07 or L13. The order of the W2, 07, and L13 genes is clearly inverted in the distal euchromatic segment of K10 which is homologous to the terminal 0.3 of the long arm of N10. The linear order is R L13 07 W2 Sr2. Only the R locus occupies the same position in the two homologues. An alternative order of R L13 W2 07 Sr2 in K10 can be ruled out since a single break in the dicentric bridge between L13 and W2 would produce a DfK10 chromosome missing the W2, 07 and Sr2 loci. DfK10(F) is deficient for W2 and Sr2 but not for 07. Our studies with the six DfK10 isolates involving translocations are not as extensive as those with the terminal deficiencies but they are in no way in disagreement with the conclusions drawn from the simple deficiencies.

Although DfK10(F) and DfK10(I) are both deficient for W2 and Sr2 but not for L13, they are not identical. They differ in pollen transmission. DfK10(F) is frequently male transmitted, producing in F2 progenies kernels homozygous for Df(F). These kernels have an aleurone layer mosaic for colored-white spots and give rise to albino seedlings. Df(I) is rarely, if ever, transmitted through the pollen. The two deficiencies also differ in the amount of recombination between the R locus and the tip of the deficient chromosome in DfK10(F)/K10 and DfK10(I)/K10 compounds. In testcrosses of DfK10(F) R - / K10 r Sr2 heterozygotes there was 15% recombination between R and the end of the Df(F) chromosome while in DfK10(I) R - / K10 r Sr2 compounds there was less than half as much crossing over.

Of some interest is the fact that the frequency of crossing over between R and the tip of the deficient chromosome in DfK10(C)/K10 heterozygotes is ca. five percent and most of the exchanges take place in the homozygous differential segment

with its three small knobs. The extent of this segment in DfK10(C)/K10 meiocytes, in which crossing over distal to R can occur, is physically as long as the R-Sr2 interval in N10 homozygotes where there is 36% recombination. One is forced to conclude that recombination is low per unit length of chromatin in the differential segment. Unfortunately, this chromatin is not populated with mutant loci and its origin is unknown.

DfK10(K) has the shortest deficiency. It lacks only Sr2 and is pollen transmissible but the amount of recombination between R and its tip in DfK10(K)/K10 heterozygotes is only 19 percent. This interval is approximately twice as long at pachynema as the R-Sr2 region in N10/N10 bivalents where the recombination value is 36 percent. Either the large heterozygous K10 knob is depressing crossing over in regions adjacent to it or there is a high exchange frequency in the tip of normal 10L, where none can occur in DfK10(K)/K10 individuals since it is in a hemizygous condition. This question is being resolved.

Apparently some misunderstanding exists regarding the architecture of abnormal 10 found in races of maize. All of the abnormal chromosomes 10 observed by Kato in different races of maize had the same structure in that the segment in 10L with the three small knobs (our differential segment) was not adjacent to the K10 knob but was separated from the knob by a stretch of euchromatin. In K10/N10 pachytene bivalents, the differential segment is paired with the distal 0.3 of the long arm of N10. Kato found that some races of teosinte have an abnormal 10 with the differential segment next to the knob while others possess the maize type. The two kinds of abnormal 10 present in teosinte may differ by a single paracentric inversion but this has not been demonstrated to be so. Since teosinte crosses readily with maize to give fertile hybrids it is not obvious why the second type of abnormal 10 found in certain teosintes has not undergone introgression into sympatric races of maize.

M. M. Rhoades and Ellen Dempsey

The induction of mutable systems in plants with the high-loss mechanism

In recent years we have been occupied with the study of the mechanism responsible for the loss of segments of chromatin from knobbed A chromosomes at the 2nd microspore mitosis. We were able to show that loss resulted from the rupturing or breaking of dicentric bridges. These dicentric bridges were the consequence of a delayed replication of the heterochromatic knobs. Any knobbed A chromosome was subject to loss. We also demonstrated that faulty replication of knobs only happened in microspores with two or more B chromosomes. An unexpected bonus or spin-off from these investigations on chromatin loss, in which bridge breakage played a cardinal role, was the appearance of three mutable systems. We did not anticipate their occurrence and our experiments were not designed to reveal their presence but crop up they did, somewhat to our annoyance, since they diverted our full attention from the main thrust of our studies. However, we could not completely disregard them and a considerable body of information has been obtained in recent years. This report is a summary statement of our progress to date in analyzing these mutable systems, all of which proved to involve a controlling and a responding element.

Origin: As stated previously, three new mutable systems were discovered in stocks undergoing the high-loss phenomenon. The first of these involved an a-m responding allele (tentatively designated a-m-rh to distinguish it from the a-m responders of McClintock, Neuffer and Peterson) and a mutator designated Mrh. Mutation from A to a-m occurred in an F1 (30418) from a cross of the high-loss stock with a high-knob strain. The F1 individual had 4 B chromosomes and numerous knobs (including K10) so it was well endowed with heterochromatin. It was homozygous for the A allele. The mutation was detected when the F1 was used as male

parent in a cross to an a tester. All progeny kernels were colored as expected save an exceptional kernel which had a white endosperm with colored dots. The embryo proved to be a/a-m-rh; it was heterozygous for the new a-m allele. The mutator Mrh has been found in plants with the high-loss background, so its precise time and place of origin are unknown.

A similar situation was found with the second system, which involves a mutable bz allele in 9S. The mutator, Mut, was carried in the high-loss stock but its presence was not recognized until a mutation arose at the Bz locus to give a bz allele which was mutable with Mut. It is designated as bz-m-rh. This mutation occurred in a derivative of the same F1 plant described above. Following five generations of inbreeding and one outcross to an a tester, a plant arose which proved to be heterozygous for the new responding allele. Subsequent tests of one of the intermediate generations revealed a plant containing Mut that continues to produce Bz → bz mutations, presumably to mutable alleles. Since the Bz/Bz parent was crossed to a bz-m mut tester, it has not yet been possible to determine whether the recurring bz mutations are to stable or mutable alleles.

The third system involves the responder, bz2-m in chromosome 1, originating in Neuffer's stocks and obtained from the Maize Coop. We found a new mutator, called Ac2, which interacts with bz2-m, as does Ac in Neuffer's stocks. Ac2 was detected in a cross of the 30418 F1 male parent with the Coop. bz2-m stock (a stable bronze without mutators). The source of the Ac2 is not yet clear; the few plants of high-loss background that we tested did not contain Ac2 but it may have been present in the high-knob stock.

The fact that all three of the mutable systems were associated, either in the occurrence of the mutator or the inception of new responders, with strains containing a large amount of heterochromatin may be of some significance. The 30418 F1 carried 4 B's as well as numerous knobs, including knobs on chromosomes 3, 4, and 1. Crosses of the 30418 F1 as male to female parents with recessive genes on those chromosomes showed that the high-loss phenomenon producing chromosome breaks was occurring in the 30418 male parent. Instability in the genome caused by repeated cycles of breakage initiated at the second microspore division may have been responsible for the origin of the new mutable systems. Such an origin was postulated by McClintock and supported by her studies and those by Doerschug. They found new Dt-like mutations arose in a dt plant which had a chromosome 9 undergoing the breakage-fusion-bridge cycle.

Phenotypes and dosage effects: The unstable phenotypes detected in the high-loss pedigrees show some similarities: in all three cases, reverse mutations in the presence of the controlling gene occur late in endosperm development giving fairly uniform small dots of full color on a colorless or bronze background. With increasing doses of the mutator, dots are more frequent in number but the time of mutation is not affected. In the a-m, Mrh system, kernels with three doses of responder and three doses of mutator are almost completely colored since the numerous dots are confluent, while in the bz2-m, Ac2 system such kernels have an average of more than 300 dots per kernel. The latter system has been most thoroughly analyzed; counts of dots per kernel gave the following results:

<u>Pedigree number</u>	<u>Genotype</u> ⁺	<u>Average dots/kernel</u>
34616 & 34611	Ac Ac ac	8.2 & 14
34616	Ac Ac Ac	303
34628	ac ac Ac	0
34614	ac ac ac	0
35418, 35497 & 35491	ac ac Ac Ac*	0.7, 2.5, & 3.7

+ All are bz2-m/bz2-m/bz2-m but vary in dosage of Ac2.

* Two Ac alleles contributed by male. Second Ac arose by transposition.

The action in aleurone cells of Ac2 on bz2-m is strikingly different from the interaction of McClintock's Ac with the same responding allele. Ac2 in a single dose usually gives no Bz dots while her Ac induces early reversions to Bz resulting in a coarse pattern when one dose is present. With two doses of Ac, there are many late mutations and with three doses, mutations are few and very late (Neuffer, MGCNL 28 & 29). The virtual absence of Bz dots in kernels with one dose of Ac2 was at first considered to be due to inactivation of Ac2 in the male gametes. We no longer subscribe to this view. It is apparent from the huge difference in mutation frequency between Ac2/Ac2/ac2 and Ac2/Ac2/Ac2 endosperms that the Ac2 contributed by the pollen parent is not inactive. Male gametes with two doses of Ac2 arise by transposition and kernels with two paternally derived Ac2 alleles have a low rate of mutation. The data presented above could be interpreted as indicating a lessening of Ac2 activity when Ac2 is introduced through the male. However, modifying genes affecting mutation rate are known to exist. The reported difference between kernels with two maternally contributed Ac2's and those with two Ac2 alleles of paternal origin may only reflect the effect of different constellations of modifiers. In support of this possibility is the average dot number of 12 found in kernels of another strain which possess two paternal Ac2's. There is no compelling evidence of pollen inactivation of Ac2. One dose of Ac2 simply does not produce the threshold necessary to induce mutability of the bz2-m allele and the exponential increase found in going from two to three doses represents a synergistic dosage relationship.

The A, Bz and Bz2 loci all affect mature plant and aleurone pigmentation. Plants carrying the a-m Mrh and bz2-m Ac2 systems were examined for sectors of A or Bz2 tissue in the stalk, husks or anthers. No Bz2 or A sectors were found and it was concluded that mutations are not induced by Mrh or Ac2 throughout the life cycle but appear to be restricted to the endosperm and germinal tissue. In bz-m Mut plants, very fine Bz stripes were found in the anther wall of plants carrying appropriate plant color factors, but no stripes were observed on the stalk or husks.

Linkage of mutator genes: If Mrh undergoes transposition to a heterologous chromosome or to a new site in the same chromosome, obviously it cannot be assigned a permanent position in the maize genome. That transposition takes place is indicated by the finding of kernels with sectors differing markedly in number of mutations to A, but we have not yet determined its frequency. However, transposition is low enough that no difficulty is encountered in obtaining an accurate map position in a specific chromosome. Immediately following the detection of Mrh we placed it in the long arm of chromosome 9. Since Mrh can only be scored in kernels homozygous for a-m or heterozygous for a-m and a, the most informative matings are those testcrosses where all of the resulting kernels possess the a-m allele. Data from crosses of a-m/a-m or a-m/a; Sh Wx Mrh/sh wx mrh females by a-m/a-m; sh wx mrh/sh wx mrh males are summarized below:

	(0)	(0)	(1)	(1)	(2)	(2)	(1-2)	(1-2)	
	Sh	sh	sh	Sh	sh	Sh	Sh	sh	
	Wx	wx	Wx	wx	wx	Wx	wx	Wx	
	Mrh	mrh	Mrh	mrh	Mrh	mrh	Mrh	mrh	Total
No. kernels	937	946	260	236	277	253	71	50	3030

Recombination values: $\frac{Sh}{Wx} - \frac{Mrh}{mrh} = 20.4\%$
 $\frac{Wx}{Mrh} - \frac{mrh}{mrh} = 21.5\%$

Linear order is Sh - Wx - Mrh
 Coincidence = 0.9

The waxy translocation stocks derived by Anderson and Longley were used in placing Mut and Ac2. Mut shows 32% recombination with wx in the backcross involving T2-9b (breakpoint 2S.18) and no linkage with waxy in backcrosses involving the other translocation stocks. More precise location of Mut on chromosome 2 awaits results of our crosses with chromosome 2 tester stocks. Ac2 crosses routinely give a low but variable percentage of kernels with no dots even when the mutator is introduced through the female parent. This complication must be kept in mind in the analysis of linkage data. Nevertheless, linkage of Ac2 on chromosome 8 became evident in backcrosses with T8-9(6673) (breakpoint 8L.35), where a recombination value of 19.8% was found for Ac2-wx in one test involving 526 bronze kernels. In a second test, an apparent recombination of 23.3% in a population of 304 bronze kernels was reduced to 12.5% when only the positive mutable class was analyzed. Although the recombination frequencies were variable, the data indicate linkage of Ac2 on chromosome 8. To further locate Ac2, tests were made with the ms8 gene in chromosome 8. Plants homozygous for bz2-m and heterozygous for Ms Ac2/ms ac2 were obtained. Self-pollinations of these gave a 1:1 ratio for bronze mutable and bronze stable kernels (653:669). The bz mutable kernels are either Ac2/Ac2/Ac2 or Ac2/Ac2/ac2 in constitution while the bz stable kernels are ac2/ac2/Ac2 or ac2/ac2/ac2. As noted earlier, one dose of Ac2 does not elicit response from bz2-m. Both classes of seed were planted and the ensuing sporophytes scored for male sterility to give the following data:

<u>mutable bronze</u>		<u>stable bronze</u>	
327 <u>Ms</u>	0 <u>ms</u>	170 <u>Ms</u>	152 <u>ms</u>

The ratio of Ms:ms is close to the expected 3:1. If Ac2 and Ms8 are completely linked, all plants from bz mutable kernels would be male fertile while a 1 Ms:1 ms ratio is expected from the bronze stable class of kernels. This proved to be the case. More significant is the fact that no male sterile plants came from bronze mutable kernels. The conclusion is inescapable that Ac2 and ms8 are very closely linked. The tight linkage of ms8 and Ac2 will be useful in determining the location of newly transposed Activators and in differentiating between the separation by crossing over of two tightly linked Ac's and a change in state. Tests with TB-8a have shown that ms8 is distal to the breakpoint at .7 in 8L. Our data with the T8-9(6673) show Ac2 is approximately 20 units from wx. If wx accurately marks the breakpoint at .35 in 8L, the consensus from all available data is that Ac2 lies in the distal .3 of the long arm of chromosome 8.

Interaction of mutator systems: The chart below summarizes the interaction of several maize mutators belonging to two-unit systems with a number of responding alleles. It is based partly on data presented in a similar chart (see Peterson, Maize Breeding & Genetics, 1978) and partly on tests performed in this laboratory. The + sign indicates a positive interaction and the - sign no response.

Mutator	a alleles			<u>Ds</u> ‡	<u>bz2-m</u>	<u>bz-m*</u>
	<u>a-m-rh*</u>	<u>a-standard</u>	<u>a-m, a-m(r)</u>			
<u>Mrh*</u>	+	-	-	-	-	Testing
<u>Dt</u>	-	+	-	-	-	-
<u>En=Spm</u>	-	-	+	-	-	-
<u>Ac=Mp</u>	Testing	-	-	+	+	Testing
<u>Ac2*</u>	-	Testing	-	-	+	-
<u>Mut*</u>	-	(-) to be confirmed	-	-	-	+

‡ The Ds tested is located between C-I and Wx on chromosome 9.

* New controlling and responding elements reported here.

In most cases, there is a high degree of specificity in these systems, i.e., a particular responder at a given locus is activated by only one mutator and a particular mutator affects only one responder. The exceptions identified in the chart are in the horizontal row opposite $\underline{Ac=Mp}$ and in the vertical column under $\underline{bz2-m}$. \underline{Ac} interacts with \underline{Ds} and with the $\underline{bz2-m}$ allele, while $\underline{bz2-m}$ is induced to mutate by both \underline{Ac} and $\underline{Ac2}$. Without information supplied by $\underline{Ac2}$, it might be concluded that a standard \underline{Ds} element is present at the $\underline{bz2-m}$ allele--i.e., $\underline{bz2-m} = \underline{Bz2 Ds}$. The differential interaction of the two responders (\underline{Ds} and $\underline{bz2-m}$) with $\underline{Ac2}$ (+ with $\underline{bz2-m}$ and - with \underline{Ds}) indicates that the suppressive element at the $\underline{Bz2}$ locus is not identical to a standard \underline{Ds} . Furthermore, the mutators \underline{Ac} and $\underline{Ac2}$ differ in their specificities. Unlike \underline{Ac} , $\underline{Ac2}$ has no effect on standard \underline{Ds} , as indicated in the above chart, or in the $\underline{wx-m1}$ ($\underline{Wx Ds}$) kernels obtained from the cross of $\underline{wx/wx-m1}; \underline{Bz2/bz2-m}; \underline{Ac2/ac2}$ female parents by $\underline{wx/wx}; \underline{bz2-m/bz2-m}; \underline{Ac2/Ac2}$ males. Both the $\underline{bz2}$ stable kernels (one dose of $\underline{Ac2}$) and the $\underline{bz2}$ mutable (three doses of $\underline{Ac2}$) had stable waxy endosperms. $\underline{Ac2}$ in one or three doses obviously did not interact with the \underline{Ds} element associated with $\underline{wx-m1}$ to give $\underline{wx} \rightarrow \underline{Wx}$ reversions.

Tests with $\underline{wx-m9}$ ($\underline{Wx Ac}$) revealed that no change in endosperm phenotype occurred in $\underline{wx/wx/wx-m9}$ kernels having four doses of $\underline{Ac2}$ (obtained by using a transposed $\underline{Ac2}$). In summary, $\underline{Ac2}$ has no effect on \underline{Ds} in its standard position in chromosome 9 between \underline{Sh} and \underline{Wx} , fails to interact with the \underline{Ds} element in $\underline{wx-m1}$ and likewise does not affect the \underline{Ac} gene in the autonomous $\underline{wx-m9}$ ($\underline{Wx Ac}$) mutable system. It will be instructive to see if McClintock's \underline{Ac} controls autonomous mutability induced by transposition of $\underline{Ac2}$ if indeed such occurs. Our present evidence leads us to conclude that $\underline{Ac2}$ is not a modification or change in state of the \underline{Ac} studied by McClintock but represents an independent controlling element which shares with \underline{Ac} only the ability to induce mutations of $\underline{bz2-m}$. Additional evidence supporting this conclusion is given below in summary statements.

1. One dose of \underline{Ac} elicits interaction from a responding gene while one dose of $\underline{Ac2}$ is ineffective.
2. A dosage effect on time of mutation is present with \underline{Ac} but does not occur with $\underline{Ac2}$.
3. \underline{Ac} is active in mature plant tissues; $\underline{Ac2}$ is not.

Transposition: Our observation that $\underline{bz2-m}$ kernels with one $\underline{Ac2}$ have no dots, that those with two doses of $\underline{Ac2}$ have a low number while those with three doses have a high mutation rate made it possible to screen readily for transpositions of $\underline{Ac2}$ by looking for exceptional kernels with a mutation rate much higher than that in sibling kernels. For example, in the cross of $\underline{bz2-m/bz2-m Ac2/Ac2}$ females by $\underline{bz2-m/bz2-m ac2/ac2}$ males, the genotypic constitution of the endosperm of F1 kernels would be $\underline{bz2-m/bz2-m/bz2-m Ac2/Ac2/ac2}$. They will have a low number of dots per kernel. The embryos of the kernels with low dot number will be $\underline{bz2-m/bz2-m Ac2/ac2}$ and in testcrosses they produce ears with a ratio of 1 \underline{bz} mutable (low dots):1 \underline{bz} stable. Occasionally, however, kernels are found with a much higher number of dots. Two or three such exceptional kernels may occur on a single ear. If they are clustered together on the ear, it is assumed that they all arose from one premeiotic mutational event. One seed from a cluster and all solitary exceptional kernels were grown and testcrossed as the female parent to determine their genotypic constitution for $\underline{Ac2}$. A ratio of 1 \underline{bz} mutable (high dots):1 \underline{bz} stable in a testcross of plants grown from these exceptional kernels indicates heterozygosity for two closely linked $\underline{Ac2}$ genes while ears with 3 \underline{bz} mutable:1 \underline{bz} stable ratios are expected if two independent $\underline{Ac2}$ genes are heterozygous. In the latter case, one-third of the mutable class should have a high dot number, since the kernels would possess four \underline{Ac} 's and two-thirds should have a low dot number, comparable to that of the mutable class in testcrosses of sibling plants with one $\underline{Ac2}$ since both would have two doses of $\underline{Ac2}$ in the endosperm. The

bronze stable class would have no Ac2. Good agreement was found between observed and expected frequencies. If, following transposition, two Ac2 alleles lie adjacent to one another in the same chromosome and there is no or little crossing over between them, a testcross ratio of 1 high dotted:1 no dots is expected. Deviation from a 1:1 ratio of high dots:no dots, accompanied by the appearance of a low-dotted class, would occur if the two Ac2's were situated in the same chromosome but not in juxtaposition and were separable by crossing over. The frequency of the low-dotted class would depend on the amount of crossing over. If testcross ratios are the sole criterion, it is not possible to differentiate between two linked Activators with 50% recombination and two which are unlinked. The possibility that the infrequent low-dotted kernels found in the testcross progeny of plants with two linked Ac2's arise not from crossing over between the two Ac2 loci but represent changes in "state" can and will be answered by following the closely linked ms8 gene.

The great majority of the kernels from the reciprocal cross of bz2-m/bz2-m ac2/ac2 females by bz2-m/bz2-m Ac2/Ac2 males have a stable bronze phenotype since they possess endosperms with one dose of Ac2. However, a low and varying percentage exhibit Bz dots ranging discontinuously in number from one to more than 200 per kernel. Upon testing, most of the kernels with low numbers of dots proved to be heterozygous for two Ac2's while kernels with a high number of dots generally were heterozygous for three, four or even five Ac2 loci. If the different Activators were randomly inherited, ascertainment of genotype was relatively precise and straightforward, but linked Activators complicated the analysis. Despite this difficulty, progress has been made. Plants have been found with three Ac2 loci which assort independently and others where two of the three Ac2's are linked and the third is unlinked.

Exceptional kernels with a very high mutation frequency have given rise to plants heterozygous for three, four and five independent Ac2 genes. A number of testcrossed ears have ratios suggesting heterozygosity for two or three independent Ac2's and two that are linked but an exact genotypic determination calls for additional testcrossing. As might be anticipated from an exponential dosage effect, the number of dots in endosperms with six or more Ac2's is often so great that the pigment from closely spaced dots coalesces to form what appears to the unaided eye as self-colored tissue.

The production of male and female gametes with as many as four or five Ac2's by a plant known to be homozygous for a single Ac2 gene indicates that there may be repeated replications (amplification) of the DNA comprising the Ac2 locus in one mitotic cycle or else there is a stepwise increase in Ac2 number during a series of mitoses. Regardless of the mechanism by which the multiple copies of Ac2 arise, they can be inserted at various positions in the parental chromosome or transposed to heterologous chromosomes. The two alternatives occur in approximately equal frequencies in those instances where the change is from one to two Ac2's (12 cases of two linked Ac2's:13 cases of two independent Ac2's).

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Presence of additional RNA and DNA during microsporogenesis of Sikkim Primitive

Abnormal meiotic patterns were found in some cells during our study of the chromosome morphology of Sikkim Primitive. These cells contained additional DNA and, in some cases, a second nucleolus. The modified Stockinger and Kellner staining procedure was used to demonstrate the presence of two nucleoli, while

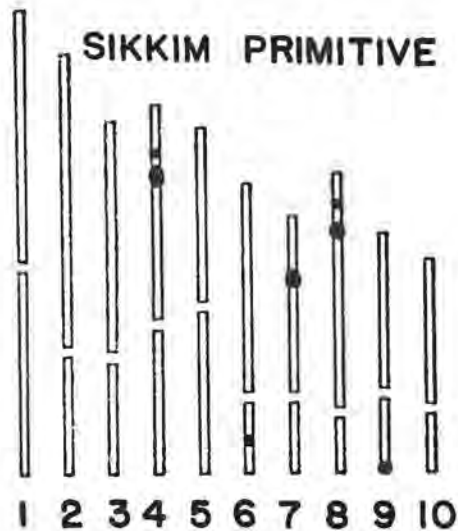
presence of additional DNA was shown with the Feulgen stain. The amount and morphology of this additional DNA is extremely variable, ranging from a tight heterochromatic knot to small and irregular fragments scattered in the cytoplasm. However, this additional DNA did not have the appearance of supernumerary chromosomes. We estimate that the cells with additional DNA occurred at an overall rate of 8% in our samples while cells which had two nucleoli were much less common. Our observations at pachytene indicate that the cells containing two nucleoli have in general two functional nucleolar organizers, one found at chromosome 6 and the second one on a small fragment of additional DNA. When present, the two nucleoli seem to be in synchrony in the sense that their size in a given cell is identical but some significant exceptions were found and most of the cells with smaller nucleoli did not show the presence of the second nucleolar organizer. It was interesting to discover the persistence of a nucleolus at metaphase. At this stage the nucleolus is rarely found associated with DNA. A vesicle, which appears to be a nucleolus, is present in some cases at anaphase where severe disruptive effects seem to be caused by its presence. Our data indicate that the abnormal cells are seen most commonly before the first meiotic division and possess in general a volume larger than the cells which appear normal. In these cells, it is not uncommon to find some irregular, non-staining structures which have not yet been characterized. The fate of the additional DNA is difficult to trace through meiosis, but we expect to find more information on this question in the mitotic study of the selfed plants. Persistent nucleoli and additional DNA levels during cell division are two characteristics which have been frequently observed in cancer cells but remain essentially unexplained events. The etiology and significance of these findings is currently under investigation in our laboratory.

John P. Peeters

Sikkim Primitive

Last year one of us reported on maize collections made while on sabbatical in India. This laboratory has continued to study the Indian landrace Murli from Sikkim and the Darjeeling region of the Eastern Himalayas. In the literature this landrace has been called Sikkim Primitive because of the resemblance to the reconstructed ancestor of P. C. Mangelsdorf. When collected in Sikkim the ears are small "Ladyfinger-like" specimens. Last year the plants did not flower for us before the killing frost because of their pronounced short day requirements but this year we did bring plants to flower in early September. These plants gave us an ear which is comparable to an eight- to ten-inch commercial open pollinated popcorn. That the small cob and slightly protruding glumes of Sikkim Primitive were not genetically fixed was a surprising finding for us. Sikkim Primitive could have any number of yellow endosperm commercial popcorns as its founder stock. In no way is Sikkim Primitive a long lost ancestral stock of maize, but a phenotypic response to a unique environment with adaptation to short day conditions of the tropics. In an attempt to fingerprint this landrace, we have worked out its knob pattern (see diagram) and would be interested in knowing if this pattern is similar to any commercial yellow endosperm popcorns.

The really interesting finding of this study is reported in the first report. It appears that this local landrace, adapted to a high elevation cloud forest region of the tropics, shows pronounced abnormalities in cell organization when grown under radically different conditions here in Boston (The Waltham Field Station, Waltham). Like most corns or teosintes collected in the tropics this collection possesses a short day photoinduction response. Grown under the long summer days, it failed to flower, but when the day length was shortened it plunged



the whole plant from vegetative growth phase to reproductive phase. The suddenness with which this happened has disrupted the timing of cellular processes and we observed in microsporogenesis double nucleoli and abnormal amounts of RNA and DNA. The question is: Can these physiologically induced abnormalities be used as a model for other abnormal cell developments? The hypothesis is that the rapid induction has stressed cell physiology and the response has resulted in increased amounts of RNA and DNA. The timing systems of the cells have in essence become unregulated and therefore abnormal.

We are interested if other researchers have observed similar phenomena when radically changing the environment of maize they have obtained from elsewhere, preferably the short day tropics. Currently, we are investigating the flint maize Tista Mendi grown in the same region to see if it possesses a similar response. This parallel habitat race from the same region should serve as a control to our hypothesis of physiological disruption as the cause of double nucleoli and other abnormalities in microsporogenesis.

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Genetics of environmental resistance and super-genes

We use a kind of modified stronger rapid-aging test to study heat tolerance in seeds. We use 48 C for 96, 104, and 120 hours within an incubator with water on the bottom. The entries are put within paper bags. After this preconditioning the seeds are put within paper dolls in a germinator in the usual way, and counted for dead and alive seeds.

A set of 18 translocations with wx and two inversions, Inv9a with wx and Inv2a with gl2, listed in MNL 40:184-185, 1966, not yet in uniform background, were used. They were crossed with IAC Maya latente, an open pollinated variety with a source of heat and frost tolerance from Michoacán 21 Comp 1-104, and a flint single cross from Brazilian Cateto. The F2 progenies were compared in split-plot design with paired waxy and non waxy seed and the F3 of glossy-2 and non glossy-2 by the

treatment described above. The method of analyzing statistically the results was that described by L. T. de Miranda (MNL 55:18-19, 1981). The results showed linkages of tolerance to heat from latente, for short, lte1, with wx T2-9b (2S.18; 9L.22) and Inv2a (2S.7; 2L.8), both in chromosome 2. With the Cateto there was very strong linkage with wx T9-10b (9S.13; 10S.40), for chromosome 10. We suggest calling it Lte2 by analogy to the known latente.

To check this last result the original MNL marker stock, g R sr2, was crossed to the component lines of the flint Cateto single cross C1 and C2. Only selfs were obtained. C1 has no plant color, and crossed to supersweet (a1 sh2) gave color. C2 has anthocyanin plant color. With the marker stock both segregated as lacking two pairs of factors: C1 gave a nearly perfect fit, 761 colorless to 976 colored, or 0.4381 colorless (expected 0.4375). C2 gave a poorer fit, 575 to 652, or 0.468 colorless, with more quantitative effects. The data are shown in Table 1, and the analysis in Table 2. We didn't measure the effect of g or sr2 in seed heat tolerance. The results show linkage of a factor for heat tolerance from Cateto with r, for which is proposed the symbol Lte2.

Table 1. Total seed of selfs from crosses of C1 and C2 with marker g R sr2, relative survival of r and R seeds measured by germination after pre-conditioning at 48 C and 100% R.U. Results from seven different experiments. Numerator, seeds alive, denominator total seed in the rep. Each line an ear.

	Hours of Pre-conditioning							
	0		96		104		120	
	r	R	r	R	r	R	r	R
C1-1	94/ 88	25/ 30	21/ 31	(7/ 34	6/ 29)*	17/ 30	8/ 28	
2	109/141	31/ 36	35/ 46	33/ 36	38/ 48	25/ 37	33/ 47	
3	134/163	38/ 41	44/ 54	38/ 47	44/ 55	33/ 46	41/ 54	
4	107/149	16/ 35	21/ 48	10/ 36	5/ 49	4/ 36	4/ 52	
5	182/209	53/ 62	50/ 72	31/ 57	29/ 73	23/ 63	17/ 64	
6	79/117	22/ 25	27/ 39	13/ 27	17/ 39	11/ 27	14/ 39	
7	56/109	14/ 20	20/ 34	11/ 16	14/ 37	9/ 20	11/ 38	
Total	761/976	199/249	218/324	143/253	153/330	122/259	128/322	
C2-1	58/ 53	17/ 28	18/ 27	(- -	- -)*	7/ 30	6/ 26	
2	78/ 81	23/ 27	17/ 27	14/ 26	14/ 28	10/ 25	9/ 26	
3	154/170	47/ 50	50/ 57	36/ 52	35/ 56	42/ 52	44/ 57	
4	96/ 91	12/ 32	10/ 30	6/ 30	10/ 32	4/ 34	4/ 29	
5	71/ 82	12/ 23	12/ 27	4/ 24	3/ 26	3/ 24	1/ 29	
6	76/105	21/ 24	25/ 36	16/ 25	18/ 34	11/ 27	20/ 35	
7	42/ 70	4/ 14	9/ 23	3/ 13	7/ 24	3/ 15	11/ 23	
Total	575/652	136/198	141/227	79/170	87/200	80/207	95/225	

*Preconditioning for 144 hours was too strong, observed in the first experiment and abandoned in subsequent trials.

Table 2. Results from table 1 fitted to the equation $X' = (100 + Y)^{1-\alpha}$ being $X' = X+Y$, and $X = \%r$ alive, and $Y = \%R$ alive. A is the antilog of $2(1-\alpha)$, or $\%$ of r germinating when RZ is one, or in logs 0 (zero).

	A	t	r
Fitted by the minimum squares method			
C1 $X' = (100 + Y)^{0.2844 \pm 0.0643}$	3.70	4.422**	0.722**
C2 $X' = (100 + Y)^{0.2686 \pm 0.1166}$	3.45	2.305*	0.477*
Fitted by the minimum products method			
C1 $X' = (100 + Y)^{0.3939 \pm 0.0609}$	6.13	6.468**	0.722**
C2 $X' = (100 + Y)^{0.5625 \pm 0.1105}$	13.34	5.090**	0.477*

* significant at $P < 0.05$.

** significant at $P < 0.01$.

The IAC Maya latente was obtained as follows. We crossed the line Michoacán 21 Comp 1-104 with adapted material and selfed two times to get pure and segregating families. The families were tested in the field against frost. Remnant seed of the plants more tolerant to frost were planted, and recombined and compared with adapted material for tolerance to heat in seeds by the test described above. We backcrossed to IAC Maya three more times, selfing two times between each backcross and testing by the heat test in seeds. Most plantings were made out of normal season and occasional frosts confirmed that heat and frost tolerance came together.

R. M. Castleberry and R. J. Lerette (Proc. 34th Annu. Corn and Soybean Res. Conf., 1979) obtained adapted latente lines from the same source as ours, using basically synchrony of pollen shedding and silking following early season drought stress as the criterion for selection. Our observations and preliminary physiological measures made in our institute agree with their conclusions.

The available information suggests that the genetics of environmental resistance up to now follows the genetics of control of distribution of anthocyanin color. Latente-1 (lte1) from Michoacán follows the R2 B super-gene and must have together at least two more loci. A factor controlling the SH-SS (Levitt's hypothesis) tolerance type is the lte1 proper, that we tested for heat and frost, a factor for abscisic acid control of stomata. Latente-2 (lte2) goes with the locus of the R1 super-gene and adds tolerance to heat. It is probably an hydrophobic protein type of tolerance, definitely different from that of Michoacán.

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Genetics of insect resistance and super-genes

A. C. Waiss et al. (J. Econ. Entomol. 72:256-258, 1979) reported that a flavone glycoside from corn silks of Zapalote Chico (ZC) had an antibiotic activity toward corn earworm, Heliothis zea (Boddie). Our source of resistance was ZC #2451, reported by B. R. Wiseman et al. (Environ. Entomol. 7:777-779, 1978) as restricting penetration in the silk channel. Sixteen translocations and one inversion marked by wx from the set listed in MNL 40:184-185, 1966, crossed and backcrossed once to IAC Maya latente before selfing, were used as markers. These

markers were crossed to ZC. This cross was then "backcrossed" to IAC Maya wx (M wx) obtained by crossing and backcrossing thrice to IAC Maya, a commercial wx cultivar imported via Japan from South Africa. The Wx and wx segregates from the same ear, preferentially in the same reps, were planted in split-plot with five reps, late in the season to suffer heavier attack. The ears were harvested unhusked and a counting was done of the number of ears with and without exit holes of larvae. Table 1 presents data for totals only, and summarizes statistical

Table 1. Underlined, our identification number. Marker involved in the cross (Zapalote Chico x IAC Maya latente with marker) x IAC Maya wx. Good stands for ears with husks intact with no hole of *Heliothis zea* (Boddie) larvae. Bad stands for exit hole present. A + sign in χ^2 indicates Wx endosperm or Zapalote Chico piece of chromosome better than IAC Mayas or Wx-wx contrast, - the reverse. Total of five reps.

Marker involved	Number of ears				χ^2	
	<u>wx</u>		<u>Wx</u>		interaction	interaction x reps
	good	bad	good	bad		
<u>2</u> wx Inv 9a (9S.70; 9L.90) ...	39	78	24	66	-1.068	8.185
<u>3</u> wx 1-9c (1S.48; 9L.22) ...	24	82	23	67	+0.227	6.101
<u>5</u> wx 1-9-8389 (1L.74; 9L.13)+ ..	27	49	30	47	+0.193	4.715
<u>6</u> wx 2-9b (2S.18; 9L.22) ...	21	106	20	80	+0.454	8.970
<u>7</u> wx 3-9c (3L.09; 9L.12) ...	45	76	24	73	-3.856*	11.673*
<u>10</u> wx 4-9 (4S.27; 9L.27) ...	26	72	18	12	-1.116	7.774
<u>9</u> wx 4-9-5657 (4L.33; 9S.25) ...	19	103	40	84	+9.389**	4.745
<u>8</u> wx 4-9b (4L.90; 9L.29) ...	48	72	23	95	-11.954**	1.540
<u>12</u> wx 5-9c (5S.07; 9L.10) ...	48	102	40	92	-0.094	5.586
<u>13</u> wx 5-9d (5L.14; 9L.10) ...	21	58	18	71	-0.949	2.114
<u>14</u> wx 6-9a (6S.79; 9L.40) ...	41	83	16	45	-0.896	9.213
<u>15</u> wx y6-9b (6L.10; 9S.37) ...	19	75	27	50	+4.748*	5.119
<u>16</u> wx 7-9a (7L.63; 9S.07) ...	28	68	26	69	-0.080	2.788
<u>17</u> wx 7-9-4365 (7 cent; 9 cent)+ ..	10	23	6	20	-0.624	2.513
<u>18</u> wx 8-9d (8L.09; 9S.16) ...	23	96	20	65	+0.526	4.639
<u>19</u> wx 8-9-6673 (8L.35; 9S.31) ...	38	77	30	76	-0.582	2.567
<u>20</u> wx 9-10b (9S.13; 10S.40) ..	17	52	18	43	+0.390	1.859

+ 5 had two reps with backcross and three with selfs, 17 had one rep with backcross and four with selfs.

* Significant at $P < 0.05$

** Significant at $P < 0.01$

analyses of the interactions. O. Ceska et al. (MNL 54:118-119, 1980) reported that in dried husks of a B P1 P-WW plants the flavone pathway was blocked by the P-WW allele. Since some of the markers had also P-WR segregating, a further decomposition of the sources of variation was possible since M wx and ZC are P-WW. This is presented in Table 2. With the exception of entry 15 the numbers differ from Table 1 because not all reps segregated for P-WW and P-WR.

In Table 1 the plus sign before χ^2 shows that there were significant differences in favor of ZC for T4-9(5657) at 4L.33 and for T6-9b at 6L.10; the minus sign before χ^2 indicated significant differences in favor of M wx for T4-9b at 4L.90 and for T3-9c at 3L.09. The significant interaction for reps in T3-9c suggests that the variety is segregating at this locus. In Table 2, using only the entries that segregated for P-WW and P-WR, appears an effect for ZC in T2-9b, 2S.18. Joining the three entries which did not have individual effect, there appears an effect for ZC in favor of P-WW, almost reaching the 5% level. The

Table 2. Same as in table 1, with the difference that only entry reps which were segregating P-WW and P-WR were used. Entries 3, 18 and 20, which didn't have individual effects were pooled to measure the effect in P-WW or P-WR present, separately. Number of reps 15, 5; 6, 3; 3, 2; 18, 2; 20, 3. The direct contrast P-WW minus P-WR was not significant although positive for P-WW in Wx and negative for P-WR in wx; that is, in the same direction as for 3' + 18' + 20' in this table.

Marker involved	Number of ears				χ^2			
	<u>wx</u>		<u>Wx</u>		interaction	interaction x reps		
	good	bad	good	bad				
<u>15</u> <u>wx</u> <u>y6</u> - <u>9b</u>	(6L.10; 9S.37)	<u>P-WW</u>	14	51	20	38	+2.568	7.070
"	(" ")	<u>P-WR</u>	5	24	7	12	+2.434	2.401
<u>6'</u> <u>wx</u> <u>2</u> - <u>9b</u>	(2S.18; 9L.22)	<u>P-WW+P-WR</u>	6	67	12	44	+4.605*	0.863
"	(" ")	<u>P-WW</u>	4	39	6	21	+2.261	1.512
"	(" ")	<u>P-WR</u>	2	28	6	23	+2.474	1.669
<u>3'</u> <u>wx</u> <u>1</u> - <u>9c</u>	(1S.48; 9L.22)	<u>P-WW+P-WR</u>	16	50	19	39	+1.105	1.319
"	(" ")	<u>P-WW</u>	13	48	17	35	+1.864	1.742
"	(" ")	<u>P-WR</u>	3	2	2	4	-0.782	1.976
<u>18'</u> <u>wx</u> <u>8</u> - <u>9d</u>	(8L.09; 9S.16)	<u>P-WW+P-WR</u>	8	32	6	17	-0.313	0.003
"	(" ")	<u>P-WW</u>	5	17	5	8	+0.991	0.436
"	(" ")	<u>P-WR</u>	3	15	1	9	-0.233	0.440
<u>20'</u> <u>wx</u> <u>9</u> - <u>10b</u>	(9S. 13; 10S.40)	<u>P-WW+P-WR</u>	16	34	15	27	+0.141	0.468
"	(" ")	<u>P-WW</u>	5	18	9	17	+0.991	4.096
"	(" ")	<u>P-WR</u>	11	16	6	10	-0.044	1.077
<u>3'+18'+20'</u>	(1S.48+8L0.9+10S.40)	<u>P-WW+P-WR</u>	40	116	40	83	+1.591	0.760
"	(" ")	<u>P-WW</u>	23	83	31	60	+3.764(*)	0.519
"	(" ")	<u>P-WR</u>	17	33	9	23	-0.312	2.036

* Significant at $P < 0.05$

(*) Nearly significant at $P < 0.05$

results suggest that the genetics of resistance follows the genes or super-genes responsible for sunlight-independent anthocyanin or anthocyanin-like (glycosides) including a specific factor, sm, controlling silk coloration. It seems that bm and bz are not involved.

For the factors of earworm resistance located in ZC we propose the symbols and indicate the probable regions as: Zer1 (Tu c2); Zer2 (P1 Bh sm); Zer3 (Br2); Zer4 (p). For M wx they should be: Mer1 (Tu c2); Mer2 (a3 a1).

The basic factors seem to be in chromosome 4 for both, in chromosome 6 for ZC, and in chromosome 3 for M wx. They are dominants or complementary dominants.

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Linkages of root characteristic from Pueblo maize II

In MNL 54:18-19 the senior author reported linkage of Su Asr1 as being $p = 43.0 \pm 2.2$. From these experiments was isolated an su su Asr1 Asr1 line. This line was pollinated by Ga-S Su from the MGCSC. In our prior work the classes were those without any seminal root or those with one or more seminal roots, and there was a significant lack of individuals of the dominant class. In the results presented here, the classes are: zero or one seminal roots, and two or more seminal roots. In eight families, three were found by a chi-square test for homogeneity to be significantly different from the mean. They were discarded for the calculation of recombination because of excess or lack in Asr classes. We have no explanation for this variation.

Family	Zero or one seminal root		Two or more seminal roots		n
	Asr1 Su	Asr1 su	asr1 Su	asr1 su	
1	166	22	120	23	331
2	138	25	86	24	273
3*	88	20	105	16	229
4	111	20	64	12	207
5*	164	24	55	10	253
6	127	22	114	22	285
7*	80	13	118	19	230
8	102	22	72	10	206
Total	976	168	734	136	2,014
Total less *	644	111	456	91	1,032

The values of p obtained by maximum likelihood were: Asr1 ga 16.0 ± 2.7 , Ga-S Su 31.0 ± 2.0 and Asr1 su 45.8 ± 2.2 . Within limits of experimental error they agree quite well among them and with values already reported elsewhere.

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Pollen grain size in perennial teosinte

In 1979 diploid and tetraploid perennial teosintes were grown from seed collected in the state of Jalisco and established in the field nearby Chapingo, state of Mexico. Pollen was collected on an individual plant basis and the size of 100 individual grains per plant was measured. For each grain two measurements were obtained, the largest diameter and that of the diameter perpendicular to the middle point of the former. The average value of these two diameters, in micrometers, was considered as the size of a particular pollen grain.

Table 1. Pollen grain size and its variation in plants of diploid and tetraploid perennial teosintes grown at Chapingo, Mexico

Plant	Number grains measured	Range in diameter (μm)	Mean diameter (μm)
DIPLOID			
II-80-1	100	53.5-72.3	65.0
II-80-3	100	57.8-74.2	65.6
II-80-4	100	57.3-73.3	63.0
II-80-6	100	60.1-75.1	68.2
II-80-7	100	57.3-70.0	63.9
II-80-9	100	59.2-77.0	68.0
TETRAPLOID			
I-80-1	100	59.2-72.8	66.8
I-80-2	100	59.2-70.4	65.0
I-80-7	100	50.7-63.9	58.4
I-80-9	100	58.2-74.2	65.9
I-80-11	100	49.8-67.2	60.3
I-80-13	100	61.5-75.6	68.5
I-80-26	100	59.2-70.4	63.9
I-80-30	100	60.6-77.0	68.7

The results given in Table 1 show some differences in average grain size among plants, within both types of teosinte. However, it seems that there is practically no difference in grain size between the two populations. The overall average difference is only of 1 μm and there is a great deal of overlapping in the size frequency distribution. Furthermore, two tetraploid plants produced smaller average grain size than the smallest average found among diploid plants.

These results are one more exception to the general rule that doubling of the diploid chromosome number increases pollen grain size. It is known that maize and annual teosinte, when artificially tetraploidized, form much larger pollen grains than the parental plants. Therefore, it would seem as if, in the case of the perennial teosinte, once the autotetraploid was originated natural selection had favored the smaller size, until the original size of the pollen grains of the ancestral diploid plants had been reached. At present it would be difficult to explain, with certainty, what kind of advantage was conferred to the tetraploid populations by this change in pollen grain size. Some experiments trying to answer these questions have already been started in our laboratory at Chapingo.

T. Angel Kato Y.

Multivalent frequencies in tetraploid perennial teosinte

Using the same group of tetraploid perennial teosinte plants mentioned in the previous work, young tassels from three stems of each plant were fixed. In each of these tassels two spikelets, located in different branches, were selected and in each spikelet 50 cells at the diakinesis stage were analyzed. A summary of the data obtained is presented in Table 2. The statistical analysis of these data

TABLE 2. Frequencies of univalents, bivalents, trivalents and tetravalents observed in diakinesis in plants of the tetraploid perennial teosinte grown at Chapingo, Mexico

Plant	No. of associations				No. cell examined	Associations per cell			
	I	II	III	IV		I	II	III	IV
I-79-2	24	2491	14	1738	300	0.08	8.30	0.04	5.79
I-79-4	63	3182	23	1376	300	0.21	10.60	0.07	4.58
I-79-5	45	2530	21	1708	300	0.15	8.43	0.07	5.69
I-79-7	208	3403	66	1197	300	0.69	11.34	0.22	3.99
I-79-9	282	3092	78	1325	300	0.94	10.30	0.26	4.41
I-79-11	341	2897	119	1377	300	1.13	9.65	0.39	4.59
I-79-19	36	2776	16	1591	300	0.12	9.25	0.05	5.30
I-79-26	55	3068	11	1444	300	0.18	10.22	0.03	4.81
I-79-29	159	2610	67	1605	300	0.53	8.70	0.22	5.35
I-79-30	31	2708	15	1627	300	0.10	9.02	0.05	5.42

showed significant differences among plants but not within plants, i.e., among spikelets of the same plant. These results seem to open the possibility for cytological selection of plants with low multivalent frequency and in this way to obtain a completely diploidized tetraploid population. This work is in process in our laboratory.

T. Angel Kato Y.

Knob constitution of perennial teosinte

Chromosome-knob constitutions were determined in several plants of diploid perennial grown at Chapingo from seed collected at San Miguel, Sierra de Manantlán in the state of Jalisco, and also observations in this regard were made in plants of the tetraploid perennial teosinte of Cd. Guzmán, Jalisco.

The tetraploid plants were almost knobless and only showed terminal small or very small (large chromomeres) knobs on the pachytene chromosomes. No attempts have been made to determine which chromosomes possessed them. Surprisingly, the diploid plants appeared to be different, since small and medium-size knobs were present in many chromosomes, all knobs being terminally located. Knobs were found on the following chromosome arms: small knobs on 1S, 2S, 2L, 3S, 4L, 5S, 5L, 6L, 7L, and 8L; medium knobs on 2S, 3S, 3L, 5L, 6L, 7L, 8L, and 9L. In neither case were abnormal chromosome-10 and B-type chromosomes nor internal knobs observed.

These preliminary results seem to suggest that the diploid perennial teosinte from the Sierra de Manantlán is similar to the southern Guatemala teosinte regarding the knobbed positions, although the latter has larger knobs than the former.

T. Angel Kato Y.

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Further studies of F1 hybrids of maize x diploid perennial teosinte

Last summer 152 F1 hybrid plants of maize x diploid perennial teosinte were grown in the field. The maize parent included three varieties: Chuen-Hwa, Tze-Tao-Shwei, 80-105. All of them came from China. When the plants were five weeks old, variations in vigor were apparent from cross to cross. For example, F1 plants of the cross Chuen-Hwa x diploid perennial teosinte were the most vigorous individuals and those of 80-105 x diploid perennial teosinte were the least vigorous group in terms of number of tillers produced as well as plant height. When the plants were 10 weeks old, 50 percent of the plants for each cross, approximately 25 clones, were cut off and weighed together. It was found that the cross Chuen-Hwa x diploid perennial teosinte also produced the largest fresh weight. On the average each clone weighed about 5000 grams, which is about six times the weights of the parental varieties. It was also observed that this cross produced the largest number of tillers per clone. One week after cuttings, almost 100 percent of the root stocks (rhizomes) regenerated into young shoots. Again the regenerations of the cross Chuen-Hwa x diploid perennial teosinte were the most profuse. In contrast, none of the parental maize stocks were capable of regenerations. Since regeneration of grasses is a reliable indicator of perennialism, it appears reasonable to say that the F1 hybrids of the above crosses are perennial. On the basis of this observation that all of the hybrids of the crosses were perennial, it seems that the perennial attribute is dominant.

Shaver (1964, 1967) found that F1 plants of 4n maize x 4n perennial teosinte were annual and he proposed that the perennialism in the 4n teosinte was governed by a single recessive gene (*pe*). If this gene became homozygous in a 20-chromosome maize by going through breeding procedures, it exerted lethal effect. In the last few years, we have crossed 4n perennial teosinte with diploid maize and the teosinte was employed as seed-parent. It was consistently observed that the triploid F1 hybrids, with two genomes from teosinte and one from maize, were perennial. During summer months, each clone produced profuse grassy tillers. Some of the clones were maintained for more than five years by transferring them to the

greenhouse in the winter. However, if they were left in the experimental field here in the Boston area, they died in the winter. Hence, the above suggests that perennialism in 4n teosinte is quantitatively inherited. A triploid plant possessing two perennial genomes manifested perennial habit.

Male inflorescences of three to five F1 hybrids of each of the aforementioned crosses were collected and fixed with aceto-alcohol fixative. By standard aceto-carmine squash technique, it was found that all the terminal knobs of the chromosomes of the teosinte parent were present. No chromosome aberrations of any kind were identified. However, univalents at diakinesis, laggards at anaphases I and II of the microsporocyte divisions were observed. When pollen fertility of the F1 plants was examined with the aceto-carmine staining procedure, it showed that more than 90% of the pollen grains were normal or fertile. In order to have advanced generations of the crosses, the F1 hybrids were backcrossed as seed parent to the maize parental variety. The percentage of seed set was above 60.

Y. C. Ting and M. K. Yu

Further studies on maize anther culture in vitro

Since the writing of our last research report (Plant Sci. Lett. 23:139-145, 1981), more experiments on maize anther culture in vitro were carried out. The medium employed was also zheng-14 and the materials were maize Dan-Sun 91 and its derivatives. Anthers, approximately 5000 in number, were inoculated. It was found that the average response, in terms of callus initiation and embryoid growth, was about 15 percent. More than 50 percent of the embryoids could grow into plantlets without any change of plant hormones in the medium. Out of the calli, 15 new lines were established. Seven of these new lines have been subcultured and maintained in the culture room for several months.

Regenerated plantlets, however, varied widely in their gross morphology. It was difficult to determine whether these variations were brought about by mutations or by adverse environments. Over 50 percent of them manifested a certain degree of chlorophyll deficiency. When they grew in the greenhouse during winter, they produced predominantly pistillate inflorescences. Fully developed kernels were occasionally obtained upon crossing these haploid plants with pollen from diploid Wilbur's Flint. Nevertheless, the percent of seed set was very low.

When male inflorescences of Dan-Sun 91 were immersed in 2,4-D solution (10 mg/l) for 6 h before inoculation, the treatment failed to yield higher percent of response to culturing than the control. This is very different from the experimental results with tomato anther culture. When pretreatment with 2,4-D was carried out, it consistently yielded better response to culturing.

It was also observed that during callus and embryoid initiations as many as 5 points of growth were found for a single anther. These growth points could exclusively lead to the development of embryoids or to the development of calli, or to the development of both calli and embryoids. There was no evidence of preference for any of them.

In order to have a high percent of survival in transplanting haploid plantlets from culture medium to the greenhouse, it was important to autoclave the soil. Furthermore, the newly transferred plantlets should be provided with 60 percent humidity, a day temperature of 65 to 70 F, and a night temperature of 55 to 60 F. Under these conditions, we obtained as high as 45 percent survival.

Margaret Yu and Y. C. Ting

Electrophoretic patterns of maize anther callus lines

Since a wide range of morphological variations among callus lines was consistently found, it seems important to know how these lines differ in their protein contents. A standard polyacrylamide gel electrophoresis (PAGE) was adopted with a concentration of 7.5 to 9.5%. The sample concentration of one gram per 2 ml of buffer gave best results. For one callus line (81-E), as many as 15 bands could be discerned. However, the protein contents of these lines are generally very low, and it is necessary to have a large amount of callus tissues and a large volume of sample solutions for one experiment. The number of electrophoretic bands varied from one callus line to the other. It seems likely that by this approach it may throw some light on the mechanism of callus development and differentiation.

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Maize anther culture

During the summer of 1980 we plated about 13,800 anthers from nine different maize genotypes. The genotypes CH1, CH2 and CH3 are derived from three Chinese hybrids (they were supplied by Dr. Rives following a visit to this country where they were reported as giving a good ability to anther culture). We used two media constituted with the inorganic salts of the N6 or Yu-Pei media complemented with glycine (2 mg/l), thiamin (1 mg/l), pyridoxine (0.5 mg/l), nicotinic acid (0.5 mg/l), casein hydrolysate (500 mg/l), activated charcoal (5 g/l), proline (100 mg/l), sucrose (120 g/l), TIBA (0.1 mg/l); pH was adjusted to 5.8-6 before autoclaving, with NaOH 0.1 N. The anthers were plated at about the middle uni-nucleate stage. The dishes were incubated at 26-28 C under light of 50 watt/m² (16 hour day).

The results are given in the table. The calluses and embryoids obtained were transferred onto the Green medium complemented with P.C.A. at 0.25 mg/l. On this medium only one embryo developed into a young plantlet, which was diploid. Following this regeneration we obtained 3 kernels on the tassel, which was a mixture of male and female flowers.

<u>Genotype</u>	<u>Number of plated anthers</u>	<u>Embryo or calluses</u>	<u>Number of plants</u>
A641 x Co158	985	0	0
A641 x Cm182	2,515	0	0
F47 x F1110	887	0	0
F47 x F1615	1,000	0	0
Co158 x W117	410	0	0
Co158 x Cm182	855	0	0
CH1	2,312	2	0
CH2	2,734	6	1
CH3	2,115	3	0

Our production of embryoid or calluses is poor in comparison with the work of Nitsch (MGCNL 54) or Brettell, but we hope that with the action of some inductor factors such as heat or cold, it will be improved. We think that before using

this technique routinely to get a mass of homozygous lines we have to do a strong effort to improve the percentage of callus and embryoid formation. Before it will be possible to use this technique on a large scale to obtain numerous homozygous lines, considerable work will be necessary to improve the callus percentage and embryoid formation.

Michel Beckert, Maurice Pollacsek and Ming Quing Cao

Analysis of genetic variation in scutellum culture

Application of tissue culture technology to the breeding of maize depends upon the ability of some genotypes to initiate and maintain calluses to regenerate and finally obtain fully fertile plants from in vitro culture. According to Green's research (1975), we carried out studies in "in vitro culture" of maize to compare the genotypic aptitudes (at the inbred level) for callogenesis initiation, callogenesis maintenance and plantlet regeneration. Significant genotypic differences were found in these three aspects (Beckert and Pollacsek, 1979, Ann. Amélior. Plantes, 29:563-581).

The analysis of our results reveals that the ability to regenerate is connected with genotype. We can group the homozygous genotypes according to their population ancestry. Therefore, our purpose is to select genotypes with a very high level of regeneration and genotypes with the ability to regenerate following long-term callus maintenance. We are going to analyze the results of a full diallel (with 8 parents) to study the different parameters of genetical variance for such traits as callus growth, long-term viability, and plantlet regeneration. In these experiments some different media are used.

Michel Beckert, Maurice Pollacsek and Ming Quing Cao

Analysis of genetic variability in regenerated inbred lines

For some strains, with which we can get the cycle of callogenesis and regeneration of fully fertile plants with normal chromosome number, we are going to study the genetic variability, after long callogenesis, within and between the progenies of the regenerated plants in comparison with normal selfed progenies from the original genotype. Biometrical traits were studied after two sexual reproduction generations to try to remove the effect connected with the seed quality or at a hybrid level in order to decrease, in an agronomic trial, the importance of environmental variation. The general protocol is described in Figure 1. The following traits were measured: number of leaves for some given developmental stage; plant and ear heights; leaf area; flowering earliness; grain yield components.

In a first experiment we compared with the check 23 regenerated plants from the line C0158 which were crossed with the line F1444 as male parent. The analysis of traits showed a varietal effect as far as the means are concerned and the comparison of the variances (within varieties) revealed heterogeneity between them. A global comparison for all traits for all varieties showed the following:

	<u>Mean lower</u>	<u>Mean = check</u>	<u>Mean higher</u>
Variance lower	0	1	0
Variance = check	0	21	1
Variance higher	0	0	0

Two regenerated plants seem to have an unusual behavior.

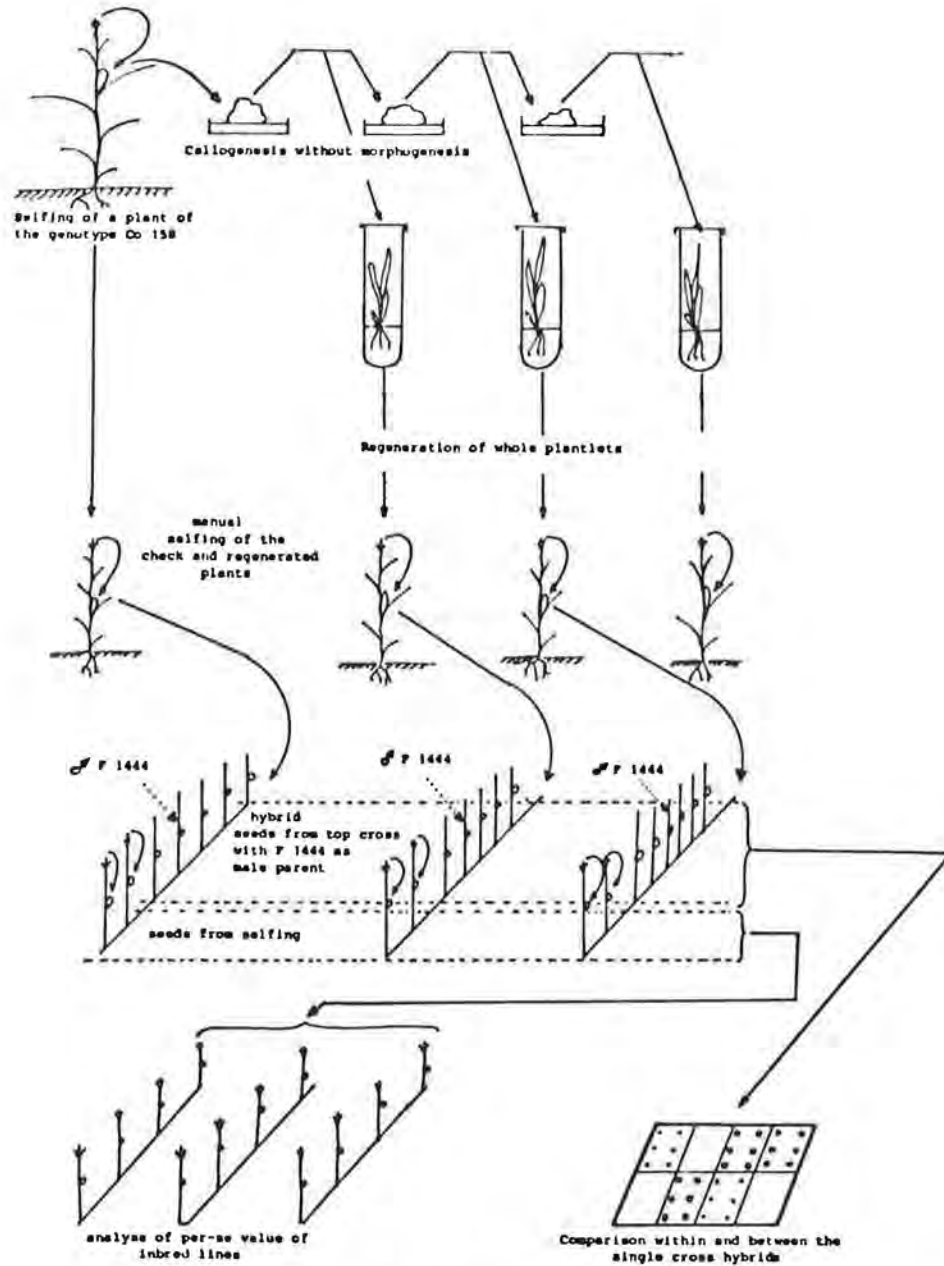


Fig. 1. General protocol for the genetical analysis of the consequences of in vitro culture technique.

We are studying now two other groups of regenerated plants with the same general protocol for the inbreds A641 and A188.

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Ds at the shrunken locus

We have continued the studies which we reported last year (MNL 55:23, 1981). Using single and double digestion with various restriction enzymes, followed by Southern blotting against our sucrose synthase cDNA, cloned in pBR322, we have constructed a restriction map around our hybridizing region with wild type DNA and with the DNAs of mutants sh-m5933 and sh-m6233. This map is divided into two parts by a BglII cleavage site within our probe. It could be shown that the map on the side corresponding to the larger part of our probe (500bp) is identical in the strains used, while differences are observed on the other side. We ascribe these differences to the presence of Ds at the Sh locus.

A comparison of the cDNA clones isolated by Burr and Burr (Genetics 98:143, 1981), by us (Geiser et al., Nucl. Acids Res. 8:6175, 1980) and by N. Fedoroff (personal communication), shows a size increase in the direction from the larger to the smaller BglII fragment. This indicated that the 5' end of the mRNA encoded by the gene is located in that part of our map, where we see the differences between wild type and mutants.

As the mutants can revert to the Sh, they must contain the complete coding sequence. From the data published by B. McClintock, we consider the possibility that the mutations are caused by deletions extending from an insertion site of Ds, towards the shrunken gene. From the data discussed above, we would conclude that the endpoints of these deletions are located upstream from the gene, possibly in its regulatory region.

We have cloned a 19kb BamHI fragment from the wild type and a 21 kb BclI fragment from the sh-m5933 in λ 1059 (Karn et al., Proc. Natl. Sci. USA 77:5172-5176, 1980). The restriction maps obtained coincide with those of the genomic DNA. We are presently trying to map the gene in the wild type DNA and to characterize the non-wild type DNA obtained from the mutant, which possibly contains part or all of Ds.

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An unstable adh1 mutant

In an attempt to isolate an adh1 mutant by the allyl alcohol selection described by Osterman and Schwartz (Genetics 83:63-65, 1976) we poisoned pollen from plants homozygous for bz2-m and containing Ac obtained from D. Schwartz. The bz2-m allele responds to Ac and is probably caused by Ds, and we hoped that the transposition to the Adh1 locus on the same chromosome would be not too infrequent. Poisoned pollen of bz2-m, Ac was used to pollinate W22. From the rare F1 seeds, we obtained only one plant, which showed a 1:1 ratio of alcohol dehydrogenase-positive and -negative pollen grains by in situ staining (Freeling, Genetics 83:701-717, 1976). This plant was selfed and the mutant was found to be viable in the homozygotes. No enzyme activity was detected in the pollen and in the scutellum, and by preliminary experiments, no material cross-reacting with maize alcohol dehydrogenase antiserum was found either.

The mutant is unstable (approximately 10^{-4} to 10^{-3} of the pollen grains of the homozygous plants were alcohol dehydrogenase-positive). Plants producing stable pollen were also observed. Segregation of the property to produce unstable pollen and of Ac showed strong but no absolute linkage between these two traits. In addition, we found a single F₂ plant (8010540-17), of which the whole F₃ progeny produced unstable pollen, regardless of the presence or absence of Ac.

These experiments will have to be extended, because the presence or absence of Ac was not in all cases determined by appropriate test crosses, but in some cases by either the phenotype of the kernels from which the plants were grown or the phenotype of the anthers producing the pollen (bronze versus variegated bronze). This is not unequivocal in all cases.

The data available are interpreted best by assuming that the unstable adh1 mutant which probably originated by the insertion of a transposable element is under the control of a regulatory element different from Ac but linked to it, and that it can be transposed from its locus to the vicinity of Adh1 (the case of plant 8010540-17).

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Prefixation of the somatic chromosomes of corn

Producing good somatic metaphase spreads is hindered by the combined difficulties of obtaining large numbers of dividing cells and of spreading and staining the chromosomes well. If good cells were routinely obtainable it would enhance the recognition of B chromosomes, the identification of translocations and aneuploids, and help with the routine counting of chromosomes. The present contribution describes a chemical prefixation technique using monobromonaphthalene and dimethyl sulfoxide (DMSO).

Over the years many methods have been developed for preparing somatic chromosomes, and they are generally made up of four stages. First, the seed is germinated and the actively growing root tip is collected. Second, a treatment prior to fixation (i.e., chemical or temperature) is used to inhibit spindle formation, preventing the congression of chromosomes to the metaphase plate. Third, fixation in any of a large range of solutions. Fourth, preparation of the root tips by hydrolysis, followed by staining.

Assuming that the material to be examined contains dividing cells it would be desirable to have a prefixation method that resulted in a quick and uniform penetration of the prefixative. Consequently, experiments were conducted utilizing several species in which the prefixative was combined with DMSO (Sallee and Kimber, in press, Cereal Res. Commun.) During these experiments it was determined that the use of DMSO along with monobromonaphthalene reduces the amount of prefixative needed and the time of treatment required, and increases the proportion of good analyzable cells.

Seeds are placed in Petri dishes lined with moist filter paper and germinated from two to three days at 30 C. Three to four cm long root tips are collected and placed in the prefixation solution consisting of five drops of monobromonaphthalene, four drops of DMSO and 100 cc tap water for two to three hours at room temperature and then transferred into glacial acetic acid for fixation. It is important that the DMSO be added to the water after the monobromonaphthalene. The minimum time of fixation is two hours; ideally, they should be left overnight. If the roots are kept longer than two days, the acetic acid should be replaced by 70% ethyl alcohol. When ethyl alcohol is used, the root tips should be rinsed in tap

water for ten minutes prior to hydrolysis. Roots are hydrolyzed for fourteen minutes in 1N HCl at 60 C. After hydrolysis the roots are placed into Feulgen stain for ten to fifteen minutes. Slides are made from squashes of small portions of the root tips in propionic orcein. Plastic cover slips proved to be preferable to glass due to the tendency of glass to break under vigorous tapping needed to ensure proper cell separations.

P. J. Sallee

A serious error in recommended method for treatment of pollen with EMS

For some years now (since 1966) we have been conducting highly successful experiments on the induction of mutants in corn pollen using mixtures of EMS and other mutagens in paraffin oil. The results far exceeded our original expectations and led to the cumulation of large numbers of mutations, the analysis of which have become a major undertaking in this laboratory and elsewhere. We reported our method and our progress from time to time in the Maize News Letter and elsewhere, beginning with MNL 42:124-125, 1968, and culminating in a publication entitled "Paraffin oil technique for treating mature corn with chemical mutagens" in *Maydica* 23:21-28, 1978. Since we were already overwhelmed by a wealth of material with new mutants, no further treatments were carried out after the early 70's. In 1979 we needed a particular kind of treated material, so we made some new treatments using our well-tried method. The treatment was a complete failure. No seed was found on the treated ears. Thinking that we might have miscalculated, we repeated the treatment again a second and a third time. To our dismay, they also were complete failures. We then remembered with mounting embarrassment, reports of other workers trying our method without success. We rechecked all our measurements and calculations and chemical sources. All were correct, but when we retested pollen germination, we found no tube growth. Killing was complete. Many anxious re-checks later, someone discovered only one change in procedure. In the successful experiments we used S/P diSPo P5210-1 glass pipets hand calibrated to 1 ml to make our stock and treatment solutions. In the unsuccessful treatments we used pre-calibrated Corning 7077 disposable 5 ml pipets. The first pipets were calibrated by taking one pipet, finding the 1 ml level, marking, measuring that level and then measuring and marking all the rest of the pipets in a box. A re-check revealed that all pipets so marked delivered 0.75 ml instead of 1 ml. Then it was clear what had happened. In our earlier experiments our stock solution was 0.75% instead of 1% and our treatment solutions were 0.75 ml instead of 1 ml of this solution in 8 ml of oil (the oil was measured in pre-calibrated large pipets). The concentration was actually $.0075/9 = 0.83\%$ instead of the published .11%. To obtain this concentration one should take 1 ml of 1% stock solution and mix with 11 ml of oil. Following this discovery we then proceeded to make new determinations of acceptable concentrations of EMS in oil for pollen tube growth and from this we identified and successfully tested new levels of concentration for good seed set and high mutation frequency. We found that one part of a 1% stock solution in 15 parts oil (.063%) gave excellent seed set and a high frequency of mutation events. With sincere apology it is recommended that our methods for pollen treatment outlined in various publications are good and can be followed with the above-described correction in concentration of treatment solution.

M. G. Neuffer

Reduced crossing over between Y1 and Dt2

The controlling element Dt2 was located on the long arm of chromosome 6 near P1. The somewhat meager data from the cross of Y Dt P1/y dt p1 x y dt p1 were as follows:

<u>parental</u>	<u>co reg 1</u>	<u>co reg 2</u>	<u>doubles</u>	<u>total</u>
103	38	7	2	150

Therefore the tentative map distances were: Y 26 Dt 6 P1. We would like to have a good kernel marker for studying Dt2 but Y1 is too far away. Therefore we attempted to produce some chromosome aberration (e.g., an inversion) that would shorten the map distance between Y and Dt2. To do this homozygous a-m, Y Dt immature pollen was treated with x-rays and crossed on a-m, y dt silks. The stage was around the 1st division of the microspore and the dose was 1200r. A total of 912 yellow dotted kernels were produced. These were planted and the resulting plants backcrossed again to a-m, y dt. From this cross 903 ears were produced and were examined superficially for cases where Y and Dt appeared to be closely linked. 44 ears showed a possible reduction in crossing over between Y and Dt. One ear, which had 62 Y Dt:41 y dt:36 Dt:0 y Dt, was selected as a good case for further study.

Yellow dotted and white nondotted kernels from a subsequent backcross were planted for material to test for gamete abortion, for female and male transmission and for material on which to do cytological tests. The results were as follows:

Gamete abortion--Plants in both classes had normal pollen and subsequent ears had full seed set indicating that there was no gamete abortion.

Female & male transmission--Reciprocal crosses of Y Dt/y dt x y dt produced the following data:

	<u>Y Dt</u>	<u>y dt</u>	<u>Y dt</u>	<u>y Dt</u>	<u>Total</u>
heterozygous female	446	435	0	0	891
heterozygous male	154	816	1	0	971

These data indicate that Y Dt linkage is very tight (the one y dt kernel could be a contamination). The transmission of this low crossover segment is normal through the female but the male transmission is reduced to 32% of normal.

Test for linkage with Y1--The low crossover (lco) Y Dt stock was crossed to a yellow dent stock (Alexander High Oil), which is presumed to have Y1 on chromosome 6. The cross was a-m, Y Dt (lco), R x A, Y dt, r (Aho). The F1 was then backcrossed to a-m, Y dt, R and the colorless kernels were separated for Y and Dt. The results were as follows:

	<u>Y Dt</u>	<u>Y dt</u>	<u>y dt</u>	<u>Total</u>
colorless kernels	82	53	48	183

These results indicate that the Y linked to Dt2 is not allelic to the Y from Aho and suggest that Y in the lco segment has been moved to a new position.

Rodney Higgins and M. G. Neuffer

Allelism tests for the virescent mutants

45 EMS-induced virescent mutants that have been located to 11 chromosome arms by the B-A translocation method were chosen to test for allelism. The objective is to determine the number of duplicate events in the target material and to identify new virescent loci. Virescent mutants were chosen because they are viable, allowing mutant by mutant crosses. If both homozygotes are not obtained then crosses of known heterozygotes or, in the worst cases, 2/3-right (unknown) heterozygotes from a segregating M2 must be used. In the latter case, several crosses are needed for each valid test. Fifty seeds from each test ear were planted in a sandbench and screened. The results showed that some mutants are alleles.

<u>Arm</u>	<u>Mutants by E#</u>	<u>Allelic</u>	<u>Nonallelic</u>	<u>Inadequate test</u>
1S	19,34,68,144,243,628,1024,1151	144,1151	19,34	the rest
1L	55,245			all
2S	453,605	453,605		
2L	350,424,576,588,1085	350,424,576,588	1085	
3L	41,54,142			all
5L	26,473,735	26,473,735		
6L	599,634,716	599,634,716		
7L	53,413,590	53,413,590		
8L	7,25,29,358,779,826	7,358,779,826	25,29	
9S	27,585,610,697,828,829	27,585,610,697	828,829	
10L	114,354,418,470	114,470	354,418	

Ming-Tang Chang

Isolation of a viable virescent mutant with high chlorophyll fluorescence

Several recessive, virescent mutants of maize were examined for high chlorophyll fluorescence during greening. Maize mutants which display high red fluorescence on irradiation with blue light have been shown to be defective in photosynthesis for all cases studied (C. D. Miles, Meth. Enz. 69:1, 1980). We have not found that this fluorescence can be induced in the case of greening, etiolated seedlings. Therefore virescent mutants which exhibit high chlorophyll fluorescence only during greening should have delayed synthesis of a chloroplast component(s) intimately involved in photosynthesis. Furthermore, we anticipate that the component(s) in question will also be critical to chloroplast biogenesis. It should be noted that there are several photosynthesis mutants in maize with high chlorophyll fluorescence which are green and are not virescent. Thus the absence of photosynthetic capacity per se does not block or slow formation of the photosynthetic apparatus.

Virescents that were screened for high chlorophyll fluorescence include:

	<u>Source</u>	<u>Fluorescence*</u>
ar; v; v2; v23;	Genetics Stock Center	1
v17; v8; v22	Urbana, Illinois	1
v*229; v*585; v*697	Neuffer	1
v5	Genetics Stock Center	2
v*-424	Neuffer	up to 5

*Normal level is defined as 1.

Of the mutants examined to date, most do not display abnormally high chlorophyll fluorescence. One which did, mutant v^*-424 , was selected for further study. The v^*-424 mutation is uncovered by TB-2L-1S and not by TB-1Sb, tentatively placing it on the long arm of chromosome 2. In seedlings which green rapidly, the phenotype is not lethal. Those which green slowly die unless rescued by sugar feeding (achieved by placing the cut edge of the second leaf to emerge in a small vial of sterile, 10% sucrose, with changes of the sucrose solution every other day). The presumed homozygotes for v^*-424 , both the rescued, slow-to-green individuals and rapidly greening individuals, grow to maturity in the field, producing small, fertile plants.

The extent of fluorescence in v^*-424 was inversely proportional to the maturity of the tissue measured:

Leaf, Position	Relative Red Fluorescence	
	v	+
1	1.0	1.0
2, tip	0.9	0.6
2, base	1.2	1.1
3, tip	2.4	1.2
3, base	5.0	0.6

The time-variability of the induced fluorescence (see Figure 1) indicates that photosystems II and I are at least partially functional (see Miles ref. above).

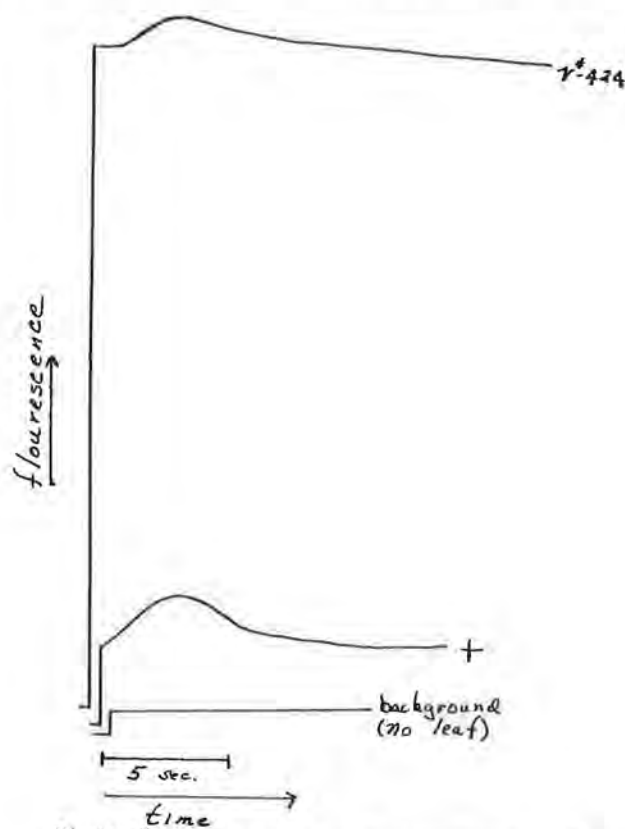


Figure 1
Time-variability of Leaf Fluorescence. The fluorescence depicted in the figure represents red light emitted by leaf sections during irradiation with blue light. Basal regions of the third leaves to emerge for normal (+) and virescent (v^*-424) siblings were employed in these studies.

Dodecyl sulfate-urea-polyacrylamide (10-18% gradient) electrophoresis revealed no significant differences between the protein compositions of thylakoids isolated from mesophyll chloroplasts of fluorescent and nonfluorescent siblings when equivalent amounts of chlorophyll are applied to gels. In all instances, the material employed was obtained from selfed heterozygotes for the v*-424 locus and segregated approximately one-fourth virescent phenotypes. Work is in progress to characterize the lesion in photosynthesis that has been inferred from the fluorescence data.

Mary Polacco and M. G. Neuffer

hcf*-19: linkage with ys3 and d; allelism of hcf*-19A and hcf*-19B

Allelism: Two distinct lethal photosystem II lesions were isolated from a single M2 progeny of Neuffer's nuclear mutant collection (Leto and Miles, MGNL 53: 38, 1979). One of these, hcf*-19A (previously designated hcf*-19g), was found to be missing a 32 kD thylakoid protein and had no observable PSII electron transport activity. The second, hcf*-19B (previously hcf*-19yg), contained the 32 kD protein absent from hcf*-19A and had partial (20-40% of normal) PSII activity. The pollen of three individuals potentially heterozygous for hcf*-19B was used to fertilize several plants potentially heterozygous for hcf*-19A. Since the parents in both cases were derived from ears of selfed heterozygotes for the respective mutations, 2/3 of the viable parent plants should be +/hcf for the hcf locus in question. Of eleven such crosses, the progeny of nine segregated approximately one-fourth hcf seedling-lethals. Therefore, hcf*-19A is allelic to hcf*-19B.

The original designations yg or g for the different hcf*-19 phenotypes have been dropped because they do not always correspond to a given phenotype. In F2 progeny from crosses of +/hcf*-19 with the inbred Mo17 or with a hybrid of inbreds Mo17 and N28 (the latter F2 progeny was kindly supplied by M. G. Neuffer), both hcf*-19 phenotypes (confirmed by gel electrophoresis) were either fully green or very slightly pale green.

Linkage: F2 progenies of a cross between d/d, ys3/ys3, +/+ and +/+, +/+, +/+, +/hcf*-19A were analyzed. Recombination values were calculated using the tables of Fisher and Balmukand (J. Gen. 20:79, 1928):

Markers	% Recombination	No. Seedlings for Given Phenotype			
<u>ys3</u> and <u>hcf*-19</u>	29 ± 3	++	+ <u>ys</u>	<u>hcf</u> +	<u>hcf ys</u>
		381	165	162	15
<u>ys3</u> and <u>d</u>	37 ± 2.4	++	+ <u>ys</u>	<u>d</u> +	<u>d ys</u>
		429	107	114	73
<u>d</u> and <u>hcf*-19</u>	58 ± 2.5	++	<u>d</u> +	+ <u>hcf</u>	<u>d hcf</u>
		421	125	115	62

The total number of individuals observed was 723. Data for 5 separate F2 progenies are combined in the table. The distributions of phenotypes were approximately equivalent for different progenies. To facilitate scoring, the hcf phenotype was equated with lethality (over 100 lethal seedlings were initially identified as hcf). Of the 15 ys3 hcf*-19 recombinants, 9 were also d.

The ys3 locus has been assigned to the long arm of chromosome 3 by analysis of crosses with B-A translocations (Beckett, MGNL 49:131, 1975) and appears to be near the centromere (Wright, MGNL 35:111, 1961). The hcf*-19B allele is uncovered by TB-3Sb (Leto & Miles, MGNL 51:58, 1977), placing this locus on the short arm of chromosome 3. Thus, the high recombination frequency observed here between d and hcf*-19A is difficult to explain. If one ignores the linkage data for d and hcf*-19, the gene order would be: d hcf19 ys3. If the assignment of hcf*-19 to the short arm of chromosome 3 is ignored, the gene order would be: d ys3 hcf*-19. A cross of cr/cr, d/d, Lg3/+ or Lg3 with +/+, +/+, +/hcf*-19 has been performed and the F2 progeny should be available for screening in late spring 1982. Crosses of hcf*-19A and hcf*-19B with TB-3Sb and TB-3La will be performed this spring in the greenhouse.

Mary Polacco and Sharisse Mills

TB-4Lb and TB-5Sc, two new B-A translocations on the long arm of chromosome 4 and the short arm of chromosome 5

TB-4Lb, a new B-A translocation on the long arm of chromosome 4, was produced by crossing a c2 tester by pollen from colored, B chromosome-carrying plants that had been x-rayed (3 minutes at 200 r/min.) as described by Lin (NL 46:193). The genes gl4, gl3, c2 and dp are distal to the breakpoint; other genes on the arm have not yet been tested.

By using the same procedure with a2 instead of c2, a new B-A translocation on the short arm of chromosome 5 has been produced. It is hereby designated TB-5Sc. Both a2 and gl17 are beyond the breakpoint; other genes on the arm will be tested to determine their position with respect to the breakpoint of the translocation.

J. B. Beckett

TB-8Lc, a new B-A translocation on the long arm of chromosome 8

In order to employ Lin's method (NL 46:193) to produce new B-A translocations, a recessive endosperm marker must be available on the arm on which translocations are desired. Until recently, no such markers were available on either arm of chromosome 8. When pro proved to be on chromosome 8 some distance proximal to v16, it seemed appropriate to use it to produce new B-A translocations, even though it might be on either arm. Accordingly, a hybrid stock segregating 2 recessive alleles at the pro locus was crossed by pollen from x-rayed plants of a colored hybrid with B chromosomes; exceptional kernels with the pro endosperm phenotype were saved. One plant grown from an exceptional kernel segregated pro when self-pollinated and when crossed onto a +/pro ear. Both pro and normal kernels from selfed and outcrossed ears segregated both normal and lethal pro seedlings, indicating frequent noncorrespondence of endosperm and endosperm genotype.

When heterozygotes of v21, which is distal to TB-8La on the long arm of chromosome 8, were crossed by 3 plants from the presumed new B-A translocation stock, 2 gave, respectively, 20 and 16 percent virescent seedlings in the progeny. Therefore, the presence of a new B-A translocation on the long arm of chromosome 8 has been established; it is hereby designated TB-8Lc. Because it is much nearer the centromere than TB-8La, it should replace TB-8La in the basic set of B-A translocations used to locate recessive genes to chromosome arm.

J. B. Beckett

Progress report on locating whp

A previous report (Modena and Coe, MNL 55:115) eliminated ten chromosomal arm segments by TB tests. During the summer of 1981 I have excluded 5S by TB uncovering, the centromeric region of 4 by a linkage test with su, and 6S and the centromeric region by a linkage test with y.

The TB observations of last year were conducted in an isolated open pollinated block. The ears on hypoploid plants were recovered. Following the suggestion of Beckett (J. Hered. 69:27-36, 1978), families from these ears, of size sufficient to distinguish a 3:1 vs. 1:1 ratio at the 5% level, were grown as two replicates. No linkages to the TB breakpoints were discerned, thereby excluding these segments: 1S proximal and centromeric; 3 centromeric; 4 centromeric; 5 centromeric; 6S proximal and centromeric; and 9 centromeric.

Tests are advancing for the remainder of the genome (1S, 2, 4L, 7S, 8S and centromeric, 10).

Stephen A. Modena

Test of the effectiveness of white pollen as a male sterile

In the summer of 1981 families segregating yellow and white pollen plants (c2/c2, +/whp and c2/c2, whp/whp) were selfed. Ears recovered were shelled and the seed was weighed.

There were 108 yellow pollen plants selfed, which yielded 14,300 kernels \pm 5%, all colorless as expected.

There were 50 white pollen plants selfed, which yielded 2 kernels: one colored and one colorless, adjacent to one another on one ear. The colored kernel must be a pollen contaminant or a back mutation. Both will be grown for genotype testing.

If yield from yellow pollen plants, selfed, is taken as an estimate of the number of potential functional gametes, then one "unlikely" kernel was set out of approximately 6,600 gametes tested.

On the other hand, the "screen" would successfully pass any functional pollen and often several pollen grains enter a single silk. Thus many more grains were tested in this experiment.

Stephen A. Modena

Examination of the white pollen sterility effect

In the summer of 1981 sib plants, some shedding white pollen (c2/c2, whp/whp) and others shedding yellow pollen (C2/c2, whp/whp), were selfed. Forty-eight hours post-pollination, silks were cut from the ears and fixed in formalin-acetic acid-80% ethanol (1:1:8) for 24 hours at room temperature. The silks were transferred to 8M NaOH for 4 to 6 hours. The silks were dip-rinsed in water and examined under the light microscope without a coverslip. The basic condition clears the silk body and heightens flavonols.

The yellow pollen grains are seen as collapsed, empty shells still attached by their germination tubes to the silk body. Here and there traces of pollen tubes are visible, aligned with the vascular bundles.

Most of the white pollen grains are half empty, still anchored to the silk body. Germination tube progress is easy to discern, as the pollen cytoplasm is an off-brown color and filled with starch grains. Within the silk body, at a distance no more than twenty pollen grain diameters, are numerous, sizable, brown, grainy "bodies" with a ballooned appearance. This is not often seen on silks with yellow pollen.

Based also upon observations from the winter of 1980/81 using silks of maize x Tripsacum hybrids, which are more suited to live viewing, I offer this interpretation. A white pollen grain germinates, penetrates the silk, and grows a short

distance, usually less than 10 pollen grain diameters. Suddenly, the tube bursts and the cytoplasm is forcibly extruded in a pulsating manner for a short time. The intra-silk "bodies" appear to be the accumulation of the contents of several burst tubes. The sterility is due to a "mass suicide" phenomenon, which involves 100% of the white pollen grains deposited on a silk.

These preliminary results were reported during the Symposium on Pollen Biology at the A.I.B.S. Meeting at Bloomington in June 1981.

Stephen A. Modena

Possible successful pollination with white pollen

In a sheltered block of c2/c2, whp/whp plants shedding white pollen, pollinations were made with verified white pollen. A "patch" entrance was cut in the husks of the ear shoot (previously shoot-bagged). A liberal amount of white pollen was blown onto the exposed silks, aiming to deposit pollen as close as possible to the junction of the silk and ovary. Then the "patch" was closed and the earshoot rebagged.

Results from 12 pollinations: four colorless kernels set, of which 3 were insect-eaten at maturity, but one survives intact. Genotype testing and scoring of pollen shed will be done in the winter greenhouse.

Stephen A. Modena

The occurrence of whp in K55 and other inbred lines

The sudden appearance of white pollen in progenies with c2 and pedigrees tracing to K55 inbred line (Coe, McCormick and Modena, J. Hered. 72:318, 1981) raises the question of the source of the whp factor. Among the likely possibilities would be that whp is present in the K55 inbred itself; the factor might occur in other inbred lines as well.

To test for whp constitution, K55 (supplied by M. S. Zuber) and several other inbreds were crossed onto white-pollen plants, then backcrossed to white pollen. Colorless (c2 c2) seeds from the backcross were planted for observation of pollen color. Each of 5 tested plants from K55 was homozygous for whp. Tests on one plant of each of 3 other white-endosperm inbreds (Ky21, Mo20W, Ky27) and 2 yellow inbreds (L289 and Mo17) identified each to be dominant (Whp).

K55 is clearly carrying whp, either homozygous or in a high frequency in the inbred. There is no evidence from tests of the other inbreds that whp is present in them. The presence of whp in K55 as a cryptic recessive, not recognized until the duplicate locus, C2, was recessive, exemplifies the difficulty of recognizing mutants at loci whose functions are duplicated elsewhere in the genome.

E. H. Coe, Jr.

Genetic interactions of c2 and whp and their duplicate function

The effects of c2 and whp on anthocyanins in the plant and in the aleurone tissue are found to be consistent with their duplicate-factor control of yellow pigments in the pollen. So are their effects on yellow fluorescence in bz anthers.

In the plant tissues, c2 Whp B R-r P1 plants have extensive anthocyanin distributed in the sheaths, husks, culm and glumes, and occasional weak pigmentation in the anthers (Coe et al., J. Hered. 72:318, 1981), while c2 whp plants, according to several observations, appear to be completely anthocyaninless. Colorless seeds from F2 progenies segregating for C2/c2 Whp/whp B/b P1/p1 segregated for plants similar to c2 B P1 (or p1), each shedding yellow pollen, and green plants, some shedding yellow and some shedding white pollen. Tests are in preparation to check

the B constitution of green plants with white pollen in these progenies. Plants with white pollen have expressed no anthocyanin in these pedigrees, or in others with R-r and b, either P1 or p1 (c2 Whp plants with R-r b p1 have so little color in the anthers that they appear green except in unusual physiological circumstances such as protracted cool conditions; c2 Whp R-r b P1 plants, on the other hand, show light color in the anthers under most conditions; consequently some uncertainty exists with regard to low levels of pigmentation in the absence of extensive observations under diverse conditions). It is not yet clear whether whp is recognizable by effects on plant color in the presence of C2, but the effects are probably not great inasmuch as anthocyanin is present in anthers and glumes in C2 whp R-r b p1 plants.

In the aleurone tissue Whp is not normally sufficient for anthocyanin development, judging from the fact that c2 Whp kernels are colorless. However, because c2 in kernels have pale color (Coe, MNL 34:91, 1960), it could be supposed either that in enhances an existing low level of synthesis (i.e., that of Whp) or that in is an active factor replacing (i.e., duplicating) c2, in which case in and whp might be allelic. Crosses were made between c2 whp and c2 gl in and self-pollinated. The F2 ears segregated about 1/4 pale (c2 in) kernels, as expected (in retrospect, 3/16 rather than 1/4). Plantings of pale seeds gave 11 + yellow: 39 gl yellow. Plantings of colorless seeds gave 42 + yellow:2 gl yellow:15 + white:6 gl white. Among gl yellow plants from the pale class self-pollinated, segregations of 3 pale:1 colorless were found (c2/c2 in/in +/whp), and progeny tests of gl white plants from the colorless class show that c2 c2 in in whp whp kernels are colorless. These interactions are consistent with the view that in is an enhancer of pigmentation, so that Whp action generates visible pigment in c2 c2 in in kernels--i.e., that C2 and Whp perform a parallel, duplicate function.

The dramatic yellow fluorescence of bz anthers, which is not blocked by recessive constitutions for a1, a2, bz2, c1, c2 or r-g (unpublished observations), is blocked in white-pollen plants. Progenies to examine this were derived in F2 from Bz bz C2 c2 Whp whp hybrids, selecting colorless kernels and plants with white pollen, crossing them by plants from bronze kernels and repeating the cross the next generation. Three progenies were derived in this way that had the pedigree Bz bz c2 c2 whp whp x bz bz C2 c2 whp whp, from which kernels of the bronze class gave only yellow-pollen plants with yellow fluorescent anthers and kernels of the colorless class gave only white-pollen plants without yellow fluorescence.

These observations, taken with the fact that yellow pollen is conferred by either C2 or Whp, support a conclusion that C2 and Whp have duplicate function in a key initial step in the flavonoid pathway that leads to yellow pigments, anthocyanins, and yellow-fluorescent compounds.

E. H. Coe, Jr.

White pollen as an enrichment screen

Since white pollen is not functional, it offers the possibility of screening for mutations to functional capability (from c2 whp to C2 or Whp, for example) in populations of pollen grains. Experiments directed to this purpose are in progress. In addition to reconstruction experiments involving mixtures of pollen, described in an accompanying article, an in situ reconstruction was initiated with c2-m1. Progenies were derived from crosses of c2-m1 with c2 whp, anticipating that c2-m1 with homozygous whp would have mostly white pollen but that a low frequency of yellow pollen would come from C2 sectors in the anthers. However, c2-m1 c2 whp whp plants invariably produce uniform yellow pollen, which has functional capability. Presumably the yellow pollen results from functioning of c2-m1 at a low level. We have further observed that aleurone and plant tissues in

c2-m1 have a light pale background with colored sectors, but that this background is colorless to the eye in c2-m1 whp constitutions. A substantial level of function is apparently necessary to produce a threshold level of anthocyanins, but a lower level is sufficient for yellow pollen and for functional capability.

A mass screening approach, with potentially high efficiency, might be to screen tassels of c2 whp bz plants under UV for yellow-fluorescent anthers, which are so brilliant that they should stand out in the field at some distance from the UV source. Mutations to C2 or Whp in such plants would be yellow-fluorescent. Since we have also noted that progenies with C2-Idf can segregate for white pollen, the expression of C2-Idf may permit enrichment screening for mutations of this allele also.

E. H. Coe, Jr. and Stephen A. Modena

White pollen as a basis for a male-sterile system

The non-functionality of white pollen permits design of a male-sterile system to replace hand or mechanical detasseling in hybrid seed production. It is in part reliant on optical-mechanical sorting of seed for the presence or absence of distinctive anthocyanin pigmentation in the aleurone and in the embryo.

We perceive the following components:

1. Source Stock, resulting from the cross of homozygous c2 whp C1 R by C2/c2 whp C1 R, is sorted each generation by aleurone color into two classes: colored (purple) and colorless (non-purple). The colorless kernels are homozygous c2 whp C1 R, and serve as the major Seed Parent in the next cycle of the maintenance of the Source Stock. These plants have white pollen and are male sterile. The colored kernels are C2/c2 whp C1 R and serve both as the Pollen Parent for the Source Stock and as a minor Seed Parent. The ears of these plants, if harvested, must be kept separate and only the colorless kernels used. The colored kernels are discarded, since they are a mixture of C2/C2 and C2/c2 genotypes. R in the Source Stock can be any R or B allele suited to sorting scheme or seed quality requirements.
2. In hybrid seed production fields the colorless kernels of the Source Stock are planted as the male-sterile seed parent for the single cross hybrid. They are homozygous c2 whp C1 R in uniform inbred background.
3. In hybrid seed production fields the pollen parent of the single cross hybrid will be homozygous c2 Whp to achieve colorless seed. It can be a standard inbred line, C2/C2, if colored seed is desired or acceptable.
4. The hybrid seed produced will be c2/c2 Whp/whp and colorless, by the main scheme here.

All current hybrids in the U.S. are r/r and usually c1/c1 C2/C2. Therefore, the Source Stock requires the greatest gene replacement effort. The seed production male parent requires one gene replacement in order to achieve a non-purple hybrid. This can be done by backcrossing and verification of the recovered genotype by crossing to an appropriate tester line for visual scoring.

If one assumes that only colorless seed (excluding pericarp color) is desired in the F1 and F2 (producers' and farmers' fields respectively), this scheme can present a potential problem from cross pollination by or onto "standard" maize, which results in colored kernel contaminants. In the seed producer's field, these colored kernels will signal that isolation measures have failed, but will permit mechanical separation of offending kernels.

For the farmer's field, what can be done? A partial solution may lie in the use of the allele C2-Idf instead of c2 when deriving the male parent line. C2-Idf is a dominant color inhibitor which suppresses C2. The single cross hybrid

would be C2-Idf/c2 Whp/whp C1/c1 R/r. C2/c2 kernels due to contaminant pollination will still show up as colored, providing a warning and permitting mechanical separation. In the farmer's field, all plants will be C2-Idf/c2 and half the kernels set by contaminating C2 pollen will be suppressed to nearly or completely colorless. Further reduction of colored exceptions can result from the use of a Pollen Parent of C2/c2 whp c1 r constitution in the final production of Source Stock for hybrid seed production.

Heterofertilization occurs at the rate of about 1%. This will cause non-correspondence between endosperm and embryo. Thus 1% of the seed parent kernels, identified by aleurone color, will in fact shed yellow pollen inappropriately. Embryo color can be incorporated into the Source Stock (for example, using R-nj, R-sc, or B-Peru). The optical seed sorting, if sensitive to wave length and low intensity, would remove most non-corresponding embryos. Alternatively, visual screening of aleurone-sorted (colorless) seed will substantially reduce this source of error. As a follow-up, these alleles in concert with C2 or Whp confer anthocyanin color to the growing plants, permitting accurate roguing.

Stephen A. Modena and E. H. Coe, Jr.

Mixed pollinations with white pollen

For white pollen to be used as a male sterile system, or for enrichment screening, the consequences of mixed pollinations need to be known. It is also important to explore the basis for the functional incapacity of white pollen, including whether yellow pollen can serve as a "helper" for white. The following experiments were conducted with these interests in mind.

Mixtures were made of roughly equal quantities of white pollen with others, with results as follows:

Cross	Ear parent	Pollen parent		No. Ears	No. kernels poss.	
		PP1	PP2		PP1	PP2
1	c2 whp y	c2 whp y	C2 sh bz wx y	3	11	414
2	c2 whp y	c2 whp Y	C2 sh bz wx y	4	4	877
3	c2 Whp y	c2 whp y	C2 sh bz wx y	17	17	3,822
4	C2 r-g y	c2 whp R Y	C2 r-g y	9	12	2,788
5	C2 r-g y	c2 whp R y	C2 r-g y	13	19	2,948
6	C2 sh bz wx	c2 whp Bz y	C2 sh bz wx y	5	3	889
7	C2 sh bz wx	c2 whp Bz y	C2 sh bz wx y	12	10	1,333

The above kernels that possibly represent transmission of white pollen (PP1) are still to be progeny tested, but it is evident that white pollen is not aided much, if at all, by admixture with yellow pollen.

Parallel pollinations were made for each of the above 7 tests with PP2 samples collected and dried overnight in a tassel bag near the heat register in the greenhouse before mixing with fresh white pollen (PP1):

Cross	Ears	Poss. PP1	Poss. PP2
1	1	0	1
2	4	0	7
3	7	1	59
4	6	0	4
5	10	3	713
6	3	0	7
7	17	2	319

The possible PP1 kernels again are to be progeny tested, but the dried pollen did not aid white pollen much, if any. The number of PP2 pollen grains surviving in crosses 3, 5 and 7 is surprising but may be an artifact of handling variations for the drying.

Pollination on two successive days, the first with white pollen and the second with yellow, was used to determine whether white pollen interferes with the receptivity of silks or ovules, as follows:

Ear parent	PP1 - day 1	PP2 - day 2	Ears	Colored	Colorless
c2 whp Y	c2 whp Y	C2/c2 whp Y	21	1,804	1,888
c2 whp Y	c2 whp Y	C2 r-g y	1	53	0
c2 whp Y	c2 whp Y	C2 sh bz wx y	1	148	2
C2 r-g y	c2 whp Y	C2 r-g y	2	0	286

Once again progeny tests are to be carried out on exceptions. The first cross indicates that the ratio of C2 to c2 is not affected by prior pollination (in this case 2 days earlier). White pollen on the others has not prevented subsequent fertilization by yellow pollen.

A further indication that white pollen does not affect the physiology of the silk is that the silks at the point of attachment to the ovule, when examined even 3 or 4 days after pollination with white pollen, do not show the collapse and pinching off that is characteristic of pollinated silks within a day after pollination with functional pollen.

E. H. Coe, Jr.

The cell lineage of the maize leaf

Clonal analyses of maize development by Coe and colleagues have demonstrated that the leaf blade is derived from a transverse ring of about 32 apical meristem cells. The number of cells in the longitudinal dimension of the leaf primordium is still unclear, however, nor is there a consensus about the number of apical cell layers that contribute to the leaf blade. Johri and Coe (MNL 55:31) have presented conclusive evidence that husk leaves are derived from at least two cell layers. Our recent results indicate that the foliage leaf is also derived from at least two apical cell layers (L-I and L-II) and from at least two rings of L-I cells.

Seedlings of the genotype Wd/wd; P1/p1; B/b; R-r/R-g were irradiated with 500 R of x-rays (89 R/min, 2 mmAl, 138 kVp, 9 mA) 5 and 8 days after sowing, and were grown to maturity in pots in the greenhouse. At the time of irradiation 5-day-old plants had 8 leaf primordia, while 8-day-old plants had 9 primordia (Fig. 1).



Fig. 1. Shoot apex at the time of irradiation. Leaf buttress a was leaf number 8 in the case of 5-day-old plants, and leaf 9 in the case of 8-day-old plants.

Only sectors in leaves initiated after irradiation were considered in this study (i.e., leaves 9 and above in the case of 5-day-old plants, and leaves 10 and above in the case of 8-day-old plants).

Four kinds of sectors were observed. Some sectors were completely white throughout the length and thickness of the leaf blade (Fig. 2). Others were similar in that they were completely white along the length of the leaf, but differed in that they had a green epidermis (as indicated by the color of guard cells) (Fig. 3). A third class consisted of sectors that were white towards the distal end of the blade, and became green towards the proximal end (Fig. 4). The epidermis of such sectors was generally white, even in regions where the mesophyll was completely green. A fourth class of sectors exhibited the reverse condition (Fig. 5). In these cases the sectors were white towards the proximal end of the leaf, greener towards the distal end, and stopped before reaching the margin. These sectors always had a green epidermis.

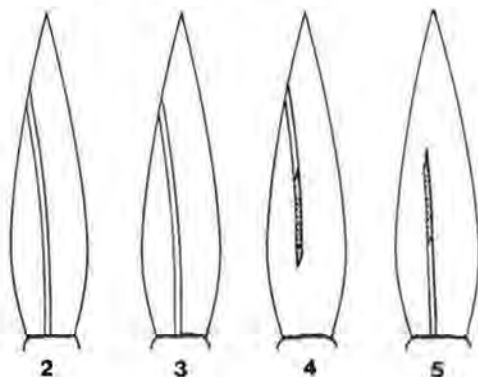


Fig. 2-5. The types of sectors found on leaves initiated after irradiation.

Sectors in which the mesophyll and epidermis are of different genotypes (Fig. 3, 4, 5) provide evidence that the leaf blade is derived from at least 2 cell layers in the apical meristem. Sectors of the type illustrated in Figure 3 represent cases in which L-I cells always divided anticlinally, new cell walls perpendicular to the leaf surface. Instances in which the L-I divided periclinally during leaf development, thereby contributing cells to the internal part of the leaf blade, are represented by the sectors illustrated in Figures 4 and 5. Note that Figure 4 represents a white sector in the L-I layer, whereas Figure 5 represents a white sector in the L-II. In response to the criticism that such sectors might have been generated after leaf initiation, it should be pointed out that they are exactly the same width (1/32 of leaf width) as pure white sectors.

That the leaf blade is derived from at least two rings of L-I cells is apparent from the fact that epidermal sectors (marked by wd, b or pl) frequently end within the leaf blade. If the leaf blade were derived from a single ring of 32 L-I cells, then sectors would always end at the base of the leaf blade, that is, each of the 32 longitudinal L-I lineages would always be of a single genotype.

In conclusion, it should be pointed out these are only minimal estimates of the number of initial cells in a leaf primordium. They are valuable in that they confirm the picture provided by histological studies, and provide yet another indication that morphogenetic phenomena in plants involve signals that act at a multi-cellular level.

R. Scott Poethig

The population genetics of chromosome aberrations in autotetraploids

Chromosome aberrations in autotetraploids create much more complex situations than they do in diploids. The relative rates of formation of various types of aberrations, the pairing configurations, the patterns of chromosome disjunction, and the fates of deficient chromosomes and unbalanced spores and zygotes are all different on the tetraploid level.

First, the difference between the relative rates of different aberrations may be explained by the table below, which gives the results of 2 break events. One break's position is given on the horizontal row and the other break's position is given on the vertical column. The symbols L and R refer to the left and right arms of the chromosome. To distinguish between homologous chromosomes, primes are used. Thus, 1L, 1L', 1L'', and 1L''' are four homologous left arms of chromosome 1. This model uses a species with $n=10$, so there are 80 arms where breaks can occur and 6,400 possible combinations (3,160 are duplicates). A diploid would have 40 entries on each side and 1,600 combinations. A haploid would have only 20 entries and 400 combinations.

		$2n$														
		n					n									
		1L	1R	1L'	1R'	1L''	1R''	1L'''	1R'''	2L	2R	2L'	10R'''			
n	$2n$	1L	A	B	D	E	D	E	D	E	C	C	C	C		
		1R	B	A	E	D	E	D	E	D	C	C	C	C		
		1L'	D	E	A	B	D	E	D	E	C	C	C	C		
		1R'	E	D	B	A	E	D	E	D	C	C	C	C		
		1L''	D	E	D	E	A	B	D	E	C	C	C	C		
		1R''	E	D	E	D	B	A	E	D	C	C	C	C		
		1L'''	D	E	D	E	D	E	A	B	C	C	C	C		
		1R'''	E	D	E	D	E	D	B	A	C	C	C	C		
		n	$2n$	2L	C	C	C	C	C	C	C	C	A	B	D	C
				2R	C	C	C	C	C	C	C	C	B	A	E	C
2L'	C			C	C	C	C	C	C	C	D	E	A	C		
10R'''	C			C	C	C	C	C	C	C	C	C	C	A		

Symmetric

- A paracentric inversion
- B pericentric inversion
- C reciprocal translocation
- D tandem duplication and internal deficiency
- E pseudo-isochromosomes

Asymmetric

- internal deficiency and acentric ring
- fragment and centric ring
- dicentric and fragment
- dicentric and fragment
- dicentric and fragment

	Relative numbers			Relative decimal frequencies		
	n	2n	4n	n	2n	4n
A	20	40	80	.0500	.0250	.0125
B	20	40	80	.0500	.0250	.0125
C	360	1440	5600	.9000	.9000	.9000
D	0	40	240	.0000	.0250	.0375
E	0	40	240	.0000	.0250	.0375

This table assumes that breaks occur at random, that broken ends unite at random, and that each arm has the same rate of breakage. Because unions take place only between broken ends that are very close together, non-random positions of chromosomes will affect the relative frequencies. If homologous chromosomes tend to be associated then there will be a greater frequency of D and E events. Also, the longer arms will have more breaks than short arms. However, the table gives an approximation.

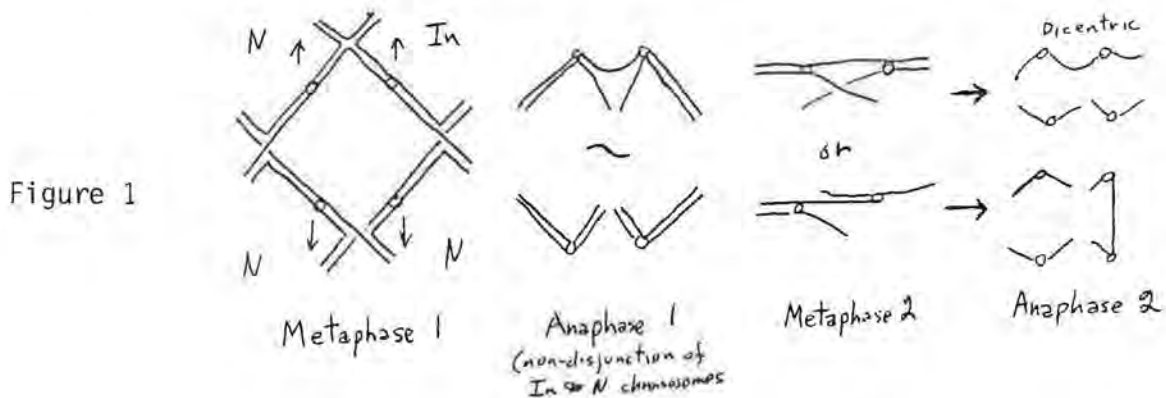
The relative frequency of reciprocal translocations is not affected by ploidy, while the frequencies of the inversions decrease and tandem duplications and pseudo-isochromosome production increases with higher levels of ploidy.

The following discussion is only a brief summary of the effects of various aberrations in autotetraploids.

Deficiencies. Most deficiencies cause lethality in haploid gametophytes and cannot be maintained in diploids. However, in an autotetraploid they can be transmitted through the gametophyte generation if they are accompanied by a complete chromosome. They persist for a time in autotetraploid populations. If there is a random mating population with an initial frequency of a deficiency (Df) of .25, after 10 generations it will decline to .0426. This assumes random chromosome pairing and an alpha value of 1/7 (equivalent to random chromatid assortment). Preferential pairing may occur in NNDfDf plants. An important pairing site may have been deleted from the Df chromosome. Preferential pairing would reduce the rate of Df elimination from the population.

Dicentric. This type of aberration is produced very frequently (C, D, and E events). However, it is very rarely found. It is believed by Steinitz-Sears and Sears (*Genetics* 42:623-630, 1957) that they generally break and give rise to two chromosomes with terminal deficiencies.

Paracentric inversions. Crossing over between normal and inversion chromosomes in the inverted region causes the formation of a bridge which undergoes a bridge-breakage-fusion cycle to form deficient or duplicate-deficient chromosomes. On the female side in diploids this bridge tends to orient chromatids so that only a complete inversion or normal chromatid goes to the functional megaspore. However, in an autotetraploid this bridge may not be resolved at the first or second division of meiosis, as shown in Figure 1.



The deficient or duplicate-deficient chromosomes are eliminated from the population. If the initial frequency of an inversion is less than 50% in a random mating population it will be eliminated. This is a result of selection against the heterozygote which fixes the gene (and chromosome arrangement) which was in the majority. The rate of elimination will depend on the frequency of crossing over in the inverted region and the amount of preferential pairing (which affects the amount of pairing between the inverted and normal segments and affects chromosome disjunction patterns). There are 12 different genotypes to be considered: NNNN, NNNIn, NNInIn, NInInIn, InInInIn, NNNDf, NNDfDf, InInInDf, InInDfDf, NNInDf, NInInDf, and NInDfDf. The Df chromosomes are of different types; some resemble the inversion chromosome and some the normal chromosome. Consequently, the expected gametic outputs of genotypes with Df chromosomes are difficult to predict because of different amounts of preferential pairing.

Pericentric inversions and pseudo-isochromosomes. Crossing over in the inversion chromosomes produces two duplicate-deficient chromosomes that are the same as pseudo-isochromosomes that are produced by radiation where opposite arms of homologous chromosomes have exchanged segments. In a diploid population a pair of pseudo-isochromosomes do not persist. They pair at meiosis and they separate. They cannot survive without each other or a complete chromosome. In an autotetraploid they can persist for a time because they may be directed to the same pole at meiosis and enter the same gamete. The elimination of a pericentric inversion from a random mating autotetraploid population (where the pericentric inversion is in the minority) is confounded by the fact that if a pair of pseudo-isochromosomes cross over in the inverted region they will restore a normal and an inversion chromosome (see Figure 2).

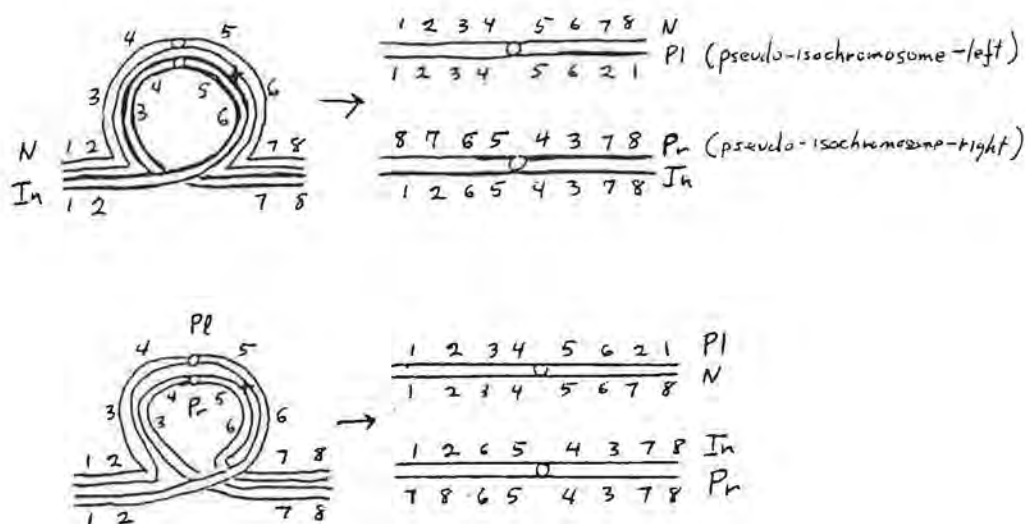


Figure 2

Eventually P1 and Pr chromosomes will be eliminated as the lethal P1P1 and PrPr gametes. As with the paracentric inversion we have a bewildering assembly of 27 genotypes (often with peculiar preferential pairing) to contend with: NNNN, NNNIn, NNInIn, NInInIn, InInInIn, NNNP1, NNPI1, NNNPr, NNPrPr, InInInP1, InInP1P1, InInInPr, InInPrPr, NNInP1, NNInPr, NInInP1, NInInPr, NNPIPr, InInP1Pr, NInP1Pr, NP1P1Pr, InP1P1Pr, NP1PrPr, InP1PrPr, NInP1P1, NInPrPr, and P1P1PrPr.

Reciprocal translocations in an autotetraploid lead to extremely complex situations. If the disjunction of chromosomes from multivalents was all alternate, then a translocation could be treated like a gene from a population genetics point of view. In maize it is about half alternate and half adjacent disjunction. Adjacent disjunction in a diploid produces unbalanced spores that abort. In an autotetraploid many types of unbalanced gametes are viable.

For the sake of brevity and clarity the genotypes of gametes and zygotes will be symbolized as sequences of 4 numbers. Thus, if the translocation was between chromosomes 1 and 2, the first number would indicate the number of chromosomes 1 and the next three numbers would indicate the numbers of 2, 1², and 2¹ chromosomes, respectively. There are 19 possible genotypes for the gametes: 1111, 2200, 0022 (balanced), 2101, 1012, 1210, 0121, 2110, 1021, 1201, 0112, 2011, 1102, 1120, and 0211 (unbalanced) and 2002, 0220, 2020, and 0202 (lethal). The non-lethal gametes would combine to form 65 zygotic genotypes. Only 5 are balanced: 4400, 0044, 2222, 3311, and 1133. Sixty other genotypes (the reader will be spared a list of them) have various degrees of imbalance.

Assuming the ends of the chromosomes paired at random the pairing configurations of NNNT (3311) plants would be 1/9 2II+IV, 4/9 II+VI, and 4/9 VIII and that of NNTT (2222) plants would be 1/81 4II, 8/81 2II+IV, 15/81 2IV, 16/81 II+VI, and 41/81 VIII. While the gametic output of quadrivalents could be computed that of sexavalents and octavalents is more difficult as the patterns of disjunction may be irregular. There would probably be frequent 5-3 separations of the chromosomes of an octavalent. Even in diploids translocation quadrivalents split 3:1 frequently. Thus we would have to consider aneuploid gametes as well. Even just considering the pairing configurations and gametic output of 65 different types of numerically euploid genotypes is a stupendous task. Also it is difficult to hypothesize the effect of competition between balanced and unbalanced gametophytes and the differential viability and reproductive success of the balanced and unbalanced sporophytes. Preferential pairing is another factor to consider.

Thus any complete theoretical treatment of the population genetics of reciprocal translocations in autotetraploids where there is adjacent disjunction is the work of a fool or a charlatan.

Tandem duplications in diploids have a low transmission rate through the pollen if they are large enough to cause imbalance affecting their competitive ability. Consequently these duplications do not become established in diploid populations. However, in an autotetraploid there is a lot of aneuploid pollen, the imbalance is reduced, and thus competition may be less intense. Also, tandem duplications can be transmitted in combination with the internal deficiency formed by the same event.

The theoretical effects of chromosome aberrations in allotetraploids also have been studied. There are many similarities and the behavior of the aberrations in allotetraploids would shed some light on their behavior in autotetraploids. Also, another approach is to obtain parthenogenetic diploids from autotetraploids that are heterozygous for aberrations. For example, it should be possible to get P1Pr plants and determine the rate of reversion to normal and pericentric inversion chromosomes.

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Preliminary characterization of certain breakpoints in B-A translocations

Cytological information has been obtained on several B-A chromosome translocations. Variable amounts of data are presented relative to one or both breakpoints in eight different B-A translocation stocks.

Good pachytene preparations were made using techniques that are somewhat different from conventional methods. Squashes were made in the usual way, but they were stained with propionic carmine for a very short period of time. This was followed with an exaggerated destaining process. In some cases, the destaining included running 45% acetic acid under the cover glass and through the cellular material of the squash. The objective was to obtain very light or understained material. These preparations were then observed with phase contrast microscopy. The combination of light staining and phase contrast usually resulted in good views of the pachytene chromosomes. In some cases, excellent observation could be made of chromomeres. In addition, the centromeres were often pronounced with this technique. This latter observation can be a very important factor in the characterization of B-A translocation stocks, since measurements have to be made, in most cases, without the aid of cross-configurations. It is feasible that other cytological studies may be accomplished using these techniques; in particular, cytological mapping of the maize genome with high resolution. Also, heteromorphic non-sister chromatids could occasionally be discerned in these preparations. The entire technique is presently being quantified.

The determination of breakpoints in hyperploid stocks with translocations between B and A chromosomes was found to be a different kind of challenge than breakpoint analyses of A chromosome translocations. Good cross-configurations were almost impossible to find. Most pachytene cells showed separate associations of BABA and ABA figures, rather than a complex of 4 chromosomes. A second concern in translocation breakpoint analyses has always been an inability to place a measure of confidence on the estimates because ratios cannot be handled by the usual statistical applications.

Attempts have been made to overcome some of the problems mentioned above. Firstly, the cytological technique described above was used in which greater resolution of chromomere patterns and centromeres was possible. Photomicrography was then employed to rapidly gain a recording of each figure encountered. Negatives were developed in a manner to enhance contrast. The negatives were placed in an artigraph instrument (generally used in cartography) and projected onto a large white surface. The very large images that were gained by this procedure could be directly analyzed. A cartographic map measurer was used to measure the various chromosome regions that could be recognized with some confidence. In most cases, the points of chromosome exchange could be discerned. This was facilitated by the very heterochromatic condition of the B chromosome and good resolution of chromomeres, centromeres, and other cytological landmarks. Although the measurements of the chromosome regions on the enlargements were in centimeters, they can be considered as arbitrary units since the objective is to gain ratios. All measurements were made at identical instrument settings. The technique is fast, and it has fairly good precision.

Several different mathematical treatments can be used to gain confidence intervals: (1) use of extremes; (2) standard error of the difference between means; and (3) the approximate variance of the error of the ratios. In this report, the use of extremes has been used. This calculation is the most conservative method, giving the largest confidence interval. All three methods have been

TABLE I

CUMULATIVE DATA FOR BREAKPOINT ANALYSES											
Trans- location	Chromo- some	Arm	Region	N	Max.	Min.	Range	Mean	Standard Deviation	Standard Error	2X Standard Error
5La	B	L	centromere to breakpoint	14	15.2	10.8	4.4	13.1	1.4288	0.3819	0.7638
5La	B	L	breakpoint to terminus	8	4.8	2.0	2.8	3.0	0.9055	0.3204	0.6408
5La	5	L	breakpoint to terminus	7	24.4	17.2	7.2	21.1	2.3940	0.9049	1.8098
3Ld	B	L	centromere to breakpoint	1	--	--	--	8.7	---	---	---
3Ld	3	L	breakpoint to terminus	1	--	--	--	6.9	---	---	---
9Sd	B	L	centromere to breakpoint	14	14.7	10.1	4.6	12.1	1.5198	0.4062	0.8124
9Sd	9	S	breakpoint to terminus	12	12.8	8.6	4.2	10.8	1.2479	0.3602	0.7204
3Sb	B	L	centromere to breakpoint	4	14.1	8.5	5.6	10.6	2.5305	1.2652	2.5304
3Sb	3	S	breakpoint to terminus	2	10.2	7.2	3.0	8.7	2.1213	1.5000	3.0000
6Lc	B	L	centromere to breakpoint	18	15.4	9.4	6.0	12.4	1.7763	0.4187	0.8374
6Lc	6	L	breakpoint to terminus	2	32.0	24.2	7.8	28.1	5.5154	3.9000	7.8000
6Lc	6	L	centromere to breakpoint	1	--	--	--	3.4	---	---	---
6Lc	B	L	breakpoint to terminus	4	7.0	3.9	3.1	5.2	1.3292	0.6646	1.3292
10Lb	B	L	centromere to breakpoint	16	12.6	9.0	3.6	10.2	0.9425	0.2356	0.4712
10Lb	10	L	breakpoint to terminus	11	22.9	10.3	12.6	15.3	3.8181	1.1512	2.3024
10Lb	10	L	centromere to breakpoint	1	--	--	--	11.9	---	---	---
10Lb	B	L	breakpoint to terminus	1	--	--	--	4.8	---	---	---
9Lc	B	L	centromere to breakpoint	13	6.4	4.7	1.7	5.2	0.4776	0.1325	0.2650
6Lb	B	L	centromere to breakpoint	5	8.6	6.2	2.4	7.5	1.0756	0.4810	0.9620
N	B	L	whole arm	28	20.4	13.6	6.8	16.4	1.5737	0.2974	0.5948
N	9	L	whole arm	4	28.0	23.0	5.0	24.8	2.2174	1.1087	2.2174
N	9	S	whole arm	3	13.4	9.7	3.7	11.8	1.9140	1.1050	2.2100
N	10	L	whole arm	5	28.2	18.0	10.2	23.1	3.7627	1.6827	3.3654
N	10	S	whole arm	5	9.4	5.1	4.3	8.0	1.7065	0.7632	1.5264
N	7	L	whole arm	1	--	--	--	31.0	---	---	---
N	7	S	whole arm	1	--	--	--	11.6	---	---	---
N	6	L	whole arm	5	38.2	24.7	13.5	30.4	5.7374	2.5659	5.1318
N	6	S	whole arm	14	7.8	4.8	3.0	6.2	0.7054	0.1885	0.3770
N	2	L	whole arm	1	--	--	--	28.3	---	---	---
N	2	S	whole arm	1	--	--	--	21.1	---	---	---

discussed and applied in an example in the 1981 MGCNL. The use of the extremes method for calculating a confidence level of approximately 95% is as follows:

$$\text{Lower limit} = \frac{\left[\begin{array}{l} \text{mean distance of the centromere} \\ \text{to the breakpoint} \end{array} \right] \text{ minus } \left[2 \times \begin{array}{l} \text{standard} \\ \text{error} \end{array} \right]}{\left[\begin{array}{l} \text{mean distance of the centromere} \\ \text{to the chromosome end} \end{array} \right] \text{ plus } \left[2 \times \begin{array}{l} \text{standard} \\ \text{error} \end{array} \right]}$$

$$\text{Upper limit} = \frac{\left[\begin{array}{l} \text{mean distance of the centromere} \\ \text{to the breakpoint} \end{array} \right] \text{ plus } \left[2 \times \begin{array}{l} \text{standard} \\ \text{error} \end{array} \right]}{\left[\begin{array}{l} \text{mean distance of the centromere} \\ \text{to the chromosome end} \end{array} \right] \text{ minus } \left[2 \times \begin{array}{l} \text{standard} \\ \text{error} \end{array} \right]}$$

Table 1 lists the cumulative data gained thus far for breakpoint analyses. Table 2 displays a summary of some of the breakpoint positions based on these

TABLE 2
SUMMARY OF CERTAIN BREAKPOINT
POSITIONS FOR SEVERAL B-A TRANSLOCATIONS

<u>Trans- location</u>	<u>Break in B</u>	<u>B-95% Confidence Interval</u>	<u>Break in A</u>	<u>A-95% Confidence Interval</u>
9Sd	.74L	± .08	.08S	± .24
10Lb	.62L	± .05	.34L	± .17
6Lc	.76L	± .08	.11L	without replication
5La	.80L	± .08		insufficient data for chromosome #5 breakpoint
3Sb	.65L	± .18		insufficient data for chromosome #3 breakpoint
9Lc	.32L	± .03		insufficient data for chromosome #9 breakpoint
6Lb	.46L	± .08		insufficient data for chromosome #6 breakpoint
3Ld	.53L	without replication		insufficient data for chromosome #3 breakpoint

data. A total of 203 measurements were made to generate the calculations in these tables. Efforts are presently being made to gain additional measurements where data are completely lacking or insufficient.

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A maize line with high tillers and ears

MET (multiple ears and tillers) maize was collected from a nationwide maize collection in Korea. It is an open pollinated flint type of maize. The MET has been grown by Korean farmers for food purposes for a long period of time. The seeds are maintained by mass selection at the Plant Genetics and Breeding Laboratory, Dept. of Agronomy, College of Agriculture, Chungnam National University. The MET was identified from the cooperative maize breeding project of the University and the Office of Rural Development (ORD).

The MET population is characterized by having seven to 12 ears on three to four tillers per plant, provided that optimum plant space is allowed. It is very unlikely that the MET might be derived from a teosinte cross or other source of exotic germplasm. It has been grown traditionally by a small group of farmers in a small village in Korea. The average plant height at harvest is about 230 cm under Korean conditions (N38°). The average plant height of tillers with usable ears is about 200 cm. The average ear size is 9 cm long and 3.5 cm wide. All tillers are borne at the nodes of the main stem below ground level. The average weight of 100 kernels is 14 g. The total dry matter of a plant is about 850 g when planted in 60 cm x 40 cm spacing. The MET is late maturing, about 15 days later than the single cross hybrid, Mo17 x B73. The MET should be useful in breeding programs for silage crops and genetic study. [Ed. note: Dr. Choe has sent two color prints of this material, which will be sent to any cooperator on request.]

Bong-ho Choe and Keun-yong Park

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Light and temperature-related behavior of coleoptiles and epicotyls

Several lines of corn we have been examining for sensitivity to light and temperature show remarkable differences which may have some use in corn breeding. Since the coleoptile and epicotyl (first internode) are responsible for getting the shoot through the soil, these two tissues and their responsiveness could be selected for in simple seedling tests which take less than a week to run. The behavior of these two tissues has been quite spectacular among certain lines and has been used routinely in freshman classes to demonstrate light sensitivity in plant tissues. Table 1 gives some of the preliminary data we have collected on several commercially available lines--mostly sweet corns.

Seeds were germinated on 9" x 9" x 2" glass-covered stainless steel pans lined with toweling and facial tissue moistened with 70 cc of tap water. Plants were grown 60 cm from a light source supplied by fourteen 200 W cool white fluorescent lamps supplemented by twelve 50 W incandescent bulbs. Continuous light conditions were maintained for the seven-day test period. 20 coleoptiles and epicotyls were measured and recorded as average values in Table 1. Since different wavelengths have been reported to be effective in the control of seedling growth, plants were grown under plastic filters of blue, green, yellow, red and far-red along with controls under white light and complete darkness for the seven-day period. Duplicate experiments were run in growth chambers set for 21 C and 27 C.

	Light Conditions							Temp. C.	Tissue
	Blue	Green	Yellow	Red	Far-red	White	Dark		
MM	3.5	4.3	3.6	4.2	4.1	2.4	1.9	21	Coleoptiles
475	3.7	4.6	3.5	4.3	5.0	2.2	3.6	27	
Silver	3.7	3.7	2.1	2.5	2.0	2.8	0.7	21	Coleoptiles
Queen	4.3	4.0	4.0	4.0	4.0	3.1	1.6	27	
Sprite	3.1	2.2	2.5	3.1	4.5	1.9	4.1	21	Coleoptiles
	3.2	3.3	2.6	3.1	4.4	2.0	4.5	27	
Gold	3.6	3.3	3.0	3.4	4.4	1.8	1.6	21	Coleoptiles
Cup	3.1	3.5	2.6	3.2	4.4	1.8	3.8	27	
Sun	3.3	3.2	2.3	2.8	4.3	1.7	2.3	21	Coleoptiles
Dance	3.3	3.3	2.6	2.8	4.4	1.4	4.0	27	
Blitz	2.1	1.9	2.4	2.2	2.7	1.9	0.9	21	Coleoptiles
	3.3	3.1	2.7	2.9	3.9	1.9	1.9	27	
MM	1.2	1.6	0.8	1.6	4.8	0.5	7.3	21	Epicotyls
475	1.5	1.6	0.7	1.7	4.4	---	11.4	27	
Silver	3.0	3.6	1.3	2.2	4.8	0.6	2.8	21	Epicotyls
Queen	3.1	3.7	1.8	3.4	9.3	1.0	9.9	27	
Sprite	1.3	0.4	0.6	1.1	4.8	---	12.2	21	Epicotyls
	1.1	1.2	0.7	1.2	4.0	---	14.3	27	
Gold	1.4	1.1	0.6	0.9	3.6	---	6.4	21	Epicotyls
Cup	1.0	1.3	0.7	1.1	3.5	---	7.2	27	
Sun	1.2	1.2	0.9	1.1	4.4	---	7.6	21	Epicotyls
Dance	0.9	0.9	0.7	1.0	3.6	---	10.8	27	
Blitz	1.1	0.6	1.2	1.7	5.1	0.5	5.2	21	Epicotyls
	1.7	1.6	0.9	1.4	3.7	---	7.4	27	

Table 1. Lengths (cm.) of coleoptile and epicotyl tissues after seven days under different light conditions.

Different levels of temperature sensitivity can be seen for dark-grown seedlings for both coleoptiles and epicotyls. Under filters, Silver Queen showed the greatest temperature differences for coleoptile elongation while, except for far-red, little temperature-related variation was noted for its epicotyls. Far-red filters permitted the greatest amount of elongation though generally less than that of dark-grown seedlings. Most coleoptiles show elongation in the dark; Silver Queen is exceptional since elongation appeared to be inhibited under dark conditions. In yellow light most epicotyls show reduced elongation. MM 475, a local field-corn hybrid, shows quite vigorous coleoptile elongation under all filters while the epicotyls show considerable inhibition for the same filters. In this hybrid it would seem from seedling performance that the coleoptile is responsible for getting the shoot above ground under reduced light conditions.

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DIMBOA and aphid resistance

In the summer of 1981, field experiments were carried out to determine the levels of resistance in corn to the corn leaf aphid. Thirteen widely used corn inbreds were planted in two locations in Southern New Hampshire. Sufficient natural infestations of the corn leaf aphid occurred in both locations, and a visual rating scale and index system used by Long et al. (Crop Science 17:55, 1977) was used to measure the levels of resistance.

A modified version of the procedure developed by Argandona et al. (Phytochem. 19:1665, 1980) was used to determine the hydroxamic acid content in the corn tassels. A significant correlation ($r = .63$) was found between hydroxamic acid concentration and resistance to the corn leaf aphid in the thirteen corn inbreds.

Evaluations of several lab procedures which quantify hydroxamic acid content including the rapid procedure are also being performed. Recent results suggest the presence of interfering compounds in corn stem extracts obtained using the rapid procedure. Also, certain environmental conditions (i.e., soil medium, light intensity) under which the corn is grown have been found to affect the results obtained using the rapid procedure.

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Pollen yield reduction in opaque-2 maize

The opaque-2 gene (o2) in maize has been the subject of a great deal of study in relation to its effect on the improvement of protein quality in maize endosperm. Researchers regard the reduction of zein synthesis as the primary effect of this gene.

The mechanism by which the reduction of zein is brought about in the mutant has not yet been established. Most of the current research on the mechanism of zein synthesis in opaque-2 seems to be based on the assumption that the primary effect of this gene is to reduce the production of zein. While this lower zein synthesis is the most striking effect of the opaque-2 gene, this is only one of the most obvious alterations caused by this mutation. Evidence indicates that the opaque-2 locus is regulatory and that, besides regulating the synthesis of zein, it has a wide range of pleiotropic effects on the plant. For instance, the opaque-2 mutant changes the following characteristics of a normal plant: (1) reduces the synthesis of zein and yield (probably due to a lower rate of starch synthesis); (2) delays germination (about 2 days) and pollen shedding; (3) accumulates more water in the developing endosperm; (4) increases the production of albumins and globulins and ribonuclease activity in immature endosperms. Hence, the opaque-2 gene alters several traits in the maize plant. In addition to these abnormalities conditioned by the opaque-2 gene, we have detected an alteration not previously described, which affects the production of pollen. We found lower yield of pollen and, as other authors have shown, reduction in the weight and circumference of the cob. The development of pollen grains occurs before the beginning of zein synthesis. The data presented in this report indicate that the opaque-2 gene (o2) begins to exert its effect before the synthesis of zein.

Because of all these changes caused by the opaque-2 gene, we wondered at which level this gene exerts its primary effect. For instance, could the opaque-2 gene affect the structures (the tassels and the ears) where both the male and female gametophytes are formed? This question originated as a result of our research with the opaque-2 mutant, and our observation that opaque-2 tassels do not produce as much pollen as their normal counterparts. To verify our previous experiences, we planted normal and opaque-2 seeds (both in the W64A background) at the University of California, Davis (where plants are grown under irrigated conditions) at two different dates. Pollen was collected from these plants by placing paper bags on the tassels at around 6:30 A.M. and collecting the pollen at around 1:00 P.M. These collections were made for several days, until no further pollen shedding was noticed. The collected pollen was immediately weighed.

After the tassels had finished shedding pollen, they were removed from the plant at the height of the last leaf on the stem. The tassels were dried for 6 weeks at room temperature. The total weight and the moisture content of each tassel were determined. The secondary spikes were separated from the main spike (continuation of the stem); their respective weights were also determined, and the numbers and lengths of the secondary spikes found in each tassel were recorded.

The ears of the plants from which pollen collections and tassel measurements were made were harvested 65 days after pollination. The kernels were shelled and the cobs were dried for 8 weeks at room temperature, and moisture determinations were made at the end of this drying period. The weight and the circumference of each cob were recorded. The data were analyzed using the t test for difference between two means with unequal sample sizes.

The results of the pollen collections were as follows:

<u>Date of Collection</u>	<u>+/+</u>	<u>o2/o2</u>
First Planting (23 plants)		
6/15	1,085	----
6/16	2,100	1,040
6/17	3,304	1,059
6/18	<u>2,651</u>	<u>704</u>
TOTAL	9,140	2,803
Second Planting (52 plants)		
6/26	5,491	----
6/27	7,890	600
6/28	4,396	1,337
6/29	4,163	2,262
6/30	1,697	1,670
7/1	390	1,580
7/2	<u>----</u>	<u>1,028</u>
TOTAL	24,027	8,477
GRAND TOTAL	33,167	11,280

The differences in the amounts of pollen collected between normal and opaque-2 are significant: there is a drastic reduction in the pollen grains produced by opaque-2. For each mg of pollen produced by opaque-2, its normal counterpart yielded 3 mg. The results of the second planting verify this observation; the reduction of pollen in opaque-2 was the same as in the first planting.

To determine the cause for the lower production of pollen in opaque-2, several measurements of weight and length of the tassel structures were made. The results presented in Table 1 are the means of 30 tassel measurements for normal and 35 for

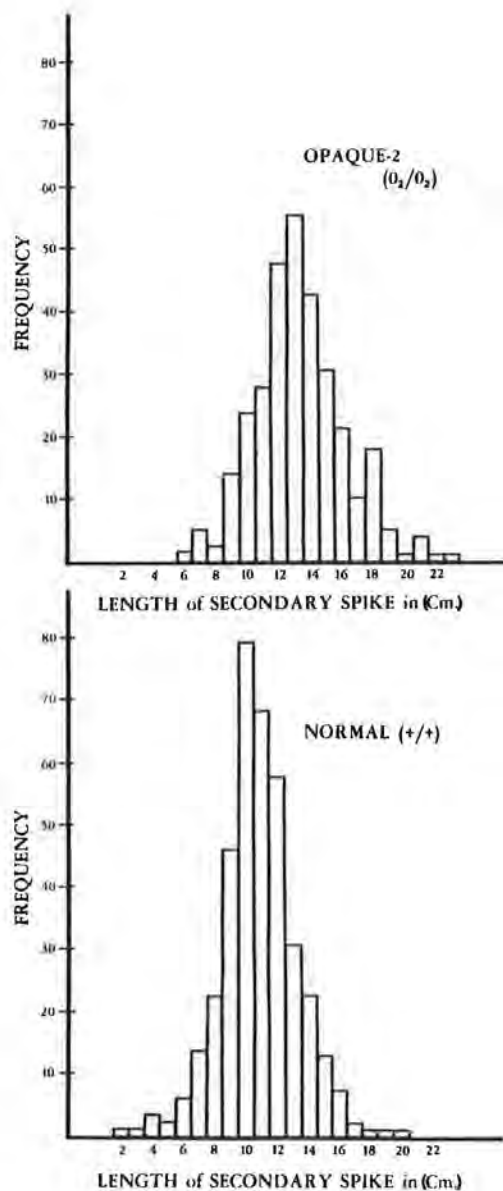
Table 1. Tassel and cob measurements of normal (+/+) and opaque-2 (o2o2) maize in W64A background.

	Normal (+/+)			Opaque-2 (o2o2)			Difference between the means of normal and opaque-2		
	M	\bar{x}_1	S ₁	n ₂	\bar{x}_2	S ₂	$S_{\bar{d}}$	t	p
Total tassel weight (mg)	30	3,660	485	35	3,173	413	111	4.4	0.001
Main spike (mg)	30	1,354	127	35	1,493	202	43	3.2	0.01
Secondary spike (mg)	30	2,306	420	35	1,680	338	100	6.2	0.001
Length of secondary spikes (cm)	545	10.8	2.4	297	13.3	2.9	0.2	12.5	0.001
Secondary spikes per tassel (No.)	545	18.2	4.8	297	8.5	2.6	0.3	32.3	0.001
Circumference of cob (cm)	29	106.1	4.6	29	95.9	4.5	0.8	12.7	0.001
Weight of cob (g)	29	30.8	3.7	29	24.1	4.4	0.7	9.0	0.001

Weights corrected for moisture content. Moisture content at the end of six weeks after harvest for normal was 11.0 percent, and for opaque-2, 10.8 percent.

opaque-2. When the means of the total tassel weight of normal and opaque-2 are compared, the difference is highly significant. On the average, the weight of a normal dried tassel exceeds an opaque-2 tassel by 487 mg. A similar analysis of the weights of the main spike also shows highly significant differences, however, opaque-2 was 139 mg heavier. A comparison of the weights of the secondary spikes reveals that normal exceeds opaque-2 by 656 mg. This difference is also highly significant, $P < 0.001$, and the difference in this category is even greater than in the two previous comparisons (the variances for each of the three categories are homogeneous). The normal secondary spikes weigh more because there are more than in opaque-2. In normal maize an average of 18 secondary spikes were present, while in opaque-2 only 8 secondary spikes were found. Pollen grains are produced in the spikelets, most of which are located on the secondary spikes, and the drastic reduction of pollen in opaque-2 seems to be due to the lower number of secondary spikes in the mutant.

The frequency distribution of the lengths of the secondary spikes is shown in Figure 1. In both genotypes, the lengths of the secondary spike have a normal distribution and their variances are homogeneous. However, they have different



means; opaque-2 has fewer and longer secondary spikes than normal. On the average, the length of a secondary spike in normal maize is 10.8 cm and in opaque-2 is 13.3 cm. The difference between these means is also highly significant. The increase in length of the secondary spikes of opaque-2 may have occurred as a result of the fewer secondary spikes produced by opaque-2 tassels. In relation to pollen production, however, the increase in the length of the secondary spikes of opaque-2 did not compensate for the fewer numbers of secondary spikes found in the mutant tassels. The reduction in the numbers of secondary spikes (from 18 in normal to 8 in opaque-2) seems to be one of the main causes for the severe reduction in pollen yield in opaque-2.

The differences between normal and opaque-2 cobs are highly significant for circumference and weight. Previous studies have detected similar reduction in cob weight in hybrids carrying the opaque-2 gene (P. S. Baenzinger and D. V. Glover, Agron. Abstr. 1975; D. Makonnen, and L. F. Bauman, Euphytica 25, 1976).

The reduction in the number of secondary spikes and in the circumference and weight of the opaque-2 mutant cobs implies that the opaque-2 gene (*o2*) affects the development of the structures where the male and female gametophytes are formed. This development takes place before the formation of the endosperm tissue and much before the synthesis of zein (zein synthesis has not been detected until about 10 days after fertilization). From the data presented in this paper, we must conclude that the primary effect of the opaque-2 gene (*o2*) is not in the reduction of zein synthesis.

Microscopic observations of normal and opaque-2 pollen grains do not reveal any obvious differences in size or shape. Therefore, we suggest that the lower production of pollen by opaque-2 tassels is not due to the reduction in size of the pollen grain, but to a reduction in the number of pollen grains. A study is underway to determine the number of pollen grains produced per floret, which should give us a better understanding of the reduction of pollen grain yield in opaque-2.

Last summer (1981), pollen was collected from normal and opaque-2 plants in Illinois High Protein (IHP) background. IHP normal plants yielded 3.5 times more pollen than IHP opaque-2, which leads us to believe that our original observation of lower pollen production in W64A02 is correct, and that the reduction is not due to the genetic background but due to the opaque-2 gene itself.

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Cloning and analysis of genes of the zein multigene system

Genetic and biochemical evidence indicates that the zein proteins are coded by a large number of genes, although estimates of the exact number still vary greatly. In order to analyze the organization and expression of these genes it is necessary to isolate genomic clones that will contain only one or a few zein genes. We have previously reported the isolation and characterization of one such zein gene (Mol. Gen. Genet. 182:440-444, 1981) but we now have several new and larger clones.

Zein coding sequences were identified from a maize DNA bank of Eco RI restricted seedling DNA of variety A619 cloned into lambda gt WES. cDNA clones specific for either the 19,000 or 21,000 dalton protein classes were used as probes for selecting the genomic clones. This method has yielded so far about 50 zein genomic clones with maize inserts varying in size from less than 2 thousand to nearly 14 thousand base pairs. After subcloning into pBR 328 we have analyzed 6 of these fragments in detail: 2 for the 19,000 dalton proteins and 4 for the 21,000 dalton size class.

The following types of analyses have been employed to gather information about the zein genes.

(a) Electron microscopic observation of hybrids formed between the genomic clones and endosperm poly rA⁺RNA (R-loops) and the cDNA clones (D-loops): These experiments demonstrate that all clones contain a complete zein gene with no detectable intervening sequences.

(b) Mapping of restriction endonuclease recognition sites has shown that the zein genes contain few sites relative to their flanking sequences. It has also been found that all 6 cloned fragments differ from each other.

(c) Hybridization of the genomic clones with restriction endonuclease treated and electrophoretically fractionated total maize DNA (Southern analysis) has revealed patterns similar to those shown when the cDNAs are used as hybridization

probes. Each of the genomic clones gives the pattern characteristic for its specific cDNA. This indicates that the flanking sequences do not contain major repetitive sequences. It has also been found that the sequences flanking the zein genes show some homology between clones but there are also some areas that do not cross-hybridize.

It will be necessary to analyze further clones in order to obtain a more general picture of zein gene structure and organization. It is significant that of 6 clones analyzed by us, all differ, to varying degrees, from one another. The identification and interpretation of the mechanisms controlling zein expression will therefore probably be complex and involve considerable further effort.

J. Pintor-Toro, P. Langridge and G. Feix

Transcriptional studies of the zein system

The transcription of the zein genes presents an interesting regulatory phenomenon since not only are the genes expressed at very high levels but their expression is also developmentally and tissue specific. The use of cDNA clones specific for the major zein protein classes has enabled the quantitation of mRNA levels during development and the identification of precursor forms of the zein mRNAs.

Total RNA isolated from kernels of a growing maize ear was electrophoretically fractionated on agarose gels under strictly denaturing conditions (2.2 M formaldehyde) and transferred to nitrocellulose. Once immobilized on nitrocellulose the zein specific RNAs were identified by hybridization with ³²P-labeled zein cDNA probes (Northern technique). The mature zein mRNA was readily seen at its expected size of about 1,000 bases but several other bands were also strongly labeled. These had sizes of about 1,600 bases and greater. The existence of the first of these precursor mRNAs (1,600 bases long) was subsequently confirmed by electron microscopic analysis of RNA:DNA hybrids formed between poly rA⁺RNA and a genomic clone containing a zein gene. It is an interesting feature of these precursors and possibly indicative of specialized transcriptional control of zein synthesis that the precursor forms of the zein mRNA are present in concentrations approximately equivalent to those of the final sized mRNA.

The Northern technique has also been used to analyze zein mRNA over the developmental period of zein synthesis in both wild-type and opaque-2 mutant endosperms. The opaque-2 mutation reduces the amount of the zein proteins synthesized, particularly the 21,000 dalton proteins. It has now been possible by this method to demonstrate that levels of hybridizable RNA for both major zein protein classes correspond with levels of the proteins in the endosperm. Furthermore, this method has enabled us to exclude processing of the precursor and specific degradation of the zein mRNAs as sites of action of the opaque-2 mutation. It, therefore, appears that the biochemical lesion in opaque-2 is a transcriptional event rather than the previously postulated translational modification. The mechanism and role of the opaque-2 mutation on the transcription of zein genes will require further analysis of the structure and organization of these genes in normal and mutant plants.

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Transposition event in tissue cultured cells of S-cms genotype in maize

The S type of cytoplasmic male sterility (S-cms) is characterized by the presence of two unique plasmid-like mitochondrial DNAs designated as S1 and S2 (Pring and Levings, 1977, PNAS 74:2904). Cytoplasmic mutations leading to restoration of male fertility were associated with integration of these plasmid-like molecules into high molecular weight mt DNA (Levings et al., 1980, Science 209: 1021). Molecular analyses of such high frequency mutations as well as physical characteristics of these plasmid DNAs suggest similarities with transposable elements. Indeed, Levings et al. (1980) referred to these mutations as transpositional events. We report here a similar type of transpositional event involving S1 and S2 under tissue culture propagation of callus tissue. Correlations of this event with a certain callus morphology is of special significance.

Callus cultures were initiated from immature embryos of WF9 inbred line carrying the S-cms trait, according to Green and Phillips (1975, Crop Sci. 15:417). Plants were regenerated from scutellar calli after six months of culture. A portion of the callus was also maintained as a callus line. This callus culture is now approximately two years old and retains its original compact and highly organized morphology (referred to as 'organized' callus); however, plant regeneration from it appears no longer possible. This callus culture has also been used to select and isolate a total of four independent friable types of callus cultures during the past two years, as done previously in the Black Mexican Sweet line (Chourey and Zurawski, 1981, TAG 59:341). The friable and the 'organized' calli have been maintained on the same medium and under identical growth conditions for the analysis for S1 and S2 DNAs.

The 'organized' callus, four independent isolates of friable callus and the leaves of ten plants regenerated from the initial scutellar callus were analyzed for the presence/absence of S1 and S2 DNAs according to Kemble (1980, TAG 57:97). These plasmid-like DNAs were readily detectable in leaf extracts by UV fluorescence of ethidium bromide stained agarose gels. Similar analysis of 'organized' and friable calli extracts, however, showed only trace amounts of S1 and S2 DNAs in the former and no detectable levels in the latter. Southern hybridization using nick-translated radioactive probes of cloned S1 and S2 DNA sequences revealed significant hybridization corresponding to the position of S1 and S2 in the 'organized' callus. However, no hybridization was seen at the position corresponding to S1 and S2 in extracts obtained from the four friable isolates. Instead, these extracts showed intense hybridization at the position of high molecular weight mt DNA, indicative of transposition of S1 and S2 into the mt DNA. It is noteworthy in this analysis of tissue cultured cells that altered callus morphology (i.e., friable in contrast to 'organized' type) was associated with the transpositional event of these DNAs. The significance of such a correlation remains unclear.

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Protoplast culture in maize

Three cell suspension cultures have been obtained from three independent isolations of friable callus cultures originating from immature embryos of WF9 inbred line carrying the S-cms trait. Protoplasts are readily obtained from these cell lines under conditions described previously (TAG 59:341). These protoplasts would

regenerate cell wall as visualized by the appearance of calcofluor fluorescence after about 48 hours of culture. However, no sustained cell divisions have been seen so far in these cells under conditions in which protoplasts derived from Black Mexican cell line divide and proliferate to give cell colonies.

Prem S. Chourey

The isolation and characterization of a Sh1 candidate clone

The shrunken-1 (Sh1) locus in maize, which codes for the enzyme sucrose synthetase, is of major interest because of its interaction with the Ac Ds controlling element system and its tissue-specific pattern of expression. The determination of the site(s) of integration of Ds in relation to the structural region of Sh1 requires work at the DNA level. In addition, DNA technology will facilitate the study of the tissue-specific pattern of expression of Sh1 since this can be investigated using DNA probes.

A first step towards understanding gene function and expression is the determination of gene structure. Accordingly, we have isolated and characterized Sh1 genomic candidate clones. The candidate Sh1 clones were isolated by screening a Black Mexican corn genomic DNA library contained in the Eco RI site of the Charon 4A lambdaoid bacteriophage vector (Sheldon, 1980, Ph.D. Thesis, University of Wisconsin). To locate candidate Sh1 clones, the DNA library was screened with the insert of the cloned cDNA probe prepared from sucrose synthetase coding mRNA and generously provided to us by Nina Fedoroff. Screening yielded six purified Sh1 candidates from a library of more than 10^6 clones. Of these six clones, one (17.6) was chosen for further analysis.

For verification purposes, the candidate Sh1 clone 17.6 was analyzed using the Southern transfer technology. DNA from 17.6 was digested with various restriction enzymes, electrophoresed through agarose, transferred to nitrocellulose, hybridized under our normal conditions at 69 C in 6 X SSC Denhardt's solution to the cDNA probe SS-8 provided by Nina Fedoroff, washed at 69 C in 3 X SSC, and autoradiographed. The results of the Southern analysis are presented in Table 1, which shows the sizes of the fragments from 17.6 which hybridized to SS-8. It is important to note that Bgl II and Pst I each produce two fragments in 17.6 that hybridize to SS-8. Since Bgl II and Pst I each cut the cDNA probe into two fragments (Fedoroff, unpublished and confirmed by us) this result implies that the 17.6 genomic clone and the SS-8 cDNA probe are similar in structure around their Bgl II and Pst I sites. As a further test to verify that 17.6 has regions of good homology with SS-8, hybridization was done under stringent conditions. In this experiment 17.6 DNA was digested with Bgl II, electrophoresed through agarose, transferred to nitrocellulose, hybridized according to our normal procedure, washed in 0.1 X SSC (a stringent condition) and autoradiographed. The same two fragments hybridized to SS-8. This experiment strongly implies that the candidate clone either contains the Sh1 gene or is closely related to it.

We also compared the restriction fragment data from our 17.6 genomic clone to the genomic Southern data generated by Burr and Burr (1981, Genetics, 98:143) and the Starlinger group (Döring et al., Mol. Gen. Genetics, in press). In these comparisons it should be noted that there are at least two important differences. The wild type genotype probed by the above two groups is believed to be the progenitor of some sh1-mutable alleles whereas we used Black Mexican. Secondly, the lengths of the cDNA probes differ. Lengths of 620 and 285 nucleotides were reported by Döring et al. and Burr and Burr, respectively. Fedoroff has estimated the size of the insert in SS-8 to be 700 to 750 nucleotides. From restriction maps of SS-8, using Hind III fragments of pBR 322 as molecular weight markers, we calculate a total length of 700 nucleotides for the SS-8 insert.

Table 1: Hybridizing Restriction Fragments from Genomic Clone 17.6

These results were obtained by using the technique of Southern. Molecular weights were estimated using a marker mix of lambdaoid bacteriophages (Blattner et al., 1977, Science 196:161) and a computer program that fits a polynomial to the marker mix data (Sheldon, unpublished).

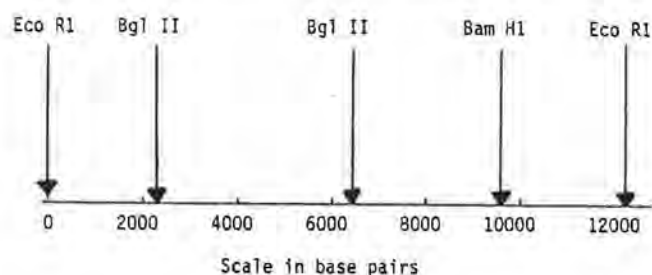
	<u>Fragment Sizes in Base Pairs X 10³</u>	
Bam HI	24*	(approximate)
Bam HI/Eco RI	10	
Bgl I	12	(approximate)
Bgl I/Eco RI	5.6	
Bgl II	7 ⁺	4
Bgl II/RI	5.5	4
Eco RI	12	
Hind III	2.3	<1
Hind III/Eco RI	2.3	<1
Pst I	1	<1
Pst I/Eco RI	1	<1
Sst I	2	
Sst I/Eco RI	2	

* The left site involved in forming this fragment was located in the left end of the Charon 4A vector.

⁺ The right site involved in forming this fragment was located in the right end of Charon 4A (Blattner et al., 1977, loc. cit.)

In fair accordance with the data of the other groups, we find relatively large Bam HI/Eco RI and Eco RI fragments which hybridize to our probe. (The large Bam HI fragment in Table 1 consists partly of vector.) The smaller size of our Eco RI fragment may be expected because of the polymorphism in Eco RI fragments reported by the Burrs. Also in agreement with earlier data, we find a 2 kbp Sst I fragment and a small (less than 1 kbp) Hind III fragment.

Although the Burr and Starlinger groups probed the same genotype, differences in resultant restriction maps were reported. For example, Döring et al. reported an 11.0 kbp Bgl I/Eco RI fragment but the Burr map predicts a 5.5 kbp Bgl I/Eco RI fragment. We observed a 5.6 kbp fragment. We do, however, find differences with the Burr map. We place a Bam HI site within the 5.5 kbp Bgl II/Eco RI fragment whereas the Burr map shows the Bam HI site outside of this fragment. This difference in maps could, theoretically, be due to polymorphism.

Figure 1: Restriction Map of Genomic Clone 17.6

This preliminary restriction map was constructed using some of the data in Table 1. The region that hybridizes to the cDNA probe SS-8 is contained between the Bgl II site at 2200 bp and the Bam HI site at about 10000 bp.

As mentioned above, analysis of the relatively large Fedoroff cDNA probe reveals internal Pst I and Bgl II sites. As predicted, we observed two bands hybridizing to the probe when the genomic clone was restricted with Pst I or Bgl II (Table 1). Unexpectedly, Hind III digests yield two hybridizing bands. Neither Fedoroff nor we can find an internal Hind III site within the SS-8 insert. It is conceivable that mutational alterations could have created or abolished this internal Hind III site. It is also conceivable that the site of Hind III action is in an intron. Finally, we can not definitely rule out, at this time, the possibility that we have cloned a gene very similar in structure to Sh1.

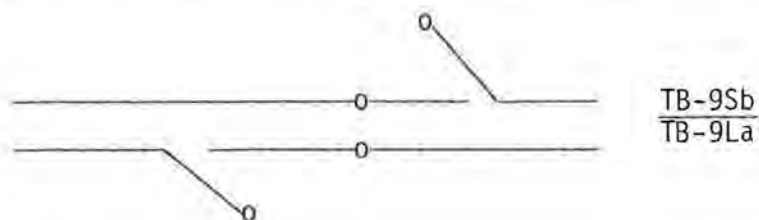
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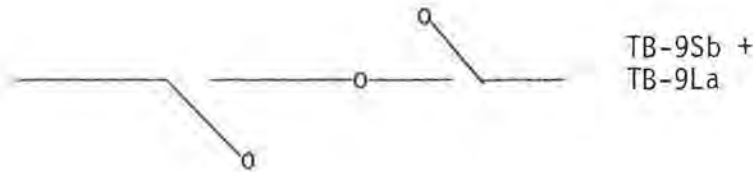
A new method of duplicating chromosome segments using B-A translocations

The nondisjunction of B-A chromosomes provides a useful method for varying the dosage of specific chromosome segments of corn. However, the method is only applicable to A chromosome regions that are distal to B-A translocation break-points. Proximal regions cannot be duplicated by nondisjunction. Nevertheless, it is possible to duplicate proximal regions using B-A translocations. The procedure requires the production of double translocation chromosomes carrying one B-A interchange in the long arm and one in the short arm.

A double translocation was derived for chromosome 9 using TB-9Sb and TB-9La. The two translocations were combined in a single plant, producing a double heterozygote with one 9-B from each translocation and one B-9 from each. Complete pairing in meiosis is as follows:



A double translocation was extracted from crossing over between the two 9-B chromosomes:



The proper combination of crossing over plus meiotic disjunction required to produce the double translocation occurs infrequently. Therefore, a selective method for identifying it was necessary. The simplest method of selection might be identification of double nondisjunction using 9S and 9L markers. However, in this case a modified TB-9Sb was used that eliminated the need for 9L markers (Translocation 2150; see Ch. 44 in Maize Breeding and Genetics, 1978). The translocation lacks the distal tip of the 9-B chromosome, which contains a factor required for nondisjunction. In the double heterozygote (TB-9Sb-2150/TB-9La), crossing over can produce a double translocation which contains the missing nondisjunctional factor. Only the double translocation is capable of giving nondisjunction of the B-9 from TB-9Sb. The double heterozygote was crossed as male to a bz bz tester and kernels with the bz phenotype were selected. These contained the double translocation. Some selection in progeny crosses was required to make certain that the B-9 from TB-9La had not been lost by nondisjunction.

Once the double translocation was established, it was crossed as male to a bz bz wx wx B B Pl Pl tester to select double hypoploid plants lacking both B-9's. Progeny with the bz plant phenotype were selected, since they lacked the B-9 from TB-9Sb. Among these plants, root tip chromosome counts were used to select plants with the 9 9-Ba+b constitution. Meiotic samples from 9 9-Ba+b plants frequently showed a lack of pairing between the chromosomes at diplotene. This is not surprising considering the evidence of Burnham et al. (Gen. 71:111-126, 1972) that corn chromosomes initiate pairing near their distal ends. The 9 and 9-Ba+b have no ends in common. The frequent lack of pairing in the double hypoploid allowed recovery of the 9-Ba+b chromosome in crosses of the type 9(wx) 9-Ba+b(Wx) X wx wx. Migration of the univalent 9 and 9-Ba+b to the same pole resulted in viable Wx progeny (9 9 9-Ba+b). By itself, the 9-Ba+b is lethal due to deficiencies.

In 9 9 9-Ba+b plants, the central region of chromosome 9 has been duplicated. This duplication may be useful in experiments which involve gene dosage. Recent tests of transmission of 9-Ba+b indicate that a homozygous duplication of the chromosome can also be produced. The Wx allele is a precise marker of the 9-Ba+b chromosome since it virtually never recombines with the normal 9. It is located at the distal tip of the 9-Ba+b short arm. Several 9(wx) 9(wx) 9-Ba+b(Wx) plants were crossed as male and female to a wx wx tester. Other such plants were crossed as male to a wx wx tester and self pollinated. Transmission of Wx in these crosses is given in the following chart:

Family Numbers of Cross	Type of Cross	Classification		
		Wx	wx	%Wx
4069-1 x 4063	Duplication as ♀	53	175	23%
4122 x 4069-1	Duplication as ♂	25	244	9.3%
4069-3 x 4064	Duplication as ♀	50	193	21%
4059 x 4069-3	Duplication as ♂	33	349	8.6%
4069-2 ♂	Duplication selfed	67	132	34%
4059 x 4069-2	Duplication as ♂	25	287	8.0%
4206-1 ♂	Duplication selfed	90	168	35%
4122 x 4206-1	Duplication as ♂	31	226	12%
4206-4 ♂	Duplication selfed	86	199	30%
4122 x 4206-4	Duplication as ♂	19	311	5.8%

The extra (Wx) chromosome is transmitted at a reasonable rate through both male and female parents. It should, therefore, be possible to select 9 9 9-Ba+b 9-Ba+b plants from the self pollinations. Selection can be made by identifying plants with all Wx pollen. It is also possible that the homozygous duplications will be stable and breed true. This would be expected if preferential pairing between structurally similar chromosomes is 100% and bivalents are always formed. Whether or not a stable 22-chromosome line can be produced depends on the mechanism of chromosome pairing. A stable line should be possible if chromosome pairing: (a) always begins in distal chromosome regions, and (b) synapsis of internal regions always follows, and (c) independent pairing of proximal regions always occurs last. Once a homozygous duplication line is established, its stability will be tested using Wx transmission tests and cytological examinations of bivalent pairing at diplotene.

W. R. Carlson

Further evidence for a role of centromeric heterochromatin in B chromosome nondisjunction

The high loss phenomenon was described by Rhoades et al. (PNAS 57:1626-32, 1967). It was found that B chromosomes cause A chromosomes to undergo a faulty nondisjunctional process at the second pollen mitosis, which results in chromosome breakage. The event only occurs in a certain genetic background and is believed to depend on non-replication of heterochromatic knobs. Rhoades and Dempsey (Gen. 71:73-96, 1972) interpreted the high loss phenomenon as evidence for a role of heterochromatin in B chromosome nondisjunction. Specifically, they proposed that centromeric heterochromatin of the B fails to replicate at the second pollen mitosis, resulting in nondisjunction. Evidence was later presented showing that identical types of heterochromatin are present in both A chromosome knobs and centromeric heterochromatin of the B (Pryor et al., PNAS 77:6705-9, 1980).

Independent support for a role of centromeric heterochromatin in nondisjunction was presented recently by Carlson and Chou (Gen. 97:379-89, 1981). They reported work with the B-A translocation B-9Sb and a B-9 isochromosome derived from it. The isochromosome was found to be a pseudoisochromosome because of asymmetry in the centromeric region. One arm of the isochromosome contains a complete B-9 (except the short arm) whereas the other arm has little or no centromeric heterochromatin. The pseudoisochromosome (also called the original isochromosome) is

unstable, and six telocentric derivatives were obtained from it. Four of the telocentrics came from the isochromosome arm that lacks centromeric heterochromatin while two arose from the opposite arm. The ability to carry out nondisjunction at the second pollen mitosis was extremely low in telocentrics lacking centromeric heterochromatin but not in those which contained it.

Since the comparisons made between telocentric B-9's with or without centromeric heterochromatin were not carried out using similar genetic backgrounds, an inbreeding procedure was begun. Inbreeding of the telocentrics will take several generations. However, a comparison of inbred isochromosome stocks can be made now. The original isochromosome contains centromeric heterochromatin as already described. A second isochromosome that lacks centromeric heterochromatin was also isolated (Gen. 97:378-89, Fig. 8). The isochromosome lacking centromeric heterochromatin (also called the new isochromosome) was derived by misdivision of a telocentric B-9, which in turn came from the original isochromosome.

Both the original isochromosome (+ heterochromatin) and the new isochromosome (- heterochromatin) were inbred for four generations to a $\underline{Bz} \underline{Bz} \underline{wx} \underline{wx}$ tester line. From each inbred line, four plants carrying an iso B-9 were selected. Crosses were: $\underline{bz} \underline{bz} \underline{wx} \underline{wx} \times 9(\underline{bz} \underline{wx})$ 9-B(\underline{Wx}) iso B-9($\underline{Bz} \underline{Bz}$). The results of classifying three ears per male parent are given below:

Male Parent	$\underline{Bz} \underline{wx}$	$\underline{Bz} \underline{Wx}$	$\underline{bz} \underline{Wx}$	$\underline{Bz}/\underline{bz} \underline{Wx}^*$	$\underline{Bz}/\underline{bz} \underline{wx}^*$
Original Iso					
4418-3	1,108	137	14	14	2
4418-4	985	106	27	11	3
4418-9	1,124	120	29	12	3
4418-11	1,079	141	44	20	6
	<u>4,296</u>	<u>504</u>	<u>114</u>	<u>57</u>	<u>14</u>
New Iso					
4417-6	1,056	191	6	10	1
4417-7	1,053	208	5	24	1
4417-9	1,178	194	4	15	4
4417-11	1,174	115	5	15	3
	<u>4,461</u>	<u>708</u>	<u>20</u>	<u>64</u>	<u>9</u>

*Kernels were classified as chimeric ($\underline{Bz}/\underline{bz}$) only if (a) more than one recessive sector was present, or (b) a single recessive sector was present which covered at least 1/4 of the endosperm. Small single sectors are not included in the table, since their identification becomes subjective at very small sizes.

In the crosses of both the original isochromosome and the new isochromosome the rate of \underline{Wx} transmission was well below 50% (13.5 - 15%). Selection against \underline{Wx} resulted from genetic imbalance of the 9-B(\underline{Wx}) iso B-9($\underline{Bz} \underline{Bz}$) chromosome combination. The more successful meiotic products contained 9($\underline{Bz} \underline{wx}$).

Comparative nondisjunction rates of the isochromosomes were determined as $\underline{bz} \underline{Wx}$ kernels (class II nondisjunction) per total \underline{Wx} . For the original isochromosome, nondisjunction was 16.8% (114/679) whereas for the new isochromosome the rate was 2.5% (20/794). The difference seen here again shows that centromeric heterochromatin has a strong effect on nondisjunction rate.

Chimeric (Bz/bz) phenotypes were recorded in the table because of their frequent occurrence in isochromosome crosses. A previous comparison between the original isochromosome and the standard B-9 showed a more than 10-fold difference in the rate of Bz/bz kernels (Gen. 97:379-89, 1981). In the table above, rates of chimeric kernels for the original isochromosome and the new isochromosome are 8.4% (57/679) and 8.1% (64/794) respectively. The control groups containing a normal 9(wx) gave rates of 0.3% (14/4335) and 0.2% (9/4485). Clearly, the isochromosomes show unusually high rates of endosperm instability. In addition, the rates of instability are quite similar between the two isochromosomes, suggesting that centromeric heterochromatin may not affect these rates. However, a true isochromosome with centromeric heterochromatin on both sides of the centromere has not yet been tested for rate of chimeric kernels. (True isochromosomes were recently characterized by Brannen--Ph.D. thesis, U. Iowa, 1980). As proposed earlier (Gen. 97:379-89, 1981), the absence of the B short arm in isochromosomes may produce frequent isochromosome misdivision at the second pollen mitosis, resulting in unstable telocentrics being transmitted to the next generation.

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Variable penetrance and expressivity of grassy tillers, gt

A line homozygous for grassy tillers in a B37 background, supplied by Dr. W. C. Galinat, was used as a pollen parent on seven inbreds: Oh43, Oh51A, R53, W64A, W117, W153R and W18213N. The penetrance and expressivity of the grassy tiller phenotype was variable both among and within the seven F₂ populations.

When Oh43 was the maternal parent, gt behaved as a single recessive gene. A pooled ratio of 126+/-:46 gt/gt for nine F₂ lines was observed. This fit the expected 3:1 ratio, $\chi^2 = 0.28$ (Table 1). The F₂ generations of other crosses did

Table 1. The total, pooled and heterogeneity χ^2 assuming a 3:1 ratio for normal to grassy tiller plants in crosses between a gt/gt and seven inbreds.

	Number of F ₂ Families	Total χ^2 (3:1)	df	Pooled χ^2 (3:1)	df	Heterogeneity χ^2	df
Oh43	9	15.71	9	0.28	1	15.43	8
Oh51A	10	61.99***	10	46.09***	1	15.9	9
R53	8	14.29n.s.	8	7.2**	1	7.088	7
W64a	9	58.46***	9	34.87***	1	23.591**	8
W117	9	49.201***	9	6.517*	1	42.684***	8
W153R	3	13.769**	3	.867	1	12.922**	2
W182BN	4	55.50***	4	55.71***	1	0.21	3

not segregate for grassy tillers in a 3:1 ratio. An excess of grassy tiller plants was observed in the cross with W182BN. The ratio of 38 normal to 54 grassy tiller plants was a poor fit to a 3:1 ratio ($\chi^2 = 55.71***$). A deficiency of grassy tiller types as well as within-cross heterogeneity was observed in the F₂ of the cross between gt/gt and W64a. The pooled ratio for 9 ear to row families was 187 normal:14 grassy tiller plants, a poor fit for a 3:1 ratio ($\chi^2 = 34.867**$). While six of the 9 F₂ families contained 0 grassy tiller plants out of 20 plants per family, in one F₂ family 10 of 20 plants had grassy tillers. A significant

heterogeneity χ^2 was found among the 9 segregating families (heterogeneity $\chi^2 = 23.591^{**}$). Of the seven crosses observed, six deviated significantly from a 3:1 ratio either in total χ^2 or pooled χ^2 or both (Table 1). Three of the seven were also found to have significant heterogeneity χ^2 's.

The differences in penetrance were related to differences in the expression of the grassy tiller trait among the 7 crosses. In the F₂ generations in which there was abundance of grassy tiller plants, such as the W182BN crosses, the grassy tiller phenotype was extreme. There were many tillers per plant and they were quite robust, at times reaching 75% of the height of the main culm. In those plants which were apparently gt/gt, from the crosses that had too few grassy tiller plants, the grassy tiller trait was expressed weakly. Only a few tillers were present in such plants and the tillers were usually a foot or less in height.

The variability in the penetrance and expressivity of the grassy tiller types did not seem to be due to any obvious environmental effects. It appears more likely that other genes may be suppressing or enhancing the expression of the grassy tiller allele. Considering the amount of variability in the expression of gt/gt observed in just seven crosses, selection for desirable phenotypes of gt/gt, especially for silage purposes, should be possible.

W. F. Tracy and H. L. Everett

Selection for male sterile versions of A632 in Pr-cytoplasm

Selection for stable male sterile versions of Minn A632 in Pr-cytoplasm has resulted in an increase in the number of fully male sterile plants in the population. After seven generations of selection, 91% of the plants in the population exerted no anthers (Table 2). Moreover, most of those plants which fell into the 2-3 group were functionally sterile. They exerted only a few malformed anthers 7 to 14 days after silking. Many of the exerted anthers contained no pollen.

Table 2. The change in the percentage of male sterility in a population through 8 cycles of selection.

<u>Year grown</u>	<u>Generation</u>	<u>Rating Scale</u>				<u>Total number of plants observed</u>
		<u>1</u>	<u>2-3</u>	<u>4</u>	<u>5</u>	
1977-78	1	8%	10%	82%	0	125
1978	2	15%	32%	53%	0	552
1978-79	3	50%	38%	12%	0	110
1979	4	29%	41%	30%	0	189
1979-1980	5	No Data				
1980	6	47%	30%	23%	0	406
1980-81	7	74%	26%	0%	0	120
1981	8	91%	9%	0%	0	500

1=Fully male sterile

5=Normally fertile

Some of the selected families exerted no anthers in the winter nursery in Florida during 1980-81 or when grown at Aurora, NY in 1981. Those families seem quite stable and it may be possible to select for lines which are fully male sterile under most environmental conditions.

W. F. Tracy, H. L. Everett and V. E. Gracen

The effect of growth chamber temperatures on the stability of male sterility induced by Pr-cytoplasm in the inbred A632

Two selections of Minn A632 in Pr-cytoplasm, A632-Pr-rf, a male fertile selection, and A632-Pr-ms, a male sterile selection, were grown under two temperature treatments. The day length was set at 14 hours and the day temperature at 29 C in both treatments. The night temperature was 18 C in one treatment and 10 C in the other. Two replications were done per treatment.

When grown under warm night temperatures, 16 of 16 A632-Pr-rf plants were fertile, while 15 of 16 plants of the same genotype were fertile under cooler nights (Table 3). No differences in the degree of male fertility were observed

Table 3. The number of completely male sterile plants versus the number of fertile plants under two temperature regimes, 29°C days and 18°C nights or 29°C days and 10°C night. The days were fourteen hours long.

<u>Growing Temperature</u>		29°-18°C					
		Run 1		Run 2		Total	
<u>Genotype</u>	<u>Source</u>	<u># Sterile</u>	<u># Fertile</u>	<u># Sterile</u>	<u># Fertile</u>	<u># Sterile</u>	<u># Fertile</u>
A632-Pr-rf	80:2354	0	8	0	8	0	16
A632-Pr-ms	80:2332	7	1	8	0	15	1

<u>Growing Temperature</u>		29°-10°C					
		Run 1		Run 2		Total	
<u>Genotype</u>	<u>Source</u>	<u># Sterile</u>	<u># Fertile</u>	<u># Sterile</u>	<u># Fertile</u>	<u># Sterile</u>	<u># Fertile</u>
A632-Pr-rf	80:2354	1	7	0	8	1	15
A632-Pr-ms	80:2332	2	6	1	7	3	13

between the two treatments. Differences were observed, however, when A632-Pr-ms plants were grown under the two different treatments. Under warm nights only 1 of 16 plants was fertile, however 13 of 16 were fertile under cool nights (Table 3). Those A632 Pr-ms plants which were fertile exerted only 1% of their anthers, under either treatment. They were far less male fertile than the A632 Pr-rf plants, which exerted 75 to 100% of their anthers.

The differences due to temperature and genotypes were found to be highly significant. The genotype by temperature interaction was also highly significant (Table 4).

Table 4. The analysis of variance of the effect of night temperature on the stability of male sterility induced by Pr (C) cytoplasm in Minn A632.

Source df	SS	F	F Value Needed
Temperature 1	15.13	33.75**	29.46 > .01
Genotype 1	36.13	74.69**	29.46 > .10
Run 1	.13	.28 n.s.	29.46 > .01
Gxt 1	21.12	46.58**	29.46 > .01
Error 3	1.36		

While it is clear that different night temperature affected the expression of male sterility induced by Pr-cytoplasm Minn A632, it is unclear which parameter does so. The change from male sterility to weak fertility might be induced by the night temperature, the mean temperature, or the day-night temperature difference. However, due to the generation time of Minn A632, identifying the major factor would require a number of growth chambers over a number of years.

W. F. Tracy, H. L. Everett and V. E. Gracen

Revised restoration ratings for Cornell's Cytoplasm Bank

The accompanying chart lists the most recent visual ratings for fertility restoration for the 36 inbreds in 44 cytoplasms which are included in Cornell's Cytoplasm Bank. These numbers are based on ratings given the last two times these plants were grown out, namely 1975, when the entire set was rated by C. E. Manchester, and 1980 and 1981, in each of which half the set was rated by V. E. Gracen. The origin of the bank is described in the two references at the bottom of the chart (Gracen and Grogan, 1974, and Beckett, 1971). Each inbred/cytoplasm combination represents a minimum of five backcrosses and in a few cases as many as 14 backcrosses to the inbred, the average being 8 to 9.

To properly interpret the ratings, several important facts must be kept in mind:

1) Although every effort has been made to plant, harvest, shell, and package this material carefully, mistakes are possible. Given the thousands of opportunities for error, we feel that rather few have in fact been made and that these are usually easily traced. Nevertheless, anyone undertaking critical work with this material should check its authenticity with as many as possible of the techniques now available: (a) restoration patterns (available in the chart); (b) susceptibility to *H. maydis*, Race T or its toxin, a characteristic of members of the T group; (c) presence of the S-bands, which seem completely correlated with members of the S group (see Kemble and Bedbrook, *Maydica* 24:175, 1979, or Kemble, *TAG* 57:97, 1980); (d) presence or absence of other low-molecular-weight nucleic acid species (Kemble and Bedbrook, *Nature* 284:565, 1980); (e) patterns of mitochondrial and/or chloroplast DNA digested with restriction endonucleases (Pring and Levings, *Genetics* 89:121, 1978); (f) in vitro translation products of mitochondrial DNA (Forde et al., *Genetics* 95:443, 1980). In a few cases, the restoration data and the biochemical tests are not well correlated (see the problem of B, D, and ME cytoplasms in the accompanying article on diversity for restoration within the S group).

2) Even though enough backcrosses have been made to each inbred to make the plant types very uniform within a single inbred, nuclear heterogeneity persists. This heterogeneity has at least two causes: (a) linkage slows the return to homozygosity in a backcrossing program, even when the recurrent parent is completely homozygous; (b) no corn inbred is likely to be homozygous for all genes. Thus, even the recurrent parent is diverse in nuclear background (see the accompanying article about A632-Pr by W. F. Tracy). The result of this nuclear heterogeneity is that some of the diversity in restoration patterns within a group (C, T, or S) may be due to nuclear rather than to cytoplasmic differences. Residual nuclear diversity is the most likely cause of the bifurcated plants which occasionally appear in EK cytoplasm in Cornell's bank. The original source of EK cytoplasm was a male-sterile bifurcated plant in the variety "Early King" (A. L. Hooker, personal communication). The bifurcation has been carried along as an occasional adjunct to the sterility. Preliminary analysis of the bifurcation shows it to have a nuclear rather than a strictly maternal inheritance pattern.

3) Environment has a strong influence on the degree of male sterility and thus on the fertility ratings. A cytoplasm/inbred combination may be rated a "1" in one location or year and a "3" in another. S-type male steriles are especially

variable. Our chart reflects ratings made in one location (Aurora, New York) during two seasons. In cases where an inbred-cytoplasm combination showed variability for restoration, the maximum rating is given.

P. H. Sisco, V. E. Gracen, C. E. Manchester and H. L. Everett

Availability of seed from Cornell's Cytoplasm Bank

The entire cytoplasm bank described in the previous article has been renewed in the 1980 and 1981 seasons. Sample lots are available on request and a charge will be made for company orders. Address inquiries to:

Dr. Herbert L. Everett
409 Bradfield Hall
Cornell University
Ithaca, NY 14853
(607) 256-3103

or

Dr. Vernon E. Gracen
418 Bradfield Hall
Cornell University
Ithaca, NY 14853
(607) 256-3103

C. E. Manchester, J. Kelly, W. F. Tracy, H. L. Everett and V. E. Gracen

NY821LERf--A universal restorer of fertility

There are at least two inbred lines which contain restorer genes for all the known types of cytoplasmic male sterility. One of these is Ky21. The other is one of our New York lines, NY821LERf. In our work with S restoration, we have found that NY821LERf not only restores S steriles but that it also seems to have more than a single restorer gene. A single gene should give 50% pollen restoration in an F1 between a sterile and a restorer, and our restorer line MS64-7 does give 50% restoration with most S-types in such crosses. NY821LERf gives between 60 and 80% restoration, depending on the sterile used. With the aid of waxy translocation stocks supplied by the Maize Stock Center and by Dr. John Laughnan, we are attempting to map the restorer gene(s) for S restoration in the New York inbred.

Because of the special features of this line, a note on its origin seems warranted. NY821LERf stands for NY821, Long Ear, Restorer of Fertility. The inbred NY821 (which is sterile in most T, C, and S types) was the recurrent parent while an older inbred, NY16, was the source of fertility. NY16 was developed from the open-pollinated variety Weber Yellow Dent.

We have made F1 hybrids between stock seed of NY16 and the S-types to determine the restoration capability of the older inbred. Surprisingly, the version of NY16 which we used had only a single-gene pattern for S restoration, similar to that of MS64-7. It is possible that our own stock seed of NY16 represents a modification of that earlier inbred. We also have a stock of NY16 from Penn State, which is rather different in plant type and will be used in further tests.

P. H. Sisco, H. L. Everett and V. E. Gracen

"LBN"--A newly-designated member of the S group of male-sterile cytoplasm

Routine screening for the presence of the S bands in our cytoplasm bank (Kemble and Bedbrook, *Maydica* 24:175, 1980) showed that one cytoplasm/inbred combination, W182BN(L), has two additional nucleic acid species found neither in any other member of the S group in a W182BN background nor in L cytoplasm in any other inbred in our cytoplasm bank. We do not know when these bands first appeared in W182BN(L), since all seed prior to the ninth backcross of that line has been

discarded. Analysis of these nucleic acid species has shown them to be double-stranded RNA's. We have named these unique RNA bands LBN1 and LBN2. If they are linear in conformation, we can estimate their size to be 2.9 and 0.84kbp, respectively. The cytoplasm in which these two bands are found should be differentiated from others called "L," and we have therefore designated it "LBN" for "L cytoplasm in inbred W182BN."

The only unique characteristic of W182BN(LBN) which we have been able to detect, other than the presence of these RNA species, is an extra degree of male sterility apparent in crosses. In 1975 most of the S group in W182BN was pollinated with C0192, an inbred which partially restores S-types. The F1's resulting from these crosses were rated in 1975 and again in 1981 for fertility.

W182BN(B and D) x C0192:	plants rated 5 (fully fertile)
W182BN(most S types) x C0192:	plants rated 3 (anthers exerted, but little normal pollen)
W182BN(LBN) x C0192:	plants rated 1 (no anthers exerted)

Because of its high degree of sterility, W182BN(LBN) was used after 1975 as a source of S-type cytoplasm for several new inbreds. Again using Kemble and Bedbrook's "Rapid Assay," we have analyzed LBN cytoplasm in the ten inbreds in which at least three backcrosses to the inbred have been completed. This analysis has shown:

1) The dsRNA's characteristic of LBN cytoplasm are present in all ten inbreds through all the backcrosses. These RNA's would therefore seem to be 100% seed transmissible.

2) The amount of these RNA's present is influenced by the nuclear background. In nine of the inbreds, the intensity of the two LBN bands decreased during the backcrossing process. In one of the inbreds, 2132, the LBN bands have been maintained in full intensity through 5 backcrosses. To determine whether the band intensity could be recovered, W182BN and 2132 were crossed as males onto each of the nine inbreds in which the band intensity had been reduced. In each case, the amount of the LBN1 and LBN2 bands was increased in the F1 hybrids.

P. H. Sisco, M. Zaitlin and V. E. Gracen

B and D cytoplasm, atypical members of the S group

Gracen and Grogan in their 1974 paper comparing the restoration patterns of male-sterile cytoplasm left B and D cytoplasm unclassified because of their unusual restoration patterns (Agron. J. 65:654). In 1980, two British groups, using assays for specific DNA species and in vitro translation products of mitochondrial DNA, grouped B and D with the S-type cytoplasm (Kemble et al., Genetics 95:451, and Forde et al., Genetics 95:443). We have confirmed the report that B and D cytoplasm have the "S-bands" which are found in all other S-types. In addition, studies of pollen restoration in sterile x restorer crosses have shown that the restoration pattern of B and D is gametophytic, another characteristic of the S group.

Nevertheless, B and D are unusually fertile in several inbred backgrounds. For example, in inbred CrS4HLA in Cornell's cytoplasm bank, all S types except B and D have no fertile pollen, whereas B and D are fertile enough to be selfed. Even in those inbreds in which B and D are sterile--Ay191-71, C0150, NYD410, and W64A--differences in restoration capacity exist. When these four inbreds in the sterile cytoplasm are crossed as females with restorers, the F1 plants in B and D cytoplasm have a greater percentage of fertile pollen than do the F1 plants in the

other sterile cytoplasms. The difference is statistically significant ($P < .05$). It is theoretically possible that the unusual restoration pattern of B and D is due to residual nuclear restorer genes, but this is unlikely. B and D are different in every inbred line we have tested for restoration, and up to 12 backcrosses have been made to each of these lines. We therefore suspect that some part of the cytoplasmic genome differentiates these two cytoplasms from other members of the S group.

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More on Aberrant Ratio

Since the first reports of Aberrant Ratio (AR) by Sprague and McKinney (Genetics 54:1287-1296, 1966; Genetics 67:533-542, 1971) there have been divergent opinions regarding the cause of this phenomenon. Although much of the data suggests that a gene regulatory mechanism is responsible, other bases have been proposed.

In the 1981 Newsletter, Nelson presented evidence on Aberrant Ratio involving the A and Su loci in stocks he obtained from Sprague. In two cases of the A a* type distortion (an excess of colorless kernels) in sib matings between plants from colored and colorless kernels, Nelson found that segregation of c in one case and c2 in the second accounted for the observed ratios. In a third instance which produced 61% colored kernels in sib crosses of plants presumably A a and a a, all other known aleurone color loci proved to be homozygous dominant. These plants occurred in a line which showed the normal 1:1 ratio of colored and colorless progeny in the previous generation. Nelson indicated that a gametophyte factor could account for these observations.

In another case involving distorted ratios of the Su su* type at the sugary locus, Nelson conducted self pollinations of presumably Su su plants as well as outcrosses to Golden Cross Bantam. In those cases where an excess of sugary kernels occurred in the self progeny, an excess was also observed in the outcross although the excesses were greater than expected in the self than in the outcross populations.

In the following generation of one of these plants which gave 37% sugary in the self progeny, reciprocal crosses were made between plants of nonsugary kernels from the selfed ear and individuals from the Golden Cross Bantam line. In these crosses, ratios of approximately 1:1 were obtained when the nonsugary plants were used as females but excesses of sugary occurred when they were used as males. Nelson pointed out that a factor on chromosome 4 affecting pollen viability could account for these results.

For the past two summers we have been examining the Aberrant Ratio phenomenon in some of the preexisting lines as well as in stocks generated from virus-infected plants in this laboratory. One stock obtained from Sprague (75:287-6 x 286-1) exhibited ratio distortions of sugary. In 1980, paired reciprocal crosses were made between sibling plants from Su and su kernels. Although seed set on these ears was low, the data were more than sufficient to draw some conclusions. The results of these pollinations are presented in Table 1. In six of the cases, there was an excess of the dominant phenotype in progeny of Su plants used both as males and females while in the remaining five, a 1:1 ratio occurred in all crosses. In five of the six instances where excesses of the Su phenotype were observed, the deviations were greater in progeny of the male flowers than in those of the female, while in one example the opposite was true.

Table 1. Data from paired reciprocal crosses of Su and su plants of an AR line obtained from Sprague.

CROSS (<u>Su</u> x <u>su</u>)	<u>Su</u> ♀ x <u>su</u> ♂				<u>su</u> ♀ x <u>Su</u> ♂			
	<u>Su</u>	<u>su</u>	% <u>Su</u>	<u>p</u> value*	<u>Su</u>	<u>su</u>	% <u>Su</u>	<u>p</u> value*
5149-3 x 5152-1	82	50	62	<0.01	88	41	68	<0.001
5149-5 x 5150-1	132	105	56	>0.05	180	85	68	<0.0001
5149-11 x 5150-2	96	75	56	>0.05	75	34	69	0.0001
5153-2 x 5151-1	47	35	57	>0.05	101	55	65	<0.001
5153-3 x 5152-2	58	37	61	0.03	201	67	75	<0.0001
5153-4 x 5152-5	42	14	75	<0.001	136	57	70	<0.001
5149-2 x 5152-1	76	67	53	>0.05	80	86	48	>0.05
5149-4 x 5150-2	36	40	47	>0.05	116	90	44	>0.05
5149-8 x 5152-3	58	54	57	>0.05	94	101	48	>0.05
5153-1 x 5152-4	47	49	49	>0.05	91	106	46	>0.05
5153-5 x 5152-5	66	62	51	>0.05	50	44	53	>0.05

* p values based on expected ratio of 1:1.

In the summer of 1981, further tests were conducted with progeny of the cross of 5150-1 x 5149-5 which generated 68% Su kernels. In these crosses paired reciprocal pollinations were made between plants from Su and su kernels of this stock and in addition, outcrosses of the Su individuals as males to an unrelated sugary tester were carried out. The results are listed in Table 2. In the first six cases, an excess of Su kernels occurred in progeny of both paired reciprocal crosses and in the outcross, where data were available. In seven of the cases, ratios were about 1:1 but in one instance (5516-5), an excess of the recessive phenotype was observed.

Table 2. Progeny of paired reciprocal crosses and outcrosses of plants from the cross 5150-1 x 5149-5 (Table 1).

Su parent (From 5150-1 x 5149-5)	Paired Reciprocal Crosses								Outcrosses of Su parent			
	<u>Su</u> <u>su</u> ♀ x <u>su</u> <u>su</u> sib				<u>su</u> <u>su</u> ♀ x <u>Su</u> <u>su</u> sib				<u>su</u> <u>su</u> ♀ (unrelated) x <u>Su</u> <u>su</u>			
	<u>Su</u>	<u>su</u>	% <u>Su</u>	<u>p</u> value*	<u>Su</u>	<u>su</u>	% <u>Su</u>	<u>p</u> value*	<u>Su</u>	<u>su</u>	% <u>Su</u>	<u>p</u> value*
5515-13	145	75	66	<0.0001	36	24	60	>0.05	-	-	-	-
5516-2	-	-	-	-	184	58	76	<0.0001	79	44	64	<0.01
5516-6	209	183	53	>0.05	193	122	61	<0.0001	277	135	67	<0.0001
5516-8	75	59	60	>0.05	212	68	76	<0.0001	234	96	71	<0.0001
5516-14	34	10	77	<0.001	142	64	69	<0.001	-	-	-	-
5516-28	83	31	73	<0.0001	165	72	70	<0.0001	-	-	-	-
5516-5	110	193	36	<0.0001	83	189	31	<0.0001	48	77	38	0.01
5515-4	146	164	47	>0.05	25	42	37	<0.05	61	53	54	>0.05
5515-11	96	104	48	>0.05	28	30	47	>0.05	-	-	-	-
5515-12	94	104	47	>0.05	60	49	55	>0.05	-	-	-	-
5516-22	123	123	50	>0.05	37	36	51	>0.05	175	179	49	>0.05
5516-26	111	90	55	>0.05	219	182	55	>0.05	155	140	53	>0.05
5516-27	38	31	55	>0.05	35	32	52	>0.05	88	87	50	0.05
5516-29	96	115	45	>0.05	96	104	48	>0.05	-	-	-	-

*p values based on expected ratio of 1:1.

In most progenies which showed an excess of either the dominant or recessive phenotype, the deviations from a 1:1 ratio were greater when a plant was used as a male parent than when it was crossed as female. These kinds of transmission patterns are like those observed when a small chromosomal deletion has occurred. Rhoades & Dempsey in their studies of Inversion 3a (Amer. Jour. Bot. 40:405-424, 1953) found that some ovules possessing a chromosome 3 lacking the terminal 5% of 3L did function but that pollen grains carrying the same deficiency could not.

achieve fertilization in competition with normal grains. In studies of x-ray induced alterations at the A locus, Stadler and Roman (Genetics 33:273-303, 1948) found that deletions (a-X1, a-X2 and a-X3) were always transmitted at a higher frequency through ovules than through pollen. And, in a similar study involving markers on the short arm of chromosome 9, deficiencies including bz alone, both sh and bz, and one encompassing the c bz region all passed through the female gametophyte at a higher frequency than through the male parent (Mottinger, Genetics 64:259-271, 1970; and E. H. Coe, MNL 55:27, 1981).

The results of these studies along with the occurrence of ovule and pollen abortion caused by duplicate deficient chromosomes in translocation and inversion heterozygotes indicate that most chromosomal segments contain genes essential for normal gametophyte development. They also demonstrate that the pollen is affected to a greater extent by small chromosomal lesions than the embryo sac.

Assume for the sake of argument that in the stocks exhibiting ratio distortions of the sugary alleles, a small segment of chromosome 4 has been deleted or repressed. (Functionally, the results would be the same.) Then the results could be explained by the scheme diagrammed in Figure 1. According to this hypothesis,

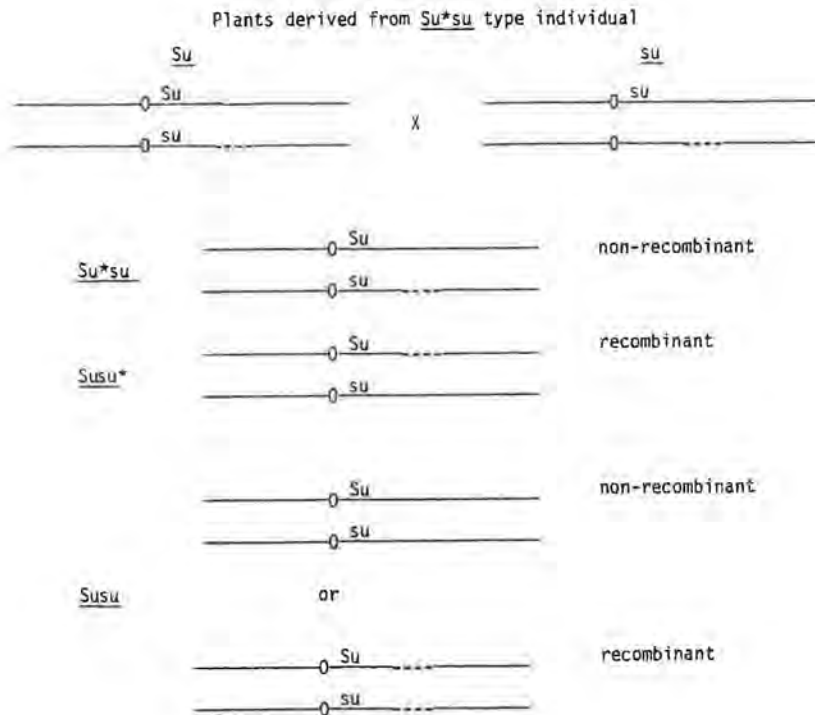


Figure 1. Chromosomal complements of plants from AR stocks exhibiting ratio distortions and normal distributions of sugary alleles. The dashes represent chromosomal segments containing male and female gametophyte genes which have been deleted or repressed.

the Su* su type of distortion in the second generation would require that a normal chromosome 4 carrying the Su allele come from one parent while a chromosome 4 containing su and the affected segment be contributed by the other.

This can be tested since plants, presumably Su su, from lines exhibiting both the Su* su and Su su* type distortions have been crossed with an unrelated su tester which should not carry a deleted or repressed segment on chromosome 4. In the progeny of these heterozygotes backcrossed to the sugary stock, certain types of distortions should not occur. The chromosomal constitutions of these plants and their offspring are depicted in Figure 2. This theory predicts that distorted

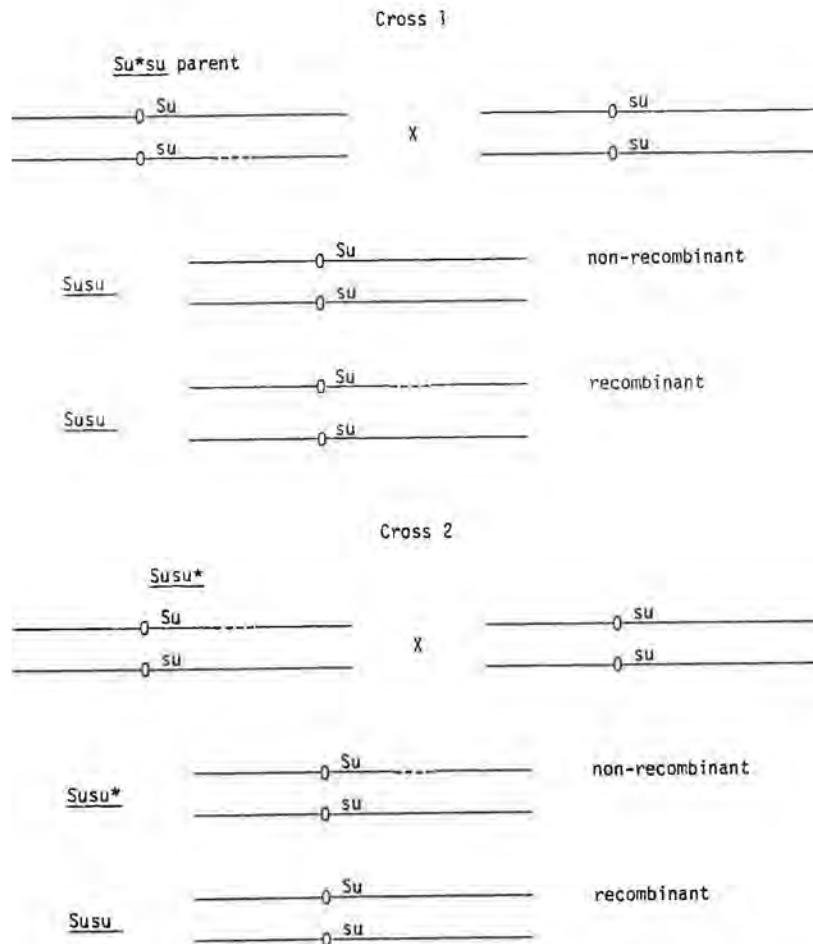


Figure 2. AR types generated from crosses between Su*su or Susu* individuals and an unrelated sugary tester stock.

ratios of the Su* su type would not occur in the following generation of an Su* su or Su su* plant mated with a normal sugary tester. If they do, then other explanations, or modifications of this one, must be put forth.

Other observations from these crosses may also be possible. Since the AR stocks have been outcrossed to an unrelated line, the resulting hybrids should be considerably more vigorous than the former plants maintained by sib matings. If seed set is good in the backcrosses, then when the putative heterozygotes from the AR stock are used as females, ears showing off ratios should exhibit ovule abortion at frequencies which would account for the difference in the number of dominant and recessive kernels, if the deletion/repression model is correct.

In addition to the AR stocks obtained from Sprague, ears exhibiting off ratios involving both sh and bz have been generated from virus infected plants in the summer of 1980 in Berkeley and in this laboratory. Plants of Mike Freeling's Adh1-S Adh2-P line, which are Sh Bz Wx pr A A2 c r in genotype, were infected with BSMV and crossed reciprocally to a stock of the genotype Adh1-F Adh2-N sh bz wx pr A A2 C R. The Adh1-S2P line is particularly susceptible to the virus and under the proper conditions plants with severe symptoms extending throughout all the leaves and which shed adequate amounts of pollen can be obtained. Over 2,000 F1 seeds from these crosses were planted in the summer of 1981 and the resulting plants were self pollinated and backcrossed as females to the sh bz wx tester. Ears from 174 of these plants have been analyzed to date and three exhibit ratios involving both sh and bz which deviate significantly from the expected values. The data from these three plants are presented in Table 3. Since the F2 progeny from the self pollinations segregate for colored and colorless kernels because of c and r, and since many of the colorless individuals cannot be distinguished from

Table 3. Data from ears exhibiting ratio distortions of involving shrunken and bronze.

	self pollinated				x <u>sh bz</u> tester					
	<u>Sh</u>	<u>sh</u>	% <u>Sh</u>	$\frac{p}{\text{value}}^*$	<u>Sh Bz</u>	<u>Sh bz</u>	<u>sh Bz</u>	<u>sh bz</u>	% <u>Sh</u>	$\frac{p}{\text{value}}^{**}$
5409-1	343	83	81	<0.01	123	1	4	172	41	<0.01
5409-3	183	41	82	0.02	28	5	0	22	60	>0.05
5414-1	343	87	80	0.01	110	3	1	74	60	<0.01

* $\frac{p}{\text{value}}$ based on expected ratio of 3:1.

** $\frac{p}{\text{value}}$ based on expected ratio of 1:1.

bronze seeds on these ears, only the Sh and sh phenotypes are recorded. On the backcross ears where colorless is not expressed, data on phenotypes of both shrunken and bronze alleles are listed. In all three cases, a significant excess of kernels expressing the dominant phenotype occurs on the F2 ears while in the backcross progeny, different results are obtained. In one example (5409-1) there is a significant deficiency of Sh seeds while in the second (5409-3) there is an excess, but the population is too small to derive any meaningful data. In the third instance (5414-1), there is a significant excess of Sh kernels. On the first and third backcross ears where populations are adequate, there is also a deficiency or excess, respectively, of Bz kernels.

To account for the data on off ratios of shrunken and bronze alleles by hypothesizing a mechanism in which gametophyte factors reduce the transmission of a particular homolog, a model must be presented which is more complex than the one used to explain the distorted ratios of sugary alleles. Since the sh and bz phenotypes are deficient on the three self-pollinated ears and in one of the backcross progeny, gametophyte factors could have been segregating in the sh bz tester stock. However, since so few plants were observed showing off ratios, the factors would have to be expressed only infrequently. And, to accommodate the deviations in opposite directions of the self and backcross ears of plant #5409-1, a means of transferring these factors from one homolog to the other somatically (as suggested by Brink et al., Science 159:161-170, 1968, to explain paramutation) must be invoked. The same kind of element could have arisen in the virus-treated parent and in the F2 plant, been transferred to the opposite homolog.

In addition to questions arising from these data, others must be answered as well. For example, in the studies reported by Nelson in which the c and c2 alleles were found to be segregating as well as a in AR lines obtained from Dr. Sprague, where did they come from if the original parents were a A2 C C2 R and A A2 C C2 R?

All of the data collected so far in various laboratories can be explained by a mechanism or mechanisms involving gene regulation; but clearly, a great deal more data are needed before the controversy surrounding the phenomenon called Aberrant Ratio can be resolved.

We thank Dr. Sprague for kindly providing stocks exhibiting Aberrant Ratio.

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Mutations of Adh induced by barley stripe mosaic virus

Except for the initial studies on the effects of barley stripe mosaic virus (BSMV) in maize in which mutation rates were analyzed (Sprague et al., Science 141:1052-1053, 1963), reports involving putative genetic effects of the virus have dealt with distorted Mendelian ratios. Attempts to formulate genetic mechanisms for which the virus may be responsible from these kinds of data suffer because when off ratios occur, they could be caused by factors which do not directly affect the gene(s) being followed. As discussed in the previous report, elements modifying the transmission rates of whole chromosomes could be responsible. One way to surmount this problem is to screen for mutations at particular loci in the F1 generation of virus-infected plants.

One locus ideally suited for this kind of analysis is Adh. The gene products are identifiable electrophoretically, several alleles exist with different migration rates and, as reported by Schwartz and Osterman (Genetics 85:63-65, 1976) and Freeling and Cheng (Genet. Res. 31:107-130, 1978) Adh-negative mutants and mutants with low levels of Adh expression can be selected by allyl alcohol treatment of pollen.

Attempts to recover virus-induced mutants of Adh were begun in the summer of 1980 in Berkeley. Plants of the stock Adh1-S Adh2-P (also c c, r r, pr pr) from Freeling's lab proved to be quite susceptible to infection by BSMV, strain ND-18. When grown in 6" pots under reduced sunlight in a lathhouse and in the relatively cool temperatures (70-80 F days, 60-65 F nights) of the Berkeley summer, a substantial number of plants expressed viral symptoms in all leaves and shed adequate amounts of pollen.

Pollen from 36 of these plants was treated with allyl alcohol at concentrations which would allow the survival of grains possessing low levels of ADH, and applied to silks of plants homozygous for Adh1-F Adh2-N C R and pr. The Adh2-P, c, r and pr alleles of the male parent served as contamination markers in these crosses as no other plants of that genetic makeup were grown at Berkeley in 1980. To reduce the possibility of stray pollen alighting on silks of the female parent, all pollinations were conducted in an isolation field. Tassels of the virus-treated plants were bagged in the afternoon, placed in a room devoid of other corn and transported to the field the following morning.

Approximately 300 pollinations were made, and it is estimated that about 2,000 grains were applied in each cross. Of 309 kernels on the resulting ears, three were identified as putative mutants. In electrophoretograms of scutellar extracts, two exhibited no Adh1-S band and one showed a low level of S expression. All three, in subsequent analyses with root tissue, proved to contain Adh2-P and also carried the c, r and pr alleles, thus confirming the virus-treated plants as their source. These mutants have been designated Adh1-S-v1, Adh1-S-v2 and Adh1-S-v3. When treated with an ADH-specific stain, the pollen of plants grown from the three

mutant kernels all segregated approximately 1:1 for purple and white grains, confirming the presence of the mutant alleles. Samples collected from non-mutant plants of the same cross stained all purple.

Adh1-s-v1, in addition to the Adh null phenotype, also expressed knotted in the F1 plant. In some allelic combinations Kn is less than 0.1 units away from the Adh locus (Freeling, personal communication). Although Kn produces a dominant phenotype, the mutant allele may in fact be a null and the knots due to a dosage effect of the kn gene product. If this is the case, then the Adh1-s-v1 mutant is either a deletion of the two loci or both markers are suppressed.

Of 54 F2 scutella subjected to electrophoretic analysis, 6 showed no ADH band and thus were probably S-v1 null homozygotes. Of the remaining 48, all exhibited only the F band. In an F2 population of 54, 13.5 S-v1 homozygotes are expected. Hence, the deficiency ($P = 0.02$) may indicate a reduced transmission of this mutant through the gametophyte stage, but more data are needed to confirm this.

Adh1-S-v2 expressed a low level of ADH in the F1 scutellum. Although pollen of the plant from this mutant kernel segregated approximately 1:1 purple and white grains when stained for ADH activity, expression of the S allele in F2 scutella appeared normal in a population of 83 heterozygotes. The nature of this mutant, whether it be unstable, expressed at different levels in various tissues, or what, awaits further analyses.

In a small sample of F2 kernels, Adh1-S-v3 has exhibited only a null expression. The frequency of S-v3 homozygotes in the F2 is as expected; hence its transmission through the gametophyte stage appears normal.

Freeling (Nature 267:154-156, 1977) reported that the spontaneous forward mutation rate for Adh is less than 10^{-7} . Hence it appears safe to conclude that these mutants are of viral origin. Further studies with these variants at both the genetic and molecular level should shed substantial light on the manner in which BSMV is interacting with the maize genome.

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Further analysis of some recent data

Data provided by Hake (1980, The genome of Zea mays: Its organization and homology to related grasses, Ph.D. dissertation, Washington University, St. Louis) have been used here to draw two modified Wagner tree diagrams. Hake used DNA/DNA hybridization and restriction analysis to measure relationships of two maize races, three races of Mexican annual teosinte and one population each of Z. diploperennis, Z. luxurians and Tripsacum laxum. Four "drivers" were used, here labeled Midwest Dent maize ("M"), Z. mexicana - Balsas race (central Guerrero; "B"), Z. diploperennis (Jalisco; "D") and Z. luxurians (SE Guatemala; "G"), and there were 1-10 "tracers" per driver. Some results are especially curious: Z. luxurians is as close to Tripsacum laxum ("T") as to Ladyfinger popcorn ("P") and Balsas teosinte, while Balsas teosinte and Midwest Dent maize are over twice as far from T. laxum as from each other (Table 1).

Table 1. Distances between test materials, derived from Hake (1980) by multiplying T_m values by 10 and averaging the three M-M values.

Driver	Tracer							
	P	M	C	B	H	D	G	T
M	32	(4)	9	27	14	40	42	88
G	37	--	32	36	29	34	--	38
B	33	--	2	--	19	46	--	80
D	--	--	--	--	--	--	--	61

Classical Wagner tree diagrams are unable to adequately account for the variation, so a modification has been tried. Upward sloping, compound branches indicate divergence of taxa, and each segment accounts for a part of the total divergence. Horizontal lines are mathematical shortcuts between taxa necessitated seemingly by hybridization and do not count toward divergence (Diagrams A and B).

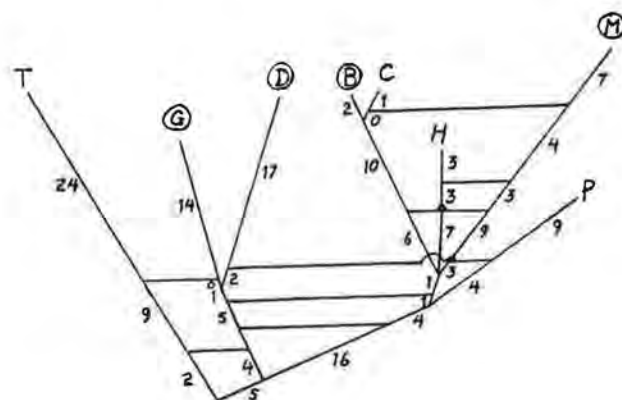
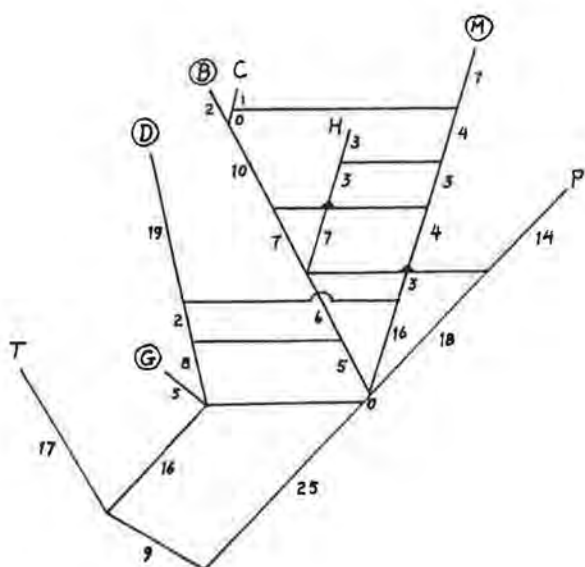


Diagram A. The drivers are circled, though *Z. diploperennis* was used as a driver only once. C is Chalco teosinte (State of Mexico) and H is Huehuetenango (NW Guatemala), both annuals.

Diagram B. As Diagram A except *Z. diploperennis* and *Z. luxurians* share a branch with *Zea* rather than *Tripsacum*.

Once values have been assigned to each of the diverging branch segments, a table (matrix) of modelled distances for that diagram is calculated, and a table of differences between the two distance matrices is calculated (Tables 2-5). The lengths of branch segments are readjusted to minimize the values in the last table.

Table 2. Modelled distances on Diagram A.

Driver	Tracer							
	P	M	C	B	H	D	G	T
M	32	--	8	26	14	40	42	88
G	37	--	34	35	29	34	--	38
B	33	--	3	--	18	46	--	81
D	--	--	--	--	--	--	--	62

Table 4. Modelled distances on Diagram B.

Driver	Tracer							
	P	M	C	B	H	D	G	T
M	32	--	8	26	14	40	42	88
G	37	--	33	34	29	33	--	38
B	33	--	3	--	18	39	--	80
D	--	--	--	--	--	--	--	43

Table 3. Differences between Tables 1 and 2.

Driver	Tracer							
	P	M	C	B	H	D	G	T
M	0	--	+1	+1	0	0	0	0
G	0	--	-2	+1	0	0	--	0
B	0	--	-1	--	+1	0	--	-1
D	--	--	--	--	--	--	--	-1

Table 5. Differences between Tables 1 and 4.

Driver	Tracer							
	P	M	C	B	H	D	G	T
M	0	--	+1	+1	0	0	0	0
G	0	--	-1	+2	0	+1	--	0
B	0	--	-1	--	+1	+7	--	0
D	--	--	--	--	--	--	--	0

So far it has been much easier to account for the results using Diagram A, where G is an offshoot of a T-G branch long separated from maize and Mexican annual teosinte, but which later interacts with proto-maize once. Diagram B overly shortens the B-D distance, and calls for two hybridizations of G with proto-maize between two hybridizations of G with T, all quite early. The closeness of G and T here is reflected in their sharing terminal chromosome knobs, inflorescence form and stem structure, so it might be proposed that SE Guatemalan teosinte, G, be separated from Zea.

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Analysis of combining ability of A344 line mutants

Two sets of sublimes of the inbred A344 were included in the experiment. The check set was composed from 10 sublimes (CP1 - CP10), randomly selected from the check population. The experimental set included the check subline CP1 and 9 NMU induced macromutants (MP1 - MP9; 3 semi-dwarfs, 2 dwarfs, two-tassel, prolific,

tall stalk, tassel seed-2 forms). The major phenotypic changes were controlled by recessive genes in all mutants. The lines of each set were diallel-intercrossed within their sets in one direction. The parental forms as well as hybrids were included in trials utilizing one-row plots, 4.9 m², with 20 plants in the plot, with six replications. The combining ability for grain yield and plant height was analyzed according to Griffing (Method 4, Model 1). The results are the following:

1) Mutants were lower than the check sublimes in grain yield (19.7 q/ha vs. 27.0 q/ha).

2) Hybrids including mutants also show a marked grain yield reduction. The mean grain yield of the control set was 34.7 q/ha, and that of the mutant hybrids was 29.8 q/ha. However, the minimal yields within each set were practically the same, 25.0 q/ha and 23.8 q/ha, respectively. The range of variability was reduced from 23.0 q/ha to 17.0 q/ha.

3) The reduction of the mean plant height of mutant hybrids was significant in comparison with that of the control set (130.2 cm vs. 138.4 cm).

4) The grain yield and plant height variation in the set of hybrids obtained by crossing the control sublimes were mainly determined by general combining ability (GCA). For the first trait the mean square deviation due to GCA ($M_g = 80.1$) more than by five times exceeded the mean square deviation value under the influence of the special combining ability (SCA) ($M_g = 14.8$) (Table 1), and for the second trait it exceeded more than two times (Table 2).

Table 1. The analysis of variance components for grain yield.

Check Sublines	GCA Effects	Variance			Mutant Sublines	GCA Effects	Variance		
		GCA	SCA	GCA/SCA			GCA	SCA	GCA/SCA
CP1	2.98	8.8	10.6	0.83	CP1	0.03	0.0	5.3	0.00
CP2	-3.17	9.8	7.7	1.27	MP1	2.95	8.5	22.9	0.37
CP3	-2.28	5.0	3.1	1.61	MP2	0.78	0.4	10.0	0.02
CP4	-3.85	14.7	9.6	1.53	MP3	3.60	12.8	17.1	0.74
CP5	5.95	35.2	23.9	1.47	MP4	0.05	0.0	7.1	0.00
CP6	-2.26	5.0	8.2	0.60	MP5	0.27	0.5	10.0	0.05
CP7	2.92	8.7	15.9	0.54	MP6	0.18	0.0	12.8	0.00
CP8	0.68	0.4	16.8	0.02	MP7	-2.42	5.7	8.4	0.67
CP9	-1.26	1.5	13.7	0.10	MP8	-2.90	8.2	9.2	0.89
CP10	0.29	0.2	9.5	0.02	MP9	-2.54	6.1	7.8	0.78

$$M_g = 80.1 \quad M_s = 14.8 \quad M_e^* = 1.20$$

$$s.e. = g_j - g_i = 0.55 \text{ q/ha}$$

$$M_g = 38.2 \quad M_s = 15.2 \quad M_e^* = 1.60$$

$$s.e. = g_j - g_i = 0.65 \text{ q/ha}$$

Table 2. The analysis of variance components for plant height.

Check Sublines	GCA Effects	Variance			Mutant Sublines	GCA Effects	Variance		
		GCA	SCA	GCA/SCA			GCA	SCA	GCA/SCA
CP1	-5.88	34.2	29.0	1.17	CP1	-3.58	12.3	35.2	0.34
CP2	-1.62	2.2	15.7	0.14	MP1	2.15	4.1	63.4	0.06
CP3	2.87	7.8	10.0	0.78	MP2	6.60	43.0	10.2	4.21
CP4	1.10	0.8	14.4	0.05	MP3	4.26	17.5	114.0	0.15
CP5	4.12	16.6	50.2	0.33	MP4	12.76	162.4	59.4	2.73
CP6	-0.59	0.0	17.0	0.00	MP5	12.83	164.0	45.8	3.53
CP7	-4.83	22.9	22.5	1.01	MP6	7.89	61.7	84.5	0.73
CP8	2.71	6.9	17.5	0.39	MP7	-15.31	233.9	36.5	6.40
CP9	1.57	2.0	27.0	0.07	MP8	-11.03	121.0	155.4	0.77
CP10	0.55	0.0	36.9	0.00	MP9	-16.57	273.9	30.2	9.06

$$M_g = 83.3 \quad M_s = 31.4 \quad M_e^* = 3.78$$

$$s.e. = g_j - g_i = 0.96 \text{ cm}$$

$$M_g = 975.6 \quad M_s = 78.0 \quad M_e^* = 5.18$$

$$s.e. = g_j - g_i = 1.13 \text{ cm}$$

5) The grain yield reduction is explained by the reduction of the effects of favorable additive factors (Table 1). The GCA mean square value was reduced twofold in mutant hybrids comparing with the check set. The effect of specific factors was maintained at the same level.

6) In the control set the subline CP1 enters the best group for the GCA effect, and in the crosses with mutants it did not express its potential (Table 1).

7) Twofold increase in plant height variation range in the set of mutant hybrids is explained by a significant rise in effect of additively acting genetic factors.

8) The M3 plants both heterozygous and homozygous for dominant alleles of oligogenes did not differ in grain yield or stalk height. The hybrids from crossing CP1 and CP8 with a homozygous mutant and non-mutant plants from M3 families were also similar in these traits. It proves that oligogenes in a heterozygous state did not affect the vigor of A344 line plants.

From the published data we see that not all oligogenes in a heterozygous state express influence on the plant vigor. Many investigators consider that the differentiation of the inbreds is essentially associated with the accumulation of micro-mutations. Palenzona (MGCNL, 1968, 42:12-14) stated that an autodiploid inbred under the influence of spontaneous mutagenic factors showed a change of combining ability for plant height on account of additive genes.

We figure out two mechanisms leading to the essential change of additive genetic effect expression.

Hypothesis I. The polygenes acting additively differ from the polygenes acting non-additively in higher mutability.

Hypothesis II. Both polygene types display similar mutability. However, a maize genotype contains an insignificant number of loci that are able to interact specifically.

Let us divide the additive factors into two groups according to their resistance to mutagenic agents: (a) stable additive genes (SAG); and (b) mutable additive genes (MAG). This assumption is based on the widely accepted view about the "hot points." Let us assume further that the genes showing the controlling effect on the combining ability for grain yield are more stable than those controlling the plant height.

In this case all above mentioned peculiarities of the grain yield variability may satisfactorily be explained by an increase in concentration of recessive alleles of the MAG group in the mutant hybrid population. The frequency of undesired recessive alleles of minor genes was higher in mutants compared with check sublimes. As a result of it the undesired effect of recessive genes is seldom compensated by the dominant genes of the partner in the cross. Respectively, the range of variability in plant height increased under the influence of new genes from the SAG group and new non-additive genes.

The genotypes of the check sublimes utilized in this experiment have been developed under the influence of the following phenomena: (a) the long-term process of accumulation of spontaneous mutations, (b) the gene recombination process, (c) genetic drift. The valuable information does not allow a direct assessment of the factors governing the formation of specifically acting gene complexes. However, comparing the GCA and SCA variances in the check sublimes and induced mutants we see that this process proceeded independently of the direction and intensity of variability of additive factors.

Thus, the analysis of the experimental data shows differences in quality and relative autonomy of additively and non-additively acting polygenes. In 1942 G. F. Sprague and L. A. Tatum found the phenomenon of relative independence of selection for GCA and SCA. It seems that the hypothesis of different nature of

the genes controlling these traits may help in explanation of selection independence.

Acknowledgement: These experiments were realized due to the valuable help and supervision of my teacher, the late Academician M. I. Hadjinov.

A. Mashnenkov

Synthesis of macromolecules in corn kernel mutants

All possible combinations of recessive and dominant alleles of o2, su2, Sup genes were obtained in the W70 inbred (Doklady VASHNIL, n11, 1981). Six phenotypes differing in endosperm pattern protein content and isoelectric focusing spectrum bands of zein were selected:

- (I) O2/- Su2/- Sup/- flint-floury
- (II) o2/o2 Su2/- sup/sup floury
- (III) o2/o2 Su2/- Sup/- modified flint-floury
- (IV) O2/- su2/su2 Sup/- flint-glassy
- (V) o2/o2 su2/su2 sup/sup glassy-floury
- (VI) o2/o2 su2/su2 Sup/- glassy

According to kernel weight they are distributed in the following succession: I > IV > III > II = VI > V; to density IV > I > III = V = VI > II; to protein concentration in endosperm I = III = IV = VI > II = V. No differences were found in protein concentration in whole kernels.

Alleles O2 and Sup intensify zein synthesis in I and III endosperms by 3.9 and 2.2 times respectively against genotype II. The partial reduction of the protein fractions albumins, globulins and glutelins is attributed to O2 allele effect. Sup gene displays an evident locally specific action. It occurs only in association with the o2 allele. Zein synthesis intensifies in outer flint portions of endosperm only due to the reduction of glutelin-3 fraction. Therefore an equal amount of albumins and globulins is typical for II and III endosperms. Allele su2 impedes zein synthesis and hastens glutelin-3 accumulation.

The total IEF spectrum of zein is represented by 42 components. In the endosperm of the triple homozygote V o2/o2 su2/su2 sup/sup, 16 components as a minimum have been detected. All these components are present in all endosperms, eight of them being strong and keeping their concentration, vs. weak ones varying in quantitative manifestation. Substituting su2 for Su2 in genotype II, the number of distinguished peaks in density diagrams rises to 18 due to the occurrence of protein in the middle part of the spectrum. Suppressor and allele O2 increase the number of components to 24 in genotypes III, VI and I, respectively. Up to 8 additional peaks appear in the opposite parts of the spectrum: anode and cathode.

The most valuable set of zeins is synthesized following the interaction of O2 and su2 alleles in genotype IV. Among 39 components all 24 components of genotypes III and VI are observed, but 2 components of endosperm I are absent. In the anode part of the spectrum seven absolutely new proteins in small amounts are synthesized. The behavior of su2 allele associated with O2 allele is far from the expected. It unblocks the accumulation of 17 zein proteins of acid nature in particular. In the presence of suppressor, su2 allele does not affect the number of components.

At 21 days after pollination in the endosperms of the lines, there were no differences in the concentration of total DNA, RNA, poly-A-RNA and activity of ribonuclease. With the intensification of zein synthesis and increase of zein diversity from 16 to 24 components, the process of organic matter accumulation intensifies by 1.35 times in the kernel of W70 line.

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Cytogenetic studies on *Zea perennis*

Zea perennis (*Euchlaena perennis*) was introduced from Mexico into Argentina in 1962, and up to now has been maintained by clonal propagation. *Zea perennis* has a chromosome number of $2n = 40$. According to Longley (J. Agr. Res. 28:673-682, 1924) all the chromosomes were bivalents, but he afterwards (J. Agr. Res. 48:789-806, 1934) pointed out that it was an exceptional case since *Zea perennis* was an autotetraploid with 10IV.

From the study of 150 cells at metaphase I, it has been determined that the meiotic configurations most frequently found were 10II + 5IV (55% of the cells) and 12II + 4IV (20.74%), with very few monovalents and trivalents. The average of chiasmata per cell was 15.69 in bivalents and 18.87 in tetravalents, totaling 34.56. The pachytene chromosomes had a tendency to stick, identification being difficult. They had very small terminal knobs in one or the other arm, or in both, or they were knobless. At anaphase in 87.5% of the cells 20 chromosomes migrated to each pole; and in the remaining 12.5%, 19 chromosomes migrated to one pole and 21 to the other. The grains of pollen were small, exhibiting a fertility of 89%.

Up to now it has been considered that *Zea perennis* is an autotetraploid. However, the meiotic configuration most frequently found was 10II + 5IV. In order that this would take place, the chromosomes could have different grades of evolution, since 5IV might have been differentiated in pairs, losing their homology, to give 10II. On the other hand, with 5IV they could not as yet have differentiated.

In hybrids between *Zea mays* ($2n = 20$) and *Zea perennis* ($2n = 40$) and reciprocals ($2n = 30$), the meiotic configuration most frequently observed was 5III + 5II + 5I. In hybrids between *Zea diploperennis* ($2n = 20$) and *Zea perennis* ($2n = 40$) and reciprocals ($2n = 30$), the meiotic configuration most frequently observed was 5III + 5II + 5I.

From the foregoing it can be asserted that the chromosomes of *Zea mays* and *Zea diploperennis* are homologous with *Zea perennis*, leaving 5 chromosomes without pairing. These could be chromosomes that had been differentiated, or they could have a different origin from the chromosomes of *Zea mays* and *Zea diploperennis*, and for this reason they do not pair. Thus, from all accounts *Zea perennis* could not be an autotetraploid; with some of the chromosomes differentiated, it is then an auto-allopolyploid.

María del Carmen Molina

Cytogenetic studies on interspecific hybrids between *Zea diploperennis* and *Zea perennis*

A hybrid between *Zea diploperennis* Iltis, Doebley and Guzman ($2n = 20$) and *Zea perennis* Reeves and Mangelsdorf ($2n = 40$) has been obtained. It has $2n = 30$ chromosome number, with the exception of one plant whose chromosome number is $2n = 40$.

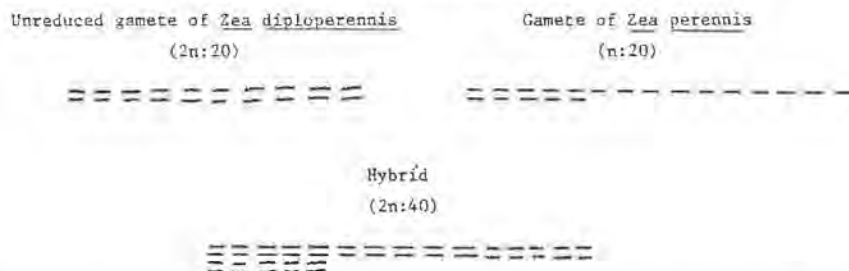
The meiotic configuration most frequently observed at metaphase I in *Zea perennis* has been 5IV + 10II; in *Zea diploperennis*, 10II; and in the hybrid with $2n = 30$, 5III + 5II + 5I. If the phenomenon of autosyndesis is discarded it can be assumed that all the chromosomes of *Zea diploperennis* pair totally or partially with the chromosomes of *Zea perennis*, leaving 5 chromosomes of *Zea perennis* without pairing. The average of chiasmata was 17.25 and at pachynema some translocations

and inversions were observed. The fertility of the pollen varied from 37 to 83%, obtaining only 2% of viable seed.

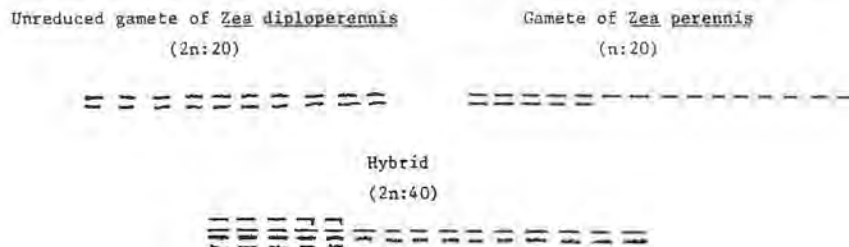
The hybrid with $2n = 40$ probably could have originated from an unreduced gamete of Zea diploperennis (female progenitor). The meiotic configurations most frequently found were 5IV + 10II; 4IV + 12II; 3IV + 14II; 6IV + 8II. Very few monovalents and trivalents were observed. The bivalents and tetravalents were open-ring. At pachynema the chromosomes paired totally or partially. The average of chiasmata was 26. The fertility of the pollen was 58.4%, obtaining 80% viable seed.

If it is considered that the chromosomes of Zea diploperennis and Zea perennis in the hybrid ($2n = 30$) are paired with the configuration 5III + 5II + 5I, it would be expected that the resulting configuration in the hybrid ($2n = 40$) would be 5IV + 5III + 5I. In spite of that assumption and as there have been very few monovalents and trivalents observed, it is supposed that some of these alternatives would take place:

1) Autosyndesis of the chromosomes of Zea perennis



2) Non-homologous pairing of Zea diploperennis chromosomes and Zea perennis ones.



3) A possible combination of cases N° 1 and N° 2.

It is difficult to individualize the chromosomes of Zea diploperennis (unreduced gamete) to Zea perennis ones in chromosome pairing. However, it is important to take into account that because the hybrid has 40 chromosomes, a chromosome balance would take place, obtaining regular meiotic divisions and 80% viable seed. In opposition to that already described, in the hybrid between the aforementioned species ($2n = 30$), the contrary took place; therefore the fertility was 2%.

María del Carmen Molina

Preliminary studies on the hybrid Zea mays x Tripsacum dactyloides

We attempted to hybridize Zea mays with Tripsacum with the object of studying the chromosome affinity between such species and the possible incorporation of some characters of the wild species into Zea mays. For this purpose, 100 ears of

Zea mays ($2n = 20$) were pollinated with Tripsacum dactyloides ($2n = 72$), developing 400 shriveled seeds. Since they had not germinated by themselves, the mature embryos were cultured "in vitro." Development of some plants was obtained but only 3 of them survived after transplantation. Two were confirmed as hybrids because their somatic chromosome number was 46.

Morphologically the hybrids are similar to Tripsacum with slow growing at the beginning but with rapid development afterwards, flowering two years after implantation. Cytogenetic studies were carried out, obtaining the following results. In Tripsacum the meiotic configurations most frequently found were 26II + 5IV; 24II + 6IV; in Zea mays, as is known, 10II; and in the hybrid, 18II + 10I. In the hybrid, apparently the chromosomes of Tripsacum would pair by autosyndesis and the monovalents would correspond to Zea mays. In exceptional cases, 1 or 2 trivalents were observed, and frequently more than 10I were found. In Tripsacum, as well as in the hybrid, the chromosomes had large terminal knobs. Other chromosomes had joined themselves obtaining 6 or 7 "conglomerations" of knobs. In anaphase different numbers of chromosomes migrated to each pole, leaving some lagging chromosomes. The fertility of the pollen was 60%, but apparently viable seed could not be obtained.

From these preliminary studies on the hybrid Zea mays x Tripsacum dactyloides it can be concluded: (a) It is very difficult to obtain this hybrid, due to the fact that most of the developing seeds are the product of a parthenogenetic multiplication of the pollen of Tripsacum (patroclinous plants) and not true hybrids. (b) In the hybrid, in the chromosomes of Tripsacum dactyloides, the phenomenon of autosyndesis can occur, forming bivalents. (c) The chromosomes of Zea mays and Tripsacum dactyloides have not much homology since only occasionally were they partially paired.

María del Carmen Molina and Teresa Pilar Rosales

Polyacrylamide gel electrophoretic analysis applicable to the lysine-threonine potential selection method: A possible prediction of the genotype dependent behavior of maize lines

A polypeptide screening in 7% polyacrylamide gels by the technique of Davis (Ann. N. Y. Acad. Sci. 121:404-427) was carried out with the proteins of the shoots derived from embryo cultures of the mutant floury-a line 79-7660 (a lysine-threonine 1mM insensitive line) and normal BP line 79-7511 (a lysine-threonine 1 mM partially insensitive line).

The embryos were cultured in tubes containing the Murashige-Skoog medium with (or without = control) lysine-threonine 2.5 mM (strong inhibitory medium). After 10 days of culture at 28 C in the dark, the soluble proteins of the shoots were extracted in a chilled mortar and pestle using two volumes of buffer tris-glycine 0.1 M, pH 8.3, containing 40% sucrose. The homogenates were centrifuged at 44,630 xg, 20 min. All the procedures were at 4 C. A sample of 0.3 ml of the supernatant (approximately 120 mg of fresh material) was taken and poured into each acrylamide tube.

Other embryos of the same lines were subjected to the same kind and time of inhibition and then were transferred to a control medium, lysine-threonine free, and were analyzed by the same means after 10 days in such "rescue" medium.

Comparing the polypeptide pattern of control with treated derived shoots we have found in both maize lines several alterations. Although the pattern of alteration was different in both lines, the phenomena of lack, unfold and synthesis of polypeptides in the treated derived shoots were usually observed. The extent and kind of alteration in each line was exactly the same in the five repetitions of the experiment carried out. But the most important fact is that several polypeptides altered by the effect of lysine-threonine reverted to the

control pattern when the seedlings were transferred to a "rescue" medium (lysine-threonine free). The results shown in Figure 1 were one of the most frequently observed. This result is shown here because in both lines, floury-a and BP, it was observed jointly.



Figure 1. Densitometer tracings.

- A- Soluble protein pattern of rescue derived shoots.
- B- Soluble protein pattern of treated derived shoots.
- i.- Unfold polypeptide
- C- Soluble protein pattern of control derived shoots.

The densitometer tracings of the Rf region from 0.40 to 0.60 of the treated shoots differ from those of the control in the unfold of the polypeptide located between the two major polypeptides of the soluble shoot extract, at Rf = 0.52. The densitometer tracing of the shoots derived from seedlings transferred to a "rescue" medium has shown a pattern similar to that of the control, with the re-synthesis of the unfold polypeptide.

These results were also observed in other Rf regions of the gel, in which the polypeptide pattern of the shoots derived from control and "rescue" seedlings were equal, and both were different from the polypeptide pattern of the shoots derived from treated seedlings.

The soluble protein alterations caused by lysine-threonine would probably have an epigenetic basis, and would not be heritable. However, we suggest that the extent of the alteration is genotype dependent. We have suggested previously (see MNL 55:54-55) that the lysine-threonine effect has secondary effects on several metabolic processes. The soluble protein alterations shown here would be one of such effects. In the same communication we also suggested that a genetic material inducible (or selectable) by the primary lysine-threonine effect but stable to the secondary lysine-threonine effects would be the best for selection purposes.

The results obtained by Phillips et al. (Crop Sci. 21:601-607) in their "rescue test" could also be genotype dependent. Those materials inhibited or not in lysine-threonine medium and recovered when transferred to lysine-threonine free medium would be the best, according to our point of view, for selection of possible variants resistant to lysine-threonine.

We suggest that by means of the electrophoretic analysis of the shoot and/or root soluble proteins of the control and treated derived seedlings and using an appropriate similarity index (S), it would be possible to predict the behavior of any strain in the "rescue" attempts. Those lines with higher "S" between the electrophoretic pattern of the soluble proteins derived from control and treated seedlings would be recovered in the "rescue" attempts. Such hypothesis is to be tested with large maize strains in our laboratory.

Miguel Angel Rapela

Study on seedlings derived from whole kernels in lysine-threonine medium

Two maize lines, floury-a 79-7660 and BP 79-7511 (see MNL 55:53 and 54), were screened for resistance to lysine-threonine 5 mM inhibition by evaluating seedlings derived from whole kernels. With the aim of testing the new hypothesis of Phillips et al. (Crop Sci. 21:601-607) on our mutant and normal lines selected by the growth of their embryo cultures in lysine-threonine medium, the following procedure was used. Thirty kernels were selected from the middle part of the ear and surface-sterilized for 20 min in 2.5% sodium hypochlorite 1:8 v/v. Murashige-Skoog medium (without changes), containing 0.2% sucrose and 0.8% agar, and either 0 (control) or 5 mM lysine-threonine was autoclaved and poured into test tubes (10 ml per tube). Fifteen kernels of each strain were tested in the inhibitory medium (one in each tube) and the rest were used as control. The incubation period was 6 days, at 28 C in the dark. The experiment was repeated twice.

Table 1. Treated/control ratios.

Parameter	Floury-a	BP
Primary root length	0.60	0.75
Shoot length	0.75	0.86
Seedling fresh weight	0.69	0.84
Roots fresh weight	0.58	0.76
Shoots fresh weight	0.78	0.90
Seedling dry weight	0.72	0.90
Roots dry weight	0.65	0.84
Shoots dry weight	0.83	0.92

As shown in Table 1, we have tested 8 parameters. Among them, the primary root length (treated/control) is the most representative of the extent of the inhibition. Seedling growth was inhibited to a different degree in both lines. However, either the mutant floury-a or the normal BP line could be considered resistant, because the root length in lysine-threonine medium exceeded 50% of the root length of control, according to the classification of Phillips et al.

Surprisingly, the normal BP line showed the higher degree of resistance. Such a result is somewhat contradictory with our previous determinations of the floury-a and BP excised mature embryos in treated 2.5 mM/control test, in which the floury-a embryos showed a higher degree of resistance compared with BP ones.

It is interesting to note, however, that contrary to the results obtained by Phillips et al., the resistance of the floury-a and BP line was not only expressed when seedlings were derived from whole kernels but also in seedlings derived from excised embryos.

The Phillips hypothesis of the high methionine/lysine or methionine/(lysine + threonine) ratios could explain our experimental results with the floury-a and BP seedlings derived from whole kernels. However, such hypothesis does not explain the behavior of such seedlings derived from excised embryos.

We have previously suggested (MNL 55:54) that the free amino acid pool could be affecting the lysine-threonine response. Such hypothesis has also been proposed by Phillips et al. (Crop Sci. 21:601-607). Either the floury-a or the BP line has a higher free amino acid pool. Such a pool could affect the lysine-threonine response either on the seedlings derived from whole kernels or on the seedlings derived from excised embryos. However, the finding that the endosperm tissue was not required to provide resistance suggests an alternative explanation. That is, probably the floury-a gene or a gene closely linked with it could affect some regulatory enzyme of the aspartate family of amino acids and produce desensitization to the lysine-threonine effect (MNL 55:55-57). Further tests will be performed in this way.

Miguel Angel Rapela

Preliminary cytological observations on the effect of lysine-threonine in floury-a maize root tip cells

We have studied the cytological behavior of the mutant floury-a line 79-7660 subjected to the lysine-threonine 2.5 mM strong inhibitory medium. Each experiment consisted of the culture of 15 excised mature embryos of the floury-a line in tubes with the Murashige-Skoog medium containing lysine-threonine 2.5 mM, and 15 embryos in lysine-threonine free medium (control). After 10 days of culture at 28 C in the dark, two embryo derived seedlings were taken from the 5 repetitions of the experiment carried out. Thus, a total of 20 (10 treated, 10 control) seedlings were analyzed cytologically. Root tips from the differentiated seedlings were prefixed at 4 C in saturated paradichlorobenzene for 4 hours, transferred to Farmer's solution, fixed for 24 hours at room temperature, and stored in 70% ethanol. Chromosome screening was made in metaphase cells from squashed root tips stained with acetic carmine and observed by optic microscopy.

We have not obtained good photographs due to the low quantity of metaphase cells found. Such low numbers could probably be due to the dark culture of the "in vitro" material.



Figure 1. Chromosomes in root tip cells of floury-a maize: A--control derived root tip cells; B--treated derived root tip cells.

As shown in Figure 1 (A and B), either in the treated or in control derived root tips, we have found normal chromosome number and normal pairing. All the cells have 10 pairs of chromosomes with normal meiotic behavior. At anaphase I, the two members of the bivalent also separated normally. We have found an increase in the nuclear and cell size. Such kind of phenomenon was similar to that produced by colchicine. Nuclear division was not observed in such cells. Probably the increase in the nuclear size could be due to an increase in the DNA content. In all the cells studied, the phenomena observed were the same. Then, apparently the increase in the nuclear and cell size and the lack of nuclear division were not due to the lysine-threonine effect, because such phenomena were observed either in treated or in control derived root tips.

In conclusion, up to now we have not found in the floury-a root tip cells any effect directly associated with the exogenous supply of lysine-threonine.

Miguel Angel Rapela and María del Carmen Molina

Nuclear DNA content in F₁ hybrids between perennial teosinte and maize (Gaspé line)

Nuclear DNA contents of meristematic root tip cells were determined in F₁ hybrids between perennial teosinte and maize (Gaspé) and in its parents by microspectrophotometric measurements on 2C anaphase nuclei, at 560 nm. The DNA amounts of Gaspé, perennial teosinte and F₁ are graphically demonstrated in Figure 1.

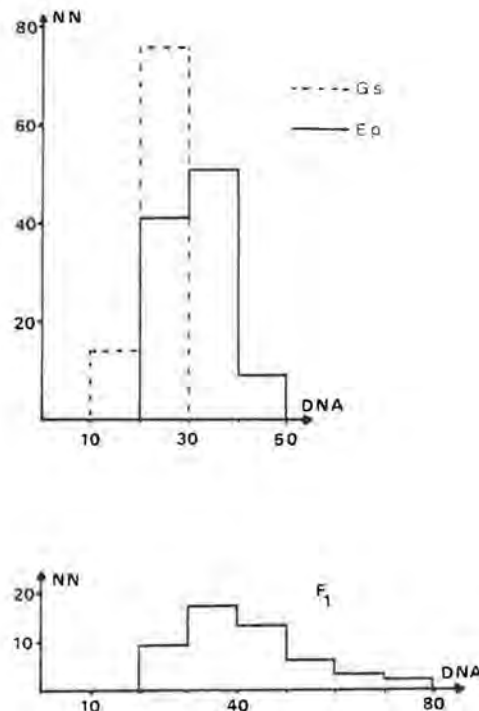


Fig. 1. Distribution of nuclear DNA content in Gaspé (Gs), perennial teosinte (Ep) and the F₁. NN--number of nuclei.

The nuclear DNA content, nuclear area and DNA density values were as follows:

	Mean DNA (A.U.)	Mean nuclear area (μ^2)	DNA density (DNA/area)
Per. teosinte	31.6 \pm 5.9	42.98 \pm 9.6	0.7564 \pm 0.16
Gaspé	23.2 \pm 3.3	31.94 \pm 6.2	0.7482 \pm 0.15
F1	41.4 \pm 12.5	39.91 \pm 11.3	1.046 \pm 1.07

The nuclear DNA content and DNA density were represented in arbitrary units and the nuclear area was represented in μ^2 .

Perennial teosinte had approximately 50 percent more DNA per 2C nucleus than Zea mays. Highly significant differences were found in DNA content between parents and with respect to the F1. For nuclear area significant differences were found between the F1 and Zea mays. The two parents did not differ in DNA density, but the F1 had more DNA density than both parents and significant differences were found. Close correlations were found between nuclear area, DNA density and DNA content:

	r (Area & DNA)	r (DNA/area & DNA)	N
Gaspé	0.288**	0.414**	90
Per. teosinte	0.435**	0.467**	100
F1	0.814**	0.40**	50

The DNA content of F1 plants is higher than the DNA content of both parents. This fact is due to a higher DNA density, perhaps due to differential polynemy. The high DNA content in F1 plants was greater than expected. This fact leads us to suppose that amplification of DNA in F1 would explain this phenomenon, perhaps associated with heterosis shown by the hybrids.

Ida Graciela Palacios

Nuclear DNA content in F2 plants of a hybrid between perennial teosinte and maize.

We describe the segregation of nuclear DNA content among the F2 progeny of hybrids between perennial teosinte with a higher DNA content (31.64 A.U. and $2n = 40$) and Zea mays (Gaspé) with lower DNA content (23.21 A.U. and $2n = 20$).

These hybrids were obtained by Magoja (MNL 52:37, 1978). The mean nuclear DNA content of the F2 progeny is 41.08 A.U. and so far corresponds to the analysis of 15 plants. Comparing this value to the mean DNA content in the F1 progeny of 41.35 A.U., it does not differ significantly.

The F2 nuclear area is $46.44 \mu^2$, which is significantly greater than the F1 area ($39.9 \mu^2$). From the nuclei analyzed, the densities (DNA content/nuclear area) were determined establishing for the F2 a value of 0.9401, which is lower than that obtained in the F1 (1.046).

The preliminary results so far obtained on F2 plants allow us to believe that due to chromosomal recombination and reorganization, the DNA content may have a range of variation that may exceed its progenitors' extremes (Fig. 1).

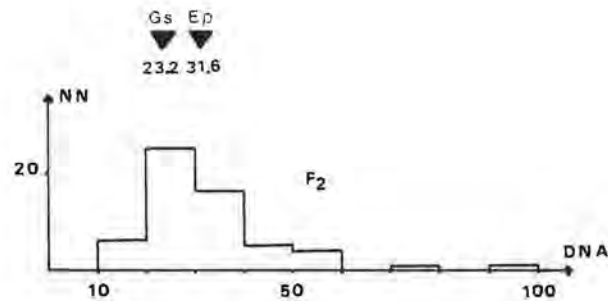


Fig. 1. Distribution of nuclear DNA content in F₂ plants. NN--number of nuclei; Gs and Ep--means of parents.

The aim of this paper is to be able to relate morphological and physiological characters to the DNA content in segregating individuals.

Ida Graciela Palacios

Heterosis in maize-perennial teosinte hybrids

The hybrids between perennial teosinte (*Euchlaena perennis* = *Zea perennis*) and maize (*Zea mays*) show a surprising vigor that draws our attention. In order to quantify the differences between the hybrids and their progenitors, a series of measurements were made in an F₁ population (88 plants) coming from the same number of hybrid seeds obtained when crossing perennial teosinte with the Gaspé line. The same characters were taken in the progenitors and hybrids in order to enable the comparison. The characters studied were flag leaf length (FLL) and flag leaf width (FLW). The leaf length (LL), leaf width (LW) and the sheath length (SL) were measured in the most developed leaf of each stalk, usually the fourth or fifth counting from the apex of the F₁ plants. The ratio length/width (L/W) and the leaf area (LA) = $3/4 L \times W$ were calculated. The tassel central spike length (TCEL), the length of lowermost tassel branch (LLTB), the number of tassel branches (TB), the plant height (PH), the number of nodes per tiller (N/T), the number of productive nodes (PN), the number of ears in the uppermost node (EUN), the number of ears per tiller (E/T), the number of fruit cases per ear (FC/E) and the number of tillers per plant (T/P), were measured. The stalk diameter (SD) was measured just below the most developed leaf. The comparisons between the values of the F₁ and their progenitors are shown in Tables 1 and 2.

F₁ plants are generally much more similar to perennial teosinte than to maize, but with a more exuberant development. They are completely perennial, blooming annually and for the characters of the inflorescence the teosinte is completely dominant. From these results it can be inferred that in general F₁ plants differ significantly, with higher values for the characters studied than those of their progenitors.

Many characters which in the hybrids are expressed in a superlative degree may not, perhaps, be an expression of heterosis because they do not condition a greater biologic efficiency. Among these, the greater height and dimension of their leaves could be included. Nevertheless, the high prolificity that they show is a potential productive capacity that exceeds that of their progenitors. It is called "potential productive capacity" because it is conditioned by its fertility. If prolificity and sterility are not related characters, it is

Table 1: Differences between means of *E. perennis* and the F_1 (*E. perennis* x Gaspé). NS-- no significant; **-- significant at 1% level; ***-- significant at 0.1% level.

	<i>E. perennis</i>			F_1			t	sig.
	\bar{x}	s^2	N	\bar{x}	s^2	N		
FLL (cm)	6.6	3.58	23	20.9	14.44	85	-17.43	***
FLW (cm)	1.1	0.20	23	3.0	0.25	85	-16.52	***
LL (cm)	47.0	26.52	24	43.1	26.01	85	3.30	**
LW (cm)	2.8	0.16	24	4.1	0.16	85	-14.07	***
L/W	16.9	4.60	24	10.5	1.96	85	17.42	***
LA (cm ²)	99.9	493.80	24	133.6	515.29	85	-6.46	***
SL (cm)	11.3	1.06	24	11.0	1.21	85	1.19	NS
TCEL (cm)	9.7	0.67	24	17.6	2.89	85	-5.09	***
LLTB (cm)	9.8	2.27	24	12.9	6.76	85	-5.57	***
TB	3.7	1.87	24	5.8	3.61	85	-5.05	***
PH (cm)	160.2	239.74	24	191.8	201.64	86	-9.45	***
SD (mm)	5.2	0.32	24	5.3	0.36	85	-0.72	NS
N/T	15.3	3.94	24	11.8	0.81	85	12.44	***
PN	3.2	0.47	24	4.7	0.64	85	-8.36	***
EUN	1.8	0.27	24	4.8	2.56	85	-9.03	***
E/T	5.3	4.35	24	25.7	90.25	85	-10.42	***
FC/E	5.1	0.71	129	9.3	1.14	85	-30.24	***

Table 2: Differences between means of Gaspé and the F_1 (*E. perennis* x Gaspé). NS-- no significant; ***-- significant at 0.1% level.

	Gaspé			F_1			t	sig.
	\bar{x}	s^2	N	\bar{x}	s^2	N		
FLL (cm)	20.4	21.16	20	20.9	14.44	85	-0.50	NS
FLW (cm)	3.0	0.49	20	3.0	0.25	85	0.00	NS
LL (cm)	38.4	29.16	20	43.1	26.01	85	-3.67	***
LW (cm)	4.1	0.25	20	4.1	0.16	85	0.00	NS
L/W (cm)	9.3	1.37	20	10.5	1.96	85	-3.55	***
LA (cm ²)	110.0	1407.00	20	133.6	515.29	85	-3.64	***
SL (cm)	11.7	8.53	20	11.0	1.21	85	1.76	NS
TCEL (cm)	11.0	3.24	20	17.6	2.89	85	-15.45	***
LLTB (cm)	8.2	3.03	20	12.9	6.76	85	-7.68	***
TB	5.9	6.76	20	5.8	3.61	85	0.20	NS
PH (cm)	57.4	234.09	20	191.8	201.64	86	-37.58	***
SD (mm)	8.6	1.21	20	5.3	0.36	85	18.48	***
N/T	6.5	0.45	20	11.8	0.81	85	-24.73	***
PN	1.9	0.35	20	4.7	0.64	85	-14.45	***
EUN	1.0	0.00	20	4.8	2.56	85	-10.58	***
E/T	1.9	0.35	20	25.7	90.25	85	-11.14	***
T/P	3.9	2.79	20	26.2	64.16	88	-12.34	***

possible to select prolific and fertile individuals. The hybrids' prolificity is very high and it is because of a higher number of productive nodes (PN), spikes per node and of spikes per stalk (E/T).

From the results obtained it can be inferred that the highest heterosis in the hybrids between perennial teosinte and maize is the high prolificity that they show.

Jorge Luis Magoja and Gabriela Nora Benito

Specific relationship of storage proteins in maize and its wild relatives

The fractionation of maize endosperm proteins by means of the Landry-Moreaux technique has proven to be very useful as, for example, to establish the regulating action of the different endosperm mutants.

A single gene can modify the protein pattern as it occurs with those which repress the synthesis of zein. Not considering these mutants, it can be said that in the normal maizes the protein pattern of the endosperm is in general relatively constant, there being a reasonable similarity. As a result of what has been said, the protein pattern of the endosperm may be considered as a specific characteristic that may be used to distinguish maize from its wild relatives. This idea has been discussed before (MNL 55:62-63, 1981), where it was pointed out that the main distinction in the storage proteins among the Maydeae is far more due to the action of the regulatory genes than to structural units.

The protein fractions of the endosperm of maize (*Zea mays*), diploperennial teosinte (*Z. diploperennis*), perennial teosinte (*Euchlaena perennis* = *Zea perennis*) and tripsacum (*Tripsacum dactyloides*), shown in Table 1 were obtained according to the Landry-Moreaux technique. The seeds of diploperennial teosinte were provided by Julián Cámara Hernández.

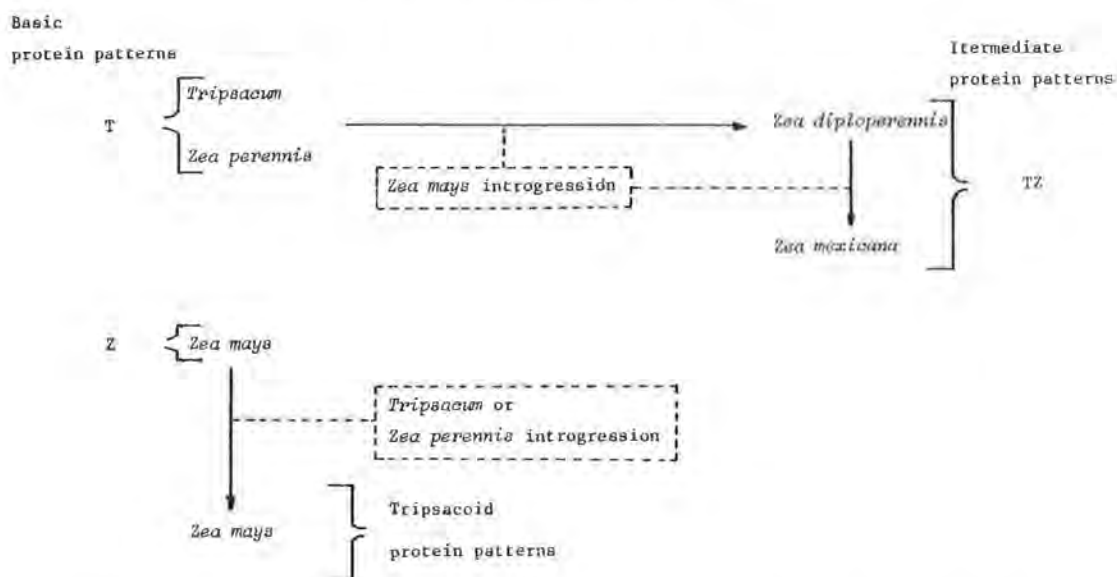
Table 1. Endosperm protein pattern of maize (*Zea mays*) (Z.m.), *Zea diploperennis* (Zd), *Zea perennis* (Zp) and *Tripsacum dactyloides* (Td). SS-- saline (albumins, globulins); Z-- zein; G₁-- glutelin-1; G₂-- glutelin-2; G₃-- glutelin-3.

fraction	Soluble nitrogen (percent of total)			
	Zm(*)	Zd	Zp	Td
SS	5.7	3.1	1.8	1.8
Z	51.9	67.1	57.1	55.2
G ₁	12.3	9.2	20.7	26.9
G ₂	9.6	7.5	3.3	5.1
G ₃	13.0	10.0	9.2	9.7
protein %	12.0	27.0	21.0	27.4

(*) average of ten normal lines.

As mentioned before (MNL 55:60-61, 1981), it can be stated that the protein fractions of perennial teosinte and *Tripsacum* distinctly differ from those of maize, because they show a low proportion of albumins and globulins (approximately one-third), a high level of glutelin-1 (approximately double) and a low proportion of glutelin-3. The patterns of perennial teosinte and *Tripsacum*, which happen to be similar, and distinctly different from the one of maize, are designated as protein pattern "T," and that of maize, as protein pattern "Z" (see Figure 1).

Figure 1: Differentiation of protein patterns between Maydeae species.



The protein pattern of *Z. diploperennis* has characteristics which are intermediate to those of "T" and "Z". *Diploperennial* teosinte has a low proportion of albumins and globulins, but it is between those of maize and perennial teosinte, a proportion of glutelin-3 similar to perennial teosinte or *Tripsacum*. As was mentioned before (MNL 55:60-61, 1981), the high relationship of glutelin-1 (which is characteristic of perennial teosinte) dominates in the progenies of the hybrids between this species and maize, whereas the low proportion of albumins and globulins and of glutelin-3 (also characteristic of perennial teosinte) acts as partially dominating.

From the results obtained, it can be determined that an intermediate protein pattern (TZ), as the one *Z. diploperennis* has, might derive from that of *Z. perennis* by germplasm introgression of maize, as the high ratio of G₁ could only take place by successive backcrossings of the hybrids with maize. Consequently we present the hypothesis that the intermediate protein patterns (TZ), which are characteristic of *Z. diploperennis* according to the results of other authors as well, and of annual teosinte (*Z. mexicana*) can have originated by maize introgression in *Z. perennis*. If the protein pattern is a character which can be considered specific, it is probable that *Z. mexicana* and *Z. diploperennis* are species with *Z. mays* introgression; and perhaps *Z. perennis* is the sole species closely related to maize which is not contaminated with its germplasm, that is to say, which was early distinguished in the evolutive process of the Maydeae.

It can also be inferred that the introgression of germplasm of *Z. perennis* or *Tripsacum* into maize may give rise to tripsacoid protein patterns, which are characterized by having a low ratio of albumins and globulins and a high ratio of glutelin-1. Especially the high ratio of glutelin-1 could indicate the introgression of *Tripsacum* or perennial teosinte in maize, as we have found in some maizes (results not published).

Jorge Luis Magoja and Angel Alberto Nivio

Genetic control of seed proteins by a spontaneous defective kernel mutant of maize

A spontaneous mutation which conditions defective kernels was found in the WK-01 red flint line. The mutation conditions kernels of small size, their approximate weight being 27% of the normal kernels of the same ear.

The characteristic of these kernels is that they present a floury endosperm, a reduced embryo and endosperm, a pale yellow color and the pericarp separated in some parts from the aleuroniferous layer. The progeny of normal kernels, heterozygous for the mutation, is characterized by the segregation of normal kernels and defective kernels in a ratio 3:1, respectively. The results obtained in the analysis of 5 F₂ ears (see Table 1) allow us to infer that the character of the

Table 1. Segregation of de*-7601

Ear N°	F ₂ kernels		x ² for 3: 1 ratio	P
	Normal	defective		
1	185	69	0.278	0.50-0.70
2	212	89	3.349	0.05-0.10
3	208	57	1.722	0.10-0.20
4	182	63	0.067	0.70-0.90
5	207	65	0.176	0.50-0.70

defective kernel is controlled by a single recessive gene, which is referred to as de*-7601. The kernels that have the gene de*-7601 have an average weight of 72.0 mg, whereas the normal ones have an average weight of 267.5 mg. In the normal kernels the endosperm represents 85.6% of the complete kernel's weight, while in the defective ones only 77.8%. The germ represents 10.8% of the normal kernel's weight but in the defective ones 9.9%. These results show that both the endosperm and the germ are affected by the mutation, reducing their proportions in the defective kernel.

The gene de*-7601 is lethal, because although 50% of the defective kernels germinate, the seedlings die in different developmental stages.

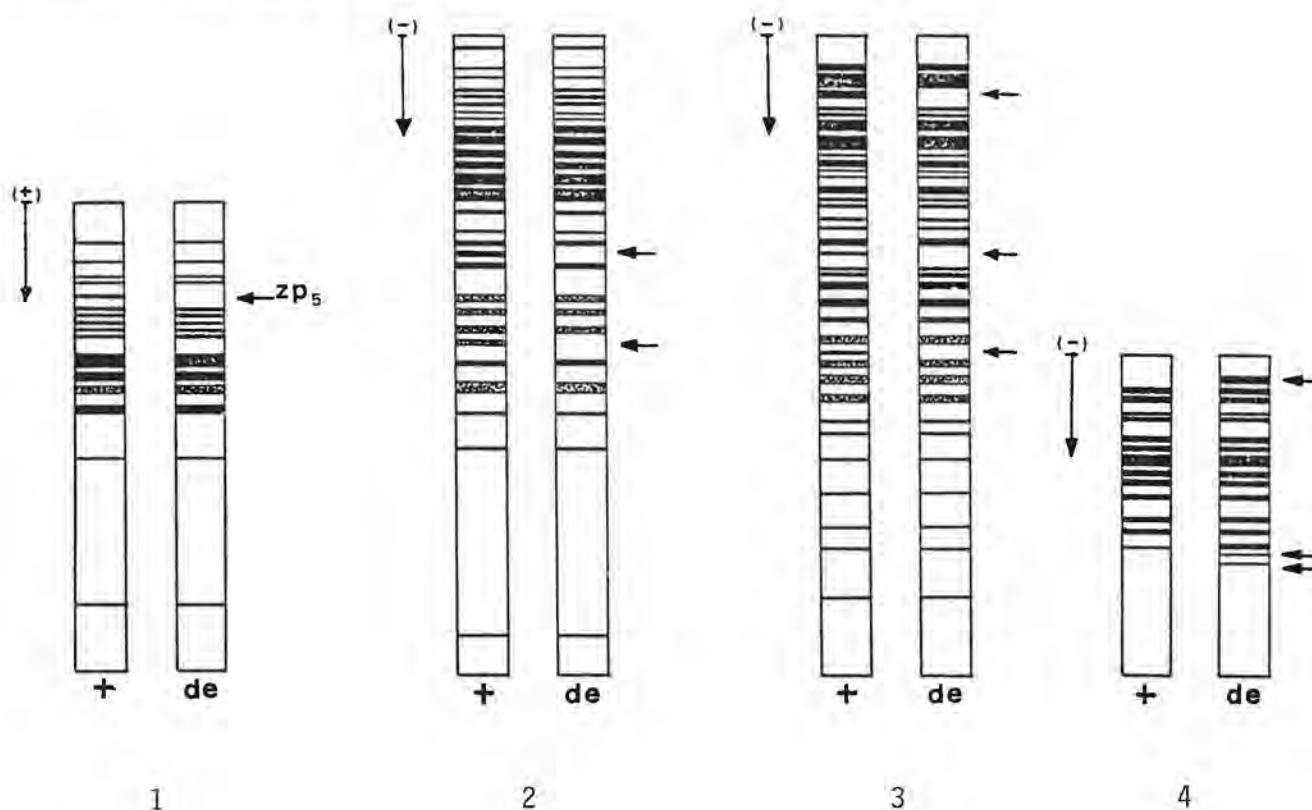
The purpose of this work is to determine the effect of gene de*-7601 over the kernel proteins. For this reason a comparative analysis was performed between the defective kernels and their normal equivalents, which have the same genetic background.

Table 2. Endosperm protein pattern of normal inbred (WK-01) and its defective kernel mutant version. SS-- saline (albumins, globulins); Z-- zein; G₁-- glutelin-1; G₂-- glutelin-2; G₃-- glutelin-3.

fraction	soluble nitrogen (percent of total)	
	Normal	defective
SS	5.6	15.0
Z	55.8	35.4
G ₁	13.9	17.4
G ₂	6.4	7.8
G ₃	16.9	22.1
protein %	9.8	8.8
lysine (g/16gN)	2.5	3.2

The storage proteins of the endosperm were fractionated according to Landry-Moureaux (1970), and the results are shown in Table 2. The protein pattern of the defective kernels notably differs from that of the normal kernels and is characterized by a lower proportion of zein and a higher proportion of albumins, globulins and glutelin-3.

The mutant *de*-7601* acts as other known regulatory genes which repress the synthesis of zein and conditions a higher proportion of albumins, globulins and glutelin-3. These modifications consequently bring about an increase in the lysine level (see Table 2). In order to establish whether there are any differences in the molecular composition of the greatest storage protein, the zein, this was fractionated by means of electrophoresis in polyacrylamide gel. The polypeptide pattern of zein in *de*-7601* is similar to that of normal kernels, but the fifth (zp5) polypeptide is completely repressed as it does not appear (see Figure 1). Comparing the soluble proteins of the endosperm separated in polyacrylamide gel, two components are also absent in the polypeptide pattern of *de*-7601* (see Figure 2)



Figures 1-4. Polyacrylamide gel electrophoretic patterns in normal and defective kernels:

- 1) zein polypeptides (zp)
- 2) soluble endosperm proteins
- 3) soluble germ proteins
- 4) germ lipoproteins

It was also considered interesting to study the polypeptide patterns of the germ proteins. In Figure 3, the patterns of the soluble proteins of the germ, which are the ones that are found in a higher proportion, are compared. In this

case the kernels which carry de*-7601 do not synthesize, in the germ, three polypeptides which are found in normal germs.

In the germ, the molecular patterns of the lipoproteins were also analyzed, these being a group of proteins characteristic in the maize germ, because in the endosperm they are found in low proportion. In Figure 4 it can be seen that, contrary to what occurred in the previous cases, where the molecular patterns of the defective kernel proteins showed the lack of some polypeptides, three new polypeptides are synthesized in the lipoproteins of the germs that have the gene de*-7601.

Several modifications occurred in the defective kernels, the most evident of which is the great reduction in the size of the kernel. In this particular case the normal development both of the endosperm and the germ is blocked, as a result of which the seed loses its viability or at least is incapable of originating a normal plant, as these die early in their development.

Apart from the modifications caused by the mutants which condition defective kernels, there are the modifications that are presented in this paper and which are exercised on the kernel proteins.

The results obtained allow us to infer in the first place that at least for this particular case, the locus de*-7601 is a regulatory gene which controls the synthesis of the endosperm proteins, deeply altering the normal protein pattern. Nevertheless, there also exist differences as regards the level of structural genes in the polypeptide patterns. In general, the blockage in the synthesis of some of the polypeptides which are perhaps essential enzymes for the normal development and viability of the kernel, leads us to suppose that there is a strong probability that the mutation of the defective kernel affects several strongly linked genes, at least one of which is a regulatory element and several of which have structural functions.

Jorge Luis Magoja

High-quality protein maize with normal genotype: Inheritance of lysine content

Previous reports (MNL 52:37, 1978 and 55:66, 1981) presented the finding of high-quality protein maize (high content of lysine) without the use of mutants which modify the structure of the endosperm, such as opaque-2. These maizes, obtained by the selection of the progeny as a result of crossing a normal line with floury-a of high lysine, have normal phenotype (hard endosperm) and are of "normal genotype" because they do not have in their genetic background any floury mutant.

The purpose of this work is to report preliminary experiences, performed with the aim of interpreting the inheritance of the lysine level in these normal maizes.

A line of maize (red flint), with high level of lysine in the endosperm (3.2g/16gN) was crossed with another normal line of low content (2.3 g/16gN). The segregation among 112 F₂ kernels, analyzed by its lysine content in the endosperm is shown in Figure 1. This kind of segregation can be interpreted as the effect of dosage in the endosperm, of two independent genes which accumulate their individual effects. According to this criterion 1/16 of the F₂ kernels would have no active allele, 3/16 two, 4/16 three, 3/16 four, 2/16 five and 1/16 six--the last ones being of the same high level of lysine as the progenitor of high lysine.

The segregation in the lysine level found among F₂ kernels can be explained by the effect of cumulative dosage for two major genes which segregate independently. These two genes would be the ones that were previously referred to as lysine-1 and lysine-2 (MNL 52:37, 1978). Actually, the real situation is that these genes repress by the effect of dosage, in a greater or lesser extent, the synthesis of

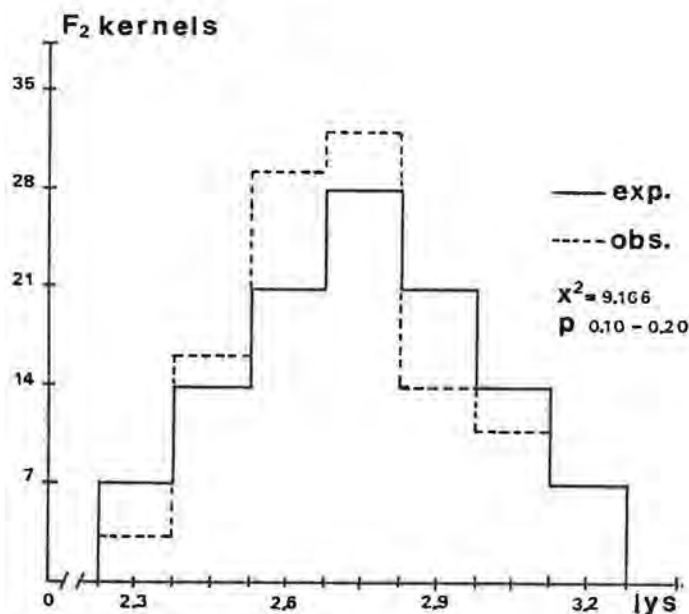


Figure 1: Segregation for lysine content (endosperm) between 112 F2 normal kernels of the same ear.

zein (MNL 55:66, 1981). Consequently, the level of lysine varies, the content for this amino acid in the absence of repression being normal, and maximum when the individual repressive effects of the six active alleles are combined. The intermediate lysine levels, between the progenitor extremes, are a consequence of intermediate levels of zein (Magoja et al., Mendeliana V (2), 1981, in press).

The results presented here are incomplete because they were performed in only one F2 ear. Actually we are studying other F2 segregations and also the backcrosses with the same line with high lysine content and "normal genotype" employed in the current paper, but also with other selected lines with greater lysine content (3.6 g/16gN). These lines are of the red flint type of maize.

We can consider from the results obtained up to now that, in the inheritance of the level of lysine in these normal maizes, at least two major genes might be involved, but perhaps there might also exist small effects of other modifiers. It can be considered from the experiments performed that the lysine level of maize endosperm can be improved without the use of mutants that modify the structure of the endosperm.

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Analysis of the heat shock response in maize

We have conducted a detailed investigation into the effects of temperature stress on gene expression in maize. Young (3-5 day old) plumules of Oh43, subjected to a brief shift in incubation temperature, exhibit a striking change in polypeptide synthetic patterns--from the production of a broad spectrum of different proteins to the new and/or enhanced synthesis of a small group of "heat-shock" proteins (HSPs). One-dimensional (1D) SDS-PAGE and fluorographic analysis of the

newly synthesized polypeptides reveals the enhanced synthesis of polypeptides with molecular masses (M_r) of 108, 89, 84, 76, 73 and 18 kilodaltons (kd), and the apparent depressed synthesis of a major polypeptide with $M_r = 93$ kd (actively synthesized in the controls) following a one-hour shift from 27 C to 41 C. The six M_r classes of HSPs resolve into at least 18 spots by 2D IEF-SDS PAGE; some spots clearly show new or enhanced synthesis relative to the control while others show no apparent differences in intensity between control and heat-shocked samples.

Seedlings germinated and grown at 27 C require temperatures at or exceeding 35 C for detectable synthesis of these HSPs. The response is rapid; by 15 minutes following a shift to 41 C, enhanced synthesis of some of the HSPs is noted. When heat shocked seedlings are returned to 27 C, the polypeptide synthetic patterns recover to the control pattern after six to eight hours.

Although recovery of polypeptide synthetic patterns occurs following heat shock, we were interested in knowing if other cellular processes in the seedlings had been affected to prevent continued development of the plants. Five-day-old seedlings were heat shocked for one hour at 35 C, 41 C, 44 C, or 50 C, returned to 27 C for at least six hours and then transplanted to our nursery. While emergence was less than 100% in all cases due to stress from transplanting, there was a clear reduction in the number of emergent seedlings treated at 44 C and a complete absence of emergent seedlings from a treatment at 50 C. Emergence for each heat shock temperature as a percentage of the controls was as follows: 35 C (94%), 41 C (82%), 44 C (28%), 50 C (0%). Casual examination revealed no phenotypic differences among emergent seedlings. They were monitored to maturity. Our results show no differences in the heat-shock response between the various treatments. Some minor changes in band position in other regions of the gels suggest that we may be detecting some genetic variability in the sampling population.

We have also isolated total RNA from individual shoots of both control (27 C) and heat-shocked (41 C) seedlings, translated these RNAs in vitro in a rabbit reticulocyte lysate system, and analyzed the translated products by 1D and 2D PAGE. The results clearly demonstrate that 5 of the 6 M_r classes of HSPs noted in vivo are also translated in vitro and that differences in polypeptide number and intensity can be detected by 2D IEF-SDS PAGE between the in vivo and in vitro translated products. We are exploiting these apparent differences to examine the level of regulation of HSP induction.

The characterization of the heat shock response as described here provides a system for investigating: a) the induction of gene activity by environmental stress, b) the effect of tissue source or genotype on this response, and c) the influence of transcriptional and/or translational control on the regulation of gene expression in maize.

C. L. Baszczynski, D. B. Walden and B. G. Atkinson

Studies on the heat shock response in different tissues and genotypes of maize

The response of maize to heat shock has been examined in several tissues and genotypes to determine the extent to which these 'factors' influence the response. Polypeptides from 3-5 day old shoots, and primary roots, and 25-30 day old leaves were radioactively labelled, extracted and separated by 1D and 2D PAGE as described elsewhere (Baszczynski et al. 1982, *Canad. Jour. Biochem.*, April). The results show that all three tissue types, although differing in control polypeptide patterns, respond to heat shock in a similar manner with the new and/or enhanced synthesis of the six M_r classes of HSPs described in the previous contribution (this Newsletter). Roots, however, do not show a marked depression in the synthesis of the 93 kd polypeptide as noted in the shoots and leaves.

When returned to the control temperature (27 C), shoots exhibit almost complete recovery after two hours while roots and leaves take longer to recover.

A comparison of different genotypes, including Oh43 (an inbred dent), Gaspe Flint (an inbred flint) and PX-11 (a commercial hybrid, Northrup-King) indicate that the genotypes differ in Coomassie blue-stained gels and fluorograms of polypeptide patterns when plants are grown at the control temperature of 27 C and show a similar response when subjected to an elevated temperature. The genotypes differ largely in the relative amounts of synthesis of the heat shock polypeptides.

We are currently examining a series of the temperature-sensitive, virescent mutants in a common background (Oh43) to determine if differences in the greening temperature optima among the virescents (Hopkins and Walden, J. Hered. 68:282-286, 1977) are reflected in their differential capacity to synthesize heat shock proteins.

C. L. Baszczynski, J. G. Boothe, D. B. Walden and B. G. Atkinson

A possible injury response observed in polypeptide patterns from treated maize leaf tissue

Many experimental procedures require the manipulation of tissue in order that the particular aspects of interest be made more readily available for study. When such procedures are employed it is important to determine whether or not they will affect the results obtained and if so, take this into account when evaluations are being made. Recent evidence has shown that even relatively minor manipulation of some tissues may result in readily observable changes in the patterns of protein synthesis (Currie and White, 1981).

We report some preliminary results with maize protoplasts which indicate that alterations in the patterns of newly synthesized polypeptides are associated with the preparation of the protoplasts from maize leaf tissue. The plant material employed was a commercial hybrid (PX-11) grown under standard greenhouse conditions. Protoplasts were obtained from 25-33 day old plants utilizing a modified version of a method developed earlier in our laboratory (Meadows and Walden, MGCNL, 1978). Young, folded leaf tissue was excised, macerated and placed in a freshly prepared osmoticum of 0.4 M mannitol. This tissue was subsequently transferred to an enzyme solution consisting of 0.4 M mannitol, 5 mM $\text{CaCl}_2 \cdot 2\text{H}_2\text{O}$ and 0.5% cellulase pH 5.7, in which it was allowed to incubate in the dark at 27 C, with shaking over a 3-1/2 hr period with two changes of enzyme solution. Following enzymatic digestion, the crude protoplast fractions were combined, centrifuged at low speed and the pellet resuspended in a wash medium of 0.4 M mannitol and 5 mM $\text{CaCl}_2 \cdot 2\text{H}_2\text{O}$.

The protein synthetic capabilities of freshly isolated protoplasts were examined through the addition of various concentrations of ^{14}C -leucine for different lengths of time. Following labelling, the protoplasts were rinsed in wash medium, pelleted and protein extracted through homogenization in plant extraction buffer (PEB - Hughes, Baszczynski and Ketola-Pirie, MGCNL, 1981) using a glass homogenizer with Teflon pestle. Incorporation of the radioactive precursor was determined through scintillation counting of TCA precipitated samples. The results revealed an almost complete absence of protein synthesis in these cells. In contrast to this, intact leaf tissue showed a high degree of incorporation when labelled under similar conditions in glass distilled water and homogenized as described above, after previously grinding the tissue with a ceramic mortar and pestle. A similar analysis performed with tissues taken at various stages in the course of protoplast preparation revealed a steady decrease in the protein synthetic capabilities associated with an increasing degree of tissue manipulation.

Separation of polypeptides extracted from intact, macerated and enzyme treated leaf tissue was achieved through SDS polyacrylamide gel electrophoresis of protein samples on 10-20% linear gradient gels (U. K. Laemmli, 1970). Examination of the stained gel patterns showed no significant differences between tissues of the various treatments. However, certain changes in the patterns of newly synthesized polypeptides were observed between the different treatments when fluorographic analysis was undertaken. These alterations were most evident in a few higher molecular weight bands and appear to represent either changes in the amounts of certain polypeptides synthesized and/or novel protein synthesis.

It is possible that some of these differences observed may in fact reflect the synthesis of repair proteins initiated as a result of cell injury. If this is the case, this phenomenon may be viewed as a stress type response. Investigations are currently underway to further characterize this response and also to compare it with those elicited by various other forms of stress.

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The increase of UFGT activity in *in/in/in* endosperms

We have observed that homozygosity for intensifier (*in*) markedly enhances the amount of UDP glucose-flavonoid glucosyl transferase (UFGT) activity in mature endosperms of three maize genotypes (A1, A2, Bz1, Bz2, C1, C2, R, Vp; a1, A2, Bz1, Bz2, C1, C2, R, Vp; and A1, A2, Bz1, bz2, C1, C2, R, Vp) that are competent to produce the enzyme. Dooner and Nelson have previously demonstrated normal UFGT levels in *a1* and *bz2* mutants (Biochemical Genetics 15:509-519, 1977). The specific activities of UFGT for the different genotypes are given in Table 1. All comparisons were made in a W22 background and for ears grown in Madison during the summer of 1981. Similar results were obtained for *a1, in* vs. *a1, In*, or *bz2, in* vs. *bz2, In* genotypes grown in 1980. Enzyme levels were higher for *in* genotypes

TABLE 1 THE EFFECT OF ALLELIC STATE AT THE *In/in* LOCUS ON UFGT LEVELS IN MATURE ENDOSPERMS OF VARIOUS GENOTYPES

Genotype	Phenotype	Specific Activity ^a	Milliunits/Endosperm ^{-1b}
<u>A1, A2, Bz1, Bz2, C1, C2, R, pr, In</u>	red	.492 ± .117	329 ± 79
<u>A1, A2, Bz1, Bz2, C1, C2, R, pr, in</u>	very dark red	1.090 ± .103	688 ± 50
<u>bz2In</u>	bronze	.620 ± .190	377 ± 115
<u>bz2in</u>	red	1.67	738
<u>a1In</u>	colorless	.666 ± .100	585 ± 88
<u>a1in</u>	colorless	1.21 ± .220	808 ± 146
Ear 1 <u>bz2; In/In/in</u>	bronze	.591 ± .080	386 ± 51
<u>bz2; in/in/in</u>	red	.878 ± .080	791 ± 79
Ear 2 <u>bz2; In/In/in</u>	bronze	.744 ± .203	518 ± 141
<u>bz2; in/in/in</u>	red	.800 ± .120	866 ± 130

a $\mu\text{moles isoquercitrin mg protein}^{-1} \text{ hr}^{-1}$, mean \pm SD for 3 assays

b nmoles isoquercitrin hr^{-1}

on the basis of specific activity (both as μ moles isoquercitrin formed per hr per mg protein and nmoles isoquercitrin formed per hr per endosperm). Furthermore, the difference in enzyme activity of In versus in genotypes is not due to the presence of an inhibitor in the In stocks. When the extracts from In and in endosperms were mixed, the UFGT activities were additive.

The effect of intensifier on UFGT levels was verified in comparisons within ears between bz2, In/In/in and bz2, in/in/in kernels (Table 1). The amount of UFGT was higher for in/in/in kernels as compared to In/In/in kernels. However, there is no apparent dosage effect for in on UFGT activity; this is in contrast to the results of Reddy and Peterson (Can. J. Genet. Cyt. 20:337-347, 1978), who reported that heterozygotes for intensifier had pigment levels intermediate between those of the two homozygous conditions.

Coe concluded that genetic evidence favors placing intensifier prior to A1 in the anthocyanin synthetic pathway (In, A1, Bz1, A2, Pr; Am. Naturalist 91: 381-85, 1957). The increased synthesis of an enzyme (UFGT) later in the biosynthetic sequence than intensifier may result from induction of Bz1 due to increased flow of intermediates through that pathway or may be caused by some other regulatory mechanism controlled by intensifier and acting on the genes for anthocyanin biosynthetic enzymes. It is not, however, due to the synthesis of an inhibitor in In stocks.

The dominant alleles C and R are also required for anthocyanin formation in the aleurone. Recessive mutations at C and R and the dominant C-I mutation result in much reduced levels of UFGT (Dooner and Nelson, Biochemical Genetics 15:509-519, 1977). They concluded that products of C and R are required for the induction of UFGT. In contrast, it is the recessive allele of intensifier that elevates UFGT activity. If, as Dooner and Nelson have speculated, UFGT may be precursor-inducible, these results suggest that homozygosity for in may divert greater quantities of intermediates through the last steps of the anthocyanin biosynthetic pathway at the expense of other flavonoid compounds.

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Response of the proline requiring mutants to D-proline

The proline requiring mutants (pro) of Zea mays, identifiable on the basis of their abnormal endosperm morphology, are seedling-lethal monogenic recessive mutants. Their growth is resumed if L-proline is added to the medium; other amino acids as well as proline precursors do not restore mutant growth (Gavazzi et al., Theor. Appl. Gen. 46:339-345, 1975 and Racchi et al., Plant Sci. Lett. 13:357-364, 1978). All mutants so far isolated appear functionally allelic when crossed inter se.

Little is known about the metabolism of "non protein" amino acids in plants (Robinson, Life Science 19:1097-1102, 1976). D-alanine is the D-isomer most frequently found in higher plants, followed by D-tryptophan and a few others, while the presence and transformation of D-proline (as far as I know) has not yet been reported.

Here results are presented on the growth of pro mutant embryos in presence of D-proline. A partial phenotypic repair is observed (Fig. 1) for all the pro alleles, except for pro1-1121, on media enriched with 2mM D-proline, while at a higher concentration (4mM D-proline) there is a complete repair. Non-mutant control siblings are not affected in their growth by the D-proline addition to the minimal media. This observation is in contrast to the inhibitory effect

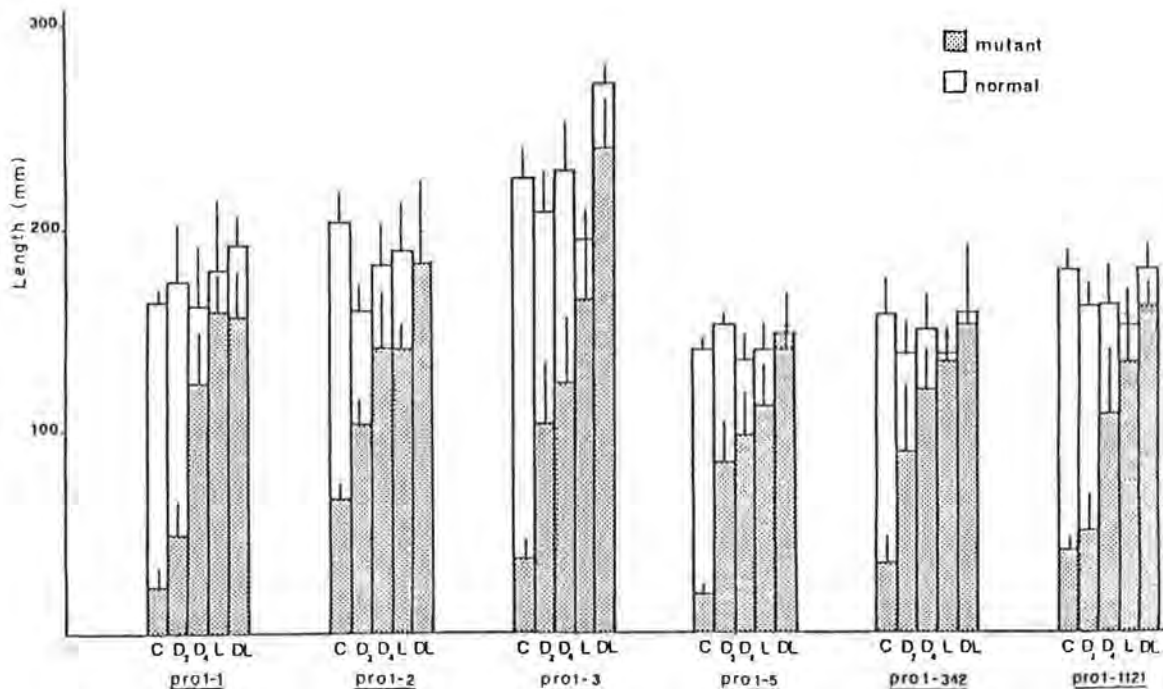


Fig. 1. Growth of *pro1* mutant and normal embryos on minimal (C) or supplemented media containing 2mM D-proline (D₂), 4mM D-proline (D₄), 2mM L-proline (L) or 2mM D-proline plus 2mM L-proline (DL) after 14 days of culture at 25°C with 12 hours photoperiod. The values are mean of at least ten determinations. Confidence limits (at 5% level) are shown.

previously reported (Robinson, 1976) for other D-amino acids in higher plants. D-isomers of nearly half the protein amino acids, when fed to plants, are converted to N-malonyl derivatives, to the corresponding alpha-ketoacids and to the L-isomer. Two different routes could be postulated for the conversion to the L-isomer: a direct racemization or a specific oxidation followed by a transamination of the resulting alpha-ketoacid. The only report about a possible direct racemization is that of Miura and Mills (Plant Physiol. 47:483-487, 1971) on D-tryptophan in cell cultures of tobacco.

The observation that *pro* mutants are repaired when fed with D-proline could be explained by assuming the presence of a proline racemase which converts D-proline directly to the L-isomer. The other possible route, through the corresponding alpha-ketoacid, is incompatible with the observations. In this respect proline requiring mutants might be of value in elucidating this area of amino acid metabolism.

Chiara Tonelli

Gametophytic expression of beta-glucosidase in maize pollen

Many beta-glucosidase variants have been described in maize sporophytic tissues, such as coleoptile, roots, and seedling. The enzyme is a functional dimer, controlled by a single locus with many alleles (C. W. Stuber et al., Bioch. Gen. 15:383-394, 1977). The locus has been localized on chromosome 10 (A. J. Pryor, MGCNL 52:14, 1978). In this note we present data on the distribution of the enzyme, as determined by starch gel electrophoresis, in pollen and immature endosperm tissues.

Extraction from pollen was obtained only with addition of Triton X to the buffer (Tris-Cl 0.1 M pH 8 + Triton X 1%), thus suggesting that the enzyme is wall-bounded. No bands are visible when electrophoresis of pollen extracts is carried out with L-His-citrate buffer system pH 6.3 (C. W. Stuber et al., Bioch.

Gen. 15:383-394, 1977). However, when a different buffer system is used (J. G. Scandalios, Bioch. Gen. 3:37-79, 1969) a single gametophytic band appears in one hour, while only very faint bands from sporophytic tissues are obtained after 24 hr. The staining mixture was the same for both gel types: Fast Blue BB 65 mg, 6-Br-2-naphthyl-beta-d-glucopyranoside 30 mg, in 100 ml phosphate buffer 0.5 M, pH 6.5

The pollen band was the same for all lines examined, independently of their sporophytic variant. Twelve lines were examined without finding any gametophytic variant. The same band is also clearly visible in pollen extracts from beta-glucosidase null plants (seeds furnished courtesy of Dr. M. M. Goodman). Direct staining of in vitro germinated pollen from null plants by means of the previously reported reaction mixture confirms beta-glucosidase activity both in grain and pollen tube.

It thus appears that different genes for beta-glucosidase are expressed in pollen and in sporophytic tissues.

Immature endosperms from some lines were analyzed for the presence of the enzyme. Neither the electrophoretic system suited for the sporophyte, nor the one used for the pollen, were able to reveal beta-glucosidase activity. It might well be that the enzyme is not expressed in the endosperm, or that another enzymatic form is involved in this tissue. Further analysis is aimed at elucidating this point.

C. Frova, M. Sari Gorla and E. Ottaviano

pr expression in endosperm culture

The feasibility of somatic cell genetics implies the expression of genetic markers at the cellular level. In our laboratory we have begun to analyze the in vitro expression of genes involved in anthocyanin biosynthesis (Gavazzi and Racchi, Maydica 26:175-184, 1981). Here I report preliminary observations on the expression of a homozygous A C R pr stock in tissue culture.

Callus was induced from ten-day-old endosperms and maintained under continuous light (3000 lux) at 26 C. The medium used was R. M. (Linsmaier and Skoog, 1965) supplemented with thiamine (0.1 mg/l), sucrose (30 g/l) and 2-4D (2 mg/l).

Explants turned brownish during the first month of culture and maintained the color through successive passages. After six subcultures, small pieces of callus were transferred to a liquid medium (MS supplemented with yeast extract (2 g/l) and 2,4-D (1 mg/l)) on a rotary shaker (120 rpm) and grown under continuous light (12000 lux) at 25 C. Pigment concentration of cultures on liquid and solid media was then compared to that of the aleurone layer.

The determinations (expressed as A 530 nm/mg fresh weight) accomplished on extracts in methanolic 1% HCl are the following:

	<u>Value*</u>
Aleurone	0.080
Callus grown on solid medium	0.012
Callus grown on liquid medium:	
extraction after 30 days culture	0.027
Callus grown on liquid medium:	
extraction after 60 days culture	0.001

*Values are means of at least three determinations

Growth on solid rather than liquid medium appears the more suited to pigment development.

Thin layer chromatography of aglycones obtained from callus using formic and Forestal as developing solvents showed, in addition to pelargonidin and cyanidin, two yellow pigments not present in aglycones obtained from aleurone. Characterization of these compounds is in progress.

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Interaction between French or North American maize lines and two cytoplasm for male sterility

We try to characterize and use the cytoplasmic male sterile variability of maize for our breeding program and are presently producing hybrids with C and El Salvador (ES) cytoplasm.

For 69 lines combined with a male sterile Cor ES tester the sterility reaction is generally similar for both cytoplasm. However, the reactions of hybrids with F487, F534, F568, W705 A appeared dubious.

During the 1981 season, isogenic combinations were compared in two environments, Montpellier (MTP) and St. Martin-de-Hinx (SMH) (LBS = Late Break of Sterility):

Male sterile tester	Cytoplasm	dubious male line	F 487	F 534	F 568	W 705 A
F7C	MTP		Sterile	Sterile	Sterile	Sterile
	SMH		Sterile	Sterile	Sterile	Sterile
F7ES	MTP		Sterile	Sterile	Sterile	Sterile
	SMH		Sterile	Sterile	Sterile	Sterile
F515C	MTP		LBS	Sterile	Sterile	Sterile
	SMH		LBS	Sterile	Empty Anthers	Sterile
F515ES	MTP		LBS	Sterile	Sterile	Sterile
	SMH		LBS	Sterile	Empty Anthers	Sterile
F546C	MTP		LBS	Sterile	LBS	Sterile
	SMH		LBS	Sterile	Empty Anthers	Sterile
F546ES	MTP		LBS	Sterile	LBS	Sterile
	SMH		LBS	Sterile	Empty Anthers	Sterile

In conclusion, the reactions seem unambiguously identical for the two cytoplasm, which could not be differentiated by fertility restoration.

Conversely, 5 lines chosen for a clear restoration reaction with ES and C male sterile tester were studied in F2 and backcrosses with these testers in Montpellier in 1981.

The phenotypes were noted in accordance with Lijingxiong and Duvick (phenotypes 0, 1, 2 classified as "Sterile," phenotypes 3, 4, 5 classified as "Fertile").

	Segregation Generation	Pedigree	Segregation observed		Segregation for one dominant gene		χ^2	Probability
			Fertile	Sterile	Fertile	Sterile		
CH 663-8	F2	A239C \times CH663-8	181	71	3	1	1,35	> 0,2
	"	A239ES \times CH663-8	194	61	3	1	0,16	> 0,5
	BC1	A239C ² \times CH663-8	38	32	1	1	0,51	> 0,3
	"	A239 ² ES \times CH663-8	39	43	1	1	0,20	> 0,5
F 602	F2	F546C \times F602	167	49	3	1	0,62	> 0,3
	"	F546ES \times F602	220	58	3	1	0,06	> 0,8
	BC1	F546C ² \times F602	44	45	1	1	0,01	> 0,9
	"	F546ES ² \times F602	43	50	1	1	0,53	> 0,3
F 720	F2	F515C \times F720	204	69	3	1	0,01	> 0,9
	"	F515ES \times F720	186	76	3	1	0,95	> 0,3
	BC1	F515C ² \times F720	39	50	1	1	1,36	> 0,2
	"	F515ES ² \times F720	49	40	1	1	0,91	> 0,3
F 722	F2	A239C \times F722	215	68	3	1	0,14	> 0,7
	"	A239ES \times F722	184	63	3	1	0,03	> 0,8
	BC1	A239C ² \times F722	39	32	1	1	0,69	> 0,3
	"	A239 ² ES \times F722	41	40	1	1	0,01	> 0,9
FR 64 A	F2	F546C \times FR64A	184	83	3	1	5,27	> 0,01
	"	F546ES \times FR64A	183	79	3	1	2,28	> 0,1

All these crosses appear to segregate for one dominant factor of restoration. However, the fit is not very satisfactory for FR64A.

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Allelic variation at the amylose-extender locus: Levels of soluble starch branching enzymes

Characterization of starch branching enzymes and starch synthases from homozygous Ae and ae-Ref kernels have shown ae-Ref kernels to lack detectable quantities of starch branching enzyme Iib (Boyer and Preiss 1978, BBRC 80:169). Two additional starch branching enzymes, I and Iia, as well as two fractions of soluble starch synthase, were found to be similar from kernels of the two genotypes. The soluble starch branching enzymes and starch synthases have not been examined in kernels homozygous for five additional ae alleles. These alleles are of spontaneous origin (ae-M1 and ae-B4), or arose in stocks carrying the Ac-Ds system of controlling elements (ae-il) or in stocks after EMS treatment (ae-EMS1 and ae-EMS3). All the alleles are recessive and homozygous kernels phenotypically indistinguishable. W64A backcross conversions for all of the alleles were provided by Douglas Garwood. Enzymes were purified from 22-day-old kernels and

characterized as described (Boyer and Preiss, *Plant Physiol.* 67:1141, 1981). No differences were found for the properties of branching enzymes I and IIa or the soluble starch synthases from kernels homozygous for different *ae* alleles. Based on DEAE-cellulose chromatography, extracts from kernels homozygous for any of the recessive alleles contained no branching enzyme IIb:

Kernel Genotype	Branching Enzyme ($\mu\text{mole}/\text{min}/\text{kernel}$)		
	I	IIb	IIa
Ae	1.04	1.70	0.86
ae-Ref	1.24	0.00	1.00
ae-il	1.06	0.00	0.67
ae-EMS1	1.02	0.07	0.50
ae-EMS3	0.84	0.05	0.96
ae-M1	0.61	0.12	0.53
ae-B4	0.89	0.00	1.02

In some purifications, the low levels of branching enzyme activity found in IIb fractions were due to contaminating branching enzyme IIa. The variation in levels of branching enzymes I and IIa between genotype does not appear to be allele related. Because purification of branching enzyme was required before different branching enzymes could be identified and quantified, the variation between purifications of the same genotype was as great as the variation between genotypes. Therefore, all the recessive *ae* alleles examined fall into one class, characterized by the deficiency of starch branching enzyme IIb.

Karen D. Hedman and Charles D. Boyer

Soluble starch synthases and starch branching enzymes from developing seed of teosinte

Previously three fractions of soluble starch branching enzyme and two fractions of soluble starch synthase were found in developing maize kernels using DEAE-cellulose chromatography (Boyer and Preiss, *Carbohydr. Res.* 61:321, 1978). The enzymes from two teosintes have now been examined. Seed from self-pollinated Galinat's northern teosinte (annual) and *Zea diploperennis* (perennial) were collected at 20 days after pollination, quick-frozen and stored at -80°C until used. Soluble starch synthases and branching enzymes were purified as described in detail elsewhere (Boyer and Preiss, *Plant Physiol.* 67:1141, 1981).

DEAE cellulose chromatography of extracts from both teosintes separated the starch synthases into two fractions and the branching enzymes into three fractions. A typical DEAE-cellulose profile is presented in Figure 1. DEAE-cellulose fraction I (column fractions 4-13) contained branching enzyme I. DEAE-cellulose fraction II (column fractions 36-64) contained branching enzyme IIb and citrate-stimulated starch synthase activities. DEAE-cellulose fraction III (column fractions 65-88) contained branching enzyme IIb and primer-requiring starch synthase activities. Characterization of concentrated DEAE-cellulose fractions revealed no differences between the teosinte enzymes and the maize enzymes previously reported. The teosinte branching enzymes are similar to maize branching enzymes in respect to chromatographic properties and relative activities in two different assay procedures. The teosinte starch synthases are similar to the maize enzymes in

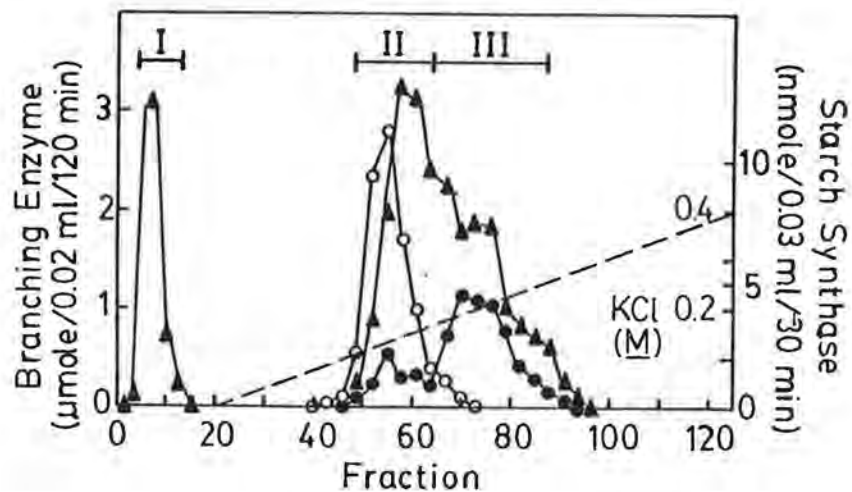


Figure 1: Chromatography of *Zea diploperennis* starch synthases and branching enzymes on DEAE-cellulose; branching enzyme activity (▲), citrate-stimulated starch synthase activity (○) and primed starch synthase activity (●). The dashed line shows the position of a KCl gradient.

regard to chromatographic properties, activity in the presence of citrate (no primer) and relative reaction rates in reaction mixtures containing different glucan primers. Although electrophoretic variation was not ruled out, these studies suggest that the catalytic properties of soluble starch synthases and branching enzymes are conserved in *Zea*. Additional races of teosinte will be examined in the future to explore this hypothesis.

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Fluorescence banding of constitutive heterochromatin in *Zea*

C. G. Vosa and D. J. Mogford (MNL 54:95, 1980) described one technique to locate knob regions in mitotic chromosomes of maize with the use of fluorochrome 'Hoechst 33258' following B.S.G. procedure. A simpler and direct procedure to obtain fluorescence bands for maize and teosinte chromosomes is described below. The experimental procedure adopted here does not involve any alkali digestion.

Pretreatment: Excised root tips were pretreated in 0.002 M 8-hydroxyquinoline at 14-18 C for 3.5 hr.

Fixation: In 1:3 acetic-alcohol at 10 C for 12-24 hr. After rinsing with 70% alcohol the roots were preserved in it.

Maceration: In 1:1 mixture of 1N HCl and 45% acetic acid at 60 C for 45-60 sec.

Washing: In distilled water for 10-15 min with 3-5 changes.

Squash: Root tips were squashed in a drop of 45% acetic acid.

Air drying: Cover glasses were flipped off after freezing in liquid nitrogen and slides were air dried and stored for 24-48 hr.

Staining: Before staining, the slides were immersed in 95% alcohol for 2-5 min and air dried. The preparations were stained in 5-10 μg/ml aqueous solution of

'Hoechst 33258' for 15-20 min. After a brief rinsing in distilled water, slides were mounted in 1:1 mixture of glycerol and water.

Observations: Fluorescent microscopic observations were made with a Leitz photomicroscope equipped with HBO 200W hg-lamp. Fluorescence was monitored and examined with exciting filters BG 12, BG 3 in combination with barrier filter OG 1. Photographs were taken on Kodak Tri-X pan film.

Remarkably clear fluorescence bands were observed in both maize and teosinte chromosomes. The terminal bands in Guatemalan teosinte may be seen in Figure 1.



Fig. 1

Fig. 1. 'Hoechst 33258' banded chromosomes of Guatemalan teosinte. Fluorescence bands have shown one to one correspondence both in number and position with C-bands.

The success may lie in the right choice of fresh roots, maceration in 1:1 mixture of 1N HCl and 45% acetic acid, and alcoholic treatment before staining. Maceration in HCl and acetic acid mixture produced the brightest bands. Satisfactory results were also obtained when roots were macerated in cellulase-pectinase enzyme mixture. Other maceration procedures did not yield satisfactory results.

J. K. S. Sachan, K. R. Sarkar, R. P. Sharma and Andy Pereira

Plant type of 'Sikkim Primitives'

Describing and rechristening a popcorn variety of Sikkim as 'Sikkim Primitive' (SP 1 and SP 2), Dr. N. L. Dhawan (MNL 38:69-70, 1964) is credited for arousing great interest in Himalayan maize among the concerned scientists of the world. A project on cytogenetic study of Himalayan germplasm of maize and its cultivated and wild relatives has been taken up by us under the auspices of the Indian Council of Agricultural Research (ICAR). Presently, we have a rich haul of maize germplasm from Sikkim, Nagaland, Meghalaya, Tripura, Mizoram, Manipur and North-Western Bengal. The germplasm can be broadly classified into waxy, sugary, floury, flint, dent and popcorn types.

Dr. H. G. Wilkes, who during his visit to our laboratory (1978-79) had the opportunity of studying our collections, has rightly emphasized the importance of studying Indian germplasm (MNL 55:13-15, 1981). We have collected through our personal trips sixteen 'Sikkim Primitive' types from various ethnic groups and different altitudes (4000-7000+ ft) of the North-Eastern Himalayas.

Experiments comprising thirteen primitive types were grown in a replicated trial at a height of 4500 ft in Kumaon Hills to compare the extent of variability among different populations. The data on five randomly selected plants were recorded. The accompanying table gives a general idea about the plant types of different collections.

Table: Morphological features of "Sikkim Primitive" types from N.E.H.

Collections	Plant height (cm)	Inter-node number	Number of shoots	Number of ears	Number of inter-nodes below the ear	Number of inter-nodes above the ear	Height below the ear (cm)	Height above the ear (cm)	Peduncle length (cm)	TASSEL		
										Tassel length (cm)	Number of primary branches	Number of secondary branches
SIKKIM												
S-18	201	17	6	5	9	3	115	20	10	29	11	4
S-23	248	19	6	5	11	3	160	17	9	28	22	2
S-27	234	19	6	5	11	3	169	14	8	27	28	6
S-44	235	18	6	5	10	3	169	14	8	29	23	6
NAGALAND												
N-1	132	15	5	5	8	3	85	13	7	29	21	2
N-3	244	15	6	5	11	2	140	10	8	30	15	5
N-4	212	17	6	5	10	2	142	7	8	32	16	2
MEGHALAYA												
M-1	210	17	5	5	10	4	110	8	11	36	32	15
M-15	113	13	6	5	5	4	48	7	6	18	27	4
M-25	216	16	5	4	3	4	84	40	10	40	34	14
TRIPURA												
T-1	235	18	5	5	10	4	126	17	7	29	19	1
T-2	236	16	7	6	9	1	134	6	4	31	9	2
T-26	267	19	10	7	9	1	126	5	3	27	10	1

Various collections of 'Sikkim Primitive' types seem to be derivatives of a single widespread popcorn variety grown and selected by different ethnic groups in remote and isolated pockets of the North-Eastern Himalayas. Tillering habit was not observed in any of the collections. All collections have the unique tendency of bearing ears at the top (Fig. 1). On an average, five ears of almost equal size per plant have been recorded. As many as eight ears have been noted in one of the collections from Tripura (T-26). The increase in the number of ears is associated with the decrease in the number of internodes between the topmost ear and peduncle. Plant types resembling the prototype of wild 'hypothetical maize,' and gynocious plants have also been recovered from the populations (Fig. 2 and 3).



Fig. 1



Fig. 2



Fig. 3

- Fig. 1. Typical 'Sikkim Primitive' plant showing internode pattern and bearing habit.
- Fig. 2. Nagaland strain (N-3): Terminal inflorescence resembles Galinat's reconstruction of prehistoric wild corn (P. C. Mangelsdorf, *Corn: Its Origin, Evolution and Improvement*, 1974).
- Fig. 3. Tripura strain (T-26): Gynoeceous plant--no tassel.

It is interesting to note that in 'Sikkim Primitives' many of the ears bearing male inflorescences at the tip are all self fertilized. Such ears are completely covered with long husks, and thus silk does not come out of the husk. Apparently there is no indication of inbreeding depression in these populations.

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Localization of Gdh2 to chromosome 10

From the F2 of 4Co82 x H25, approximately 12.5% recombination was observed between Glu, the structural gene for beta-glucosidase, and Gdh2, one of two structural genes for glutamate dehydrogenase (Gdh1 is on chromosome 1; Genetics 96:697-710).

Cross: Glu-2 Gdh2-S/Glu-7 Gdh2-F

<u>Gdh2</u>	<u>Glu</u>			<u>Totals</u>
	<u>2/2</u>	<u>2/7</u>	<u>7/7</u>	
<u>S/S</u>	18	4	0	22
<u>S/F</u>	7	37	8	52
<u>F/F</u>	<u>0</u>	<u>3</u>	<u>17</u>	<u>20</u>
<u>Totals</u>	25	44	25	94

Glu has been localized to chromosome 10 by Pryor (MGCNL 52:14) and by Stuber et al. (Genetics 95:225-236).

M. M. Goodman and C. W. Stuber

Further linkage studies with isozyme loci assigned to chromosome 1

We recently reported recombination values for several multiple point testcrosses involving Gdh1, Phi, Adh1, Pgm1, and Mdh4, all on the long arm of chromosome 1 (Genetics 96:697-710). We also routinely score most of our materials for Cat3, reportedly localized to the same chromosome arm by Roupakias et al. (TAG 58:211-218). In the course of determining the amount of recombination between Cat3 and isozyme loci spanned by Gdh1 and Mdh4, we found that Cat3 was at most loosely linked to any of these loci. We used a genetic stock obtained from Ed Coe to make two four-point testcrosses for Cat3, Gdh1, Phi, and Adh1. The same male parent (2323-17) was used for crosses with 4Co82 and 1873-4 (the latter is one of our standard tester stocks derived from Co125 x H25). The male parent had genotype Cat3-9/12, Gdh1-F/S, Phi-4/5, and Adh1-F/S. The male gametes transmitted are listed in Table 1. To test for Cat3 linkage to Mdh4, we used a testcross of Phi-3/4, Pgm1-A9/A16, Mmm-M/m, Mdh4-D8.5/D14.5, Cat3-C9/C12 as male onto W629A. The male parent had been experimentally introgressed

Table 1. Numbers of recombinant and parental gametes transmitted in a four-point testcross for Adh1-Phi-Gdh1-Cat3.

	Region 1		Region 2		Region 3		Total
	<u>Adh1</u>	<u>Phi</u>	<u>Gdh1</u>	<u>Cat3</u>			
Male parent tested*	<u>S</u>	<u>4</u>	<u>S</u>	<u>C9</u>			
	<u>F</u>	<u>5</u>	<u>F</u>	<u>C12</u>			
Region:	Parental Combinations	Single Recombinants		Double Recombinants			
Numbers Observed†	0	1	2	3	1,3	2,3	
	21	0	1	22	5	2	51
	35	5	0	15	3	2	60
Totals	56	5	1	37	8	4	111
Recombination % ± S.E.		Region 1	Region 2	Region 3			
		11.7 ± 3.1	4.5 ± 2.0	44.1 ± 4.7			

* Allele symbols listed from left to right correspond to loci spanning Regions 1 to 3 above. Locus abbreviations omitted to conserve space.

† Each entry in upper row begins with upper left allele listed under parent tested (viz. Adh1-S). Each entry in lower row begins with lower left allele (viz. Adh1-F).

with teosinte (source: race Central Plateau, collection K69-2) and the segment of 1L on which Pgm1-A16 and Phi-3 resided was of teosinte origin, while the Mdh4-D14.5 to Mmm-M segment was of maize origin. Use of such a stock might be expected to decrease the amount of recombination between Mdh4 and Cat3 if there were any lack of chromosome homology between maize and teosinte in a region of 1L spanned by the two loci. The results are listed in Table 2. Clearly Cat3 is not closely linked to Mdh4. The recombination values for Regions 2 and 3 correspond closely to those estimated earlier (7.8 ± 2.8 vs. 4.4 ± 1.1 and 24.4 ± 4.5 vs. 19.0 ± 2.1). For Region 4, however, there is a striking discrepancy. Here we

Table 2. Numbers of recombinant and parental types for the five-point testcross for Cat3-Mdh4-Mmm-Pgm1-Phi.

	Region 1		Region 2			Region 3		Region 4	
	<u>Cat3</u>	<u>Mdh4</u>	<u>Mmm</u>	<u>Pgm1</u>	<u>Phi</u>				
Cross tested*	<u>C9</u>	<u>D14.5</u>	<u>M</u>	<u>A16</u>	<u>3</u>	<u>x C9 - D12 - m - A9 - 4</u>			
	<u>C12</u>	<u>D8.5</u>	<u>m</u>	<u>A9</u>	<u>4</u>				
	Parental Combinations	Single Recombinants			Double Recombinants		Totals		
Region:	0	1	2	3	4	1,2	1,3	1,4	
Numbers Observed†	11	16	2	8	1	2	0	0	43
	19	13	1	5	0	2	9	1	47
Totals	30	29	3	13	1	4	9	1	90
Recombination % ± S.E.		Region 1	Region 2	Region 3	Region 4				
		47.8 ± 5.3	7.8 ± 2.8	24.4 ± 4.5	2.2 ± 1.6				

* Allele symbols listed from left to right correspond to loci spanning Regions 1 to 4 above. Locus abbreviations omitted to conserve space.

† Each entry in upper row begins with upper left allele listed under cross tested (viz. Cat3-C9). Each entry in lower row begins with lower left allele (viz. Cat3-C12).

have only 2.2% recombination, while a similar cross involving standard inbred lines resulted in 19.5% recombination. Presumably this discrepancy is caused by lack of sequence homology between maize and this accession of teosinte, perhaps as a result of an inversion covering most of Region 4. The only evidence these data hold for Cat3 belonging to this linkage group is the lack of C9-D8.5-m-A16-3 type double-crossover gametes in Table 2. About 5 were expected, none were observed. Yet 9 of the complementary type double-crossover gametes (C12-D14.5-M-A9-4) were observed.

M. M. Goodman and C. W. Stuber

Further variation encountered for Mmm

Mmm on 1L simultaneously affects the electrophoretic mobilities of the products of all three of the structural MDH loci, Mdh1 on chromosome 8, Mdh2 on chromosome 6, and Mdh3 on chromosome 3 (Genetics 94:153-168; 95:425-442). The common allele (Mmm or Mmm-M) is present in most inbred lines, genetic stocks, breeding materials and races of both maize and teosinte. A recessive allele (mmm or Mmm-m), found occasionally among both U.S. and Latin American stocks, results in faster migration of all the mitochondrial MDH homodimers and heterodimers. The faster migration appears to be coupled to lower enzymatic activity (K. Newton, personal communication). Another allele, intermediate to Mmm-M and Mmm-m in gene action and in phenotypic response, has been discovered in a collection of Cuzco.

The newly discovered allele, which we are calling m2, is recessive to M, but dominant to m. When Mmm-m2 is homozygous or heterozygous with Mmm-m, all the mitochondrial MDH homodimers and heterodimers no longer migrate as single distinct circular or oval bands. Instead, each such band is elongated, extending from its usual position to approximately the position it would hold if Mmm-m were homozygous (on our gels this is about 1 mm). No loss in overall activity is evident, in contrast to Mmm-m.

Table 1. Recombination data for Mmm-m2 and Mdh4 on chromosome 1 of maize.

Cross:	<u>Mdh4-D12</u>	<u>Mmm-m</u>	x	<u>Mdh4-D8.5</u>	<u>Mmm-m</u>
	<u>Mdh4-D12</u>	<u>Mmm-m</u>		<u>Mdh4-D12</u>	<u>Mmm-m2</u>
		Parental Types		Recombinant Types	Totals
<u>Mdh4-D8.5</u> :		50		2	52
<u>Mdh4-D12</u> :		49		3	52
Totals		99		5	104

Recombination % \pm S.E. = 4.8 \pm 2.1

In small-scale linkage tests we have seen no recombination between Mmm-m2 and Mmm-m, and Mmm-m2 maps to the same region of 1L as does Mmm-m (Table 1), approximately 5 map units from Mdh4. Specifically, Mmm-m was mapped to 4.4 \pm 1.1 recombination units from Mdh4, while these data suggest virtually the same result, 4.8 \pm 2.1 recombination units.

M. M. Goodman and C. W. Stuber

Isozyme (allozyme) genotypes for 406 publicly available inbred lines

In an earlier report (MGNL 55:126, 1981) we reported that a compilation of the isozyme genotypes for 342 inbred lines of corn was available from our laboratory. We have assayed an additional 64 lines and are including a complete listing of the data for the 406 lines as Table 1.

In Table 1, designations used for the alleles are those used in our laboratory scoring. Alleles at a locus are ordered by their relative migration distances. For the MDH loci, larger numbers correspond to greater anodal migration. For the other loci, larger numbers correspond to slower migration. The symbol N denotes a recessive null allele. The column with the heading "MMM" denotes the presence or absence of an allele that affects the migration distance of the mitochondrial MDH bands. An M in this column denotes the presence of a rarely found recessive migration modifier allele at this locus, whereas a blank in this column denotes the usually found dominant allele at this locus.

Because most lines are homozygous, we have listed only a single number at each locus. For those lines that are segregating at one or more loci, the most commonly found allele is shown followed by an asterisk (*). Also, in a few cases where the locus was not fixed, two equally frequent alleles were found. In these cases, the alleles are followed by an ampersand (&) and the line is listed twice as (A) and (B) versions of the line.

C. W. Stuber and M. M. Goodman

Mitochondrial DNA variation in Latin American maize races

Maize cytoplasms can now be partially classified using molecular techniques for the isolation and characterization of organelle DNAs. Restriction endonuclease cleavage fragment analysis of mitochondrial (mt) and chloroplast DNAs has demonstrated variation between and within the major cytoplasmic groups N, C, S and T (Pring and Levings, *Genetics* 89:121-136, 1978; Pring et al., *Crop Sci.* 20:159-162, 1980) and also among several races of teosinte (Timothy et al., *Proc. Natl. Acad. Sci. USA* 76:4220-4224, 1979).

In the current survey, mtDNAs from 93 Latin American maize races were examined electrophoretically for the presence or absence of plasmid-like elements and were also subjected to analysis using both BamHI and EcoRI endonucleases. Eighteen races contained plasmid-like mtDNAs. One of these, Conico Norteño, was previously reported (Weissinger et al., *MGCNL* 55:84-86, 1981; Weissinger et al., *Proc. Natl. Acad. Sci. USA* 79:1-5, 1982) to contain mtDNAs indistinguishable from the S-1 and S-2 plasmid-like elements of the cms-S cytoplasm. That study also reported the discovery of two other plasmid-like mtDNAs, R-1 and R-2 (previously designated S*-1 and S*-2), which we have now found in seventeen different races, all but one of which are South American.

BamHI digestion of mtDNAs produced ten different fragment patterns, while EcoRI digestion produced eight distinct electrophoretograms. Races were placed into one of eighteen groups, each group being defined by a particular combination of BamHI and EcoRI patterns and the presence or absence of plasmid-like elements. Seven races produced patterns identical to those produced by one or another of the major cms groups (C, T and S) with each group being represented by at least one race. Races from Meso-America and some South American races with Meso-American affinities were separated from other South American races. South American races were divided among three general classes of related groups. There was considerable agreement between these groupings and those derived by other workers on the basis of morphological and cytological affinities. We believe that this work provides the breeder and geneticist with a catalog of cytoplasmic variation of indigenous Latin American maize, and may also provide a model for future classification of maize cytoplasms.

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Zea diploperennis may have plasmid-like mitochondrial DNAs

Plasmid-like mitochondrial (mt) DNAs have been reported in two maize cytoplasms: the cytoplasmic male-sterile cms-S (Pring et al., *Proc. Natl. Acad. Sci. USA* 74:2904-2908, 1977) and the fertile RU cytoplasms of 18 Latin American maize races (Weissinger et al., *Proc. Natl. Acad. Sci. USA* 79:1-5, 1982). In each case, the plasmid-like DNAs are observed in pairs of linear molecules with one molecule being appreciably larger than the other (Fig. 1).

Restriction endonuclease fragment patterns of five RU cytoplasmic maize races differ from the standard RU pattern by the presence or absence of up to three bands, and/or the degree of fluorescence in other bands (Weissinger et al., 1982). The spontaneous reversion of cms-S maize to fertility, correlated with the disappearance of the S-1 and S-2 molecules and changes in restriction fragment patterns, has been associated with a transpositional event (Levings et al., *Science* 209:1021-1023, 1980).

Mitochondrial DNAs from 31 accessions representing all the described taxa of teosinte were examined by agarose gel electrophoresis. Five of six Zea diploperennis accessions examined possessed two molecules designated D-1 and D-2 that migrated, respectively, to approximately the same positions, although

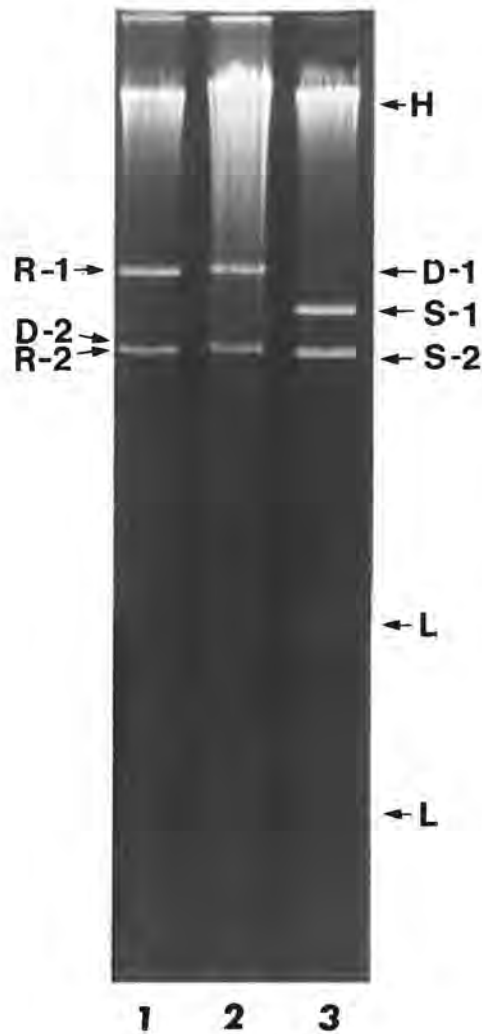


Fig. 1. Agarose gel electrophoresis of mitochondrial DNA from: (1) RU maize, (2) *Zea diploperennis* which contains the D-1 and D-2 molecules, (3) cytoplasmic male-sterile S maize. Symbols: H, high molecular weight mt chromosomal DNA; L, low molecular weight circular mtDNA (circularity not established in 2).

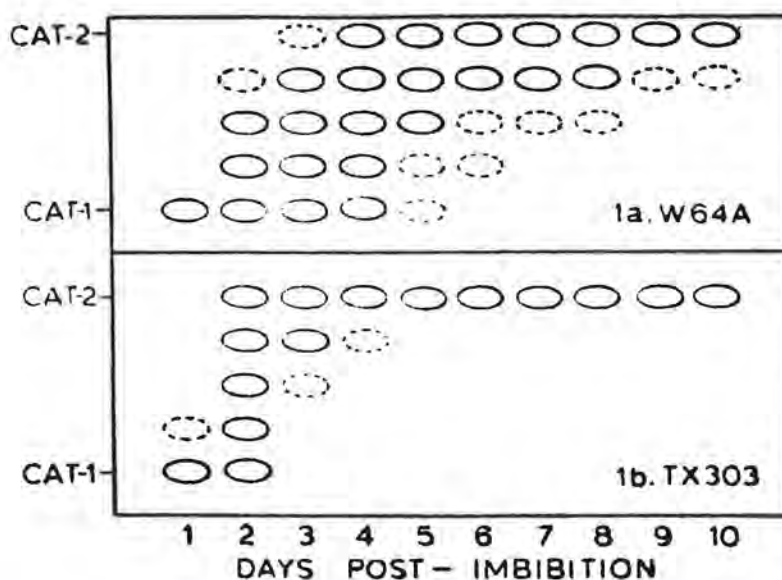
slightly higher, as the R-1 and R-2 plasmid-like DNAs found in normal maize (Fig. 1). Differences in migration were greater between D-1 and R-1 than between D-2 vs. R-2 and S-2. The S-1 and R-1 molecules are distinguished by length, a BamHI site in R-1, and unique sequences. S-2 and R-2 have not been differentiated by length, molecular hybridization, heteroduplex analyses, or restriction sites.

BamHI restriction patterns of *Z. diploperennis* reveal three pattern groups. Those accessions containing D-1 and D-2 differ by nine or more bands from accessions lacking D-1 and D-2. Similar results were obtained with EcoRI. When D-1 and D-2 are present, two BamHI fragment pattern groups occur which differ only by the presence or absence of one band. The brilliantly fluorescent bands associated with total mtDNA digestion patterns of cms-S and RU cytoplasm are not seen with restriction endonuclease digestion of D-1 and D-2 bearing cytoplasm.

D. H. Timothy, C. S. Levings III, W. W. L. Hu, and M. M. Goodman

Altered catalase gene expression in the scutellum of maize

Two distinct, genetically defined catalases of *Zea mays* are differentially expressed in the scutellum during development of the seedling (Scandalios, *Physiological Genetics*, Chapter 2, 1979). These are CAT-1 and CAT-2 coded for by the *Cat1* and *Cat2* loci, respectively. In a typical line (e.g., W64A), CAT-1 is expressed in the dry seed and early days post-imbibition. CAT-2 production becomes evident on days 2 to 3 post-imbibition, when a 5-banded zymogram pattern is observed (Figure 1a). As development of the seedling proceeds, CAT-1 activity declines while CAT-2 activity increases. This results in a "shift" in the zymogram pattern from CAT-1 expression to CAT-2 expression (Figure 1a).



A naturally occurring mutant line (Tx303) has recently been characterized which exhibits a more rapid shift from CAT-1 to CAT-2 expression than is commonly observed in most lines. By day 4 post-imbibition, only *Cat2* is expressed on the zymograms (Figure 1b). It has been found that this mutant line does not differ significantly from W64A for various parameters related to CAT-2 expression. For instance, the developmental activity profile for catalase, primarily controlled by the production of CAT-2 gene product, does not differ between the two lines. CAT-2 protein developmental profiles (determined by rocket immunoelectrophoresis) do not differ between the two lines. Also, the developmental profiles of two glyoxysomal associated enzymes (malate synthase and isocitric lyase) do not differ between the two lines. The glyoxysomes were investigated because maize catalase is known to be associated with these organelles (Scandalios, *J. Hered.* 65:28, 1974). These data suggest that the regulation of the levels of CAT-2 protein in the scutellum of the two lines is similar. Therefore, the more rapid shift observed in the mutant line is probably due to a regulatory effect on the levels of CAT-1 protein likely by a more rapid degradation of CAT-1 protein.

Preliminary genetic analysis indicates that the trait is likely controlled by a single dominant gene. This and linkage studies are currently underway.

Joel M. Chandlee and John G. Scandalios

Purification of the chloroplast-associated superoxide dismutase of maize

Superoxide dismutase (SOD) isozymes are associated with the cytosolic, mitochondrial and chloroplastic fractions of maize seedlings (Baum and Scandalios, *Differentiation* 13:133, 1979). The isolation and characterization of the cytosolic and mitochondrial superoxide dismutases has been recently reported (Baum and Scandalios, *Arch. Biochem. Biophys.* 206:249, 1981). The chloroplast-associated isozyme, SOD-1, is encoded in the nuclear gene *Sod1* and is distinct from the cytosolic isozymes (SOD-2, SOD-4) though it is similar to these proteins with respect to its molecular weight, subunit composition, metal content and sensitivity to cyanide (Baum and Scandalios, *Differentiation* 13:133, 1979; Baum and Scandalios, *Arch. Biochem. Biophys.* 206:249, 1981; Baum and Scandalios, *J. Hered.*, in press, 1982). SOD-1 has been purified to homogeneity using conventional purification techniques (Table 1). The purity of the isozyme preparation was determined by

TABLE 1
PURIFICATION OF SOD-1

	units/ml	total units	mg protein	units/mg protein	fold purification
Crude extract	109	114,265	3,580	32 ^a	—
45% - 65% (NH ₄) ₂ SO ₄ fraction	1,844	51,639	359	144	4.5
G-100 Sephadex chromatography	465	31,140	56	556	17.4
DEAE-Sephacel chromatography	148	16,619	5.4	3,078	96.2
Hydroxylapatite chromatography	2,000	4,000	.366	10,930	341.6

^a Assayed according to the method of Beauchamp and Fridovich (1971)

non-denaturing and sodium dodecyl sulfate (SDS) polyacrylamide gel electrophoresis (PAGE). The holoenzyme molecular weight (M_r) of SOD-1 was estimated to be $33,500 \pm 1,500$ by G-100 Sephadex chromatography. The subunit molecular weight of SOD-1 was estimated to be approximately 14,500 by SDS-PAGE on 15% gels. It was concluded that SOD-1 is a dimer with a molecular weight of 31,000 - 33,000 and that it is composed of apparently equal subunits. Like the cytosolic isozymes, SOD-1 appears to have at least one intrachain disulfide bond per subunit.

J. A. Baum and J. G. Scandalios

Amino acid analysis of cytosolic superoxide dismutases of maize

Two cytosolic superoxide dismutases, designated SOD-2 and SOD-4, have been reported in maize (Baum and Scandalios, *Differentiation* 13:133, 1979). These two proteins have been purified to homogeneity and partially characterized (Baum and Scandalios, *Arch. Biochem. Biophys.* 206:249, 1981). SOD-2 and SOD-4 are virtually indistinguishable on the basis of a number of biochemical properties including

their sensitivities to various inhibitors such as KCN, NaN₃, H₂O₂ and diethyldithiocarbamate. In addition, the two isozymes are recognized identically by antibodies raised against SOD-4. However, the nuclear gene *Sod4* affects SOD-4 expression but has no obvious effect on SOD-2 expression, suggesting that SOD-2 and SOD-4 may be encoded in distinct structural genes (Baum and Scandalios, J. Hered. in press, 1982). This study has been extended to include an amino acid analysis of the two superoxide dismutases.

Duplicate lyophilized samples of SOD-2 and SOD-4 were hydrolyzed in 0.7 ml 6N HCl under nitrogen in 1 ml vials (Pierce Chemical Co.) at 145 C for 2, 4 and 8 hr. The contents were neutralized and dried in a sodium hydroxide vacuum dessicator and analyzed with a Durrum 500 amino acid analyzer. The labile amino acids, threonine and serine, were estimated by extrapolation to zero-time hydrolysis. For the other amino acids, the estimates obtained for the 2, 4 and 8 h hydrolysis periods were averaged together because the recoveries of each amino acid were statistically the same for each sample ($\pm 10\%$). SDS-polyacrylamide gel electrophoresis was performed on a 13.5% gel according to the method of Laemmli (Laemmli, Nature 222:680, 1970).

Table 1
Amino Acid Compositions

	Maize SOD ²	Maize SOD ⁴	Cupro-zinc superoxide dismutases						
			bovine erythrocyte ^b	wheat Isozyme I ^c	wheat Isozyme II ^c	Green Pea ^d	Spinach ^e	chicken ^f	Neurospora crassa ^g
Aspartic acid ^h	38.3 \pm 0.4	38.2 \pm 0.5	35	28	28	45	35	32	56
Threonine	17.0 \pm 0.2	24.2 \pm 0.3	26	53	30	30	28	18	26
Serine	20.3 \pm 0.2	21.9 \pm 0.3	20	15	12	14	10	14	14
Glutamic acid ^h	29.8 \pm 0.6	21.2 \pm 0.4	24	21	26	19	20	23	20
Proline	15.5 \pm 0.6	17.0 \pm 0.5	14	19	19	14	17	12	14
Glycine	68.0 \pm 0.9	65.0 \pm 0.8	50	55	43	56	42	50	39
Alanine	26.8 \pm 1.0	29.6 \pm 0.5	21	28	25	21	23	22	20
half-Cystine	3.4 \pm 0.3	3.6 \pm 0.8	6	6	4	6	4	14	3
Valine	30.8 \pm 1.2	27.8 \pm 1.1	28	31	34	21	28	29	22
Methionine	0.8 \pm 0.1	0.7 \pm 0.1	1	2	0	0	2	4	0
Isoleucine	11.4 \pm 0.2	12.1 \pm 0.2	17	13	10	20	6	14	13
Leucine	18.3 \pm 0.3	19.9 \pm 0.3	20	22	31	21	22	16	11
Tyrosine	4.4 \pm 0.4	2.1 \pm 0.3	7	0	0	0	0	2	2
Phenylalanine	6.2 \pm 0.1	7.0 \pm 0.1	10	7	6	9	6	8	6
Histidine	15.7 \pm 0.4	17.6 \pm 0.1	16	19	15	18	14	14	11
Lysine	10.8 \pm 0.2	12.3 \pm 0.3	22	10	8	10	13	20	12
Arginine	5.4 \pm 0.2	5.2 \pm 0.6	10	8	10	6	7	8	9
Tryptophan	--	--	0	--	--	0	0	0	0

^aThe number of residues was calculated for 32,000 g of enzyme.

^bKeele, B.B.Jr., McCord, J.M., Fridovich, I. (1971) *J. Biol. Chem.*, 246:2875.

^cBeauchamp, C.O., and Fridovich, I. (1973) *Biochim. Biophys. Acta* 317:50.

^dSawada, Y., Ohyama, T. and Yamazaki, I. (1972) *Biochim. Biophys. Acta* 268:305.

^eAsada, K., Urano, M. and Takehashi, M. (1973) *Eur. J. Biochem.* 36:257.

^fWeisinger, R.A. and Fridovich, I. (1973) *J. Biol. Chem.* 248:3582.

^gMisra, H.P. and Fridovich, I. (1972) *J. Biol. Chem.* 247:3410.

The amino acid compositions of the two proteins are shown in Table 1 along with amino acid composition data for several copper and zinc-containing superoxide dismutases purified from other eukaryotes. SOD-2 and SOD-4 differ considerably in their glutamic acid, threonine, and tyrosine content, indicating that the two isozymes are distinctly different proteins. Similarities among all of the superoxide dismutases in Table 1 can be observed, reinforcing the notion that they may be conserved in their structure. More definitive information on the evolution of these proteins could be obtained through determination of their amino acid sequences.

Although no attempt was made to determine tryptophan, it is clear that copper and zinc-containing superoxide dismutases are generally deficient in this amino acid. The higher content of glutamic acid in SOD-2 could account for the difference in electrophoretic mobility observed between the two proteins on non-denaturing gels as well as for their chromatographic behavior on anion-exchange columns.

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Relationships between husk number and several characters in dent maize hybrids

Simple correlations between husk number and several characters in 63 dent maize single cross hybrids (450-600 FAO maturity) grown in performance trials in 1981, as well as simple correlations between husk number of the hybrids and husk number of their parent inbreds are estimated. Average husk number varies from 6.6 to 14.3 (9.4 mean) in hybrids studied, from 7 to 18 (9.2 mean) in female, and from 6 to 14 (9.2 mean) in male inbred parents.

Significant correlations are established between husk number and stalk node number from the soil surface to the ear ($r = -.80^{***}$), and between husk number and number of days from planting to silking of the hybrids ($r = .37^{**}$). Simple correlation coefficients estimated in 1981 between husk number and ear diameter ($.37^{**}$), kernel row number ($.49^{**}$), ear height from the soil surface ($-.64^{***}$), and kernel moisture percent at harvest time (.12) are similar to those estimated in 1979, $r = .38^{**}$, $.27^*$, $-.23$, and $.11$, respectively (Georgiev and Mouhtanov, 1980, Genetics and Plant Breeding, n. 3, p. 180-190, Sofia).

Husk number in hybrids correlates better to the husk number mean of the two inbred parents ($r = .65^{***}$), than to the husk number of the female ($r = .48^{**}$) and male inbreds ($r = .36^{**}$).

The above data may be useful for maize breeders in improvement of the kernel drying rate in new hybrids.

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Identification of two cytoplasmic male sterile sources from the People's Republic of China

At least 31 independently discovered sources of cytoplasmic male sterility have been classified by one or more means into one of three types: C, S, and T (Beckett, Crop Sci. 11:724, 1971; Gracen and Grogan, Agron. J. 66:654, 1974; Forde et al., Genetics 95:443, 1980; Kemble et al., Genetics 95:451, 1980). None

of the 84 sources mentioned by Duvick (Adv. Genet. 13:1, 1965) was known to have arisen as a mutation within a homogeneous population of N (nonsterile) cytoplasm plants. We were interested, therefore, in characterizing two additional sources of cytoplasmic male sterility that had been discovered in China.

One source (designated Chi31cms) was identified in 1974 as one of several male sterile plants in a population designated Luo Synthetic 1 originally derived from 10 USA lines and 2 China lines. From 1974 to 1976 this source was maintained by crosses with fertile plants of Luo Synthetic 1. In 1977 backcrossing with the inbred, Chi31, was begun and our material was analyzed after the third backcross.

The second source (designated Mo17cms) was found in 1976 as a single male sterile plant within a population of the inbred line C103 which, according to records in China, was not uniform. This line was advanced by crosses with Mo17 from 1976 through 1979.

In 1980 these two male-sterile lines and their respective maintainer lines were grown at St. Paul, MN. We inoculated leaves of 5-6 week-old plants with pathotoxin from Helminthosporium maydis race T. Lesions developed on leaves of the Mo17cms line and on Wf9T check plants but not on any other lines. This suggested that Mo17cms belonged to the T cytoplasm group. We also crossed the two sterile lines with Wf9 lines carrying nuclear genes for fertility restoration of T, S, and C cytoplasm as indicated in Table 1.

Table 1. Fertility restoration in progeny of cytoplasmic male sterile lines crossed by fertility restoration testers.

Sterile line	Tester line	Progeny	
		Sterile	Fertile
<u>Mo17cms</u>	Wf9T Rf ₁ /rf ₁ , Rf ₂ /rf ₂	18	23
	Wf9S Rf ₃ /rf ₃	35	0
	Wf9C Rf/rf	32	0
	Wf9N rf/rf	32	0
<u>Chi31cms</u>	Wf9T Rf ₁ /rf ₁ , Rf ₂ /rf ₂	38	3
	Wf9S Rf ₃ /rf ₃	0	40
	Wf9C Rf/rf	36	0
	Wf9N rf/rf	39	0

Fertile progeny from Mo17cms were obtained only in crosses with the T cytoplasm restorer line. The 1 fertile:1 sterile segregation indicated that Mo17cms was homozygous dominant at either the Rf1, or Rf2 locus. Wf9S Rf3/rf3 restored fertility to all the progeny in crosses to Chi31cms as expected if only pollen carrying the Rf3 allele were functional. We have no explanation for the 3 fertile plants in progeny from Wf9T tester crosses. These fertility restoration data indicated that Mo17cms belonged to the T class and Chi31cms belonged to the S class.

We confirmed this classification by comparing mitochondrial DNA restriction endonuclease digestion patterns of the 2 sources with those of Wf9N, C, S and T cytoplasm controls (Fig. 1). The XhoI restriction pattern of Mo17cms (lane C) was indistinguishable from that of Wf9T (lane B) and the Chi31cms (lane G) and Wf9S (lane H) patterns both clearly show the S1 and S2 plasmid-like DNAs characteristic of the S cytoplasm group (Pring et al., PNAS 74:2904, 1977).

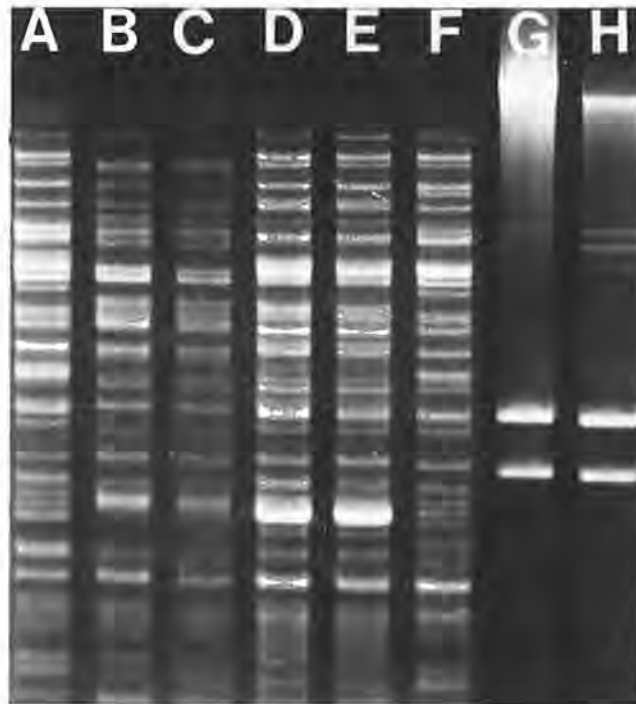


Fig. 1. Comparison of mitochondrial DNAs from two male sterile sources with N, C, S and T cytoplasm controls. Lanes A-F, digestion with XhoI; Lanes G and H, no digestion. A = Wf9N; B = Wf9T; C = Mo17cms; D & G = Chi31cms; E & H = Wf9S; F = Wf9C.

Records on the identity and sources of the original lines comprising Luo Synthetic 1 are no longer available but it is possible that one line could have been S cytoplasm with restorer genes. Subsequent segregation of rf3 alleles could have uncovered the S cytoplasm male sterility. S cytoplasm has not been used in China for hybrid production. It is also possible that the T cytoplasm source in C103 resulted from a seed mixture some time prior to 1976. Therefore, it is very likely that Chi31cms and Mo17cms trace back to other S and T sources, respectively, and not to new independent events. It remains of interest, however, to determine whether the same cytoplasmic event(s) leading to male sterility can occur repeatedly and independently.

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Characteristics of T-cytoplasm revertants from tissue culture

Brettell and colleagues (Theor. Appl. Genet. 58:55, 1980) reported that male-fertile plants were obtained among plants regenerated from tissue cultures of male-sterile T-cytoplasm corn. The male-fertile plants also were resistant to *Helminthosporium maydis* race T toxin and were regenerated from control cultures not exposed to toxin.

Table 1. Plants regenerated from A188T tissue cultures expressing immediate and/or heritable changes in male sterility and toxin susceptibility.

Line designation	Phenotype ^{a/}	Progeny segregation		
		Cross	Fertile, resistant	Sterile, susceptible
T*1	Fertile, susceptible	⊗	109	2
T*2	Fertile (sector), susceptible	⊗	115	1
T*4	Sterile, susceptible	XA188N ^{b/}	8	11
T*5	Fertile (sector), resistant	XA188N	56	0
T*6	Fertile, resistant	XA188N	58	0
T*7	Fertile, resistant	XA188N	48	0
T*8	Fertile, resistant	(No seed obtained)		
T*9	Fertile, resistant	(No seed obtained)		

a/ One or two leaves were inoculated with toxin.

b/ From tassel seeds on a completely pistilloid tassel.

Among 162 plants regenerated from A188T cultures we have now obtained 8 plants (Table 1) which expressed an immediate altered phenotype for male sterility and toxin susceptibility and/or segregated for such alterations in the maternal progeny.

The results indicate that the following situations can arise in regenerated plants from unselected A188T tissue cultures: (1) the entire plant phenotype may be altered, e.g., fertile tassel, resistant leaves and uniform progeny from the ear (T*6 & 7 and possibly T*8 & 9); (2) the tassel may be sectored for fertility and the leaves may be resistant (T*5) or susceptible (T*2); (3) the tassel may be fertile, the leaves susceptible and the ear may segregate (T*1 & 2); and (4) the plant may be uniform but progeny from tassel seeds may segregate (T*4). In this last case, the tassel was entirely pistilloid and had no lateral branches, therefore, male-fertility in the altered sector likely did not have the opportunity to be expressed in the regenerated plant. Brettell observed one plant that was male fertile and toxin susceptible but did not report data on progeny from that plant. The progeny data shown here support the notion that even though male fertility and toxin resistance can be separated spatially in a regenerated plant (probably as the result of a heterogeneous multicellular condition early in the differentiation process), this separation is temporary and does not carry through to progeny of such plants.

Other progeny tests (data not given) in which the male-fertile, regenerated plants were used as males have confirmed that the male fertility and toxin resistance traits are cytoplasmically inherited.

Mitochondrial DNA (mtDNA) was extracted from the variant lines by the procedure of Kemble et al. (Genetics 95:451, 1980) and digested with various restriction enzymes. Fig. 1 shows the patterns obtained when mtDNAs from A188N and A188T controls and the six variants were digested with XhoI. Examination of ethidium bromide-stained gels revealed that a fragment of about 6.6Kb was missing in five of the six variant lines compared to the A188T control pattern. This alteration is the same as found in previous analyses of nine other tissue culture-derived lines selected for toxin resistance (Gengenbach et al., Theor. Appl. Genet. 59:161,

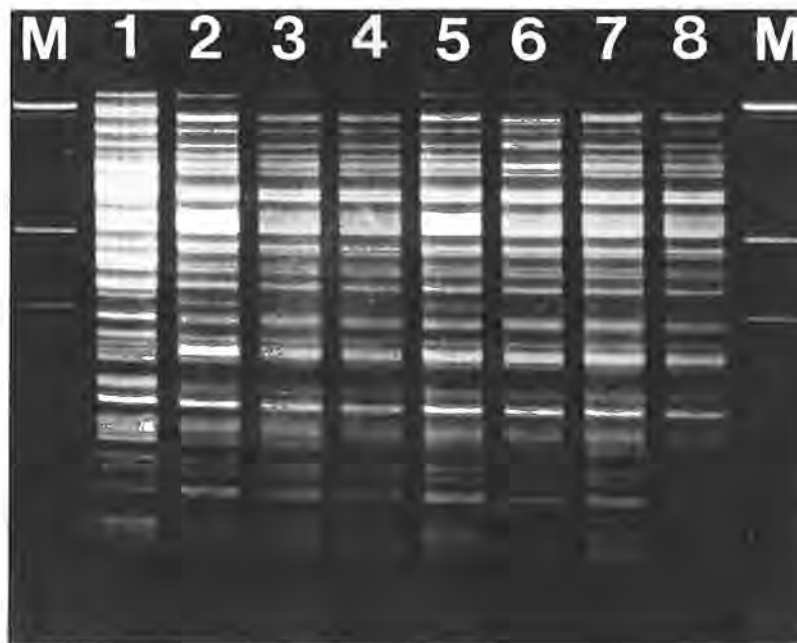


Fig. 1. XhoI restriction endonuclease digestion patterns of mtDNA from A188N and A188T control and regenerated variant lines. M = λ DNA markers of 23.5, 9.6 and 6.6 Kb from the top; 1 = A188N; 2 = A188T; 3 = T*1; 4 = T*2; 5 = T*4; 6 = T*5; 7 = T*6; 8 = T*7.

1981; Pring and Gengenbach, unpublished). One male-fertile, toxin-resistant line (T*4) has not exhibited any detectable differences from the A188T control pattern. T*4 mtDNA has a band at the comparable position as the 6.6Kb fragment in A188T mtDNA, but small alterations may not be resolved by this level of comparison. T*5 also exhibits alterations in higher molecular weight fragments indicating the possibility of additional rearrangements in this line.

We have compared mtDNA from 34 male-sterile, toxin-susceptible lines derived from plants regenerated from unselected A188T cultures. None of these lines had detectable alterations in the 6.6Kb fragment (not shown). Thus, 14 of 15 male-fertile, resistant lines but 0 of 34 male-sterile, susceptible lines regenerated from T cytoplasm cultures at Minnesota have an alteration in a specific portion of the mtDNA. We think the circumstantial evidence is strong enough to propose that the 6.6Kb fragment is responsible for the male-sterile, toxin-susceptible phenotype of T cytoplasm corn and that alterations in this sequence (e.g., base substitutions, new restriction sites, rearrangements or deletions) could result in a male-fertile, toxin-resistant phenotype.

Burle Gengenbach and Paul Umbeck

Partial synchronization of maize cells in liquid suspension culture

Mass isolation of somatic metaphase chromosomes might be possible from maize cells growing as a liquid suspension culture. Increasing the mitotic index of the culture should facilitate chromosome isolation procedures. Black Mexican Sweet Corn (BMS) suspension cells, kindly provided by C. E. Green, were grown for four days at 26 C in a modified Murashige and Skoog liquid medium (MS) prior to various

treatments. Six general procedures for attaining partial synchronization were tested.

1. Temperature alternatives: Cell suspension cultures were shifted to 4 C for 10, 20, 36, or 42 hours or 35 C for 36 hours and then returned to 26 C.

2. Carbohydrate starvation: Cultures were transferred to a sucrose-free medium for 27, 35, 45 or 56 hours.

3. Double thymidine block: Thymidine (1 ug/ml medium) was added for 16 hours. Cultures were then washed with fresh media and grown for 8 hours. Thymidine was added again (1 ug/ml medium) for 8 hours. Cultures were then washed again.

4. 5-FudR synchronization: 5-Fluorodeoxyuridine (0.5-2 ug/ml medium) and uridine (1 ug/ml) were added to the medium for 20 or 24 hours. After washing with fresh media, thymidine (1 or 2 ug/ml) was added.

5. Hydroxyurea synchronization plus colchicine: Hydroxyurea (3-10 mM) was added to the medium for 24 or 36 hours. Cultures were then washed and subcultured into a conditioned medium (80% fresh, 20% used medium) with or without 0.02% colchicine.

6. Mitotic arrest with colchicine: Cells were cultured in the MS medium plus 0.02% colchicine.

After the treatments, cultures were maintained at 26 C on a shaker in the medium specified at the end of each procedure. Samples (1 ml) were taken every 1-2 hours, fixed in 3 parts 95% ethanol:1 part glacial-acetic acid, squashed and stained in propionic carmine.

The first four procedures did not significantly alter the mitotic index (M.I.) from the untreated control. The last two procedures, hydroxyurea plus colchicine and colchicine alone, resulted in a 2-2.5 fold increase in the M.I. (Fig. 1). We

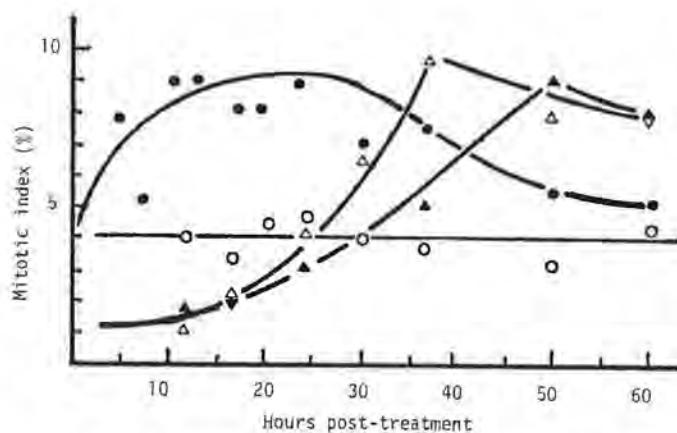


Fig. 1. Frequency of mitotic cells in BMS cell cultures; 0-0 untreated control cells; ●-● 0.02% colchicine treated cells; △-△ 3mM hydroxyurea and 0.02% colchicine treated cells; ▲-▲ 3mM hydroxyurea treated cells. All hydroxyurea treated cultures have been washed and transferred to the fresh MS medium after treatment.

presume that the hydroxyurea blocks the active cells (probably a low percentage) at the end of G1. Upon release, the cells stay in G1 for several hours. This could be due to the transfer procedure or a hydroxyurea effect. The hydroxyurea without colchicine procedure required a longer time (50 hours) to reach the maximum effect compared with the hydroxyurea plus colchicine procedure. The reason for this is not clear. The "mitotic arrest with colchicine" procedure resulted in a mitotic index of 9% after 10 hours compared with less than 4% in the untreated control.

Table 1. Effect of 0.02% colchicine on ploidy level of BMS suspension culture. Cells in metaphase were used to determine the ploidy level.

Ploidy level	Hours post-treatment																				
	4	8	12	14	16	20	24	28	32	36	40	44	48	52	56	60	72	84	96	108	200
2X	88*	96	85	74	94	81	89	95	96	87	80	84	40	38	36	49	58	47	76	68	50
4X	12	4	10	16	4	16	11	5	2	11	13	16	58	52	60	50	36	47	24	32	50
8X	0	0	5	10	2	3	0	0	2	2	7	0	2	10	4	1	6	6	0	0	0
Total no. of cells	32	37	52	33	66	38	38	51	54	37	51	25	39	38	28	28	37	27	25	35	20

* Data represent % 2X, 4X, 8X cells.

Although the BMS culture is variable in chromosome number, ploidy changes were expected with the colchicine procedure. The frequency of tetraploid cells (Table 1) increased significantly at around 48 hours of treatment and generally remained high. Octoploid cells also were observed.

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Cell cycle parameters and doubling time of Black Mexican Sweet corn suspension cultures

Large quantities of purified chromosomes have been successfully isolated from animal cell cultures. The bulk isolation of plant chromosomes has been less successful probably due to difficulties in synchronizing cell division. Understanding the cell cycle parameters and other growth characteristics would facilitate achieving synchronization. We report here the doubling time and cell cycle parameters for Black Mexican Sweet (BMS) corn suspension cultures.

Doubling time: The BMS culture, kindly provided by C. E. Green, was grown in a modified Murashige and Skoog (MS) liquid medium for 4 days at 26 C on a rotary shaker. After standing for about 10 minutes, settled cells (0.5 ml) were transferred to fresh MS medium and grown for 2, 4, or 6 days at 26 C. An exponential growth pattern was apparent based on fresh weight:

Culture age (days)	Fresh weight (mg)	$\ln (wt/w_0)^a$
0	162 + 7 ^b	
2	455 + 10	1.02 + 0.20
4	917 + 145	1.72 + 0.15
6	1482 + 64	2.22 + 0.40

^a w_0 = initial fresh weight, w_t = fresh weight at time t .

^b Standard deviations based on 5 replicates.

Fresh weight was obtained by centrifuging the culture in a Beckman TJ-6 centrifuge at 1000 rpm for 5 minutes. Doubling time was estimated by using the following formulae: $y = mx + b$ and $y = \ln(wt/w_0) = kt$, where m is the slope of $\ln(wt/w_0)$ plotted against days, b the y-axis intercept, w_0 the initial fresh weight, wt the fresh weight at time t , and $1/k$ the doubling time. Based on $\ln(wt/w_0)$ values in Table 1, $y = 0.32x + 0.39$. At $x = 6$ days, $y = 0.32(6) + 0.39 = 2.31$ and, thus, $2.31 = k(6)$ or $1/k = 2.6$ days or 62 hours. Therefore, the doubling time is 62 hours.

Frequency of dividing cells: The BMS suspension cultures were grown in MS medium plus ^3H -thymidine (^3H -TdR) of specific activity 2 uCi/ml. A sample (1 ml) was collected every hour and fixed in 3 parts 95% ethanol:1 part glacial acetic acid overnight at room temperature. The fixed cells were washed twice with 0.1 M sodium acetate (pH 4.5) and digested for 2 hours with 0.5 ml cellulase (0.5%) plus pectinase (0.5%). The resulting protoplast preparation was rinsed twice with 45% acetic acid and stored in 45% acetic acid at 4 C. A drop of the protoplast preparation was placed on an acid-cleaned slide and a coverslip applied. The slide was then placed on dry ice for 5 minutes and the coverslip flipped off with a razor blade. The slide was then passed through 70% and 95% ethanol, air dried, coated with Kodak NTB-2 emulsion, exposed for 1 day at 4 C, and developed in Kodak D-19 developer.

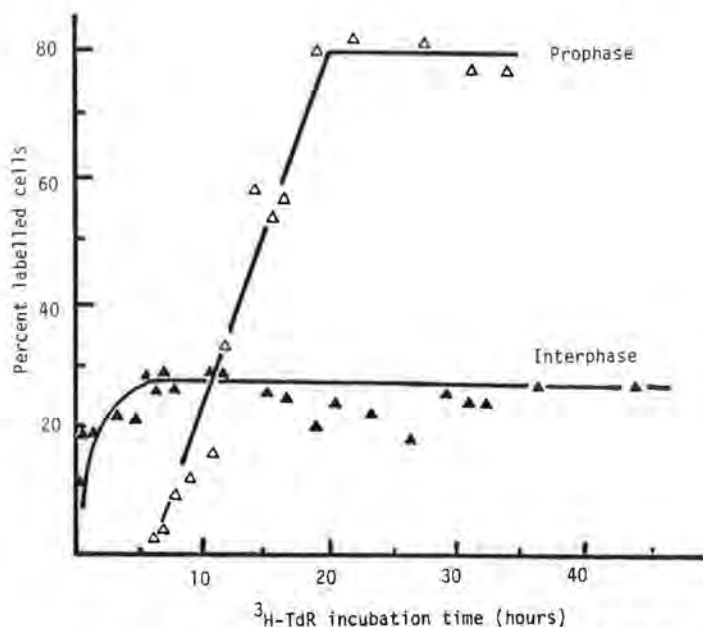


Fig. 1. Frequency labelled interphase and prophase cells after continuous growth in MS medium plus ^3H -TdR

Less than 30 minutes were required for ^3H -TdR to be incorporated into certain nuclei (Fig. 1); 9% of the nuclei were labeled within 30 minutes of incubation. Labeling of interphase nuclei reached a maximum (28%) in 5 hours. Apparently, 72% of the cells were not actively dividing. The number of labeled prophase cells reached a plateau (80%) in 20 hours. The reason for 20% unlabeled prophase cells is not known but it might reflect a prolonged G2 period for cells in G2 at the time of initial labeling. The first labeled prophase cell was observed 7 hours after labeling and the first labeled telophase cell was observed after 11 hours. The mitotic period (M), therefore, is 4 hours.

Cell cycle parameters: The BMS culture was grown for 4 days, labeled with $^3\text{H-TdR}$ for 1 hour, washed, and transferred to fresh MS medium. Samples (1 ml) were collected at various times as indicated in Figure 2 and prepared for autoradiography. Subculturing appeared to cause a growth lag of about 5 hours. The

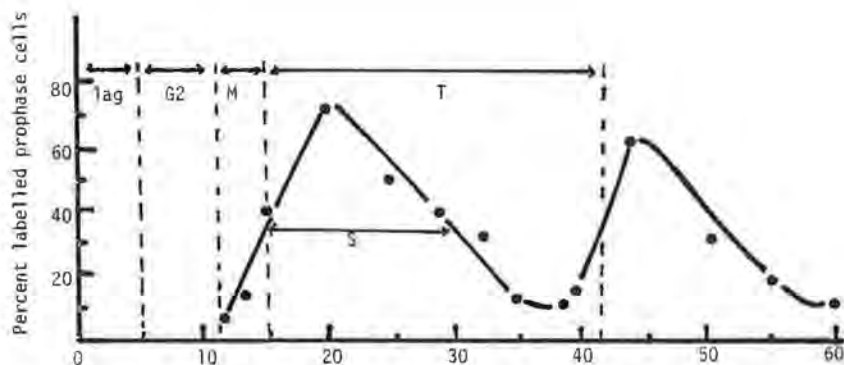


Fig. 2. Frequency of labelled prophase cells after pulse labelling with $^3\text{H-TdR}$ for one hour at 25°C

first labeled prophase cell was observed after 7 hours without subculturing after label introduction (Fig. 1) and after 12 hours with subculturing prior to labeling (Fig. 2). Based on the results displayed in Figure 2, G2 is 6 hours, S is 15 hours, M is 4 hours, G1 is 2 hours, and the total cycle time (T) is 27 hours. The 27-hour cycle time is in general agreement with the growth data presented in Table 1, assuming a doubling time of 62 hours and only 28% of the cells actively dividing.

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Evidence for disproportionate replication of ribosomal RNA genes during endosperm development

Corn endosperm development represents a complex series of genetic events with rapid physiological and morphological changes. These changes are especially striking during the period 12-18 days post-pollination. During this time, endosperm mitoses cease except in the peripheral area, starch and protein synthesis escalates, nuclear and nucleolar volumes increase, and cellular DNA amounts increase in a dramatic fashion. Results reported here indicate that rDNA amounts are not constant during endosperm development; the proportion of rDNA increases during this 12-18 days post-pollination period but subsequently decreases to near initial levels.

In 1977, 1978, 1980, and 1981, plants of the single cross Wf9 x B37 were self-pollinated and the developing ears collected periodically, frozen, and stored at -20°C . In 1978, inbreds Wf9, B37, and A619 were sampled for comparative purposes. The Wf9 material was not usable due to poor development. DNA isolation, rRNA labeling, and DNA/rRNA hybridization procedures were varied over the years in an attempt to determine whether the unexpected results were a function of the procedure or represented a developmental phenomenon. Filter saturation DNA/rRNA hybridization procedures published by Phillips et al. (Chromosoma 36:79-88, 1971) were used in 1977 and 1978. In 1980 and 1981, DNA and rRNA were isolated by

procedures described by Mascia et al. (Gene 15:7-20, 1981). In 1980 we employed an aqueous DNA/rRNA hybridization technique published by Casey and Davidson (Nucleic Acids Res. 4:1539-1552, 1977). The 1978 hybridization procedure was followed in 1981. ^3H -rRNA of specific activity $3\text{-}4 \times 10^3$ cpm/ug rRNA was isolated from 6-day-old seedlings of inbred A188 for the 1977 and 1978 experiments. ^{32}P -end-labeled rRNA used for the 1980 and 1981 experiments was extracted from Black Mexican Sweet corn suspension cultures; this RNA was kindly provided by Brenda Hunter in 1981. The specific activity was 3×10^4 to 6×10^5 cpm/ug rRNA. Only the hybridization percent can be reported; rRNA gene multiplicities cannot be estimated because the DNA content of endosperm cells changes during development.

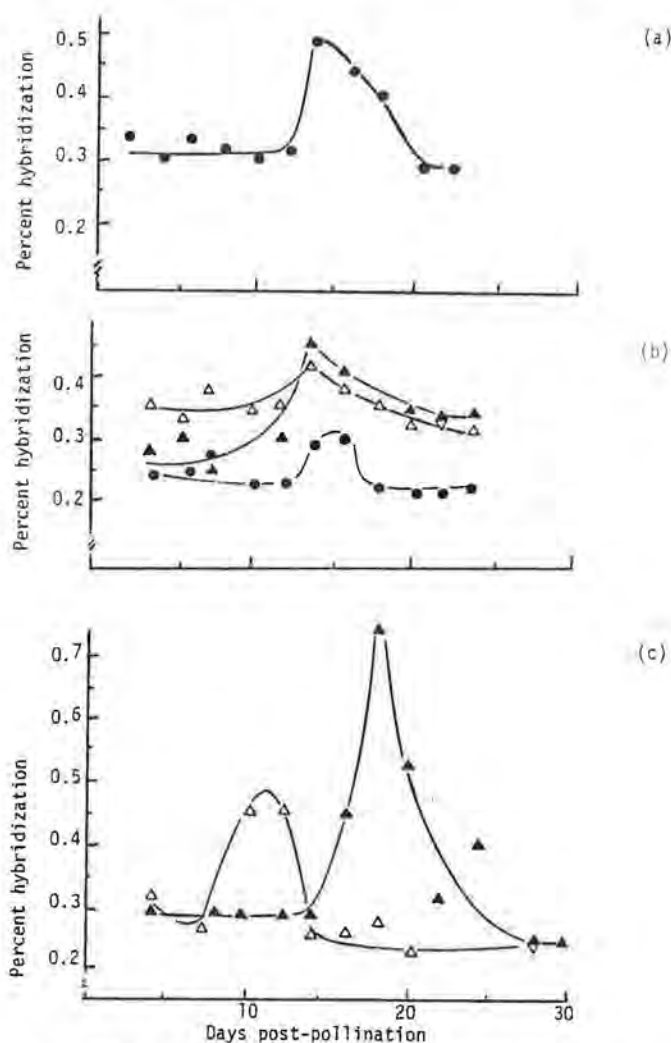


Fig. 1. Percent DNA/rRNA hybridization in inbreds A619 and B37, and single cross Wf9 x B37 (self-pollinated) obtained in the 1977 (a), 1978 (b), 1980 and 1981 (c) experiments. Each value represents 4 to 10 replicates.

Unexpectedly, the relative amount of rDNA was found to change during endosperm development. A hybridization peak occurred for all three lines (Wf9 x B37, B37, and A619); the peak was apparent in each of the four years for Wf9 x B37. Maximum hybridization values for Wf9 x B37 were 0.49%, 0.46% and 0.74% for 1977, 1978, 1980, and 1981 respectively. The reason for a higher maximum hybridization value

in 1981 is not known. The time of occurrence of the maximum hybridization value ranges from 12 to 18 days post-pollination with an average of 14-15 days. The variation may be due to varying environmental conditions in the different years.

Our hypothesis is that when endosperm cells cease dividing they continue to synthesize DNA and the rDNA is preferentially replicated, at least to some degree. As other parts of the genome continue to replicate, the proportion of rDNA is reduced. DNA synthesis during corn endosperm development might be analogous to *Drosophila* salivary gland polytenization in that different portions of the chromosomes may be replicated to different degrees. If selected regions are preferentially replicated, the multiplicity of those DNA sequences could be extremely high.

R. L. Phillips, A. S. Wang and P. N. Mascia

Regenerable maize tissue cultures derived from immature tassels

For several years it has been known that regenerable tissue cultures (cultures capable of plant regeneration) can be initiated from immature embryos of several maize genotypes. Recently, explants of other tissues such as immature tassels, ears, and nodal stem sections also have been used successfully (S. J. Molnar et al., MGNL 54:52, 1980; T. Rice et al., Propagation of Higher Plants Through Tissue Culture, Univ. of Tenn. Symp. Proc., p. 262, 1979). This study was undertaken to determine optimal conditions for culturing young tassels, and to identify genotypes which will produce regenerable cultures under those conditions.

Immature tassels were removed under sterile conditions from 6- to 7-week-old field grown plants and then cut into 1-2 mm long cross-sections. The pieces were placed on modified MS medium (1.0 mg/l 2,4-D) in 100 x 25 mm plastic Petri dishes and incubated at 26 C under a 16/8 hr photoperiod. All tissues were examined after 3 and 5 weeks for the presence of scutellar-like bodies usually indicative of regenerability for embryo-derived cultures. A positive culture response was scored if one or more pieces of a tassel possessed these bodies. Immature embryos from the same 13 genotypes (4-5 ears, 50 embryos per ear) also were used to initiate cultures; their response was scored similarly. When tassel-derived cultures were transferred to MS medium without 2,4-D, not all tissues with scutellar-like bodies produced plants capable of growing to maturity. Some genotypes (W22, W23, Mangelsdorf Multiple Tester, Coe's Stock 6) produced plants that grew up to 2" tall, but then died.

Table 1. Genotypic comparison of regenerable culture initiation from 0.1-3.0 cm tassels.

Genotype	No. of tassels	No. of positive-responding tassels ¹	% positive response	Avg. no. of responding pieces per tassel ²
A188	89	73	82.0	2.3
W22	44	4	9.1	1.5
A188 X W22	21	18	85.7	2.8
W22 X A188	52	30	57.7	2.9
A619	29	27	93.1	5.3
A188 X A619	26	26	100	10.3
A632	45	0	0	0
ND203	3	0	0	0
W23	48	33	68.8	1.8
WF9	23	13	56.5	4.0
Black Mexican	39	1	2.6	3.0
Mangelsdorf	39	11	28.2	1.8
Stock 6	39	19	48.7	2.9

¹A positive-responding tassel possessed scutellar-like bodies on any tissue when examined 5 weeks after culture initiation.

²The average number per positive-responding tassel of explant pieces that produced scutellar-like bodies.

The frequency of positive culture response from immature tassels ranged from 0% (A632 and ND 203) up to 100% (A188 x A619 (Table 1). Other types of growth responses were observed, including root formation, secretion of mucilage, enlargement of florets, and callus growth. Each genotype had a characteristic growth pattern, which was genotype specific for all tassel sizes. The average number of positive-responding pieces per tassel is another measure of genotypic response and revealed further differences among genotypes (Table 1).

The tassel response often, but not always, corresponded to the positive response rate of immature embryos. Vigor of cultures derived from either explant source was similar for each genotype.

Tassels from progeny of a self-pollinated A188 plant that had been regenerated from a tassel-derived tissue culture gave about the same positive response (85.7%) as did tassels from progeny of normally grown A188 plants (82.0%).

Further experiments with A188 tassels were performed to determine additional important factors for culture initiation. When tassels were cut into sections of varying lengths (25 tassels per treatment), the response of 3 mm sections (60.0%) was slightly lower than with 1 mm sections (72.0%). The developmental stage as measured by tassel size was an important factor (Table 2). Although explant pieces from any position in the tassel could give a positive response, pieces from the basal half of branches of 2-3 cm long tassels had the highest response rate.

Table 2. Effect of tassel size on regenerable culture initiation from A188 tassels.

Tassel length	No. of tassels	No. of positive-responding tassels ¹	% positive response	Avg. no. of responding pieces per tassel ²
cm				
0.1-1.0	26	19	73.1	1.8
1.1-2.0	30	24	80.0	2.2
2.1-3.0	33	30	90.9	2.5
3.1-4.0	15	5	33.3	1.8
4.1-5.0	15	2	13.3	1.0
5.1-6.0	9	1	11.1	1.0
6.1-7.0	4	0	0	0

¹A positive-responding tassel possessed scutellar-like bodies on any tissue when examined 5 weeks after culture initiation.

²The average number per positive-responding tassel of explant pieces that produced scutellar-like bodies.

Initiating cultures from immature embryos is mechanically easier than from tassels, but it may be useful to initiate cultures from tissues taken later in the sporophytic life cycle than embryonic stages. Plants are usually 6-7 weeks old when the tassels are 2-3 cm long; this allows time for the expression of certain genetic markers. Cytogenetically altered plants, such as haploids, monosomics, or those with deletions, can be identified via genetic markers and then cultured by the immature tassel technique. We have used this technique to establish regenerable tissue cultures from haploid and monosomic plants. (Supported in part by USDA/SEA Competitive Research Grant 5901-0410-8-0149-0 and NSF Graduate Fellowship).

C. A. Rhodes, C. E. Green and R. L. Phillips

Tests of Hooker-Russell exotic sources of cytoplasm converted to inbred A632

One of the 42 lines, my culture number B2709 in 1979 and B3175 in 1981 (PI 16164375 x B14¹⁶, x A632¹⁴), has all male sterile plants. One line, my culture no. B2714 in 1979 (PI 174990 x B14¹⁶ x A632¹⁴) is very late and 3179a in

1981 has very poor germination. The other 40 lines are being tested for the possibility that they may have a cytoplasmic restorer of genetic male-sterile #1. Thirty-four of these do not carry the cytoplasmic restorer for ms1. The other six will be tested in 1982.

Charles R. Burnham

Linkage tests of T8-9a (semi-sterile #2) with bk2 bm4 (backcross data)

Based on only 42 plants in which N vs. bk2 and semi-sterile vs. fertile plants were segregating 1:1, there were seven recombinants and 35 parental combinations, 16.7% recombination. Based on only 15 plants in which each of the three markers was segregating 1:1 there was 33.3% recombination between bm4 and the interchange point, 46.7% between bm4 and bk2 indicating the probable order is bk2 T bm4.

Charles R. Burnham

Segregation patterns in maize interchange heterozygotes

In Chapter 42 on the "Properties and uses of duplicate-deficient chromosome complements in maize," by E. B. Patterson in "Maize Breeding and Genetics," edited by D. B. Walden, the following statement appears on p. 696:

"Although individual exceptions are known, adjacent-2 and 3-1 disjunctional patterns from most translocation heterozygotes appear to be infrequent."

This is contrary to evidence presented by Burnham (Genetics 35:445-481, 1950). As stated on p. 677 of Chapter 41:

"In maize, chromosome segregation in ring configurations without crossing over in interstitial segments is two alternate:one adjacent-1:one adjacent-2. In chain-forming interchange heterozygotes, using ones with one break in the satellite of chromosome 6, adjacent-2 segregations are rare regardless of the length of the interstitial segments. The segregation is one alternate:one adjacent-1."

Since Patterson was dealing with interchanges that produce female transmissible (Dp + Df)'s they were probably chain-formers in which adjacent-2 segregations are rare.

We have exchanged letters on the above and we are in agreement.

Charles R. Burnham

A method for counting pollen abortion in heterozygotes for chromosome aberrations

The pollen from a plant heterozygous for a chromosome aberration may include several classes: 1) normal size fully filled with starch, 2) much smaller but well filled, 3) completely devoid of starch, 4) small amount of starch (less than about 1/4, scattered starch grains), or 5) larger amount of starch (about 1/2 or more, but scattered starch grains). The method outlined requires only one decision when distinguishing the number 2 class from number 1 and the number 5 class from number 4.

About 1/3 of an anther (cut with a razor blade) is teased into a small drop of dilute I₂ + KI solution to stain the starch, add a narrow cover slip (about 1/3 of a 7/8" square slip) or a similarly-sized piece of a glass slide. The drop of solution should be just large enough to fill out under the cover glass. Ring the cover glass with glycerine to prevent drying out during the counting.

The entire slide is counted, proceeding as follows: starting at one side of the cover glass, count adjacent strips lengthwise of the cover glass. Using the circular disc of notecard (a disc that has a parallel-sided window cut out), placed on top of the shelf, inside the ocular, gives a microscope field with parallel sides rather than a circular field. As the slide is moved during the counting of each strip across the field, pollen grains are not appearing at the edges of a circular field. Counts may be made as follows: on the first trip across the slide, count the total of #1 and 2 classes. Then, passing back across the same strip, count the small but well filled #2 class. The difference between the total of 1 plus 2 minus #2 gives the number of normal grains. Next, passing back across the same strip, count the completely empty grains (class #3). The remaining obviously aborted grains may be the partially filled ones (classes 4 and 5 with different amounts of scattered starch grains). Passing back across the same strip, count the total of these two classes (#4 and #5). Then, passing back across the same strip, count the number five classes. The total of 4 plus 5 minus #5 gives the number in the number 4 class.

When plants with two rings of 10 chromosomes each or a ring of 20 were treated with colchicine (see note by Ghobrial in this newsletter), the tassels that had some fertility had an additional class that was much larger than normal (also fully filled with starch), presumably with the $2n$ chromosome number. The total of this class plus 1 and 2 was counted first, then this larger class of pollen, then #2.

By noting a pollen grain at one edge of the field, the slide can be moved over to count the next strip. Count successive strips until the entire slide is counted.

This information on the different interchanges would survey the effects on starch formation and development of pollen resulting from deficiencies of different chromosome segments.

Charles R. Burnham

Effects of colchicine and colcemid seed treatment on the fertility of multiple chromosome interchange heterozygotes of corn

In a preliminary experiment (reported in Maize Genetics Newsletter 42:120, 1968), when germinating corn seeds with two rings of 10 chromosomes each were treated with colchicine, a few plants were found to have fertile pollen of large size. The purpose of this study is to produce more of these fertile plants and to study their chromosomal constitution and cytogenetic behavior. This report presents the data obtained last summer.

Five diploid stocks were used. Each stock is homozygous (designated Δ) for multiple chromosome interchanges for big rings that involve the chromosomes indicated below:

- stock #1: involves chromosomes 3, 2, 4, 9, 10
- stock #2: involves chromosomes 1, 5, 6, 7, 8
- stock #3: involves chromosomes 6, 3, 2, 4, 8
- stock #4: involves chromosomes 5, 7, 1, 9, 10
- stock #5: involves chromosomes 5, 7, 1, 9, 10, 8

F1 seeds from the following crosses were used:

- cross A: stock #1 (Δ 3-2-4, 9, 10) x stock #2 (Δ 1-5-6-7-8)
- cross B: stock #3 (Δ 6-3-2-4-8) x stock #4 (Δ 5-7-1-9-10)
- cross C: stock #5 (Δ 5-7-1-9-10-8) x stock #3 (Δ 6-3-2-4-8)

The A and B F1's have 2 rings of 10, C has a ring of 20. All three F1 types have 20 different chromosomes. About 200 F1's of each of the three crosses were soaked in water for 20 hours, then placed on filter paper and watered for 5 days with 0.2% aqueous solution of colchicine. Due to the limited quantity of colcemid, a .02% aqueous solution of colcemid was used only on F1 seed of cross A. The solutions were adjusted to pH 7.0. Tap water was used for watering the following 2 days. The seedlings were planted in sand in the greenhouse for 10 days after which they were transplanted in the field. About half the seeds survived the treatment and reached the mature plant stage.

The majority of the plants were, like their untreated siblings, very highly sterile (over 99%) with shriveled anthers that did not open. However, all the tassel branches of 8 plants (1 from cross A, 3 from cross B, and 4 from cross C) had plump anthers that shed pollen. Examination of their pollen samples with a single lens field microscope confirmed that they were partially fertile and had some large pollen similar in size to that from tetraploid plants. Tassel samples from these plants were preserved in alcohol for examination in the laboratory. Also these plants were selfed and their pollen was used to pollinate as many plants from the same cross as possible. Unfortunately it was very late in the season and seed set occurred only on one selfed plant (38 seeds, all plump and well formed).

Pollen from two separate plants from each of crosses B and C and a duplicate sample from the one plant from cross A was stained with a solution of iodine plus potassium iodide. Pollen was classified and counted under a light microscope, using the procedure described elsewhere in this newsletter. The following table lists the number of pollen grains and the percentage in each class. In one F1 tassel from cross B there were a few sterile spikelets. All anthers of these spikelets were shriveled and highly sterile like those in sterile tassels.

F ₁ from		Large pollen filled with starch	Medium size pollen filled with starch	Small size pollen filled with starch	Large size pollen partially filled	Medium size pollen partially filled	Small size pollen partially filled	Empty %
		"a"	"b"	"c"	"d"	"e"	"f"	
Cross A (A3-2-4-9-10 x A1-5-6-7-8) 1 plant	Sample 1	661	-	160	-	-	410	2900
	Sample 2	500	-	194	-	-	464	2515
	%	14.9	-	4.5	-	-	11.2	69.4
Cross B (A6-3-2-4-8 x A5-7-1-9-10)	Plant #1	747	301	26	378	416	272	928
	%	24.3	9.8	0.8	12.3	13.6	8.9	30.2
	Plant #2	1198	158	21	322	349	253	846
	%	30.0	5.0	0.8	10.2	11.1	8.00	26.9
Cross C (A5-7-1-9-10-8 x A6-3-2-1-8)	Plant #1	1174	-	146	-	-	353	483
	%	54.5	-	6.8	-	-	16.4	22.4
	Plant #2	1023	-	135	-	-	351	541
	%	49.9	-	6.6	-	-	17.1	26.4

As shown in the table, all 8 plants had pollen similar in size to that of tetraploids. The percentages varied somewhat for different plants from the same cross. The percentage of these grains (class a) was higher in both plants from cross B than that from cross A. F1 plants from cross C had a higher percentage than those of crosses A and B. Some of the pollen in classes b and c may be functional. Some of these are possibly 1n pollen. These may have 10 normal chromosomes, one or both parental multiple interchanges. Further studies will be undertaken to ascertain the nature of the observed changes in fertility. More F1 seeds from the three crosses will be treated and planted in the field early in the coming season to obtain more of these partially fertile plants and to obtain F2 seeds from all three crosses. Pollen from fertile plants will be applied to tetraploid plants as well as to the parental homozygous multiple interchange stocks and to the sterile sibs.

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ys2 a new mutant in the short arm of chromosome 1

A new mutant with yellow striped leaves appeared in the progeny of a colchicine treated plant of inbred B73. For this mutant, whose phenotype recalls iron deficiency similar to ys1 (chromosome 5, The Mutants of Maize, Neuffer et al., 1968), we propose the symbol ys2.

This yellow striped mutant is generally expressed from the third leaf up to maturity. The yellow stripes develop first in the proximal half of the leaf blade, extending later toward the tip. Environmental conditions have been observed to influence the expressivity of ys2. Because of their slower growing habit these mutants reach anthesis 6 to 7 days later than normal sibs, but the plants are fully fertile.

The analysis of segregating progenies of selfed plants indicated for ys2 a recessive Mendelian inheritance (Table 1). The shortage of homozygous types may be due to reduced embryo vitality as suggested by an inverse correlation between chi-square values and germination rates.

Table 1. Germination percentages, phenotypic frequencies and chi-square tests for ys2 segregating progenies of selfed plants

Progeny	Germination %	Observed		3:1 Expected		Total	χ^2	P value
		Ys2	ys2	Ys2	ys2			
1	86	65	21	64.50	21.50	86	.02	.90-.80
2	84	62	22	63.00	21.00	84	.06	.80-.70
3	66	80	19	74.25	24.75	99	1.78	.20-.10
4	77.3	45	13	43.50	14.50	58	.21	.70-.60
5	83	65	18	62.25	20.75	83	.48	.50-.40
6	56.6	72	13	63.75	21.25	85	4.27	.05-.02
Total	73.3	389	106	371.25	123.75	495	3.39	.10-.05

Table 2. The results of crosses between ys2 plants, used as female parents, and a set of TB-A translocations

TB-A	Ys2	ys2	Total no. of plants scored
TB-1Sb*	41	3	44
TB-1La	93		93
TB-2L"4463"	91		91
TB-3La	95		95
TB-4Sa	209		209
TB-5La	179		179
TB-6Lb	50		50
TB-7Lb	70		70
TB-9Sb	83		83
TB-9Lc	89		89

* In all cases homozygous ys2 plants were used except for a heterozygous plant ys2/* plant employed in the cross with TB-1Sb

Crosses of ys2 plants as female parents were made to a set of TB-A translocations. The results indicated that ys2 is uncovered by TB-1Sb (Table 2). Therefore this mutant appears to be located in the short arm of chromosome 1.

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Localization of Esterase-8 to the short arm of chromosome 3

Linkage studies provided preliminary evidence for the presence of a major anodal esterase locus on chromosome 3 (Goodman et al., Genetics 96:697, 1980). The E8 gene, originally defined by MacDonald and Brewbaker (J. Hered. 65:37, 1974) using null alleles, encodes a highly active, ubiquitous anodal esterase. Several electrophoretic mobility classes for E8 have now been described, including naturally-occurring (Ferl, pers. comm.; Goodman and Stuber, 35th Ann. Corn and Sorghum Res. Conf.) and EMS-induced variants (Birchler, Genetics 92:1211, 1979). Heterozygotes for two different electrophoretic forms produce a three-banded pattern in sporophytic tissues, indicating that E8 is active as a dimer. Pollen from heterozygous plants exhibits only the two homodimer bands, suggesting that the E8 present in pollen is synthesized post-meiotically.

In order to confirm the localization of E8 to chromosome 3, plants carrying the extremely slow-migrating, EMS-induced E8-S allele (Birchler, 1979) were pollinated by TB-3Sb (kindly provided by J. Beckett). Immature kernels were harvested 20 days post-pollination and embryonic (scutellar) and endosperm tissues were separately tested for E8. Of 5 tested individuals with the slow allele, one was euploid; i.e., the endosperm and embryo each carried both the paternal fast and the maternal slow forms. In the embryo, a 1:2:1 FF:FS:SS band ratio was observed, while in the triploid endosperm, the banding pattern was skewed towards the SS form, due to the fact that the maternally-derived S allele is represented twice. Three kernels had only the E8-S allele present in the endosperm and an approximate 4:4:1, FF:FS:SS band ratio in the corresponding embryos. These kernels show uncovering of the maternal form in the endosperm and the complementary duplicated condition in the embryo. One kernel exhibited the reverse of this phenotype: the maternal slow allele was uncovered in the embryo and a 1:2:1 band ratio was observed in the correspondingly hyperploid (S/S/F/F) endosperm.

When coleoptile tissue from a similar cross was examined, five tested seedlings carried the slow allele. One was hypoploid, exhibiting only the maternal, slow allozyme. Another was hyperploid as evidenced by an approximate 4:4:1, FF:FS:SS banding pattern. The remaining three were apparent euploids, with equal paternal and maternal E8 contributions. The hypoploid seedling could not be explained by self-contamination, since this individual was heterozygous for maternal and paternal alleles of Glu (chr. 10), Phi (chr. 1), Pgm1 (chr. 1), and Mdh2 (chr. 6). In the exact reciprocal cross (using the TB-3Sb plant as a female), no cases of uncovering of the E8-S allele were found.

Collectively, these data place the E8 locus in the region of 3S included in TB-3Sb and confirm the linkage of this gene to other chromosome 3 genes. Since E8 is easy to work with and the dosage of 3S can be readily discerned by its banding ratios in heterozygotes, it should serve as a useful marker for dosage studies involving the short arm of chromosome 3.

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Genetic analysis of callus growth

The genotype differences in callus induction, growth and morphogenetic potential indicate genetic determination of these characters. Therefore, we analyzed the genetic determination of somatic callogenesis in a diallel cross and studied genetic correlations between characters in vitro and in intact plants. In the diallel cross 6 inbreds with different growth rates of callus were included. Kernels of the F1 generation from the diallel cross were used both for induction of callus culture from mature embryos and for field trials.

A different degree of heterosis (in comparison with population mean, 52.7 - 198.65%) was found in growth rate of callus culture of F1 hybrids. Statistically significant differences were observed between hybrids from direct and reciprocal crossing. The highest degree of genetic variability was due to GCA, and the effect of reciprocals was also important:

<u>Source</u>	<u>df</u>	<u>Mean square</u>	<u>F</u>
GCA	5	1.1778	19.62**
SCA	15	0.8302	6.00**
Reciprocals	15	1.2726	7.8**
Residual	432	74.9167	

The correlation analysis on the level of inbreds showed strongly negative dependence between the callus weight and kernel yield ($r = -0.811^{**}$) and ear localization on the plant ($r = -0.604^{**}$). Positive correlation was found between the callus weight and the leaf area ($r = 0.729^{**}$). The correlation relationships on the hybrid level were impaired both by a different degree of heterosis effect on the in vitro characters and the characters of intact plant and by reciprocal differences occurring only in the callus weight.

The results of the diallel cross and correlation analysis indicate that the growth of callus is under genetic control of two genetic systems; one is localized in the nucleus and the other in cytoplasm.

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Influence of genetic background on callus culture

Working with 60 inbreds for callus induction from mature embryos, callus growth rate and morphogenetic response, large genotype differences were found. In the study normal inbreds and their mutant analogs were used. The analogs included endosperm mutants *o2*, *sh2*, *su1*, *su2*, *f11*, *f12*, *wx*, *du*, *ae* and morphogenic mutants *br2* and *rd*. Proliferation of callus from scutellum, mesocotyl and root was found. The origin of callus strongly depended on the genotype. The callus induction and growth rate depended more on genetic background than on the mutant. The decreased growth potential of mutant analogs was not manifested in callus culture, although it was found in embryo culture (unpublished results). The differences in callus growth rate were not even found in mutant analogs *br2* and *rd*, which reduce the height of the intact plant.

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Variability of aleurone thickness in single and multilayer aleurone types

As reported earlier (Nelson and Chang, 1974, Crop Sci. 14:374) the population "Coroico" with multilayer aleurone could be used as a genetic source in breeding for high content of essential amino acids. After crossing of this source with

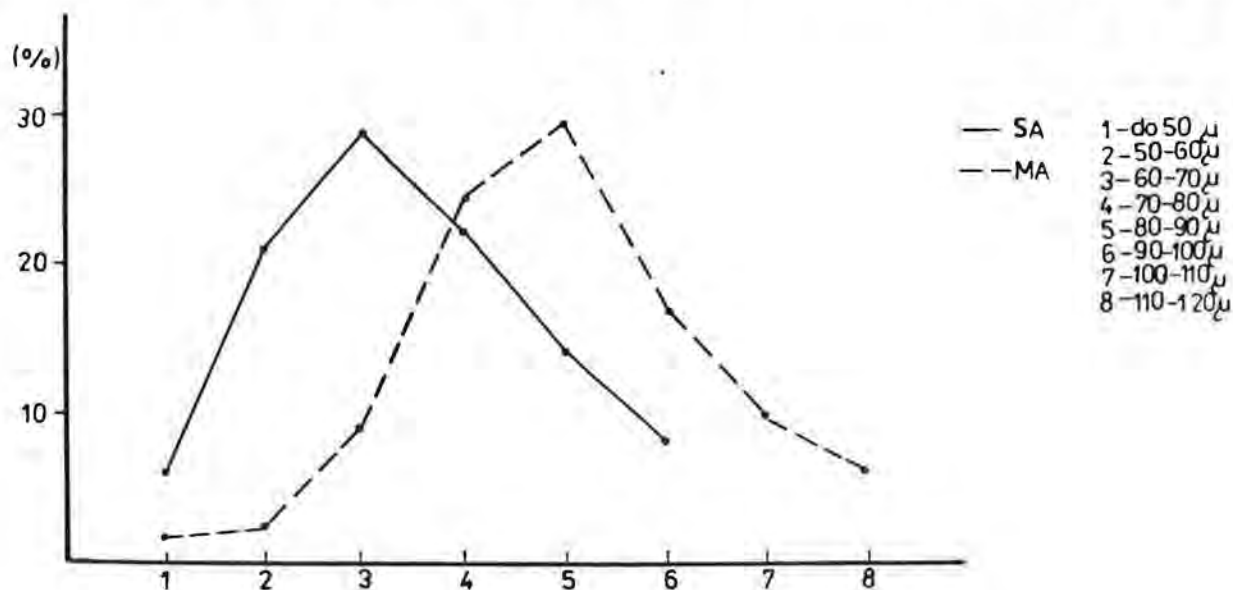


Fig. 1. Variability in aleurone thickness.

three opaque-2 inbreds the variability of the aleurone thickness in opaque-2 kernels with multilayer aleurone and with single aleurone was studied (Fig. 1).

Opaque-2 kernels with single aleurone had variability in the range 50-100 u, with the highest frequency (28.4%) in the class 60-70 u. The variability curve in opaque-2 kernels with multilayer aleurone showed a similar course, with the highest frequency (28.87%) in the class 80-90 u. The range of the curve varied from 50 to 120 u. Moreover, within the range of single aleurone variability are found 36% of kernels with multilayer aleurone, and the range of the multilayer aleurone variability contains 45% of single aleurone kernels.

Although there is a distinct difference in the number of aleurone layers between single and multilayer aleurone types, these facts indicate that differences between these two aleurone types in the thickness of aleurone are not so evident. No differences in lysine content were found either between the aleurone types or between the class range alone on a whole kernel basis.

M. Neštický and A. Piovarčí

Genetic expression of brevis-2 mutant in comparison with its normal counterpart

In diallel crossing of 5 bv2 inbreds (BIS6) and their normal counterparts, 10 bv2 hybrids and their normal counterparts were released. Results from their comparison and studies of other bv2 hybrids give some results useful for utilization of the bv2 gene in breeding programs.

Plant height of bv2 hybrids, 140-150 cm, corresponds to the optimal level of this trait for a short-stem modified plant type. In comparison with their normal counterparts the plant height was reduced by 30-50%. The growth curve showed that most expressive growth reduction was manifested between tasseling and flowering period. Contrary to normal hybrids the vegetative growth of bv2 hybrids ceased at the beginning of flowering. It seems to be favorable for ear shoot growth and development. The vegetation period of bv2 hybrids was shorter by about 8 days on average and grain moisture at harvest time 2-6% lower (3.5% average). The date of flowering of normal and bv2 hybrids was identical.

In 1979 the grain productivity of bv2 hybrids was equal to normal hybrids, and in 1980 normal hybrids significantly outyielded the bv2 counterparts (16% in average). In only a few cases, the bv2 hybrids yielded better. 1000-kernel weight of bv2 hybrids decreased by about 28%. Stalk breakage was not present in bv2 hybrids from the diallel cross.

Combining ability analysis showed that both general (GCA) and specific (SCA) combining abilities were important in conditioning of observed characters (grain yield and its components, plant height). From the GCA standpoint bv2 inbreds should be considered as totally new inbreds. The results supported use of bv2 mutant for modification of plant height.

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Induction of forward mutation in somatic cells of maize after acute or chronic exposure to ethylmethanesulfonate

Forward mutation at the yellow-green-2 (yg2) locus of Zea mays has been used as the endpoint in various studies on mutagenesis (B. V. Conger and J. V. Carabia, 1977, 46:285-296; T. Fujii, 1980, Japan. J. Genetics 55:241-245; T. Fujii, 1981, Environ. Exper. Bot. 21:127-131). We are interested in investigating the kinetics

of mutation induction in somatic cells after the test organism has been exposed to a chemical mutagen under acute or chronic treatment regimens.

Maize kernels heterozygous at the *yg2* locus were used in all experiments. The kernels were surface sterilized by soaking for 5 min in a 0.5% sodium hypochlorite solution and rinsing 10 min in running tap water. In the acute tests 10 to 25 kernels per treatment group were soaked for 72 hr in aerated distilled water at 20 C. The kernels were treated for 8 hr at 20 C in aerated solutions of ethylmethanesulfonate (EMS). The concentrations of EMS tested were: 0 (control), 1mM, 10 mM and 20 mM. Following treatment the kernels were rinsed in running tap water for 16 hr. Three kernels each were planted in soil in 10 cm diameter plastic pots. The pots were placed in a growth chamber at 20 C with a 17 hr photoperiod ($300 \text{ uE/m}^{-2}\text{sec}^{-1}$ PRR) for 20 to 25 days. The fourth and fifth leaves were scored for the presence of *yg2* sectors. Only yellow-green colored sectors with a minimum length of 1 mm were scored. A fluorescent light box and magnifying lens were used as aids in scoring.

In the chronic treatment regimen, the kernels were surface sterilized and soaked for 72 hr in distilled water as in the acute treatments. The kernels were planted in 8.5 cm diameter paper cups filled with sterile vermiculite. Each container received 150 ml of Hoagland's solution or a known concentration of EMS in Hoagland's solution. The concentrations tested were: 0 (control), 1 μM , 10 μM , 100 μM and 1 mM EMS with 10 to 25 kernels per treatment group. When the seedlings reached 3 to 5 cm in height they were transferred to foil-covered 1 liter glass jars filled with 800 ml of the appropriate control or treatment Hoagland's solution. Three to four plants were grown in each jar in a plant growth chamber at 20 C with a 17 h photoperiod ($300 \text{ uE/m}^{-2}\text{sec}^{-1}$ PRR). Water-saturated air was continuously bubbled within each jar. The jars were replenished with the control or treatment solutions during the course of the experiment and the amounts of solution added were recorded. The fourth and fifth leaves were scored for the presence of *yg2* sectors.

The results of the acute treatment experiments are presented in Table 1. Although at concentrations of EMS of 1 mM there is a slight increase in the mean number of yellow-green sectors in both leaf four and leaf five, the increase is not significant. At 10 mM EMS the increase in the mean number of sectors is apparent when compared to the control values. We analyzed the summary data for leaf four and five by linear regression and calculated the coefficient of determination (r^2). For the leaf four data within the range of 0 to 20 mM EMS the r^2

Table 1. Forward Mutation at the *yg2* Locus After Acute EMS Treatment

Experiment Number	EMS Conc. (mM)	No. Leaf 4 Scored	$\bar{X} \pm \text{SE}$	No. Leaf 5 Scored	$\bar{X} \pm \text{SE}$
4165	0	10	0.50 ± 0.22	10	0.30 ± 0.21
4165	1	10	0.70 ± 0.21	10	0.30 ± 0.15
4165	10	10	8.40 ± 1.09	10	3.70 ± 0.45
4165	20	9	16.44 ± 1.56	9	9.56 ± 0.58
4611	0	22	0.36 ± 0.14	22	0.18 ± 0.08
4611	1	20	0.40 ± 0.11	20	0.25 ± 0.10
4611	10	21	4.52 ± 0.37	21	1.14 ± 0.26
4611	20	21	11.38 ± 0.79	21	1.17 ± 0.28
SUMMARY					
	0	32	0.41 ± 0.12	32	0.22 ± 0.09
	1	30	0.50 ± 0.10	30	0.27 ± 0.08
	10	31	5.77 ± 0.54	31	1.97 ± 0.31
	20	30	12.90 ± 0.83	30	3.93 ± 0.77

was 0.99. For the leaf five data within the range of 0 to 20 mM EMS the r^2 was 0.99. Thus the increase in yellow-green sectors in both leaf four and leaf five is dose dependent and linear.

Table 2. Forward Mutation at the yg-2 Locus After Chronic EMS Treatment

Experiment Number	EMS Conc. (μ M)	No. Leaf 4 Scored	$\bar{X} \pm SE$	No. Leaf 5 Scored	$\bar{X} \pm SE$
4172	0	12	0 \pm 0	12	0.17 \pm 0.11
4172	1000	5	0 \pm 0	5	1.40 \pm 0.51
4175	0	10	0 \pm 0	10	0.20 \pm 0.13
4175	1000	11	0.91 \pm 0.21	11	0.91 \pm 0.28
4181	0	9	0 \pm 0	9	0.22 \pm 0.15
4181	1	10	0.20 \pm 0.13	10	0 \pm 0
4181	10	10	0.30 \pm 0.21	10	0.10 \pm 0.10
4181	100	10	0.90 \pm 0.31	10	0.40 \pm 0.16
4186	0	9	0.22 \pm 0.15	9	0 \pm 0
4186	1	8	0.13 \pm 0.13	8	0.13 \pm 0.13
4186	10	9	0.33 \pm 0.24	8	0.38 \pm 0.18
4186	100	9	0.67 \pm 0.24	9	0.56 \pm 0.24
4608	0	15	0.13 \pm 0.09	15	0 \pm 0
4608	1	15	0.27 \pm 0.12	15	0.07 \pm 0.07
4608	10	15	0.47 \pm 0.16	15	0.13 \pm 0.09
4608	100	15	1.07 \pm 0.25	15	0.40 \pm 0.13
SUMMARY					
	0	55	0.07 \pm 0.03	55	0.11 \pm 0.04
	1	33	0.21 \pm 0.07	33	0.06 \pm 0.04
	10	34	0.32 \pm 0.10	33	0.18 \pm 0.07
	100	34	0.91 \pm 0.15	34	0.44 \pm 0.09
	1000	16	0.62 \pm 0.18	16	1.06 \pm 0.25

The results of the chronic treatment experiments are presented in Table 2. From the summary data an increased number of sectors over control was induced in leaf four with an EMS concentration of 1 μ M. Within the range of 0 to 100 μ M EMS the frequency of mutant sectors in leaf four increased in a dose dependent and linear manner ($r^2 = 0.96$). However, a reduction in the number of sectors was observed when a concentration of 1 mM EMS was administered to the plants. For leaf five, a dose dependent and linear response ($r^2 = 0.93$) was induced by EMS in the concentration range of 0 to 1 mM. However, the first discernible increase in the mean number of sectors required a concentration of 10 μ M EMS.

The comparison of acute versus chronic treatments of EMS and the corresponding genetic effects indicate that both treatment regimens induce mutant yg2 sectors with dose dependent and linear kinetics. The data indicate that the plants are more sensitive to a lower molar concentration of EMS in the chronic treatment regimen versus the acute treatment regimen. However, this comparison does not account for the total moles of EMS that the plants were exposed to under the two treatment regimens. A dose of a mutagen should be expressed in terms of μ Moles or mMoles per kilogram of tissue. Also the rate of degradation of the mutagen must be considered when computing dosage. Finally the time of exposure should be incorporated into an assessment of dose. We are presently trying to determine these parameters and hope to use them in comparing the kinetics of mutation induction after acute versus chronic treatment conditions. (This research was funded, in part, by NIEHS Grant No. ES01895 Gen.).

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Flavonoids from pollen of *Zea mays* relatives

There are seven flavonoids of maize pollen, and they occur in approximately the following relative proportions:

<u>Compound</u>	<u>Relative proportion</u>
P1 = Quercetin 3-diglycoside	2
P2 = Quercetin 3,3'-diglycoside	4
P3 = Isorhamnetin 3,4'-diglycoside	1
P4 = Quercetin 3-diglycoside	1
P5 = Isorhamnetin 3-diglycoside	0.6
P6 = Quercetin 3,7,3'-triglycoside	trace
Q1 = Quercetin 3-glucoside	1
P1 to P6 are pollen specific.	
Q1 occurs in other tissues also	

Samples of pollen from three relatives of maize have been analyzed for a comparison of their flavonoid patterns with that found in maize; the results are tentative because of the small number of plants sampled, but they show the following:

Euchlena mexicana: The flavonoid pattern is similar in all respects to that of Zea mays. All the same compounds are present, and they occur in approximately the same relative proportions.

Tripsacum dactyloides: The compounds are the same as those in Zea mays, but there are differences in relative proportions. Q1 and P2 are the major compounds; the other glycosides occur in trace amounts only.

Coix lachryma-jobi: The flavonoid pattern is similar to that of Zea mays except that P4 and P5 are present in greater relative proportions.

O. Ceska

Unstable pigmenting factors

A few years ago Dr. Charles Burnham sent us some seed from plants that were sectoring for a dark orange pigment in the husk, cob, and pericarp. In a covering letter he happened to mention also that his a₃ stocks were 'quite variable.'

The orange pigment seems similar if not identical to the P-controlled cob and pericarp pigments that we call 'phlobaphenes.' The pigment can be produced in most plant parts, including sheaths, tassels, glumes, etc., but plants that are heavily pigmented are definitely unhappy. Some families derived from crosses segregate albino, dark purple, and orange-purple seedlings that die at an early stage. At least one family segregates for P-WR, P-WW and also the phlobaphene factor, so it is probably independent of the P locus, although P locus activity may be required for its expression. The homozygote may be lethal, at least in some stocks. The best and most uniform expression is in plants heterozygous for the phlobaphene factor, and also heterozygous Sm/sm. These plants are uniformly orange when mature, with orange anthers, tassels, stems, ligules, leaves and silks.

We obtained our a₃ seed from Dr. Ed Coe, and like Dr. Burnham, we have found 'variability.' 'Instability' would definitely be a better word to describe the changes we are recording. The variability seems not to derive directly from the a₃ locus, however, after several years of testing and retesting, the following patterns of change seem to be emerging:

The seeds that Dr. Coe provided us were from self-pollinated a3 R-g B-b/ K55 A3 r-g b plants. Some 40 different a3 r-g lines have been obtained from these seeds, and for four generations we have never recorded any unexpected plant types, either from crosses to other stocks, or from self-pollinations. In contrast, lines derived from a3 R-g/- plants not infrequently show anthocyanin sectors of several types, the most common being in the leaf blades. We now have 12 lines, of independent origin in the sense of not being closely related, that have a finely sectoried, almost diffuse leaf blade pigment, the factor responsible being inherited together with the R-g allele. The leaf blade pigment does not require a3 or B-b for its expression.

Another type of sectoring occurs in R-g/- Pl/- plants. The pericarps show occasional 'cherry'-type sectors, and in one such ear the sector included a complete kernel. The plant grown from this one seed was uniformly purple in all tissues except anthers and silks. Progeny from this plant have ranged from uniform purple, to purple with fine green sectors, to green with fine purple sectors. Again the factor responsible for the pigmentation is inherited with the R-g allele.

A third type of change is associated with the anthers. Anthers become pigmented in some R-g/- plants. This is a progressive type of change, starting sometimes as a pale 'sun-red' type of pigment and sometimes as fine longitudinal sectors on the anthers only. Seed from these plants give rise to plants with a range of anther colors, from green to full red. The full red anthered plants can in turn have progeny with anthers that range from full red to a deep mahogany, and if a plant color factor is present, the plant pigment ranges into the mahogany also.

One a3 R-g/r-g line, derived from self-pollinating an a3 R-g/W22 a r-g plant, gave a very unusual spectrum of phenotypes. Some plants were uniformly purple, some were green, and some were initially green, and remained green in the lower stem and leaves, but became progressively more purple in the upper parts of the plant so that the tassel and anthers were fully purple. On further analysis we found that the R-g allele had associated with it a leaf blade factor, and that the factor responsible for the progressively appearing pigment was inherited with the r allele. As the pigment changes include the anthers, this r allele is now effectively an r-r allele. We have grown three successive generations of plants derived from this family, and the following pattern of change seems to be fairly predictable: Green-anthered 'r-g' plants when selfed give rise to plants with a range of anther colors from green to pale to red, and the associated plant pigments range similarly from green to purple. Some plants change developmentally from green to purple. Progeny from this latter class are all purple. Progeny from purple plants range from purple to dark mahogany with some plants changing developmentally from purple to mahogany. Mahogany plants have only mahogany progeny. These pigments seem not to require either a3 or B-b, but Pl will enhance the pigments and will turn the mahogany to a bronze-necrotic. It seems that we are recording a type of change that can be registered both somatically and germinally. Dare we mention the word 'paramutation?'

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Key traits of maize expected by domestication

The key traits that separate the ears of teosinte and maize are: (1) solitary in teosinte vs. paired female spikelets in maize; (2) two-ranked in teosinte vs. many-ranked central spike (ear) in maize; (3) shattering in teosinte vs. non-shattering rachis (cob) in maize.

It is significant that all of these key traits make the maize ear more adapted as a food source for man and at the same time less adapted for survival in the wild. This is not a change in reproductive strategy expected from evolution under natural selection but under domestication.

The close cytological homology of teosinte and maize indicates a parent to progeny relationship, and evolutionary trends leading to formation of the cupulate fruit case indicate that teosinte is the parent. The simple genetic changes under domestication would be: (1) a reactivation of the pedicellate female spikelet; (2) a proliferation to many ranks of spikelets; and (3) a reduction of abscission layers between rachis segments.

Walton C. Galinat

Perennialism: Dominant or recessive?

The classification of segregants from perennial teosinte x maize hybrids for the perennial trait is difficult. Plants which are scored as annual because they appear to be dead may rest in a dormant state for a month or more and then suddenly a basal or underground shoot will emerge showing that they should have been scored as perennial. Under inadequate light intensity and stunting, this second growth will be abnormal and result in only female spikes as is also the case for stunted corn.

On a basis of scoring for evergreen stalks and its apparent association with basal shoot development, Galinat (1980) thought that perennialism is at least partially controlled by two dominant complementary genes giving a 9:7 F₂ segregation ratio. Meanwhile, scoring of an F₂ segregation by Mangelsdorf et al. (1981) indicates that trait is recessive.

One important factor that differs in the various studies of perennialism is the corn background. The corn background could be responsible for a reversal of dominance in expression of perennialism.

Walton C. Galinat

Vegetative vs. floral shoot proliferation and perennialism

Perennial shoots and multi-ranked spikelets tend to be mutually exclusive as if they were a regulatory choice between early and late proliferation plus apical dominance. That is, strong perennialism (vegetative proliferation) appears to exclude a large many-rowed ear (floral proliferation). Weak and unstable proliferation may co-exist in both vegetative and floral regions. The suggestion is that the effect of the corn background may account in part for the different results on studies of the inheritance of perennialism. If perennialism is mutually exclusive with agronomic ear development, attempts to breed a perennial grain corn would be futile.

Walton C. Galinat

Pollination technique for maintaining Vg Vg inbreds

It has been four years since seed of MA500 was distributed to the NSCBA group. Recently several breeders have had to ask for replacement supplies because they were unable to maintain MA500, which is homozygous Vg Vg, by hand pollination.

The technique for successful pollination of Vg Vg inbreds is different from that for normal inbreds. For best pollen production, the Vg tassels should not be pre-bagged up the day before but, rather, just shake the pollen out directly into the tassel bag at the actual time of pollination. If properly done, perhaps by one person not immediately working on other corn lines, little or no contamination results.

There has been a recent revival of interest in glumeless sweet corn now with the discovery that the high sugar content of sugary enhancer lines tends to just wash out and away from cut corn. Because the vestigial glume trait facilitates intact kernel removal, it is useful in reducing sugar loss from cut corn of the new high sugar endosperm types.

Walton C. Galinat

Reconstruction of the missing links between teosinte and maize

The key traits of maize, namely a many-ranked (decussate) spike and paired female spikelets, were individually transferred to teosinte in order to examine the hypotheses of a common ancestor between a wild maize and teosinte or that of intermediate steps in the presumed domestic transformation of teosinte into maize.

A joining of the fruit cases of teosinte into yokes with a decussate arrangement is non-adaptive as a wild trait because it reduces the number of dispersal units by half. It must, therefore, represent a domestic trait. When the paired condition of female spikelets in maize is transferred to teosinte, the effectiveness of the fruit case as a protective device is destroyed.

The conclusion is that, when the key traits of maize are individually transferred to teosinte, the product is no more adaptable as a wild type than that of the eight-rowed ear of the oldest maize. Furthermore, these modified teosintes are like maize in not fitting into a logical early position in the sequential steps leading toward the cupulate fruit case. They must represent transitional forms in corn's origin by domestication.

Walton C. Galinat

The ae (amylose extender) gene is a modifier of agronomic value for sugary shrunken sweet corn

The ae gene is useful in making the su sh2 double mutant fully viable with the phenotype being similar to the Su sh2 single mutant. Because the ae gene is recessive, it is only expressed in about 25% of the kernels from high sugar hybrids with Ae su sh2:Tr7 (2) where Tr7 is an extra chromosome from Tripsacum carrying the Su allele, as covered in Research Corp.--U. Mass. patent #4,051,629.

Walton C. Galinat

The cupule and spatial accommodation of different kernel types

The surface area of the dry cob is reduced by shrinkage in relation to the size and induration of its cupules. The amount of cob shrinkage determines its capacity to carry various kernel types. If the mature dry kernels on the cob become either too tightly packed or too loosely spaced out, many of them will shatter during handling.

A rigid framework of large indurated cupules is coadaptive with broad flinty, floury or flinty-dent kernels because such cupules maintain adequate surface on the cob to accommodate the non-contracting kernels. In contrast, reduced or soft cupules are adaptive when cob shrinkage must coincide with kernel shrinkage in order to prevent shattering as follows: (1) sugary or other defective seed types that can wrinkle in concordance with rachis contraction; (2) long or indeterminate kernels such as occur in Shoepeg (Pepetilla) and certain pointed popcorns such as Confitte Morocho that have spaced-out kernels that may become thin without wrinkling.

The type of kernel shattering referred to here is from the dead absorbing cells or so-called "black-eye" that occurs in most grasses and is not a normal means of seed dispersal via abscission layers.

Walton C. Galinat

The cupule, a secondary female trait of teosinte and maize, as evidence that the maize ear is derived from the teosinte ear

The cupule is a secondary sex trait of the female spike in both teosinte and the oldest known archaeological maize as well as in most stocks of modern maize. While the non-cupulate cob or reduced-cupulate cob now occurs in certain maize strains, it has evolved only in coadaptive association with certain derived kernel types. Paired female spikelets, when transferred from maize to teosinte, flatten and reduce cupule development similar to the cupules of the oldest known maize.

When sex reversal suddenly occurs during the ontogeny of growing plants of teosinte, maize and many other species, the expression of secondary sex traits changes accordingly. The synchronization is thus developmental with a somatic basis. The primary and secondary sex traits may have different genetic bases. The genes for secondary male traits also are distinct from those for secondary female traits. A change in the primary sex of the teosinte tassel could not fail to activate genes for the secondary female trait of a cupulate fruit case. Genes separate from those controlling the primary sex are known to produce secondary female traits of paired spikelets, long soft glumes, a narrow flexible rachis and reduced cupules. A change from the cupulate fruit case to the above as female secondary sex traits in teosinte would evolve by domestication from changes at their controlling loci, not that for the primary sex. Primary sex genes such as "anther ear" and "tassel seed" activate changes in the expression of the secondary sex genes accordingly in a form of controller to structural gene relationship. The common presence of the cupule in the female rachis of both teosinte and maize is evidence that the maize ear is derived from the teosinte ear, not the tassel.

Walton C. Galinat

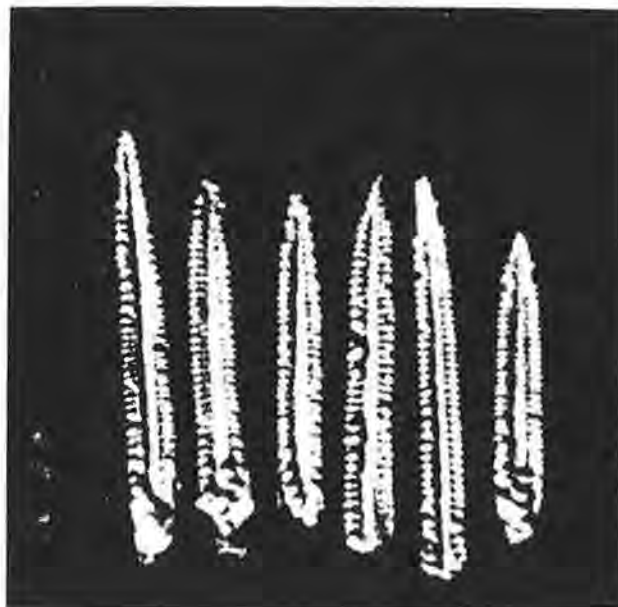
WILLIAMS, INDIANA
Williams Laboratories

Maize ear and floret response to dinitroaniline herbicides

My practice of isolating pet strains of maize in soybean fields has been complicated in recent years by a switch to preplant soil-incorporated herbicides. Some maize plots invariably end up at least partially on soil treated with dinitroaniline herbicides ("Treflan" or "Tolban") applied at the double rate recommended to suppress rhizome Johnsongrass in soybeans. The usual outcome of this herbicide incompatibility is no more than a slight reduction in maize plant vigor and seed yield.

However, three strain specific dinitroaniline herbicide responses in maize ear or floret structure have thus been brought to my attention. These are:

- 1) a marked increase in proportion of "two-squared" ears (see W. Galinat, MNL 52:60) among ears of an eight-rowed strain (see Figure),
- 2) a marked reduction in ear and tassel branching in an ra strain, and
- 3) a marked inhibition of fertile pistil production in a Pt-like, Bf strain.



All have been confirmed as herbicide dependent by split plantings of single ear populations on herbicide treated vs. untreated soil.

Responses similar to (1) and (2) can be generated by unfavorable growing conditions including drought, leaf pruning, or growth in pots. On the other hand, these same conditions tend to reduce the severity of Pt action rather than accentuate it as per (3) (see A. Williams, MNL 54:123). Specific roles for dinitroanilines in eliciting these responses will therefore be difficult to demonstrate. I plan to see what effects more delayed and direct application of "Treflan" have on these strains of maize.

Absalom F. Williams

WOOSTER, OHIO

Ohio Agric. Res. and Devel. Center and U.S. Dept. of Agriculture

Inheritance of maize chlorotic dwarf virus resistance in maize x Zea diploperennis backcrosses

Of the many hundreds of maize lines tested for resistance to maize chlorotic dwarf virus (MCDV), all have shown at least some plants with the vein clearing symptom associated with susceptibility to MCDV. Zea diploperennis was found to be resistant to MCDV by L. R. Nault, D. T. Gordon, V. D. Damsteegt and H. H. Iltis (Plant Disease 66, in press). Crosses of Z. diploperennis as the pollen parent and maize were made to transfer resistance to the latter. F1 x maize seedlings

were exposed to leafhopper, Graminella nigrifrons viruliferous with MCDV. Following a virus acquisition access period (AAP) of 48 hours, leafhoppers were allowed to feed on test seedlings for 48 hours. After 14 days seedlings were examined for vein clearing symptoms. Leaf samples were taken from symptomless plants to test for virus by enzyme linked immunosorbent assay (ELISA). ELISA is generally more sensitive and accurate than symptoms in tests for virus. Of the 107 BC1 plants, 22 appeared resistant to MCDV. This is not a significant deviation from a 3:1 ratio ($\chi^2 = 1.125$, $P = 0.30-0.20$).

Plants with negative reactions in ELISA were transplanted to the greenhouse and used to pollinate other maize plants or selfed. The second generation backcross (BC2) or selfed seedlings were exposed to viruliferous leafhoppers. Symptomless seedlings were assayed by ELISA and those that reacted negatively for MCDV were transplanted to the 1981 field or greenhouse-sunyard. The transplants were again inoculated with MCDV as older plants (some were tasseling) to insure that these plant populations did not include "virus escapes." Leafhoppers were caged in groups of approximately 10 on the youngest leaf for a period of 48 hours following a 48-hour AAP. After 14-21 days plants were again tested for MCDV by ELISA. The following results were recorded:

Generation	No. Plants		χ^2 3:1	χ^2 9:7	P
	Susceptible	Resistant			
BC2	278	100	.428		0.70-0.50
BC1-selfed	24	30		.011	0.90

These results indicate that resistance to MCDV in Zea diploperennis is controlled by two dominant complementary genes.

W. R. Findley, L. R. Nault, W. E. Styer and D. T. Gordon

YATABE, TSUKUBA, JAPAN
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In vitro germination of pollen grains from F1 plants in maize

In a series of mixed pollination experiments in maize (K. Murakami et al., 1972; M. Yamada and K. Murakami, 1978) the existence of the superiority of pollen grains from F1 plants in selective fertilization could be confirmed. It is assumed that the superiority must be corroborated by in vivo or in vitro germination experiments on pollen grains from inbred lines compared with those from the F1 plants.

In vitro germination experiments were carried out using three inbred lines (A34, CI64, and Koshu-564) and three F1 hybrids of these lines (A34 x Koshu-564, CI64 x Koshu-564, and A34 x CI64), among which three F1 hybrids had shown the superiority in selective fertilization from mixed pollination (M. Yamada, in press). The Cook and Walden (1965) artificial medium used for the germination experiment consisted of 0.6% bacto-agar containing $\text{CaCl}_2 \cdot 2\text{H}_2\text{O}$ (0.03%), H_3BO_3 (0.01%) and sucrose (0.35 M). Fresh pollen grains of each material were spread over the surface of agar medium in Petri dishes. The inoculated dishes were incubated at 24 C for one, two, and three hours three times to estimate germination. Pollen activity was interrupted by flooding the surface of the medium with a solution prepared according to P. L. Pfahler (1967). The Petri dishes were then stored at 3 C until the observations were made. Pollen germinability was determined on the screen of a Sonic Digitizer with a graph pen, which could record

automatically points, length, etc. on a disk. For observation of pollen grains, projections onto a screen with a 20 cm x 20 cm frame were measured twice in relation to the total number of pollen grains in the frame, number of pollen grains which had germinated and length of the tube.

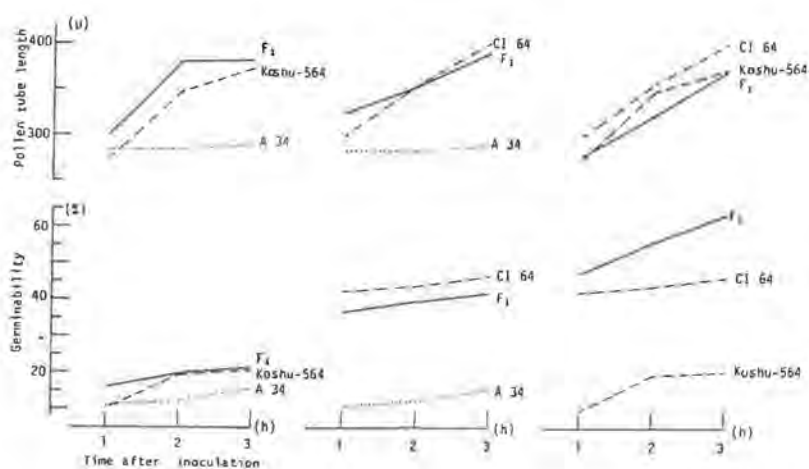


Fig. 1. In vitro germinability of pollen grains derived from inbred lines and their F1 plant in maize as well as growth of pollen tube.

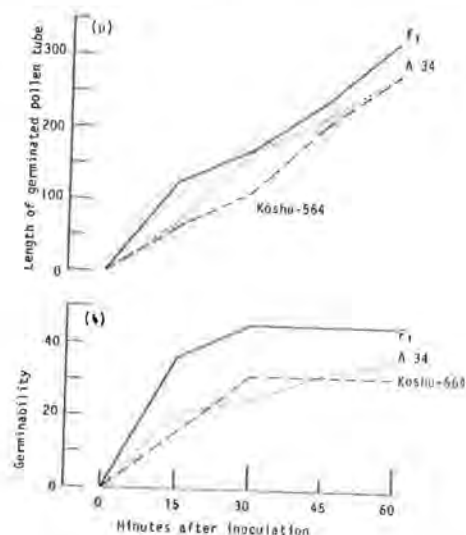


Fig. 2. In vitro germination of pollen grains (1 hour). cf. Fig. 1.

As shown in Fig. 1, pollen grains from F1 plants in each F1 hybrid showed higher germination percentages at an earlier time (A34 x Koshu-564 and CI64 x Koshu-564) and a longer pollen tube (A34 x Koshu-564 and A34 x CI64). It is well known (J. A. Goss, 1968) that germinability of maize pollen is controlled by pollen per se, the pollen tube reaching a maximum length of 500 within 3 hours. On the basis of the results shown in Fig. 1, therefore, an experiment was designed to determine the germinability of each pollen grain on the screen within the first hour after inoculation. As shown in Fig. 2, the increment of percentages of germinated pollen grains occurred within thirty minutes after inoculation. It also appeared that the elongation of pollen tubes in pollen grains from the F1 hybrid was promoted unlike that of the tubes of the inbred lines. This preliminary experiment suggests that the superiority of pollen grains from F1 plants is controlled in the first stage of germination depending upon pollen grains per se which are derived from the F1 heterozygous plant. It is necessary to carry out in vivo experiments on silks after pollination, so as to correlate the results with those obtained from in vitro germination experiments.

Minoru Yamada and Yasunobu Ohkawa

ADDENDUM

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University of North Dakota
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Recent progress in the study of maize developmental mutants

An examination of mature kernels of 17 defective kernel mutants believed to be good candidates for designation as developmental type mutants revealed that 14 of the mutants were of this type, since they were unable to form leaf primordia during kernel development. Nine of the 14 mutants are located to chromosome arm and five arms are represented (1S, 1L, 3L, 4L, and 5L). These developmental mutants are blocked at several different points in the embryonic sequence ranging from the proembryo through the transition to the coleoptilar stage (Abbe and Stein, 1954). Among the 14 mutants 12 appear to be stage specific and two mutants range in expression from the transition to the coleoptilar stage. Since all of these mutants have a defective endosperm, as well as a defective embryo, an examination of embryo-endosperm interaction is warranted to determine whether the defective endosperm is responsible for the defective condition of the embryo or whether it is the genetic constitution of the embryo itself that determines its developmental fate. Among the nine developmental type mutants located to chromosome arm, five are allelic and located on 1S. An examination of one of these mutants on 1S and the four other located mutants was performed using kernels (from the arm locating ear) that contained a genetically normal endosperm and a genetically mutant embryo and kernels that contained a genetically mutant endosperm and a genetically normal embryo. In all five cases examined, the genetically mutant embryos were defective and were not aided in their development by the presence of a normal endosperm. Data are incomplete for the reciprocal arrangement but for two mutants it is evident that a genetically mutant endosperm results in death of a genetically normal embryo after germination at the seedling stage. It appears therefore, to the extent that data are available, that it is the genotype of the embryo that determines its developmental fate, that it cannot be helped by a normal endosperm, but that, in some cases, a defective endosperm can produce seedling death. The results to date indicate that the developmental-type mutants should be valuable in studying the genetic regulation of embryonic development as well as embryo-endosperm interaction.

William F. Sheridan and M. G. Neuffer

The continuing search for maize auxotrophs

During the past growing season we screened several new defective kernel mutants as well as a group of such mutants previously identified as promising mutants in our search for auxotrophs. Two of these mutants showed superior growth on enriched medium with little or no growth on basal medium when immature embryos were cultured as previously reported (Sheridan and Neuffer, 1980, 1981). Both of these mutants (E1054 and E1532) proved (as did the previously studied mutant E1121) to require proline as a sole supplement and to be allelic to the pro-1 mutant of Gavazzi et al. (1975) and located on chromosome arm 8L. After several years of screening, in which 108 mutants have been examined by embryo culturing we have found three proline-requiring mutants in our collection of EMS induced defective kernel mutants (Neuffer and Sheridan, 1980) and all three are allelic to pro-1. None of the other 105 mutants have proven to be auxotrophs. We believe

that our screening procedure works in detecting auxotrophs and that the single-gene, recessive, defective-kernel mutants may not contain auxotrophs (except for the proline requiring mutant). It seems likely that auxotrophs occur and one possible explanation for our general lack of success is that they may require the mutation of two different loci for their expression. We have begun work on the analysis of defective kernel mutants displaying a 15:1 segregation ratio on self-pollinated segregating ears.

William F. Sheridan and M. G. Neuffer

III. ZEALAND 1982

This list is based on new data reported in the current list of Recent Maize Publications (author, year) and in the Reports from Cooperators in this issue (author, 56:page). Recombination percentages and notes show genetic locations; new alleles and new loci are also listed. I accept responsibility for any errors and misses, and await your enthusiastic corrections and new observations.

Chromosome 1

- Adh1 - 11.7 - Phil - 4.5 - Gdh1 - 44.1 - Cat3 Goodman &, 56:125
Adh1 alleles -4, -6, -N Stuber &, 56:128
adh1 allele; ADH-negative, unstable Döring &, 56:40
Adh1 alleles -S-v1, -S-v2, -S-v3; -S-v1 expresses Kn phenotype Mottinger, 56:89
Adh1 alleles -1F, -6F, -9F, -11F, -12F, -13F, -15F, -21F, -24F, -29F, -33F, -44F, -45F, -48F, -1S, -32S, -34S, -35S, -52S, -54S Woodman &, 1981
Ap1 alleles -S, -I, -F; enzyme properties dissimilar to those of Phos4 El-Metainy &, 1981
Ap1 alleles -2, -2L, -2S, -3, -4, -6 Stuber &, 56:128
Cat2 allele -null Tsafaris &, 1981
Cat3 - 47.8 - Mdh4 - 7.8 - Mmm1 - 24.4 - Pgm1 - 2.2 - Phil Goodman &, 56:125
Cat3 alleles -7, -9, -12, -12L, -N Stuber &, 56:128
Gdh1 alleles -F, -I Goodman &, 1980
K1L -- 185 bp repeat hybridizes to P100 knob Peacock &, 1981
K1S -- 185 bp repeat hybridizes to P100 knob Peacock &, 1981
Mdh4 - 4.4 - mmm1 - 19.0 - Pgm1 - 5.8 - Adh1 - 11.9 - Phil - 5.2 - Gdh1 Goodman &, 1980
Mdh4 allele -null from Bov 1032 collection Goodman &, 1981
Mmm1 - 4.8 - Mdh4; alleles Mmm1-M, -m, -m2 Goodman &, 56:126
Pgm1 alleles -A9, -A16 Goodman &, 1980
Phil alleles -2, -4, -5 Goodman &, 1980
Phil alleles -2, -3, -4, -5 Stuber &, 56:128
v* mutants uncovered by TB-1S include -E68, -E243, -E628, -E1024; intercrosses show -E144 and -E1151 are allelic, -E19 and -E34 are nonallelic; mutants uncovered by TB-1L include -E55, -E245 Chang, 56:44
ys2 yellow stripe, uncovered by TB-1Sb (previous designation of ys2, "Reported in chrom. 2" in Emerson, Beadle and Fraser, has lapsed) Pogna &, 56:153
Zer4 -- Zapalote Chico earworm resistance; association with P1-WW Miranda &, 56:32

Chromosome 2

- K2L -- 185 bp repeat hybridizes to knob Peacock &, 1981
lte1 association with T2-9b wx and Inv2a gl2 Miranda &, 56:29
Mut -- mutator of bz1-m-rh; Mut - 32 - T2-9b wx1 Rhoades &, 56:22
v* mutants uncovered by TB-2S, -E453 and -E605, are allelic; mutants uncovered by TB-2L, -E350, -E424, -E576 and -E588, are allelic; -E1085 not allelic to others Chang, 56:44
Zer3 -- Zapalote Chico earworm resistance; association with T2-9b wx1 Miranda &, 56:32

Chromosome 3

- a1 allele -m-rh Rhoades &, 56:21
E8 located by TB-3Sb; alleles -F, -S Newton &, 56:154
E8 alleles -4, -4.5, -5, -6, -N Stuber &, 56:128
Got1 - 2.3 - Me1; alleles Got1-L4, -L6 Goodman &, 1980
hcf*-19 - 29 - ys3; hcf*-19 - 58 - d1 (TB-3Sb uncovering needs confirmation); Polacco &, 56:47
 allelism between hcf*-19A (= -19g) and -19B (= -19yg)
K3L -- 185 bp repeat hybridizes to P100 knob Peacock &, 1981
Mdh3 - 9.4 - A1 - 12.5 - Et1 Goodman &, 1980
Mdh3 allele -null from Gua159 collection Goodman &, 1981
Me1 alleles -F, -R Goodman &, 1980
Mer2 -- IAC Maya earworm resistance; association with T3-9c wx1 Miranda &, 56:32
Pgd2 alleles -2.8, -5 Stuber &, 56:128
 TB-3Ld breakpoint in B at approximately L.53 Kowles &, 56:61
 TB-3Sb breakpoint in B at L.65±.18 Kowles &, 56:61
v* mutants uncovered by TB-3L include -E41, -E54, -E142 Chang, 56:44

Chromosome 4

- Asr1 - 16.0 ga1; Asr1 - 45.8 - su1 Miranda &, 56:33
K4L -- 185 bp repeat hybridizes to knob Peacock &, 1981
Mer1 -- IAC Maya earworm resistance; association with T4-9b wx1 Miranda &, 56:32
TB-4Lb uncovers gl4, gl3, c2, dp1 Beckett, 56:47
Zer1 -- Zapalote Chico earworm resistance; association with T4-9(5657) wx1 Miranda &, 56:32

Chromosome 5

- Got2 alleles -M2, -M4, -M7 Goodman &, 1980
Got3 alleles -U4, -U6 Goodman &, 1980
K5L -- 185 bp repeat hybridizes to KYS and P100 knobs Peacock &, 1981
Mdh5 allele -E15 Goodman &, 1980
Mdh5 allele -null from Tx325 Goodman &, 1981
Mdh5 alleles -12, -15, -16.6, -N Stuber &, 56:128

(Chromosome 5, continued)

Pgm2 - 16.7 - Mdh5 - 16.9 - Got3 - 1.4 - a2 - 31.6 - pr - 30.2 - Got2
Pgm2 alleles -B3, -B4, -B8
Pgm2 alleles -1, -3, -4, -8
 TB-5La breakpoint in B at L.80±.08
 TB-5Sc uncovers a2, gl17
 v* mutants uncovered by TB-5L, -E26, -E473, -E735, are allelic
 Goodman &, 1980
 Goodman &, 1980
 Stuber &, 56:128
 Kowles &, 56:61
 Beckett, 56:47
 Chang, 56:44

Chromosome 6

dep1 -- defective pistils; y1 - 37.59 - dep1
Dt2 - 26 - Y1; Dt2 - 6 - P11
Enp1 alleles -4, -6, -8, -10, -11, -12
Idh2 - 1.1 - Mdh2
Idh2 alleles -4, -4.2, -6
 K6L -- 185 bp repeat hybridizes to 3x10⁴ copies in Black Mexican; also hybridizes to KYS and P100 knobs
 K6S -- 185 bp repeat hybridizes to P100 and to 3x10⁴ copies in Black Mexican; low hybridization to KYS knob
 Mdh2 alleles -B3, -B6
 Mdh2 allele -null from Ky21
Mdh2 alleles -3, -3.2, -3.5, -4.5, -5.6, -6, -N
Pgd1 alleles -2, -3.8, -N
 TB-6Lb breakpoint in B at L.46±.08
 TB-6Lc breakpoint in B at L.76±.08, in 6L approximately .11
 v* mutants uncovered by TB-6L, -E599, -E634 and -E716, are allelic
 Zer2 -- Zapalote Chico earworm resistance; association with T6-9b wxi
 Micu, 1981
 Higgins &, 56:43
 Stuber &, 56:128
 Goodman &, 1980
 Stuber &, 56:128
 Peacock &, 1981
 Peacock &, 1981
 Goodman &, 1980
 Goodman &, 1981
 Stuber &, 56:128
 Stuber &, 56:128
 Kowles &, 56:61
 Kowles &, 56:61
 Chang, 56:44
 Miranda &, 56:32

Chromosome 7

o2 - 6.6±0.4 - De*-B30 - 0.5 - (Zp1, Zp2, Zp3); o2 - 9.0 - vp9 - 4.0 - (Zp1, Zp2, Zp3);
Zp6 - 16.6±2.3 - Zp21 - 0.8±2.2 - o2 - 12.7±1.6 - (Zp1, Zp2, Zp3) - 5.5 - Zp16;
Zp21 - 3.1 - Zp29 - 4.3 - o2
 K7L -- 185 bp repeat hybridizes to KYS and P100 knobs
 v* mutants uncovered by TB-7L, -E53, -E413 and -E590, are allelic
 Soave &, 1981
 Peacock &, 1981
 Chang, 56:44

Chromosome 8

Ac2 -- mutator of bz2-m; Ac2 - 19.8 - T8-9(6673) wx1; Ac2 - 12.5 - T8-9(6673) wx1;
Ac2 - 0 - msB
Idh1 - 22.5 - Mdh1
Idh1 alleles -4, -6, -8
 K8L -- 185 bp repeat hybridizes to P100 knob
 Mdh1 allele -A105
 Mdh1 allele -null from H25
Mdh1 alleles -1, -6, -10.5, -N
 TB-8Lc uncovers pro1, v21
 v* mutants uncovered by TB-8L, -E7, -E358, -E779, -E826, are allelic; -E25 and -E29 are nonallelic
 Rhoades &, 56:22
 Goodman &, 1980
 Stuber &, 56:128
 Peacock &, 1981
 Goodman &, 1980
 Goodman &, 1981
 Stuber &, 56:128
 Beckett, 56:47
 Chang, 56:44

Chromosome 9

bk2 - 16.7 - T8-9a - 33.3 - bm4
bz1 alleles Bz1-McC; derivatives from bz1-m4 Bz1'-1, Bz1'-2, Bz1'-3, Bz1'-4, Bz1'-5, Bz1'-6, Bz1'-7
bz1 allele -m-rh
 K9S -- 185 bp repeat hybridizes to KYS and P100 knobs
 Mrh -- mutator of a1-m-rh; Sh1 - 20.4 - Wx1 - 21.5 - Mrh
sh1 alleles -m5933-1, -m6233A2, -m6258A(McClintock); Sh1-m5933-1, sh1-m6795;
 restriction sites for SstI, BstEII, BglII, BclI, BglI, BamHI, EcoRI
sh1 alleles -m5933, -m6233; restriction sites for BglII, BamHI, BclI
 TB-9Lc breakpoint in B at L.32±.03
 TB-9Sd breakpoint in B at L.74±.08, in 9S.08±.24
 v* mutants uncovered by TB-9L, -E27, -E585, -E610, -E697 are allelic; -E828 and -E829 are nonallelic
wx1 alleles -m-7, -m-9
wx1 alleles -B3-M, -B3-W
 Burnham, 56:150
 Dooner, 1981
 Rhoades &, 56:22
 Peacock &, 1981
 Rhoades &, 56:21
 Burr &, 1981
 Döring &, 56:40
 Kowles &, 56:61
 Kowles &, 56:61
 Chang, 56:44
 Schwartz &, 56:15
 Echt &, 56:16

Chromosome 10

Gdh2 - 12.5 - Glul; alleles -F, -S
Glul alleles -1, -2, -2.5, -3, -6, -7, -9, -10, -N
 K10 gene order R1 O7 W2 Sr2, inverted relative to N10 (R1 W2 O7 Sr2)
 DFK10(C) deficient for L13 O7 W2 Sr2 and K10 knob; DFK10(F) deficient for W2 Sr2 & K10;
 DFK10(I) deficient for W2 Sr2 & K10; DFK10(H) deficient for Sr2 and K10;
 DFK10(K) deficient for Sr2 and K10
 Isr1 -- inhibitor of striate; location in (P) and (S) elements of R1
 K10 -- 185 bp repeat hybridizes to 10⁶ copies
 K10L -- 185 bp repeat hybridizes to P100 knob
 Lte2 -- Latente heat tolerance; association with r1
R1 alleles r1-g:e, R1-g:2, R1-g:3
 Goodman &, 56:125
 Stuber &, 56:128
 Rhoades &, 56:18
 Rhoades &, 56:18
 Kermicle &, 1981
 Peacock &, 1981
 Peacock &, 1981
 Miranda &, 56:29
 Dooner &, 1981

(Chromosome 10, continued)

- Rp1-Td -- resistance to *P. sorghi* (from *Tripsacum dactyloides*); Rp1-Td - 0.3 - Rp1-d
TB-10Lb breakpoint in B at L.62±.05, in 10L.34±.17
v* mutants uncovered by TB-10L, -E114 and -E470, are allelic; -E354 and -E418 not
- Unplaced
- Adr1 -- alcohol-dehydrogenase regulator; alleles -H, -L
Ap2 alleles -S, -I, -F; enzyme properties dissimilar to those of Phos4
bs1 -- barren sterile
bv2 -- brevis-2
db1 -- dichotomously branching
Rf4 -- restorer of cms-C group cytoplasm (-C, -Bb, -ES, -PR, -RB)
Sod1 -- superoxide dismutase
Sod4 -- superoxide dismutase
t11 -- tassellless
whp1 not uncovered by TB-5Sb; not linked with su1 or yl; not closely linked to
breakpoints of TB-1La, -3Sb, -3La, -4Sa, -5La, -6Lc, -9Sb, or -9Lc
- B chromosome
- Factors 1, 2, 3, 4 controlling nondisjunction
KBL -- 185 bp repeat hybridizes to 1.25×10^4 copies in knob

Assembled by Prof Ligate

Synopsis of *Zea* (Iltis & Doebley, Amer. J. Bot. 67:982-1004. 1980)

- Zea perennis (Hitchc.) Reeves & Mangelsdorf, Amer. J. Bot. 29:817. 1942.
(*Euchlaena perennis* Hitchc., Jour. Wash. (D.C.) Acad. Sci. 12:207. 1922.)
c.n. perennial teosinte
- Zea diploperennis Iltis, Doebley, & Guzman, Science 203:186. 1979.
c.n. diploperennial teosinte
- Zea luxurians (Durieu & Ascherson) Bird, Taxon 27:363. 1978.
(*Euchlaena luxurians* Durieu & Ascherson, Sitzungsber. Ges. Nat. Freunde Berlin 1876:164. 1876.)
(*E. mexicana* Schrader var. *luxurians* (Durieu & Ascherson) Haines, Bot. Bihar and Orissa pt. 6:1065. 1924.)
(*Zea mays* subsp. *luxurians* (Durieu & Ascherson) Iltis, Phytologia 23:249. 1972.)
- Zea mays L., Sp. Pl. 971. 1753.
Zea mays L. subsp. *mays*
c.n. corn, maize, Indian corn
- Zea mays L. subsp. *mexicana* (Schrader) Iltis, Ann. Rev. Genet. 4:450. 1971.
(*Euchlaena mexicana* Schrader, Index Sem. Hort. Göttingen 1832.)
(*Zea mexicana* (Schrader) Kuntze, in Post & Kuntze, Lexicon 599. 1904.)
c.n. teosinte
- Zea mays subsp. *parviglumis* Iltis & Doebley, Amer. J. Bot. 67:1001. 1980.
Zea mays subsp. *parviglumis* var. *parviglumis*
Zea mays subsp. *parviglumis* var. *huehuetenangensis* Iltis & Doebley, Amer. J. Bot. 67:1002. 1980.

This synopsis is now listed in the USDA Nomenclature File as a revision to Agricultural Handbook 505 (E. E. Terrell, Botanist, USDA Plant Taxonomy Laboratory, Beltsville, Maryland).

NEW YORK STATE COLLEGE OF AGRICULTURE AT CORNELL UNIVERSITY
 CORNELL UNIVERSITY, AGRICULTURAL EXPERIMENT STATION
 ITHACA, N. Y.

DEPARTMENT OF PLANT BREEDING

Report of a meeting held during the Genetics Congress on
 August 26th by those interested in corn genetics

- M. M. Rhoades -

October 5, 1952

To Corn Geneticists :-

Enclosed with this is a report of the meeting of corn geneticists held at Ithaca at the time of the Genetics Congress and a report of a committee provided for at that meeting.

In accordance with the action taken by the whole group and by the committee, it is requested that, as soon as convenient, you send to the undersigned a small quantity of seed of any stocks which you think may be useful to other workers now or which should be maintained for future use. As these lots of seed are received, a record of them will be made and later sent to all of you so that you may know what is available. As an illustration of combinations of genes such as should be available for distribution, a list of types now in our possession at Cornell is given below. You should not fail to send material even tho it duplicates stocks in this list

- | | |
|---|---|
| 1. lg-gl ₂ -b-v ₄ | 7. lg g a ₁ -na-ts ₄ |
| 2. Bn-ra-v ₅ | 8. p-(Ts ₂ ts ₂)-(Ff)-(Br br)-an |
| 3. Bn-gl ₁ -v ₅ | 9. A ₁ -na-cr gl ₁ -v ₅ ts ₂ -f ₁ Y-Pl |
| 4. y-Pl-al | 10. a ₁ j B-lg Y-Pl C r ^F pr |
| 5. a ₁ P sh wx lg f ₁ | 11. P-br-f-an |
| 6. A B Pl lg sh wx y | 12. P-br-f- br ₂ br ₂ |

A limited supply of trisomic seed is available for the b-lg, a-na, pr-v₂, Y-Pl, ra-gl₁, j, c-wx and r-g linkage groups. We shall be glad to supply samples of this seed to the different individuals charged with the responsibility of the various groups. If the demand is not too great we shall try to supply all requests for trisomic seed.

If your work requires some unusual set-up or if you want better material of certain types than you now have, please indicate your needs at once. These requests will then be circulated from this office. As an illustration of what is in mind here, Emerson wants an early maturing stock involving green-striped. He also desires the combination adherent-anther ear.

M. M. Rhoades
 M. M. Rhoades, Sec'y

The meeting was called to order by Dr. R. A. Emerson. Approximately 45 individuals were present.

The following resolutions were discussed and favorably acted upon:-

1. That the dropping of the second letter in bi-literal symbols to form a subscript be condemned as confusing and unsatisfactory.
2. That some place be designated as a 'clearing house' to assist in the assigning of appropriate names and symbols for characters and genes. Cornell was chosen as the institution where the records will be kept and help given in the assigning of symbols. An example of how this 'clearing house' may be expected to function is as follows:- Two individuals, A and B, are working on glossy seedlings. A reports he has 5 and B reports he has 4 new glossy seedlings. A will then be assigned from gl₄ to gl₃ and B will be assigned from gl₃ to gl₁₂. This should avoid the confusion that arises when two investigators use the same symbols for different genes.
3. That a repository be formed for the storing and disseminating of new genes and of desirable multiple factor combinations, and that a list of such genes and combinations be furnished those interested from time to time.
4. That the geneticists refrain from designating the linkage groups by numbers until the cytologists agree to the size sequence of the different members of the haploid set.

5. That a committee be appointed by Dr. Emerson to consider the problems connected with the maintenance of a central seed repository. The report of the committee follows:-

In accordance with the action acted above a committee was appointed consisting of Brink, Jones, Mangelsdorf, Stadler, and Emerson (chairman). The committee met and took action as follows:-

1. The genetics group at Cornell, with M. M. Rhoades in charge, is to act as custodian of these stocks.
2. The custodian is to receive from the several workers seed of any stocks involving new characters considered by the finder as worth saving and certainly any such characters the linkage of which is known, also particularly useful combinations of genes in the several groups, etc.

2.

3. The custodian will furnish those interested a list of the stocks received.

4. He will distribute on request small lots of particular stocks to workers having need of them.

5. The custodian will see that viable seed of these stocks is provided at least every three or four years by those charged with growing them.

6. The finder of a new character is expected to maintain the stock or to notify the custodian that he can not do so. Those assuming responsibility for particular groups will maintain stocks involving all the genes of those groups and will endeavor to build up desirable combinations of genes of the particular groups.

7. The following assignment of groups was made by the committee:-

Group 1.	P-br	Emerson
Group 2.	B-1g	Berdle
Group 3.	a ₁ -Rg	Brink
Group 4.	su-Tu	Jonas
Group 5.	pr-v ₂	Burnham
Group 6.	Y-Pl	Stadler
Group 7.	g ₁ -ra	Jenkins
Group 8.	j ₁	Sprague
Group 9.	c-wx	Eyster
Group 10.	R-g ₁	Lindstrom

Any of the above who cannot assume or continue responsibility for the group assigned him is to notify the custodian at once. It is to be understood that anyone may begin or continue work with any group whether or not it has been assigned to him. The purpose is not so much to prevent duplication as to insure that no group is neglected. It is expected, however, that when two or more are interested in the same group, they will work in close cooperation!

R. A. Emerson (chairman)

V. REPORT OF THE MAIZE GENETICS COOPERATION STOCK CENTER

There were 141 seed requests received during 1981 and 1132 seed packets sent to fill the requests. Foreign requests amounted to 21% (29) of the total requests and 18% (212) of the total seed packets sent. Geneticists accounted for 52% of the seed requests received, physiologists 27%, plant breeders 14% and educational 7%.

Each year maize stocks are sent in by maize geneticists to become part of the collection. The following stocks have been received over the past several years and have either been placed on the stock list or will be increased and placed in the next few years.

<u>Contributor</u>	<u>Genetic Stock</u>	<u>Contributor</u>	<u>Genetic Stock</u>
J. B. Beckett	sr3	E. H. Coe, Jr.	Wc-WH Bf bm4
J. Birchler	R-scm2 ; bz2 A A2 C C2 Bz		wx v g115 bk2 Bf bm4 c
	R-scm2 ; a-st A2 C C2		lg g12 B ; r-g P1 K55 ⁴
	R-scm2 ; c2 A A2 C		lg g12 b ; r-g P1 K55 ⁴
	R-scm122 ; a2 A C C2		g12 B wt ; r-g P1 K55 ⁴
	R-scm2 ; c A A2 C2		B ; r-g P1 K55 ⁴
E. H. Coe, Jr.	Ac		b ; r-g P1 K55 ⁴
	cm	O. E. Nelson	o9
	gt		o10
	NCS2		o11
	NCS3		o12
	Spm		o13
	pyd	M. M. Rhoades	Tp9 N9 N3 Df3
	bz2 a c2 a2 pr c bz wx r (Down tester)	K. W. McWhirter	o7 f12
The following stocks are partially converted to W23 and K55:		F. Salamini	Glossy mutants converted to WF9 inbred:
	d		g1
	wx d3		g12
	an		g13
	an-6923		g14
	D8		g16
	c sh wx		g18
	yg2 c sh wx	G. F. Sprague	.y8 v5 g1
	sh bz wx		
	yg2 C sh bz wx		

The Advisory Committee for the Stock Center (D. Duvick, C. S. Levings, O. E. Nelson) and E. H. Coe, USDA representative, met at Urbana in September 1981 to discuss and advise on the Stock Center. The general advice of the committee was that the chief function of the stock center should be the maintenance of maize genetic stocks and the promotion of cooperation between maize geneticists and also related disciplines. Additional functions of the stock center should be carried out as time and demand dictate. The Committee further suggested that the Director/Curator spend considerable time directing the functions of the stock center and conduct allied research activities as time and monies permit.

The following catalogue lists available stocks; translocations are listed in the 1981 News Letter. Requests for stocks and correspondence relative to the Stock Center should be addressed to:

R. J. Lambert
 University of Illinois
 Department of Agronomy
 S-118 Turner Hall
 1102 South Goodwin Avenue
 Urbana, IL 61801

Catalogue of Stocks

Chromosome 1

sr zb4 P-WW
 sr P-WR
 sr P-WW
 sr P-WR an gs bm2
 sr P-WR an bm2
 sr P-RR an bm2
 sr P-RR gs bm2
 sr P-WR bm2
 vp5
 zb4 ms17 P-WW
 zb4 ms17 P-WW rs2
 zb4 ts2 P-WW br f bm2
 zb4 ts2 P-WW bm2
 zb4 P-WW
 zb4 P-WR
 zb4 P-WW br
 zb4 P-WW br f bm2
 zb4 P-WW bm2
 ms17
 ts2 P-RR
 ts2 P-WW bm2
 ts2 P-WW br bm2
 ts2 br f bm2
 P-CR
 P-RR
 P-RW
 P-CW
 P-MO
 P-VV
 P-RR as br f an gs bm2
 P-RR br f an gs bm2
 P-RR br f an gs bm2 rd
 P-RR br f an gs bm2 id
 P-RR br f an gs bm2 v*-8983
 P-RR br f an gs bm2 v*-8943
 P-RR an ad bm2
 P-RR an gs bm2
 P-RR ad bm2
 P-WR an Kn bm2
 P-WR an ad bm2
 P-WR an bm2
 P-WR an br bm2
 P-WT = WR an bm2
 P-WR br Vg
 P-WR br f gs bm2
 P-WR br f an lw gs bm2
 P-WR br f bm2 id
 P-WW rs2
 P-WW rs2 br f
 P-WW as br f bm2
 P-WW hm br f
 P-WW br f ad bm2
 P-WW br f bm2
 P-WW br f an gs bm2
 P-WW br Vg
 as
 as br2
 as rs2
 rd Hy
 br f
 br f bm2 v*-5568
 br f Kn
 br f Kn Ts6
 br f Kn bm2
 br bm2
 Vg
 Vg an bm2
 Vg br2 bm2
 v22
 bz2 m ; A A2 C Pr
 bz2 M ; A A2 C R Pr
 bz2 ad bm2 ACR
 an bm2
 an-bz2-6923 (apparent deficiency
 including an and bz2)
 br2
 br2 bm2
 br2 an bm2
 th-8963
 Kn
 Kn Ts6
 Kn bm2
 lw
 Adh1-S
 vp8

Chromosome 1 (continued)

gs
 gs bm2
 Ts6
 bm2
 id
 nec2
 ms9
 ms12
 ms14
 mi
 D8
 L1s
 Les2
 TB-1La (1L.20)
 TB-1Sb (1S.05)
Chromosome 2
 ws3 lg g12 B
 vs3 lg g12 B sk
 ws3 lg g12 B sk v4
 ws3 lg g12 B sk f1 v4
 ws3 lg g12 B gs2 v4
 ws3 lg g12 B ts
 ws3 lg g12 b
 ws3 lg g12 b sk
 ws3 lg g12 b sk v4
 ws3 lg g12 b gs2 v4
 ws3 lg g12 b f1 v4
 ws3 lg g12 b sk f1 v4
 ws3 lg g12 b v4
 al
 al lg
 al lg g12 B sk v4
 al lg g12 b
 al lg g12 b sk v4
 al lg g12 b sk f1 v4
 lg
 lg g12
 lg g12 B
 lg g12 B gl11
 lg g12 B gs
 lg g12 B gs2 v4
 lg g12 B gs2 Ch
 lg g12 B gs2 sk Ch
 lg g12 B gs2 sk v4
 lg g12 B sk
 lg g12 B sk v4
 lg g12 B v4
 lg g12 b
 lg g12 b gs2
 lg g12 b gs2 Ch
 lg g12 b gs2 sk Ch
 lg g12 b sk
 lg g12 b sk f1
 lg g12 b sk f1 v4
 lg g12 b sk v4
 lg g12 b wt v4
 lg g12 b fl
 lg g12 b fl v4
 lg g12 b fl v4 Ch
 lg g12 b v4
 lg g12 b v4 Ch
 lg g12 mn v4
 lg g12 wt
 lg g12 b gs2 wt
 lg g12 w3
 lg g12 w3 Ch
 lg g12 Ch
 lg b gs2 v4
 lg Ch
 g12
 d5 = d*-037-0
 B gl11
 B ts
 gl14
 gl11
 wt
 mn
 fl
 fl v4 Ch

Chromosome 2 (continued)

fl Ht v4
 fl Ht v4 Ch
 fl w3
 fl v4 w3
 fl w3 Ch
 fl v4 w3 Ch
 ts
 v4
 v4 w3 Ht Ch
 v4 Ht Ch
 w3
 w3 Ht
 w3 Ht Ch
 w3 Ch
 Ht (A & B source)
 ba2
 R2 ; v A A2 C
 r2 ; r-g A A2 C
 Ch
 gs2
 Les
 2 Trip Trip2 /ws3 lg g12
 TB-1Sb-2L4464
 TB-3La-2B6270
 Primary trisomic 2
Chromosome 3
 cr
 cr d
 cr d Lg3
 cr pm ts4 lg2
 cr ts4 na
 d-Tall = d*-6016 (short)
 d rt Lg3
 d Rf Lg2
 d ys3
 d ys3 Rg
 d ys3 Rg lg2
 d Lg3
 d Lg3 g16
 d Lg3 ts4 lg2
 d Rg
 d Rg ts4 lg2
 d pm
 d yg*- (W21)
 d ts4 lg2
 d ts4 lg2 a-m ; A2 C R Dt
 d ts4
 d g16
 d lg2 a-m A2 C R Dt
 d a-m A2 C R Dt
 ra2
 ra2 Rg
 ra2 Rg ts4 lg2
 ra2 Rg g16
 ra2 ys3 Lg3 Rg
 ra2 ys3 Rg
 ra2 Rg lg2
 ra2 pm lg2
 ra2 ts4
 ra2 ts4 lg2
 ra2 lg2
 Gg
 cl
 cl ; Clm-2
 cl ; Clm-3
 cl-p ; Clm-4
 rt
 ys3
 ys3 Lg3
 ys3 g16 lg2 a-m et ; A2 C R Dt
 ys3 ts4
 ys3 ts4 lg2
 lg3
 lg3 Rg pm
 g16
 g16 lg2 A ; A2 C R
 g16 lg2 A-b et ; A2 C R Dt
 g16 lg2 a-m et ; A2 C R Dt

Chromosome 3 (continued)

pm lg2
 ts4
 ts4 na
 ts4 na pm
 ts4 ba na
 ts4 lg2 a-m ; A2 C R Dt
 ts4 na a-m ; A2 C R Dt
 ig
 ba
 y10
 lg2
 lg2 A-b et ; A2 C R Dt
 lg2 a-m sh2 et ; A2 C R Dt
 lg2 a-m et ; A2 C R dt
 lg2 a-m et ; A2 C R Dt
 lg2 a-st sh2 et ; A2 C R Dt
 lg2 a-st et ; A2 C R Dt
 na
 na lg2
 A sh2 ; A2 C R B Pl dt
 A-d31 ; A2 C R
 A-d31 ; A2 C R pr dt
 A-d31 ; A2 C R B Pl dt
 A-d31 ; A2 C R Dt
 A-d31 ; A2 C R pr Dt
 A-d31 sh2 ; A2 C R B Pl dt
 A-d31 sh2 ; A2 C R Dt
 A-d31 sh2 ; A2 C R B Pl Dt
 A-d31 et ; A2 C R Dt
 a-m ; A2 C R B Pl dt
 a-m ; A2 C R Dt
 a-m ; A2 C R B Pl Dt
 a-m sh2 ; A2 C R B Pl dt
 a-m sh2 ; A2 C R B Pl Dt
 a-m et ; A2 C R Dt
 a-st ; A2 C R Dt
 a-st sh2 ; A2 C R Dt
 a-st sh2 A2 C R B Pl Dt
 a-st sh2 et ; A2 C R Dt
 a-st et ; A2 C R Dt
 a-p sh2 et ; A2 C R B Pl Dt
 a-p et ; A2 C R dt
 a-p et ; A2 C R B Pl Dt
 a-x1
 a-x3
 a Ga7 ; A2 C R
 sh2
 vp
 Rp3
 pg14
 a3
 g5
 le
 li
 yel*-5787
 TB-3La (3L.10)
 TB-3Sb (3S.50)
 TB-3Lc (distal to 3La (3L.10)
 Primary Trisomic 3

Chromosome 4

Rp4
 Ga
 Ga su
 Ga-S
 Ga-S ; y
 Ga-S ; A A2 C R
 st
 st Ts5
 st fl2
 Ts5
 Ts5 fl2
 Ts5 su
 Ts5 la su gl3
 Ts5 su zb6
 Ts5 su zb6 o
 Ts5 su gl3 o
 Ts5 Tu
 la
 la su Tu gl3
 la su gl3

Chromosome 4 (continued)

la su gl3 c2 ; A A2 C R
 la su gl3 o
 la su bt2 gl3
 fl2
 fl2 su
 fl2 bt2
 fl2 su bm3
 fl2 su gl4 Tu
 su
 su-am
 su bt2 gl4
 su bm3
 su zb6
 su zb6 bt2
 su zb6 Tu
 su zb6 gl3 dp
 su gl4 j2
 su gl4 o
 su gl4 o Tu
 su j2
 su gl3
 su gl3 o
 su o
 su gl4
 bt2
 bm3
 gl4
 gl4 o
 Tu
 Tu-1 1st
 Tu-1 2nd
 Tu-d
 Tu-md
 Tu gl3
 j2
 j2 c2 ; A A2 C R
 j2 C2 ; A A2 C R
 j2 gl3
 v8
 gl3
 gl3 o
 gl3 dp
 c2 ; A A2 C R
 C2 ; A A2 C R
 C2-Idf (Active-1) ; A A2 C R
 dp
 o
 v17
 v23
 ra3
 Dt4 su ; a-m A2 C R
 TB-4Sa (4S.20)
 TB-1La-4L4692
 TB9Sb-4L6504 (9S.40-.83; 4L.09)
 TB7Lb-4L4698 (7L.30-.74; 4L.08)
 Primary Trisomic 4

Chromosome 5

am a2 ; A A2 C R
 lu
 lu sh4
 ms13
 gl17
 gl17 A2 pr ; A C R
 gl17 a2 ; A C R
 gl17 a2 bt ; A C R
 gl17 a2 bt v2 ; A C R
 A2 vp7 pr ; A C R
 A2 bm bt pr ys ; A C R
 A2 bm pr ; A C R
 A2 bm pr ys ; A C R
 A2 bm pr ys eg ; A C R
 A2 bm pr v2 ; A C R
 A2 bt v3 pr ; A C R
 A2 bt pr ; A C R
 A2 bt pr ys ; in A C R
 A2 v3 pr ; A C R
 A2 pr ; A C R
 A2 pr v2 ; A C R
 A2 pr na2 ; A C R
 A2 pr ys ; A C R
 A2 pr zb3 ; A C R

Chromosome 5 (continued)

A2 pr v12 ; A C R
 a2 ; A C R
 a2 bm bt bv pr ; A C R
 a2 bm bt pr ; A C R
 a2 bm bt pr ys ; A C R
 a2 bm pr v2 ; A C R
 A2 v3 pr ; A C R
 a2 bt v3 pr ; A C R
 a2 bt v3 PR ; A C R
 a2 bt pr ; A C R
 a2 bt v2 ; A C R
 a2 v3 pr ; A C R
 a2 pr ; A C R
 a2 pr ; A C R B Pl
 a2 pr v2 ; A C R
 vp2
 vp2 pr
 vp2 gl8
 vp7
 bm
 bm yg
 bt
 ms5
 v3
 td ae
 ae
 sh4
 gl8
 na2
 lw2
 ys
 eg
 v2
 y8
 ms13
 v12
 br3
 nec3
 TB-5La
 TB-5Lb
 Primary Trisomic 5

Chromosome 6

rgd po y
 rgd po Y
 rgd y
 rgd Y
 po = ms6
 po y pl
 po y Pl
 po y wi
 po Y pl
 y = pb = w-m
 y rhm
 y 110
 y 111
 y 112
 y W15
 y pb4
 y pb4 pl
 y pb4 Pl
 y ai
 y wi Pl
 Y Dt2 ; a-m A2 C R
 y pg11 ; Wx pg12
 y pg11 wi ; wx pg12
 Y pg11 ; Wx pg12
 y pg11 ; wx pg12
 Y pg11 ; wx pg12
 y pg11 su2 ; wx pg12
 y pl
 y Pl
 y Pl Bh ; c sh wx A A2 R
 y pl Bh ; c sh wx A A2 R
 y su2
 Y 110
 Y 112
 Y pb4
 Y wi pl
 Y wi Pl
 Y su2
 wi

Chromosome 6 (continued)

Pl Dt2 ; a-m A2 C R
 pl sm ; P-RR
 Pl sm ; P-RR
 Pl sm py ; P-RR
 Pl sm Pt py ; P-RR
 Pt
 w
 w14
 ms6
 2NOR ; a2 bm pr v2
 TB-6Lb
 Primary Trisomic 6

Chromosome 7

Hs o2 v5 ra gl
 In-D
 In-D gl
 o2
 o2 v5
 o2 v5 ra gl
 o2 v5 ra gl sl
 o2 v5 ra gl Tp
 o2 v5 ra gl ij
 o2 v5 gl
 o2 v5 ms7
 o2 ra gl ij
 o2 ra gl sl
 o2 gl
 o2 gl sl
 o2 ij
 o2 bd
 y8 v5 gl
 In ; A2 pr A C R
 in gl ; A2 pr A C R
 v5
 vp9
 vp9 gl
 ra
 ra gl ij bd
 gl
 gl-M
 gl Tp
 gl o5
 gl mn2
 Tp
 ij
 ms7
 ms7 gl Tp
 Bn
 bd
 Fn
 o5
 o5 mn2 gl
 va
 Dt3 ; a-m A2 C R
 V8-8647
 ye1*-7748
 TB-7Lb (7L.30)
 Primary trisomic 7

Chromosome 8

gl18
 v16
 v16 j
 v16 ms8 j
 v16 ms8 j nec
 v16 ms8 j gl18
 ms8
 nec
 v21
 Fl3
 Fl3 j
 TB-8La (8L.70)
 Primary Trisomic 8

Chromosome 9

yg2 C Bz Wx ; A A2 R
 yg2 C sh bz ; A A2 R
 yg2 C sh bz wx ; A A2 R
 yg2 C-I sh bz wx ; A A2 R

Chromosome 9 (continued)

yg2 C bz wx ; A A2 R
 yg2 c sh bz wx ; A A2 R
 yg2 c sh wx ; A A2 R
 yg2 c sh wx gl15 ; A A2 R
 yg2 c sh wx gl15 K-S9 ; A A2 R-g
 yg2 c bz wx ; A A2 R
 wd-Ring C-I ; A A2 R (temp. out of seed)
 C sh bz ; A A2 R
 C sh bz wx ; A A2 R
 C sh bz wx bm4 ; A A2 R
 C-I sh bz wx ; A A2 R
 C sh bz wx gl15 bm4 ; A A2 R
 C sh ; A A2 R
 C sh wx ; A A2 R
 C wx ar ; A A2 R
 C sh wx K-L9 ; A A2 R
 C sh ms2 ; A A2 R
 C bz Wx ; A A2 R
 C Ds wx ; A A2 R Pr y
 C Ds wx ; A A2 R pr Y
 C-I Ds Wx ; A A2 R
 C-I ; A A2 R
 C ; A A2 R
 C ; A A2 R B Pl
 C wx ; A A2 R
 C wx ; A A2 R B Pl
 C wx ; A A2 R b Pl
 C wx ; A A2 R B pl
 C-I wx ; A A2 R y
 C-I wx ; A A2 R y B pl
 C wx ar da ; A A2 R
 C wx v ; A A2 R
 C wx v ; A A2 R Pl
 C wx gl15 ; A A2 R
 C wx gl15 ; A A2 R pr
 C wx Bf ; A A2 R
 c bz wx ; A A2 R
 c sh bz wx ; A A2 R y
 c sh wx ; A A2 R
 c sh wx v ; A A2 R
 c sh wx gl15 ; A A2 R
 c sh wx gl15 bk2 ; A A2 R
 c sh wx gl15 Bf ; A A2 R
 c sh wx bk2 ; A A2 R
 c ; A A2 R
 c wx ; A A2 R y
 c wx v ; A A2 R
 c wx gl15 ; A A2 R
 c wx Bf ; A A2 R
 sh
 sh wx v
 sh wx d3
 sh wx pg12 gl15 ; y pg11
 lo2
 wx*
 wx-a
 w11
 wx d3
 wx d3 w11
 wx d3 v gl15
 wx d3 gl15
 Wx pg12 ; y pg11
 wx pg12 ; y pg11
 Wx pg12 ; Y pg11
 wx pg12 ; Y pg11
 wx pg12 bm4 ; y pg11
 wx v
 wx v gl15 bk2 Bf bm4
 wx bk2
 wx bk2 bm4
 wx Bf
 wx Bf bm4
 v
 ms2
 gl15
 gl15 Bf
 gl15 bm4
 bk2 Wx
 Wc
 bm4
 bm4 Bf
 16

Chromosome 9 (continued)

17
 ye1*-034-16
 w*-4889
 w*-8889
 w*-8951
 w*-8950
 w*-9000
 Tp9 N9 N3 Df3
 TB-9La (9L.40)
 TB-9Sb (9S.40)
 TB-9Lc
 Primary trisomic 9
 *Additional waxy alleles available
 from collection of O. E. Nelson.

Chromosome 10

oy
 oy R ; A A2 C
 oy hf2
 oy ms11
 oy bf2 R ; A A2 C
 oy bf2 ms10
 oy zn R ; A A2 C
 oy du R ; A A2 C
 oy du r ; A A2 C
 oy sr2
 oy zn
 sr3
 Og
 Og B Pl
 Og du R ; A A2 C
 ms11
 ms11 bf2
 bf2
 bf2 zn
 bf2 li g r ; A A2 C
 bf2 g R sr2 ; A A2 C
 bf2 g r sr2 ; A A2 C
 bf2 r sr2 ; A A2 C
 nl zn g R ; A A2 C
 nl g R ; A A2 C
 nl g r ; A A2 C
 nl g R sr2 ; A A2 C
 y9
 y9 v18
 nl
 li zn g r ; A A2 C
 li g R ; A A2 C
 li g r ; A A2 C
 li g r v18 ; A A2 C
 li g r v18 ; A A2 C
 ms10
 du
 du v18
 du o7
 du g r ; A A2 C
 du sr2
 zn
 zn g
 zn g R sr2 ; A A2 C
 zn g r ; A A2 C
 Tp2 g r ; A A2 C
 g R sr2 ; A A2 C
 g r ; A A2 C
 g r sr2 ; A A2 C
 g r sr2 l ; A A2 C
 g R-g sr2 ; A A2 C
 g R-g sr2 v18 ; A A2 C
 g R-g K10 ; A A2 C
 g R-g sr2 ; A A2 C
 g R-r K10 ; A A2 C
 g r-r sr2 ; A A2 C
 Rj r-r ; A A2 C
 Rj r-r sr2 ; A A2 C
 r sr2 l ; A A2 C
 R-g ; A A2 C
 r-g sr2 ; A A2 C
 r K10 ; A A2 C
 r-g ; A A2 C
 r-r ; A A2 C
 r-ch Pl ; A A2 C
 R-mb ; A A2 C
 R-nj ; A A2 C

Chromosome 10 (continued)

R-r ; A A2 C
 R-ch B P1 ; A A2 C
 R-lsk ; A A2 C
 R-sk-mc.2 ; A A2 C
 R-sk ; A A2 C
 R-st ; A A2 C
 R-st Mst
 R-st Mst o7
 R-scm2 ; bz2 A A2 C C2
 R-scm2 ; a-st A2 C C2
 R-scm2 ; c2 A A2 C
 R-scm122 ; pr A A2 C C2
 R-scm2 ; a2 A C C2
 R-scm2 ; c A A2 C2
 Lc
 w2
 w2 l
 o7
 o7 ; o2
 l
 v18
 mst
 l yel*-5344
 yel*-8721
 yel*-8454
 yel*-8793
 cm
 TB-10La (10L.35)
 TB-10Sc
 TB-10L19
 Primary trisomic 10

Unplaced Genes

dv
 dy
 el
 l4
 o9
 o10
 o11
 o13
 R8
 v13
 ws ws2
 ub
 zb
 zb2
 zn2
 l4-4923
 nec*-8376

Multiple Gene Stocks

A A2 C C2 R-g Pr B P1
 A A2 C C2 R-g Pr B p1
 A A2 C C2 R-g b P1
 A A2 C C2 r-g Pr B P1
 A A2 C C2 r-g Pr B p1
 A A2 C C2 R-g Pr B p1
 A A2 C C2 R-r Pr B P1
 A A2 C C2 R-r Pr B p1
 A A2 C C2 R-r Pr B P1
 A A2 C C2 R-r Pr B p1
 A A2 C C2 R-r Pr B P1
 A A2 C C2 R-r Pr B p1
 A A2 C C2 R Pr
 A A2 C C2 R Pr wx
 A A2 C C2 R Pr wx gl
 A A2 C C2 R pr
 A A2 C C2 R pr y wx gl
 A A2 C C2 R Pr y wx
 A A2 C C2 r Pr Y wx
 su pr y gl wx ; A A2 C C2 R
 A su pr ; A2 C C2 R
 bz2 a c2 a2 pr Y/y c bz wx r
 a su A2 C C2 R
 hm2 lg a su pr y gl i wx g
 colored scutellum
 lg gl2 wt ; a Dt A2 C C2 R
 a su pr y gl wx A A2 C C2 R
 hm hm2
 ts2 ; sk
 lg gl2 wt ; a-m A2 C C2 R Dt
 A A2 C C2 R-nj ; purple embryo S. Chase
 Stock 5 ; Hi-haploid R-r B P1

Popcorns

Amber Pearl
 Argentine
 Black Beauty
 Hulless
 Ladyfinger
 Ohio Yellow
 Red South American
 Strawberry
 Supergold
 Tom Thumb
 White Rice

Exotics and Varieties

Black Mexican Sweet Corn
 (with B-chromosomes)
 Black Mexican Sweet Corn
 (without B-chromosomes)
 Knobless Tama Flint
 Gaspe Flint
 Courdseed
 Maiz Chapolote
 Papago Flour Corn
 Parker's Flint
 Tama Flint
 Zapaluta Chica

Tetraploid Stocks

P-RR
 P-VV
 B P1
 a A2 C R Dt
 su
 pr ; A A2 C R
 y
 gl
 Y sh wx
 sh bz wx
 wx
 g A A2 C R
 A A2 C R B P1

Cytoplasmic traits

NCS2
 NCS3

Cytoplasmic steriles and Restorers

WF9-(T) rf rf2
 WF9 rf rf2
 R213 Rf rf2
 Ky21 Rf Rf2

Waxy Reciprocal Translocations

wx 1-9c (1S.48; 9L.22) * Sx
 wx 1-94995 (1L.19; 9S.20) * Sx
 wx 1-98389 (1L.74; 9L.13) W23 only
 wx 2-9b (2S.18; 9L.22) * Sx
 wx 3-9c (3L.09; 9L.12) * Sx
 wx 4-9b (4L.90; 9L.20) * Sx
 wx 4-95657 (4L.33; 9S.25) * Sx
 wx 4-9g (4S.27; 9L.27) W23 only
 wx 5-9a (5L.69; 9S.17) * Sx
 wx 5-9c (5S.07; 9L.10) W23 only
 wx 6-9a (6S.79; 9L.40) * Sx
 wx y 6-9b (6L.10; 9S.37) * Sx
 wx 7-9a (7L.63; 9S.07) * Sx
 wx 7-94363 (7 cent.; 9 cent.) * Sx
 wx 8-9d (8L.09; 9L.16) * Sx
 wx 8-96673 (8L.35; 9S.31) * Sx
 wx 9-10b (9S.13; 10S.40) * Sx

Non-waxy Reciprocal Translocations

wx 1-9c (1S.48; 9L.22) * Sx
 wx 1-94995 (1L.19; 9S.20) * Sx
 wx 1-98389 (1L.74; 9L.13) * Sx
 wx 2-9c (2L.49; 9S.33) W23 only
 wx 2-9b (2S.18; 9L.22) * Sx
 wx 3-98447 (3S.44; 9L.14) * Sx
 wx 3-98562 (3L.65; 9L.22) * Sx
 wx 4-9e (4S.53; 9L.26) * Sx
 wx 4-95657 (4L.33; 9S.25) * Sx

wx 5-9c (5S.07; 9L.10) * Sx
 wx 5-94817 (5L.69; 9S.17) M14 only
 wx 5-98386 (5L.87; 9S.13) * Sx
 wx 6-94778 (6S.80; 9L.30) * Sx
 wx 6-98768 (6L.89; 9S.61) * Sx
 wx 7-94363 (7 cent.; 9 cent.) * Sx
 wx 7-9a (7L.63; 9S.07) W23 only
 wx 8-9d (8L.09; 9L.16) * Sx
 wx 8-96673 (8L.35; 9S.31) * Sx
 wx 9-108630 (9S.28; 10L.27) M14 only
 wx 9-10b (9S.13; 10S.40) * Sx

* = Homozygotes available in both
 M14 & W23 backgrounds

Sx = Single cross of homozygotes between
 M14 & W23 versions available

Inversions

Inv.1a (1S.30-L.50)
 Inv.1c (1S.35-L.01)
 Inv.1d (1L.55-L.92)
 Inv.1L-5131-10 (1L.46-L.82)
 Inv.2a (2S.70-L.80)

Inv.2S-L8865 (2S.06-L.05)
 Inv.2L-5392-4 (2L.13-L.51)
 Inv.3a (3L.38-L.95)
 Inv.3L (3L.19-L.72)
 Inv.3L-3716 (3L.09-L.81)
 Inv.4b (4L.40-L.96)
 Inv.4c (4S.86-L.62)
 Inv.4e (4L.16-L.81)
 Inv.5-8623 (5S.67-L.69)
 Inv.6-8452 (6S.77-L.33)
 Inv.6-8604 (6S.85-L.32)
 Inv.6-3712 (6S.76-L.63)

Inv.7L-5803 (7L.17-L.61)
 Inv.7-8540 (7L.12-L.92)
 Inv.7-3717 (7S.32-L.30)
 Inv.8a (8S.38-S.15)
 Inv.9a (9S.70-L.90)
 Inv.9b (9S.05-L.87)
 Inv.9c (9S.10-L.67)

VI. MAILING LIST, ALPHABETICALLY AND GEOGRAPHICALLY

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IL 61068
IL 61001
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GREEN, M. M.	CA	95616	PATTERSON, EARL	IL	61801	STUCKER, ROBERT	MI	55108	CHAO, EDDIE	HY	12201
TSUCHIYA, T.	CO	80521	PLEHA, MICHAEL	IL	61801	THOMPSON, S.	MI	55108	ALTSCHULER, MARSHA I.	HY	12866
CUAHY, ROBIN L.	CO	80523	RHOODES, A. M.	IL	61801	WALTER, T. J.	MI	55108	MATTHEWS, DAVID L.	HY	13201
MOHL, LEO	CO	80901	SPRAGUE, G. F.	IL	61801	WANG, ANDREW	MI	55108	HEATH-PAGLIULO, S. R.	HY	14253
GREIBBLATT, I. M.	CT	06268	STEFFENSEN, D. M.	IL	61801	XIE, Y-J	MI	55108	RISCHLER, J.	HY	14644
GORDON, P. W.	CT	06340	WIEDHOLM, J. M.	IL	61801	ARMSTRONG, CHARLES L.	MI	55110	HISCHKE, C. F.	HY	14644
RICE, THOMAS B.	CT	06340	HEIDERSON, CLARION B.	IL	61820	MAAPALA, BERNARD	MI	55325	TAYLOR, A. D.	HY	14653
DAY, PETER R.	CT	06504	KOENIG, RICHARD	IL	61820	RATHS, EARL	MI	55343	MOORE, CAROL H.	HY	14653
ROBICHAUD, C.	CT	06511	SMITH, GEORGE E.	IL	61820	HOODMAN, JAMES C	MI	55402	EARLE, ELIZABETH D.	HY	14653
SHULMATH, ALAN H.	CT	06511	SLAICHO, H. H.	IL	61826	RENLEY, FRANK M.	MI	55946	EVERETT, HERBERT L.	HY	14653
SUSSEX, T. M.	CT	06520	VIAL, FRED	IL	61920	KLEESE, ROGER	MI	55967	GRACEY, V. E.	HY	14653
STROUP, D.	DC	20015	BAILEY, ZENO E.	IL	62026	KOWLES, R. V.	MI	56058	JOHNSON, ALICE	HY	14653
COMPIERFORD, J. D.	DC	20036	THOMPSON, D.	IL	62027	PURDY, J. L.	MI	56058	KELL, R. L.	HY	14653
BROGAN, C. O.	DC	20250	WILLIAMS, ROBERT E.	IL	62525	BIRDY, KEI	MI	56277	LENKE, CAROL A	HY	14653
LEWIS, HERMAN	DC	20550	BEAR, ROBERT P.	IL	62525	KELTGEN, KEITH	MI	56277	SISCO, P. H.	HY	14653
HANK, JAMES A.	DE	19711	FERGUSON, V.	IL	62567	SUTTON, LOUWE M.	MI	56283	STINSON, HARRY	HY	14653
KIM, B.-D.	FL	32306	GARWOOD, D. L.	IL	62567	CUTLER, HUGH C.	MI	63110	TRACY, W. F.	HY	14653
HANS, RUSTY J.	FL	32610	AINSHORTH, THOMAS C.	IL	62664	ULMER, R. L.	MI	63110	YODER, G. C.	HY	14653
CHOUREY, PREM	FL	32611	NAGLE, B. J.	IL	62664	BIRD, ROBERT MCK.	MO	63122	BOUGH, R. P.	OH	43011
FERL, R. J.	FL	32611	CUMMINGS, D. P.	IN	46076	HEINKE, D.	MO	63130	MIKULA, BERNARD C.	OH	43512
HAMMAN, L. G.	FL	32611	REED, J. E.	IN	46076	HOOKER, A. L.	MO	63141	GOLDMAN, STEPHEN	OH	43606
PFÄHLER, P. L.	FL	32611	VORIS, MERLE	IN	46076	OPENSHAW, STEVE	MO	63141	PRICE, S. C.	OH	44115
PRING, D. R.	FL	32611	CHU, I. Y. E.	IN	46140	TROYER, A. F.	MO	63141	LAUGHNER, BILL	OH	44234
THORNIBURY, DAVID M.	FL	32611	HARTMAN, CARL J.	IN	46383	HESBERG, K. A.	MO	63166	DOLLINGER, E. J.	OH	44691
POPEHOE, JOHN	FL	33156	JIMENEZ, J.	IN	46605	MASCIA, P. A.	MO	63166	FINLEY, WILLIAM R.	OH	44691
KANG, M. S.	FL	33436	SHAFER, JOHN	IN	46947	NORTON, E. A.	MO	63166	MAULT, LOHELL R.	OH	44691
JELLUH, MILTON D.	GA	30212	MERTENS, THOMAS R.	IN	47306	MOHAMED, A. H.	MO	64110	RAVEED, DAN	OH	45827
FLEHING, A. A.	GA	30602	BOHNER, JOSE	IN	47401	HANSEL, WM. C.	MO	64635	BALLENGER, STEVE	OH	45844
DUCLOS, ZACHARY H.	GA	31763	DEMPSEY, ELLEN	IN	47401	ALEXANDER, C. W.	MO	65001	METZGER, ROBERT	OH	45844
WIDSTROM, HEIL W.	GA	31794	DLOUHY, STEVE	IN	47401	NELSON, M.	MO	65001	BAIRETT, F.	OH	47403
JOHNSON, ELIZABETH	HI	96748	IRISH, ERIN	IN	47401	ALDRICH, R. J.	MO	65211	HAGUE, DONALD R.	OH	97403
BRENDAKER, JAMES	HI	96822	NELSON, KATHERINE	IN	47401	BECKETT, JACK	MO	65211	HERSKOWITZ, I.	OH	97403
DICHE, F. F.	IA	50010	RHOADES, M. H.	IN	47401	BRODIE, C.	MO	65211	NICHEL, KENNETH	OH	16057
DOONER, HUGO K.	IA	50010	SCHWARTZ, DREW	IN	47401	CHIANG, MING-TANG	MO	65211	AYERS, J. E.	PA	16802
HALLAUER, ARNEL R.	IA	50010	SINCLAIR, JOHN	IN	47401	COE, E. H.	MO	65211	BOYER, C.	PA	16802
PALMER, REID	IA	50010	ECHT, C. S.	IN	47405	DARRAN, L.	MO	65211	HAMILTON, R. H.	PA	16802
PENNY, L.	IA	50010	KRIZ, A. L.	IN	47405	DOYLE, G. G.	MO	65211	JOHNSON, M. W.	PA	16802
PETERSON, PETER A.	IA	50010	WILLIAMS, A. F.	IN	47406	EISENSTARN, A.	MO	65211	WRIGHT, JAMES E. JR.	PA	16802
ROBERTSON, DONALD S.	IA	50010	GOOD, RONALD L.	IN	47614	FINCHER, ROBERT R.	MO	65211	SURVER, KATHLEEN	PA	02879
RUSSELL, W. A.	IA	50010	STILES, J. I.	IN	47009	GARDNER, CANDY	MO	65211	KELLER, PATRICK	RI	02881
SILVA, JAIRD	IA	50011	MCBRIDE, A. C.	IN	47905	HELSEL, D.	MO	65211	LA MARRA, ANGELIN	RI	02881
CROSBIE, T. M.	IA	50011	ALVEY, DAVID	IN	47906	HOISINGTON, DAVID	MO	65211	MOTTJAGER, JOHN	RI	02881
SMITH, OSCAR S.	IA	50112	JENKINS, M. T.	IN	47906	KIKUODNE, GARY Y.	MO	65211	ZIMMERING, STANLEY	RI	02912
GERRISH, EVERETT E.	IA	50131	SINGLETON, KATERINA I.	IN	47906	KING, K.	MO	65211	LIU, EDWIN H.	SC	29208
ALBERTSEN, M. C.	IA	50131	ASHMAN, R. B.	IN	47907	LARSON, RUSSELL L.	MO	65211	MARWILLER, ALFRED	SC	29501
BAKER, RAYMOND	IA	50131	AXTELL, JOHN D.	IN	47907	LEHMAN, H.	MO	65211	GATTIS, RICHARD E.	SC	29500
BROWN, WILLIAM L.	IA	50131	BAUMAN, L. F.	IN	47907	LONGLEY, A. E.	MO	65211	NEELY, J. WINSTON	SC	29550
DUVICK, DONALD N.	IA	50131	CRANE, PAUL L.	IN	47907	LOWER, M.	MO	65211	KAHLER, A. L.	SD	57006
HOGLE, STEPHEN W. JR.	IA	50131	GLOVER, DAVID V.	IN	47907	HILES, DONALD	MO	65211	KRATOCHVIL, D. E.	SD	57006
PAEZ, ALIX V.	IA	50131	LARKINS, B.	IN	47907	MITCHELL, ROGER	MO	65211	HAGEL, C. M.	SD	57006
RINEHART, K.	IA	50108	MATSUMOTO, Y.	IN	47907	MODEBA, STEPHEN	MO	65211	SHAIR, D. B.	SD	57006
SENGAL, S. M.	IA	50300	ROBINSON, M. F.	IN	47907	HEUFFER, H. G.	MO	65211	WALEH, R. H.	SD	57006
WATSON, JOHN T.	IA	50315	ULLSTRUP, A. J.	IN	47907	OGHTSING, JOSEPH	MO	65211	WICKS, Z. W.	SD	57007
HOLDEN, A.	IA	50421	LOEFFEL, F. A.	IN	47923	PFAINDER, W. H.	MO	65211	WIESMEYER, HERBERT	TN	37235
WILMARTH, C. E.	IA	50520	FUNK, DW E.	IN	47951	POETHIG, R. S.	MO	65211	COHNER, B. V.	TN	37830
LINDSEY, M. F.	IA	50604	LIANG, GEORGE H.	KS	66502	POLACCO, MARY	MO	65211	CONSTANTIN, M. J.	TN	37830
REID, W. R. P.	IA	50627	PITTEINGER, THAD	KS	66502	REDEI, G. P.	MO	65211	FOARD, DONALD	TN	37830
JULSTROM, P.	IA	50665	BATES, LYNN S.	KS	66506	WALLIN, J. R.	MO	65211	HEWKE, RANDOLPH R.	TN	37830
CAMP, L. M.	IA	50665	SPLITTER, MELVIN V.	KS	67459	ZUBER, M. S.	MO	65211	MCKINLEY, C. H.	TN	37830
MADA, D.	IA	51301	GOERTZEM, KENNETH L.	KS	67871	LEE, EDWARD H.	MO	65211	WEST, BEANIS R.	TN	37901
MURPHY, T. C.	IA	52240	KARPOFF, A. J.	KY	40208	OODH, W. H.	MS	38776	JOSEPHSON, L. M.	TN	37916
BRAININ, E.	IA	52240	RAFAELL, WILLIAM S.	KY	40494	THOMAS, JOHN P.	MS	38776	UNDECK, P. F.	TX	78003
CARLSON, WAYNE	IA	52561	POHELEIT, CHARLES G.	KY	40506	DUKE, STEPHEN O.	MS	38776	HARRIS, JOHN W.	TX	78003
FOYSTER, MICHAEL J.	IA	83605	THORNBURY, D.	LA	71245	HOAGLAND, R. E.	MS	38776	BAIFER, W. C.	TX	77843
WHITHOOD, W.	IA	83605	SAXENA, K. H. S.	LA	71270	CREECH, ROY G.	MS	39762	COLLIER, J. W.	TX	77843
CHEN, SHU-HEI	IL	60053	CHRISTIAN, J. A.	LA	71270	SCOTT, GENE E.	MS	39762	SANKISSIAN, IGOR V.	TX	77843
BEIL, G. M.	IL	60115	HOCHMUTH, GEORGE J.	LA	01003	WILLIAMS, W. P.	MS	39762	SCHERTZ, K. F.	TX	77843
MAUNDER, A. BRUCE	IL	60115	JENKINS, PAUL H.	LA	01003	SCHAEFFER, JÜRGEN R.	MT	59715	SMITH, JAMES J.	TX	78285
SNYDER, JOHN R.	IL	60115	HULCAHY, DAVID	LA	01003	SIDHU, S.	MT	59715	RAMIREZ, S. A.	TX	78711
TSOTSIS, BASIL	IL	60507	STEIN, OTTO L.	MA	01003	PERKINS, K. W.	NC	27215	FABERG, A. C.	TX	78711
DODD, J. I.	IL	60554	MADDONALD, TIMOTHY	MA	01054	BARRY, EDWARD G.	NC	27514	HAGUIRE, HARJORIE	TX	78712
OGLE, CHARLES W.	IL	60637	DUNSHUIR, PANELA	MA	02115	HANGELDOORF, P. C.	NC	27607	JACKSON, R. C.	TX	79409
GARDNER, E. D.	IL	60953	SAGER, RUTH	MA	02115	BOLEN, P. L.	NC	27607	KEVIN, KEHT R.	TX	79409
HALOHE, CARL	IL	60953				BUESCHER, P.	NC	27607	THOMPSON, JACK C.	VA	23140

GIBBON, EDWARD L.	WA	24141	HAGEMANN, R.	E GERMANY	ZAINAL-ABDIN, M. I.	MALAYSTA
OSTERMAN, JOHN	WA	98195	DHAWAN, N. L.	EGYPT	BJARHASSON, HAGHI	MEXICO
STAOLER, DAVID R.	WA	98195	GHODRIAL, HELMY	EGYPT	DE LEON, CARLOS	MEXICO
KONZAK, CALVIN F.	WA	99163	RAKHA, FAROUK A.	EGYPT	JOHNSON, E. C.	MEXICO
HILAN, R. A.	WA	99163	SAYED, SAYED GALAL	EGYPT	KATO Y., T. A.	MEXICO
BAYNE, PETER D.	WI	53211	BERVILLE, A.	FRANCE	KOYASHI, JOSUE	MEXICO
ANDREW, P. H.	WI	53706	BOYAT, A.	FRANCE	OSLER, R. O.	MEXICO
BRINK, R. A.	WI	53706	COMRU, A.	FRANCE	SANCHEZ, F. M.	MEXICO
DEHOPULOS-RODRIGUEZ, J.	WI	53706	DERIEUX, M.	FRANCE	VASAL, S. K.	MEXICO
FURTEK, DOUGLAS	WI	53706	GALLAIS, A.	FRANCE	VIDLIC, ALEJANDRO D.	MEXICO
ILTIS, HUGH H.	WI	53706	KAHN, F.	FRANCE	BOYDE, S.	MOZAMBIQU
KERNICL, JERRY	WI	53706	KISS, CHARLES	FRANCE	DE JOIB, A. W.	NETHERLAND
KLEIN, ANITA S.	WI	53706	LAFOUASSE, MARYSE	FRANCE	DE WOLFF, F.	NETHERLAND
LOHNQUIST, J. H.	WI	53706	LAMBERT, A.	FRANCE	BAMMAL, R. K.	NETHERLAND
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STELLY, DAVID	WI	53706	MORA, G.	FRANCE	AOEIIYI, GIDEDH I.	NETHERLAND
STOUT, JOHN T.	WI	53706	NITSCH, C.	FRANCE	BAHADUR, KHAH	NETHERLAND
HANSEN, DALE J.	WI	53716	POLLACSEK, M.	FRANCE	CHAUHUR, A. R.	NETHERLAND
HEATHMAN, CHARLES	WI	53810	AKPOSOE, M. K.	GHANA	CHOSHAN, ALEXANDER	NETHERLAND
BURHOOD, T.	WI	54027	CHAO, CHUAN-YING	HONG KONG	HAIRIQUE, ANTOINIO	NETHERLAND
WEST, D. P.	WI	54027	BALIHT, ANDOR	HUNGARY	SEVILLA, RICARDO	NETHERLAND
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KASHA, K. J.	CANADA		KUSHIBIKI, HIDEO	JAPAN		
LOCY, R. D.	CANADA		MUKAI, YASUSHI	JAPAN		
MCHALTY, GERALD	CANADA		OHTA, Y.	JAPAN		
MOLMAR, S. J.	CANADA		OKADA, TAKASHI	JAPAN		
OAKS, ANH	CANADA		OKUNO, K.	JAPAN		
POLCHICK, PATRICIA L.	CANADA		SANO, Y.	JAPAN		
SCOLES, GRAHAM J.	CANADA		UCHIZIYA, H.	JAPAN		
SKEPAST, A. V.	CANADA		YABUNO, T.	JAPAN		
STRIBGHAN, GARY	CANADA		YAMADA, MINORU	JAPAN		
STYLES, E. D.	CANADA		BELLEY, H. M. G.	KENYA		
TAYLOR, P.	CANADA		VAN EIJHATTEN, C. L.	KENYA		
WALDEH, D. B.	CANADA		GHOE, BOIG-HO	KOREA		
GEBAUER, JUAN	CHILE		KIM, SOON KWON	KOREA		
HOVAK, F. J.	CZECH		CHANG, S. H.	MALAYSTA		
PIOVARCI, A.	CZECH		GRAHAM, K. M.	MALAYSTA		
PIWH, LUDEK	CZECH		JALANI, B. S.	MALAYSTA		
JENSEN, C. J.	DEINMARK		LEE, CHONG	MALAYSTA		
VON WETTSTEJH, D.	DEINMARK		YONG, M. S.	MALAYSTA		

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FAMILY CIRCUS

by Bil Keane



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"Slow down, Daddy. I want to see the corn."

14th STADLER GENETICS SYMPOSIUM, April 23-24, 1982

University of Missouri, Columbia, MO 65211

- Berg, D. E.--Washington University--Mechanism and Regulation of Movement of Prokaryotic Transposable Element Tn5
- Coe, E. H., Jr.--University of Missouri-Columbia--Nuclear Genes and Chloroplast Modifications in Maize
- Collins, Mary--Carnegie Institution of Washington--A Molecular Analysis of Two Unstable Mutations in Drosophila
- Dieckmann, Carol--Columbia University--Nuclear Control of the Expression of the Cytochrome d Gene in Yeast Mitochondrial DNA
- Elgin, Sarah C. R.--Washington University--Chromatin Structure and Expression in Drosophila
- Huang, Henry--California Institute of Technology--Antibody Diversity and Expression
- Mitchell, R. L.--University of Missouri-Columbia--Mr. Mendel, Please Help me Explain the Importance of Your Work to Senator Proxmire
- Orosz, L.--National Cancer Institute--Bacteriophage 16-3 of Rhizobium meliloti, and its Pattern of Recombination
- Peloquin, S. J.--University of Wisconsin--Meiotic Mutants in Potato Breeding
- Sherman, F.--University of Rochester--Regulation of the Cytochromes c in Yeast

The 1982 meeting of the AIBS (to be held at the Pennsylvania State University, August 9-13, 1982) will include two sessions entitled MOLECULAR CYTOGENETICS OF PLANTS: THE APPLICATION OF NUCLEIC ACID TECHNOLOGY TO PLANT SYSTEMATICS. These sessions are being sponsored jointly by the Botanical Society of America and the American Society of Plant Taxonomists. The morning session will be a symposium devoted to discussion of the types of nucleic acid methodology available for plant systematics studies and their general applicability to phylogenetic and other evolutionary problems. Invited speakers for this symposium are: H. James Price (Texas A&M University); Barbara Schaal (Washington University, St. Louis); Jeffrey Palmer (Carnegie Institution, Stanford University) and Richard Jorgensen (University of California, Davis). These presentations will last approximately thirty minutes each, with a further ten minutes allowed for questions and discussion.

The afternoon session will consist of shorter presentations (fifteen minute talks; five minute question periods). We hope to include both invited and contributed papers on nucleic acid studies of specific families or genera. Invited speakers include Diana Stein (Mount Holyoke College--Osmunda); Jeffrey Doyle (Washington University, St. Louis--Claytonia); Thomas J. White (CETUS, Berkeley--familial relationships among fungi); and Elizabeth Zimmer (Stanford University--Iridaceae, especially Homeria and Moraea). For the afternoon session, we plan to have a total of nine or ten speakers. Members of the BSA or ASPT interested in having their papers considered for inclusion in the MOLECULAR CYTOGENETICS session should indicate that fact along with their regular abstract submissions. Other scientists interested in participating in these talks should contact Dr. Elizabeth Zimmer, c/o Department of Biological Sciences, Stanford University, Stanford, CA 94305 (Phone 415-497-2609). Please try to contact her promptly, but no later than April 6, 1982.

