MAIZE GENETICS COOPERATION

NEWS LETTER

58

April 30, 1984

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and
U.S. Department of Agriculture
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Columbia, Missouri

This is an informal news letter by which working research information on the genetics and cytogenetics of maize is shared. Please see the Foreword for instructions.

Author and Name Indexes	
Nos. 3 through 43	Appendix to No. 44, 1970
Nos. 44 through 50	No. 50, pp. 157-180
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Nos. 54 to date	Annual in each issue
Recommended Rules of Nomenclature	No. 49, pp. 3-4

Some sources of general information on maize genetics and cytogenetics:

Emerson, R. A., G. W. Beadle and A. C. Fraser, 1935. A summary of linkage studies in maize. Cornell Univ. Agric. Exp. Sta. Memoir 180.

Neuffer, M. G., L. M. Jones and M. S. Zuber, 1968. The Mutants of Maize. Crop Sci. Soc. Am., Madison, Wisconsin.

Simmonds, N. W., ed., 1976. Evolution of Crop Plants, Chap. 37, pp. 128-136. Longman, N.Y.

Coe, E. H., Jr. and M. G. Neuffer, 1976. The genetics of corn. Pp. 111-223 in G. F. Sprague, ed., Corn and Corn Improvement, 2nd ed., Amer. Soc. Agron., Madison, Wisconsin.

Carlson, W. R., 1976. The cytogenetics of corn. Pp. 225-304 in G. F. Sprague, ed., Corn and Corn Improvement, 2nd ed., Amer. Soc. Agron., Madison, Wisconsin.

Walden, D. B., ed., 1978. Maize Breeding and Genetics. Wiley, N.Y.

Hafliger, E., ed., 1979. Maize. CIBA-GEIGY Monograph, Basle, Switzerland.

Henderson, C. B., 1980. Maize Research and Breeders Manual No. IX. Illinois Foundation Seeds, Inc., Box 722, Champaign, Illinois 61820.

Hallauer, A. and J. B. Miranda, 1981. Quantitative Genetics in Maize Breeding. Iowa State Univ. Press, Ames.

Sheridan, W. F., ed., 1982. Maize for Biological Research. Plant Molec. Biol. Assoc., Box 5126, Charlottesville, Virginia 22905.

Coe, E. H., Jr., D. A. Hoisington and M. G. Neuffer, 1982. Linkage map of corn (maize) (Zea mays L.). Pp. 377-393 in S. J. O'Brien, ed., Genetic Maps, vol. 2, National Cancer Institute, Frederick, Maryland.

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Announcement

Dr. John Drake, editor of Genetics, has asked that certain specifications regarding nomenclature be brought to the attention of maize geneticists, for use in manuscripts directed to the Journal. First, it is important that the distinction be kept clear between a symbol that is used to refer to a locus generally and a symbol used to refer to the specific allele (e.g., the reference allele) under discussion; in text, it would be desirable to use the term "locus" after the symbol when locus is meant, and "allele" whenever the usage might be ambiguous. Second, the presence of a numeral after the character symbol (e.g., a1), is preferred, for clarity and unambiguous specification. Third, chromosome numbers will routinely be italicized (i.e., underlined) in the Journal; maize historically has been an exception to this standard, and the Journal wishes to standardize this practice.

Corrigenda

In the article by Rhoades and Dempsey, MNL 57:14-17, the following four lines were inadvertently excluded from the printer's copy; please patch them into your copy at the bottom of page 14:

The remarkable feature of the chart is the restriction, with the possible exception of Ac2, of a positive interaction to responding and controlling elements belonging to the same mutable system. For example, the recessive a alleles, a-mrh, a-standard, a-m and a-mr, all produce an indistinguishable colorless aleurone in

In the map by Coe, Hoisington, and Neuffer, MNL 57:191A-191B, the position of TB-5La and b is shown between bm1 and bt1. The correct position, according to data of Beckett, is between v3 and bv1.

The article from Honolulu, MNL 57:49-51, should identify authorship to both C. Nagai and J. L. Brewbaker.

I. FOREWORD

Your "Secretary" (title assumed by previous, more humble editors) fell behind this year, and this issue was delayed accordingly - no alibi, only an apology. Over 1,000 copies of this largest-ever News Letter will be sent to maize genetics cooperators and colleagues around the world. Funds for preparation, reproduction and mailing are provided by the U. S. Department of Agriculture directly and through the Maize Genetics Stock Center. We are, all of us, grateful beneficiaries of this indispensable support and encouragement.

Mailing list revisions, to update and correct addresses and to include telephone numbers, are still in process at press time. If you have not returned the post card (or have not received the post card) confirming your name and address and providing your telephone number, please supply the necessary information for next year's list.

Fifty Years Ago in Old Zealand is not included this year (News Letters 4, 5, 6, 7 and 8, totalling 54 pages, were distributed in one year!). These issues are included in the microfilm of Nos. 1-29 and 33, available for $9.50 (simply send a check to Coe, made out to Maize Genetics). Back issues of No. 30 (1956) to date will be sent upon request.

Please note the corrigenda at the end of the Table of Contents.

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

The deadline for the next issue (number 59, 1985) is January 1, 1985. Reports normally 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 to readers. Items that may have been prepared as for formal publication, or that are not concise and specific, may be returned for reconsideration. Communications are received and assembled with minimum editing:

Tables, figures and charts must be compact, single-spaced, and ready for direct copying by the camera.
Double-space the text so that it can be retyped with greater ease.
References, if needed, should be incorporated in the text; they should be abbreviated but should include initials to facilitate indexing.

Acknowledgements first go to the Cooperators who have again contributed the richly diverse and interesting notes, and to those who have offered suggestions and corrections (not to mention patience and morale support!). The work and tending of this issue has been done by Shirley Kowalewski, who saw to most of the editing and compositional tasks in addition to the year-round office routine. Mary Nelson and Roxanne Streeter helped skillfully from their special previous experience and abilities in producing quality copy. Christopher Browne and Kathryn Chappell helped with numerous vital tasks. Help with proofing, suggestions and critiques was provided by Ming-tang Chang, Christine Curtis, Bryan Kindiger and Stephen Modena. Enhancement of this issue, through friendly terms with the computer and through development of ideas for the new emphasis on mapping, was aided by the skill and insights of David Hoisington. At University Printing Services, Don Wren has been a steady and helpful aid in insuring that this and past News Letters are done promptly and with high quality.

Quotation of the year:

"Heterozygotes for big rings are homozygous for cob." (C. R. Burnham)

With wishes for a better season than ever.

E. H. Coe, Jr.

II. REPORTS FROM COOPERATORS

ALEXANDRIA, EGYPT
Alexandria University

Peroxidase isozymes in relation to resistance to stalk-rot disease caused by Fusarium moniliforme in maize inbred lines

Six local maize inbred lines were infected under field conditions with F. moniliforme to estimate their degree of resistance or susceptibility to stalk-rot disease. On the basis of a two-year test, the inbred G-102 was found to be rather resistant while inbred G-307 was found to be susceptible.

Peroxidase isozymes of these two inbreds were examined. No differences in the number or the activity of isoperoxidase bands were detected in the extracts of 2-week-old seedlings of these two inbreds. However, in the mature plants G-102 showed three cathodal isoperoxidases, but G-307 showed only one. Furthermore, among the cathodal isoperoxidases, C3 showed specific activation in relation to infection with F. moniliforme. The densitometric scanning of the C3 isoperoxidase showed about a 7-fold increase in its activity in infected plants of G-102 when compared with the uninfected plants. Such stimulation effect was absent in the infected plants of the susceptible inbred G-307.

From the present results, it was concluded that the stimulation effect observed for C3 isoperoxidase is a resistant specific reaction, occurring in maize inbreds resistant to stalk-rot disease caused by F. moniliforme.

F. A. Rakha, A. El-Metainy and N. Sabrah

AMES, IOWA
Iowa State University

C-I changes

C-I has long been used in genetics experiments. The frequent sectoring seen when C-I is used as a male on C C is a common observation. Very likely C-I is a stable insert that only occasionally excises.

In the same isolation plot described in the report on the wx alleles (see wx report), several new C-I alleles have been rescued. There are several classes:

A. C-I-m: C-I-m836526/C = Colored with colorless sectors; C-I-m836553/C = Colored with colorless sectors. Both are basic colored alleles that change to colorless (C-I). There is a remote possibility that these are basic c alleles that are mutating to C-I colorless.

B. C: C-I836683, C-I836684, C-I836685, C-I836934, C-I836955, C-I836956, C-I836958, C-I836959, C-I836960, C-I836969, C-I836970. These 11 C derivatives are seemingly stable, but some may belong to the C-I-m category.

C. C-sh: C-sh-836882/C sh = purple colored, shrunken. This is a coincident change from C-I Sh to C sh. The female transmission is normal. As with all the others of this series, each parental source ear was C-I Sh/C-I Sh.

D. C-I-unst: C-I-unst836511, C-I-unst836513, C-I-unst836518, C-I-unst836522, C-I-unst836524, C-I-unst836811. Each of these alleles is basically C-I that is unstable, giving colored sectors. Some show changes to sh and bz indicating multiple loss events. Outcrosses to color lines show heavy loss of the C-I allele.

There are two new sh alleles from the same plot: sh-836660 and sh-836673. Along with the C-sh-836882 allele, the three sh alleles arose in the test plot at the rate of one in 1.3 x 10^6 kernels, which is a lower frequency for sh than that in the 1975 tests but close to the 1983 report (MNL57:2).

Four new c2-m alleles have been recovered and confirmed: c2-m836018, very fine, low rate; c2-m836019, very fine, low rate; c2-m836024, infrequent spots; c2-m836039, infrequent spots. These four alleles arose out of crossing block C2 C2 x c2 c2 at a rate of one per 4 x 10^5--a rate close to the rate of the previously rescued five c2-m alleles (MNL57:2).

Peter A. Peterson

wx alleles newly originated from an En-containing plot

In a search for newly originated wx alleles, an isolation plot with genetic makeup with respect to anthocyanin genes C-I C-I R R A2 A2 A A C2 C2 En En was crossed by a C sh bz wx pollen parent. Five wx kernels were found and these were rescued in confirmatory tests for the authenticity of the pollen parent and the wx phenotype. Each of these does not show mutability, but extensive tests have not been concluded that would uncover very few, late changes (such late, infrequent spotting has been found among several of our c2-m isolates but the c2-m is very readily confirmed.)

These five wx alleles are: wx-836605, wx-836608, wx-836610, wx-836616, and wx-836617. Each of these wx alleles can be distinguished from the wx tester allele in a colorimetric assay.

These five wx alleles originated at the rate of one in 7.8 x 10^5 kernels.

Peter A. Peterson and Jean Cormack

An autonomous En-controlled mutable, bz-826301

The En relationship (MNL57:2) has been confirmed by the following series of crosses:

#1 bz-m826301 x Bz a2-m(r)

#2 Colored F1 x a2 bt gave a2 variegated, a2 non-variegated

#3 a2 variegated x bz gave 44 variegated bz, 0 non-variegated bz
a2 non-variegated x bz gave 2 variegated bz, 27 non-variegated bz.

The two exceptions are being tested for seed classification verification.

A consistent low frequency of bz stables arises in testcrosses. These are being tested for En content as possible bz-m(nr) types.

Peter A. Peterson

Autonomous mutable c2-m3

McClintock reported that c2-m3 was not related to Ac, Spm or Dt (pers. comm., May, 1971). Tests with a-ruq, a-m-rh, o2-m(r) and a-m(r) are negative. No mutability is observed with these receptor alleles of the Uq, Mrh, Bg, and En systems, respectively. Thus far, c2-m3 behaves as an autonomous mutable.

Peter A. Peterson

Uq-controlled mutable allele at the c locus

c-m816667 is one of three confirmed c unstable mutants and one stable mutant from the 1980 c sh wx isolation plot. This plot contained Uq and its receptor a-ruq (Peterson and Friedemann, Maydica 26:213-249), and produced 1.85 x 10^6 kernels. c-m816667 displays spots on a colorless background. This c allele material was shown to contain one Uq when crossed with a-ruq, as follows:

$$\frac{\text{c-m Sh Wx}}{\text{c sh wx}} \frac{\text{a Uq}}{\text{A +}} \times \frac{\text{a-ruq C}}{\text{a-ruq C}}$$

The Uq relationship was established by backcrossing a-ruq spotted and colorless kernels to the c sh wx tester (Table 1, A and B, respectively). If c-m816667 is

Table 1 Uq relationship test for c-m816667. Frequency of ears showing c mutability versus colorless from the cross $\frac{\text{c-m Sh}}{\text{C Sh}} \frac{\text{a-ruq}}{\text{a}}$ (+/-) Uq by $\frac{\text{c sh wx}}{\text{c sh wx}} \frac{\text{A}}{\text{A}}$.

Cross (1982-family #'s)	c locus mutable	c locus colorless
A. a spotted selection		
c-m Sh a-ruq Uq / C Sh a +		
1. 1016	5	0
2. 1017	4	0
3. 1020	4	2
4. 1021	8	0
5. 1024	5	1
6. 1025	4	0
7. 1028	4	0
8. 1029	5	0
B. a colorless selection		
c-m Sh a-ruq + / C Sh a +		
1. 1018	0	4
2. 1019	0	5
3. 1022	0	7
4. 1023	2*	3
5. 1026	0	4
6. 1027	0	5
7. 1030	0	4
8. 1031	0	3

* Two ears from the "no Uq" selection showed c mutability.

controlled by Uq only the cross of spotted by c sh wx will express c mutability. This is confirmed in Table 1A. The colorless a-ruq selections indicate that Uq was absent. The confirmation test would then be expected to produce only the colorless c phenotype. This is largely confirmed, but there were two exceptions (Table 1B). These selections had been made from an ear which exhibited variable Uq spotting frequencies. The possibility exists that these two were a-ruq Uq with low frequency spotting rather than a-ruq, no Uq colorless. This was tested by backcrossing to a-ruq. The cross was as follows:

$$\frac{\text{c-m Sh Wx}}{\text{c sh wx}} \frac{\text{a-ruq}}{\text{A}} \frac{\text{(+/-)Uq}}{\text{+}} \times \frac{\text{a-ruq C}}{\text{a-ruq C}}$$

This cross exhibited a-ruq Uq spotting. It can be concluded that these two selections were misclassified. These should have been grouped with the selections in Table 1A.

In each of the ears of Table 1A which shows c mutability, it has been confirmed that Uq is segregating independently of the c locus. The spotting of c-m816667 is due to the interaction of c-ruq and Uq.

Elizabeth Oberthur and Peter A. Peterson

New c mutants: test of system relationship

Out of 1.85 x 10^6 kernels produced in the 1980 c sh wx isolation plot, four c mutants were obtained. In addition, there was also one c confirmed mutant obtained from a similar plot in 1979 which produced 3.1 x 10^6 kernels. Both plots contained a-ruq and Uq.

The mutants are:

c-m816665--spotted on a colorless background, Uq-controlled (see Table 2), Uq segregates independently of c locus

c-m816666--spotted on a colorless background, system relationship unknown

c-m816667--see previous note and Table 1

c-st817086--colorless, not responsive to Uq, responsiveness to other systems unknown

c-m804655--colored to colorless sectors, system relationship unknown

Table 2 Uq relationship test for c-m816665. Frequency of ears showing c mutability versus colorless from the cross

$$\frac{\text{c-m Sh a-ruq}}{\text{C Sh a-ruq}} \text{ (+/-) Uq by } \frac{\text{c sh wx A}}{\text{c sh wx A}}.$$

Cross (1983-family #'s)	c locus mutable	c locus colorless
A. a spotted selection		
c-m Sh a-ruq Uq / C Sh a-ruq +		
1. 0915	6	0
2. 0917	4	1
B. a colorless selection		
c-m Sh a-ruq + / C Sh a-ruq +		
1. 0916	0	8
2. 0918	0	2

c-m816665 is Uq related.

Currently system tests are being made for c-m816666, c-st817086, and c-m804655.

Elizabeth Oberthur and Peter A. Peterson

vp-m451, a mutable allele at the Vp locus

Viviparous-1 is a mutant that, in the homozygous state, prevents seed developmental arrest and has a colorless aleurone. Mutable viviparous kernels exhibiting small, numerous spots on a colorless background were uncovered in the progeny of a self-pollinated ear of a colored line (1980g 090-23). Crosses to vp and five aleurone testers (a, a2, c, c2, and r) confirmed this unstable as vp-m and as allelic to the Vp locus. The spotting pattern, demonstrated by single cell spots, indicates late mutation events. Some of the fully colored cells in the aleurone layer exhibit diffusion to adjacent cells.

Receptor alleles of five presently known controlling element systems were used to test the relationship of these systems to the regulatory element responsible

for the viviparous mutability. The general method of the crosses with vp-m was to cross a et with vp-m/+, A Et, and self the F1 and testcross onto a-mrh. In selfs, heterozygotes for a et +/A + vp-m segregate 3/16 spotted viviparous, but in the testcross to a-mrh give colorless with no spots. The recovery of colorless, no spot progeny, and the accompanying confirmation of the vp-m allele, demonstrate the lack of response of the Mrh receptor, a-mrh, to the regulatory element for vp-m. Similar crosses conducted with the receptor alleles for the known regulatory elements Dt, En, Fcu, and Uq were also negative.

Receptor element	System tested	Mutability
a-mdt	Dt	-
a-m1/a-m(r)	En	-
r-cu	Fcu	-
a-mrh	Mrh	-
a-ruq	Uq	-

Systems not confirmed are Bg, Ac, and several others.

Brian Scheffler and Peter A. Peterson

a-m13 and a-m16, additional Uq-responsive mutants

In the progeny of virus-affected plants showing aberrant ratio, Sprague found several mutant a alleles, some of which showed mutability. In addition to the a-ruq allele previously described (Friedemann and Peterson 1982, Mol. Gen. Genet. 187:19-29) two more, a-m13 and a-m16, displayed mutability. The new mutants, each with a distinctive pattern phenotype, as well as a-ruq, arose as separate individual events.

Tests of the relationship of these two alleles (a-m13 and a-m16), both independently controlled, to the En, Ac, Dt, Fcu, and Bg systems proved negative. Both mutable alleles, however, responded to the Uq regulator element of the standard Uq a-ruq system. In addition, the independent regulator elements of both a-m13 and a-m16 triggered the standard a-ruq allele. These tests demonstrate that both a-m13 and a-m16 belong to the Uq a-ruq controlling element system. It is interesting that each of the three independently isolated a unstables were Uq-responsive.

Andy Pereira and Peter A. Peterson

The multiple gene loss at the a-m61138-3 allele is due to En: test for the presence of Ac

The a-m61138-3 allele is autonomously mutable with En. Since this is a unique En, it will be identified as En-61138-3. This En shows a consistent low frequency of coincident loss of the a En Sh2 chromosome segment from chromosome 3 (1983 MNL). The remote possibility arose that this loss event, the first among maize mobile elements outside of the Ac Ds system, might be from the insertion of an Ac element within the a locus. To test this possibility the following experiments were executed: En-61138-3 was crossed to a C Ds sh-m stock (see Table 1). Half the F1 plants have both C Ds sh1-m and En-61138-3. If Ac was present, crosses of the F1 onto a c sh wx tester would express Ac.

Two different shrunken 1 genotypes are expected, one with En and the other with a-m-1 sh2. If Ac is present in En-61138-3, one-half of the colored shrunken kernels will be expected to show colorless sectors and sh1 to Sh1 sectors. However, in no case were colorless sectors found on colored shrunken kernels. The

data from the selfs of these F1 plants are also shown in Table 1. In six out of the twelve selfed progeny, the ratios with respect to total number of kernels were

Table 1. Phenotypic frequencies of the F_1 [$\frac{a^m En Sh_2}{a^{m-1} sh_2}$ $\frac{C\ Sh_1}{C\ Sh_1}$(En61138-3) x $\frac{A\ Sh_2}{A\ Sh_2}$ $\frac{C\ Ds\ sh_1^m}{C\ Ds\ sh_1^m}$(C Ds sh^m)] selfed progeny and the crosses of the F_1 on the cshwx/cshwx tester.

	Outcross onto cshwx/cshwx				Selfed progeny							
	Colored				Round		Shrunken				x^2-test	
							Colored		Colorless spotted			
number 1983	Round	Round & colorless sector	Shrunken (sh)	x^2-test (1:1)	Colored	Colorless spotted	sh_1	sh_2	sh_1	sh_2	9:3:3:1	9:3:4
1224-1	175		142	ns	147	65	42		12		ns	
-2	175		201	ns	228		110	108				**
-4	148	1	192	ns	142	61	46		21		ns	
-5	96		89	ns	310	4	87	80				**
-11	91		76	ns	106	41	37		17		ns	
-12	25		28	ns	225	77	60		21		ns	
-13	88	2	77	ns	191		55	80				ns
-14	151		133	ns	223	76	63		22		ns	
-15	169	2	155	ns	186		75	94				ns
1359-4	173		167	ns	198	85	75		36		ns	
-6	43		30	ns	167		50	59				ns
-9	109		93	ns	194		69	100				ns

**: 1% significant level.

ns: nonsignificant

3/16 colorless round spotted and 1/16 colorless shrunken spotted. The other 6 of the 12 tested plants giving rise to selfed progenies should be without En. This confirms that En-61138-3 is present in our male parents. At the same time, these same plants do not show C Ds losses when used as outcrosses onto the c sh wx tester. Thus, it is evident that Ac is not a component of the En-61138-3 complex. The multiple loss mutability of En-61138-3 that includes a1, sh2 and En is due to En.

This En-61138-3 a1 allele arose as an individual event from the original a-m(dense) (Peterson, Genetics 1961). This is the only allele of the En systems to display chromosome breakage events leading to the loss of chromosome segments.

Yih Ching Huang and Peter A. Peterson

Nonreciprocal, partial cross-incompatibility

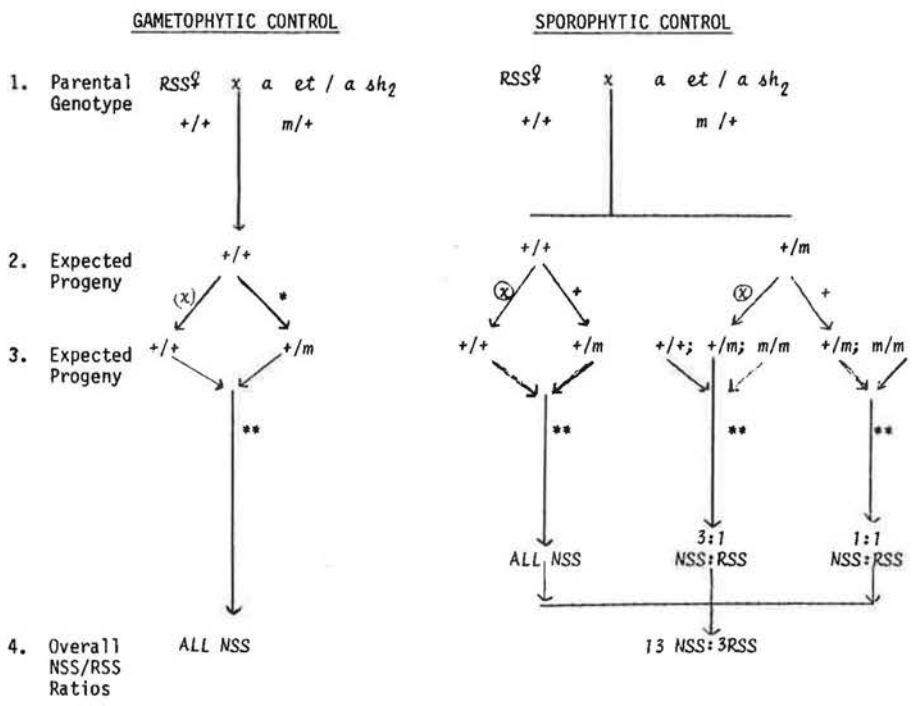
The phenotypic expression of this nonreciprocal, partial cross-incompatibility (NPC) is a reduced seed set (RSS) (Sukhapinda and Peterson, 1983). NPC occurs in our experiments only in crosses between certain parents: an En controlling element genotype or derivatives of it as a female--e.g., a-m(papu) (Peterson 1970, TAG) and a et/a et as male. A crossing design was developed to distinguish between gametophytic and sporophytic control. This is illustrated in Figure 1.

The results (Table 1) show a nonsignificant difference from the expected "all NSS" (Figure 1, line 4) if this incompatibility is gametophytically controlled.

Hence, it is concluded that the transmission of this incompatibility factor is under the gametophytic system of control.

Paul Bdliya and Peter A. Peterson

Figure 1. Diagram of crossing design used to distinguish between gametophytic vs. sporophytic control of incompatibility.



RSS♀(+/+) = confirmed reduced seed set female - a-m(papu)Sh derivative.

+ = compatible male gamete on RSS(+/+)female

m = incompatible male gamete on RSS(+/+)female

* = crossed by a et/a et (m/m), pollen homogygous for the incompatibility factor.

** = genotypes in line 3 used as males on RSS females.

Table 1 - Observed seed set (kernels per ear) in the cross RSS x various male genotypes (figure 1, line 3) for two years.

Year Tested*	Number of ears of each class (%)[a]			
	L=<25 K/ear	I=26-200 K/ear	H=>200 K/ear	Total
1982	35(10)	93(27)	212(63)	340
1983	14(7)	30(15)	151(78)	195
Total	49(9)	123(23)	363(68)	535

number in parenthesis is % of total

* = Nonsignificant difference between years

a = Nonsignificant difference from the expected 'all NSS' under gametophytic control. (figure 1 line 4)

bz-m805137 A bronze mutable of the Cy system

A bronze kernel with fully colored spots arose in the 1979 isolation plot from the cross of C Sh Bz Wx (63 1005/828) X C sh bz wx. The new bronze mutable allele has been designated bz-m805137 (P. A. Peterson, MNL 57:2). Mutability of this allele is controlled by an independent element designated Cy. In the absence of Cy, bz-m805137 conditions a stable bronze phenotype. This original isolate is a responsive allele, designated bz-rcy. In the presence of Cy a fine-high pattern is produced by the original isolate. Numerous heritable patterns characterized by a reduced frequency of mutation, and in some cases by earlier mutation, have been isolated. Several of these have been shown to be conditioned by the state of the allele.

Table 1. Test of the interaction of the Cy system with the En, Ac, Uq, Mrh, and Dt controlling element systems.

Other regulatory elements against bz-rcy

	En	Ac	Uq	Mrh	Dt
bz-rcy	-	-	-	( )	-

Other receptors against Cy

	a-m-1 a-m(r) a2-m(r)	bz-m4	a-(ruq)	a-mrh	a-mdt
Cy	-	-	-	-	-

- indicates no interaction
( ) indicates that test is still in progress

The isolate bz-rcy is not triggered to produce spots by the regulatory elements En, Ac, Uq, or Dt. Cy does not trigger mutability at En, Ac, Uq, Mrh or Dt responsive alleles. These data are summarized in Table 1. These results place bz-m805137 as a newly described system, subject to negative relationships to the Mut, Bg and Fcu systems.

Patrick S. Schnable and Peter A. Peterson

Mutable allele bz-m794226

bz-m794226 was previously uncovered from the same population (63 1005/828) as bz-m805137. Mutability at this allele has been shown to be under independent control. In the presence of the appropriate regulatory element the allele conditions a fine-high spotting pattern. In the absence of the regulatory element the phenotype is a stable bronze. No controlling element system has been tested to completion, and as such we will not designate a system.

Patrick S. Schnable and Peter A. Peterson

Anther fluorescence induced by bz

According to E. H. Coe, Jr. and M. G. Neuffer (Corn and Corn Improvement, G. F. Sprague, ed.), anther fluorescence is displayed by bz-null alleles. The basis for this fluorescence is variously ascribed as likely to quercetin (Coe and Neuffer, Corn and Corn Improvement), or not ascribed to quercetin (R. L. Larson and E. H. Coe, Jr., Biochem. Genetics 15:153-56).

We have determined the following:

Genotype	Anther Fluorescence	Reference
bz-ref/bz-ref	+	Coe and Neuffer
bz-ref/Bz	-	Coe and Neuffer
bz-m4/bz-m4	-	Coe has observed variable fluorescence--pers. comm.
bz-m4/bz-ref	+, mild	

These results are not surprising in view of H. K. Dooner's finding that bz-m4 is not a null allele (Cold Spring Harbor Symp. Quant. Biol. 45:457-462).

Patrick S. Schnable and Peter A. Peterson

A possible technique for isolating genic DNA for quantitative traits

I have found that many plant breeders feel that modern genetic engineering techniques will be useful for locating genic DNA for qualitative traits, such as disease resistance. However, they do not foresee much hope of isolating genic DNA for the genes controlling quantitative traits. Because the expression of quantitative traits involves many genes, each contributing only a small effect to the total phenotype, they feel it will be impossible to screen for the effect of a single quantitative gene. If quantitative genes are in a class by themselves and are not related to those with which the geneticist deals (i.e., qualitative genes), this might be true. To me, however, it seems unreasonable (to say nothing of wasteful) to assume a plant has two sets of loci, one for quantitative traits and one for qualitative traits, when one set could account for both patterns.

Let us, for instance, consider a trait like plant height. If one tests the inheritance of plant height in crosses between a tall and a short genotype, it would be found that plant height is indeed a quantitatively inherited trait. The geneticist, however, knows of many loci with qualitative mutants for plant height (e.g., dwarfs, brachytics, etc.). The numbers of such loci approach those suggested for the number of quantitative loci assumed. I would like to suggest that the qualitative mutants of the geneticist may be just null or near null alleles of the quantitative loci of the plant breeders. Molecular geneticists have shown that at the DNA level there is probably no one wild type allele (Johns, Strommer and Freeling, Genetics 105:733-743, 1983; Burr, Evola, Burr and Beckman, Genetic Engineering: Principles and Methods, Vol. 5, 1983). For those loci in which the DNA has been isolated, it has been determined that there is extensive polymorphism at the base pair level in the wild type alleles. All of these alleles produce active gene products resulting in what is recognized as wild types. Undoubtedly, there is variation in the relative efficiency of the gene products of these wild type alleles. If two inbred lines have different sets of wild type alleles producing functional products with different efficiencies, it seems reasonable to expect a quantitative pattern of inheritance. Thus, variation at the base pair level that does not eliminate the production of a functional gene product produces quantitative alleles. However, a mutation at one of these loci resulting in a gene product that has lost all or most of its function will result in a qualitative mutant.

If this suggested relationship is real between qualitative and quantitative genes, it would be possible to isolate the DNA for quantitative genes. If one uses Mutator or a similar system to induce a dwarf mutant, one would potentially be able to isolate the DNA for the wild type allele of the dwarf locus and, hence, the DNA for a quantitative gene for plant height.

Similar experiments could be done for genes involved in yield. Seed size is one important component of yield. Possibly the many defective seed mutants are qualitative alleles of loci responsible for the quantitative inheritance of seed size. Perhaps the photosynthetic mutants (hcf) are qualitative mutants for quantitative genes controlling photosynthetic efficiency.

This is only a brief description of a hypothesized concept of the relationship between quantitative and qualitative genes and some of the implications of this model. There are many other aspects of this relationship, which have not been considered here, but will be incorporated in a more in-depth paper that is being prepared.

Donald S. Robertson

Putative forward mutation frequencies at the y1 and wx loci in the presence of Mu

To determine the influence of Mu on the frequencies of forward mutations at the y1 and wx loci, a large isolation plot was set up in which the seeds of the female rows consisted of Y1 Y1 Wx Wx Mu plants. Tests had established that the parents of these plants had mutator activity. Y1 Y1 Wx Wx control rows consisted of the two standard stocks used in the propagation of Mu lines. The pollen rows were y1 y1 wx wx gl8 gl8. The ears of the female rows were screened for white starchy and yellow waxy seeds. Using graph paper, ear maps were made of each ear with one or more mutant seeds. The map arbitrarily started with the paired rows to the left of the paired row with a mutant seed. The butt was the bottom of the map and the tip the top. The total number of seeds was estimated by shelling and weighing all shelled seeds. Numerous samples of about 1 lb were taken and the seeds counted to obtain the number of seeds per lb. These values were then used to calculate the total number of seeds. The results of these tests are shown in Table 1.

Table 1. Putative Mu induced y1 and wx mutants.

Mutator							
Total ears	Total seeds	Total white, pale yellow and questionable pale yellow seeds		Total white and pale yellow seeds		Total wx	
		Total	Sector (counted as one event)	Total	Sector (counted as one event)	Total	Sector (counted as one event)
3257	1,768,386	419	314	406	304	51	7
Frequencies:		2.4×10^{-4}	1.8×10^{-4}	2.3×10^{-4}	1.7×10^{-4}	2.9×10^{-5}	4.0×10^{-6}
Average seeds per ear = 542.9							
Control							
4311	2,578,391	20	0	3	0	0	0
Frequencies:		7.8×10^{-6}	-	1.16×10^{-6}	-	-	-
Average seed per ear - 595.3							

The most conservative estimate of the frequency for y1 mutants in Mutator stocks is 1.7×10^{-4}. This is more than a 50-fold increase in the mutant frequency over that observed in the control. The wx mutation frequency in the presence of Mu was much lower (4.0×10^{-6}). This is low enough to be a spontaneous mutation frequency for many loci. However, it is higher than the zero

value found in a very large control population. It is obvious that these two loci have different sensitivities to Mu.

For both the y1 and wx loci there were ears in which more than one mutant seed occurred in close proximity on the ear (i.e., a sector). These are undoubtedly due to late somatic mutations. The size distributions of these sectors are shown in Table 2.

Table 2. Distribution of sector sizes for y1 and wx mutants.

	Number of Seeds in Sector													
	2	3	4	5	6	7	8	9	10	11	12	13	14....29	
Mutator - y1														
	24	6	4	2	1	2	1	2	0	0	0	0	0	0
Mutator - wx														
	0	0	0	1	0	0	0	0	0	0	0	1	0	1
No sectors in controls														

For the y1 locus the vast majority of events were singular. When sectors did occur most were small in size. This confirms earlier observations that Mu acts in late mitotic stages, or meiosis, or later. The one sizable sector (14 seeds) may be a spontaneous mutant. Most of the wx mutants, on the other hand, are found in sectors. Only four single waxy mutants were found. Could the large wx sectors be the result of spontaneous mutations not induced by Mu? This seems unlikely since large sectors would also be expected in the controls, and yet none were found. It is possible that loci not only differ with respect to Mu sensitivity, but they may also differ with respect to when in ontogeny a locus is sensitive to Mu.

One hundred three of the y1 mutator-induced mutants were grown last summer, self-pollinated and outcrossed to a yellow endosperm standard. Seeds from the selfed ears were grown at 95 F. Under these conditions the pastel alleles of y1 (y1-pas8549, y1-mut and pb1) are pale green. These alleles, when grown at 70 F, are green. Fourteen of the y1 mutants segregated pastel seedlings at 95 F and only green seedlings at 70 F. One rather strange feature of the y1 mutants was the absence of any mutable y1 seeds. In a random sample of Mu-induced seedling mutants, about 40% will be mutable. Yet in spite of screening every y1 mutant under magnification, no mutable mutants were found. However, some of the pastel mutants were mutable (similar to y1-mut). Thus, there are indeed Mu-induced y1 mutables. Why were they not obvious in the endosperm, as is the case with y1-mut, which does have a mutable endosperm? The mutable endosperm phenotype of y1-mut has rather large sectors of yellow. On the other hand, Mu-induced mutable aleurone mutants, at several loci, have very small round spots. Such a pattern may not be readily recognizable at the y1 locus if the reversions are occurring primarily in the interior of the endosperm and at a low frequency. (As expected, all the ears segregated for glossy seedlings.)

Also, no obvious mutable waxy mutants were found. However, none of the waxy mutants were stained with iodine to check for mutability. This will not be done until the mutants have been increased. However, in the next report we present evidence that at least one of the waxy mutants might be mutable (i.e., unstable).

Donald S. Robertson and Philip Stinard

"Reversion" frequency of plants homozygous for the standard waxy and y1 alleles in the presence of Mu

Definition: The term reversion in this report refers to the appearance of a seed or pollen grain with the dominant starchy phenotype in stocks that originally had only recessive waxy alleles. Reversion is not synonymous with the term "back mutation" since we have not demonstrated if the mutant waxy or y1 alleles have indeed been restored to an allele responsible for the wild phenotype (i.e., Wx or Y1). For convenience' sake, we will speak of wx and/or y1 "reversions", but such terminology refers to phenotype only and the foregoing caveat must be kept in mind.

In an isolation plot, y1 y1 wx wx Mu seeds from outcross ears of plants that had been tested and found to have Mu were used as female rows. The pollen rows were y1 y1 wx wx plants without Mu. Both pollen rows (controls) and female rows were screened for yellow waxy (y "reversions") and starchy white (wx "reversions") seeds (See Table 1). The "reversion" frequencies of both y1 and wx are slightly

Table 1. "Reversions" frequencies of standard y1 and wx allels in Mu plants as observed in seeds.

Total No. of gametes	y1 "reversions" yellow	Possible yellow	wx "reversions"
Mutator 718,972	6	6	2
Corrected values*	4+1 seed that did not germinate	0	2
Frequency	a.* 6.95×10^{-6}		2.78×10^{-6}
	b.* 5.56×10^{-6}		
Controls 1,580,460	3	3	1
Corrected values	2	0	1
Frequency	1.27×10^{-6}		0.6×10^{-6}

* All putative "reversion" seeds were planted and the resulting plants self pollinated and outcrossed to a white waxy stock to confirm the original classification. Some proved not to be "reversions", hence, the corrected values. One yellow waxy seed in the mutator stock did not germinate. Frequency (a) assumes that seed was a true "reversion"and frequency (b) that it was not.

higher in the Mu stocks. If Mu is acting at the wx and y1 loci, it would appear that the standard wx allele is more resistant to the action of Mu than the standard y1 allele.

Another way to measure the "reversion" under the influence of Mu is by determining the frequency of Wx pollen grains in wx wx Mu plants using the pollen staining technique. Waxy Mu lines were produced by crossing Mu stocks as a male to yellow waxy plants. The F1 mutator plants were again crossed to a yellow waxy. Most of the waxy seeds that resulted were wx wx Mu plants. A waxy line that has been used in our program for many years served as the source of the standard wx allele. Twelve standard waxy stocks with Mu and six standard waxy stocks without Mu (controls) were studied.

The pollen staining and counting techniques used were those described by Nelson (Genetics 60:507-524, 1968), and Yu and Peterson (Theor. Appl. Genet. 43:121-133, 1973) with minor modifications.

The results are summarized in Tables 2 and 3. The "reversion" frequency of the standard waxy stock with Mu (Table 2) was higher than that of the control (Table 3). The control pollen "reversion" frequency was higher than the control of the

Table 2. The frequency of phenotypic "reversions"of the standard waxy allele in pollen of putative Mu plants.

Plant No.	No. of waxy pollen grains	No. of non-waxy pollen grains	Total	Frequency of non-waxy pollen grains ($x10^{-5}$)
5866-1	259,253 85	114.60	259,368.45	44.18
-3	206,394.60	429.75	206,824.35	207.79
-5	243,610.95	28.65	243,639.60	11.76
-7	278,678.55	57.30	278,735.85	20.56
-8	242,837.40	57.30	242,894.70	23.59
Total	1,230,775.40	687.60	1,231,463.00	55.84
5867-2	194,705.40	28.65	194,734.05	14.71
-3	236,247.90	85.95	236,333.85	36.37
-4	250,859.40	0.00	250,859.40	0.00
-6	174,736.35	0.00	174,736.35	0.00
-7	205,105.35	0.00	205,105.35	0.00
-8	241,462.20	114.60	241,576.80	47.44
-9	206,967.60	401.10	207,368.70	193.42
Total	1,510,084.20	630.30	1,510,714.50	41.72
Total minus zero freq. plants	879,383.10	630.30	980,013.40	71.62
Grand total	2,740,859.60	1,317.90	2,742,177.50	48.06
Grand total minus zero feq. plants	2,110,158.50	1,317.90	2,111,476.40	62.42

Table 3. The frequency of phenotypic "reversions" of the standard waxy allele.

Plant No.	No. of waxy pollen grains	No. of non-waxy pollen grains	Total	Frequency of non-waxy pollen grains ($x10^{-5}$)
9109-1	276,042.75	28.65	276,071.40	10.38
-7	246,934.35	0.00	246,934.35	0.00
Total	522,977.10	28.65	523,005.75	5.48
9111-1	299,077.35	0.00	299,077.35	0.00
9112-2	255,242.85	0.00	255,242.85	0.00
9113-5	349,787.85	0.00	349,787.85	0.00
-6	316,009.50	0.00	316,009.50	0.00
Total	665,797.35	0.00	665,797.35	0.00
Grand Total	1,743,094.70	57.30	1,743,152.00	3.29

seed experiment (Table 1). A great deal of variability in the "reversion" frequency existed among the plants of the families in Table 2. In some, no

"reversions" were observed. Since Mu is lost in about 10% of Mu outcrosses, these plants with no "revertants" could be those that had lost Mu. The outcrosses of all plants will be tested for the presence of mutator activity to determine if Mu loss can account for the difference in "reversion" frequencies. The waxy Mu stock (Table 2) has a higher pollen "reversion" frequency than the same genotype studied at the seed level (Table 1). The reason for the discrepancy between these two frequencies (i.e. pollen and seed) is not known. The wx wx Mu stocks in the two studies were in different backgrounds, and thus there may be background differences. The standard control stocks, however, were the same in the seed and pollen tests, and thus the higher pollen "reversions" frequency in the pollen controls than in the seed control cannot be explained by background differences.

The evidence from both seed and pollen studies suggests that Mu may be responsible for phenotypic "reversions". In our studies, "reversion" frequencies in pollen were higher than those observed in seeds. Perhaps "reversion" in many instances is associated with a pollen lethal condition.

Donald S. Robertson and Solomon K. Sackitey

"Reversion" frequency of putative Mutator induced waxy alleles

Twelve plants from a putative Mu induced sector of 13 seeds were scored for wx "reversions" in the pollen. (Please see the previous paper for the definition of "reversion", for a description of the pollen staining techniques and for the standard waxy allele "reversion" frequencies.) The origin of these putative Mu-induced new waxy mutants has been reported in the previous article entitled: "Putative forward mutation frequencies at the y1 and wx loci in the presence of Mu".

Plants of this sector are heterozygous for new mutant Mu induced alleles and the standard wx allele. This would create a heteroallelic situation in which, in many instances, previous workers have reported results of elevated "reversion" frequencies as compared to those found in homoallelic plants. This elevated rate is thought to be due to intragenic recombination. Such an elevated frequency is not observed here (Table 1). In fact, the frequency in the heteroallelic plants is about half that seen in the wx wx Mu plants (Table 2, previous paper). Thus, the observed "reversion" frequency in these Mu plants heterozygous for the standard waxy and putative Mu-induced waxy alleles can be explained on the basis of the action of Mu on the standard allele alone (Table 2, previous paper). This suggests that the putative Mu-induced waxy allele is not "reverting" at an appreciable frequency nor is there intragenic recombination occurring.

Two plants from single seed mutants from different ears (Table 2) were screened for the Wx "reversions", and it was observed that not only did plant 8109-1 have a higher frequency than any single plant in this study, but it also was higher than has been reported for waxy alleles in any other homoallelic or heteroallelic condition. This high frequency could be due in part to the heteroallelic condition of this plant. But this is probably not the full explanation. Because mutable mutants occur in about 40 percent of all Mu-induced mutants, it could be that the allele of 8109-1 is unstable (mutable) and that this instability is expressing itself in the germ line. The somatic mutability observed for most Mu-induced mutable mutants is very late and is frequently a rare event. Thus, we probably would not recognize a mutable waxy seed visually without staining. Since we did not want to run the risk of impairing the germination of these new waxy mutants, the seeds were not stained with iodine. A selfed ear and an outcross to a y1 y1 wx wx stock did not reveal any wx seeds, but the number of seeds obtained was small. After this mutant has been increased, more extensive studies of its germ line stability as measured in both pollen and seed can be undertaken.

Table 1.. The frequency of phenotypic "reversions"of putative Mu induced waxy alleles found in plants from a 13-seed ear sector. (One seed did not germinate).

Plant No.	No. of waxy pollen grains	No. of non-waxy pollen grains	Total	Frequency of Non-waxy pollen grains ($x10^{-5}$)
8107-1	283,146.95	143.25	283,291.20	50.57
-2	342,252.90	0.00	342,252.90	0.00
-3	111,162.00	0.00	111,162.00	0.00
-4	303,317.55	0.00	303,317.55	0.00
-5	301,426.65	114.60	301,541.25	38.00
-6	305,695.50	28.65	305,724.15	9.37
8108-1	269,453.25	57.30	269,510.55	21.26
-2	293,891.70	143.25	294,034.95	48.72
-3	270,828.45	85.95	270,914.40	31.73
-4	365,609.55	28.65	365,638.20	9.37
-5	259,884.15	0.00	259,884.15	0.00
-6	247,736.55	28.65	247,765.20	11.56
Total	3,354,406.20	630.30	3,355,036.50	18.79
Total minus zero ferq. plants	2,337,789.60	630.30	2,338,419.90	26.95

Table 2. The frequency of phenotypic "reversions"of two putative Mu induced waxy alleles found on separate ears.

Plant No.	No. of waxy pollen grains	No. of non-waxy pollen grains	Total	Frequency of non-waxy pollen grains
8109-1	271,200.90	2922.30	274,123.20	0.010
8109.1-1	315,952.20	0.00	315,952.20	0.00

The other plant, from a single seed mutant 8109.1-1, was observed to be very stable. Why "reversions" to the wx phenotype in this plant did not take place is not known.

In summary, it seems that two classes of putative Mu-induced waxy alleles have been found. Those that appear to be quite stable and those that are apparently germinally unstable.

Solomon K. Sackitey and Donald S. Robertson

Stability of Mu induced mutants

Reports differ as to the stability of Mu-induced mutants. Ben Burr (pers. comm.) has found a high reversion rate of Mu-induced sh1 mutants. Freeling has reported that some Mu-induced Adh1 mutants are unstable. In the previous report, we described a putative Mu-induced wx mutant that appears to be highly unstable.

In the last few years, we have tested three putative Mu-induced mutants for their reversion rates to their respective wildtype alleles. Putative Mu-induced gl1 and gl8 mutants and standard gl1 and gl8 alleles were transferred to a y1 wx background. Each of these glossy white endosperm waxy stocks were grown in

isolation and allowed to self-pollinate. The resulting white waxy seeds were grown in the seedling bench, and the frequency of non-glossy seedlings was determined. The non-glossy seedlings were transplanted to pots and the pollen of mature plants checked to confirm that they were homozygous waxy and, when possible, either self-pollinated or outcrossed to a y1 wx stock. Any plant that did not test homozygous for waxy (and white endosperm when this test was possible) was not counted as a reversion. The results of these tests are given in Table 1.

Table 1. Back mutation of putative Mu-induced gl1 (gl1-5048) and gl8 (gl8-3134) mutants and the standard gl1 and gl8 alleles.

Allele	Total Gametes Tested	No. of Gl	Frequency of Gl
gl1	343,485	1	2.9×10^{-6}
gl1-5048	397,609	0	
gl8	493,478	0	
gl8-3134	405,222	3	7.4×10^{-6}

There is no indication that the Mu-induced gl1 mutant has a higher mutation rate than the standard allele. The gl8-3134 allele may have a slightly higher reversion frequency than the standard gl8 allele. A larger population of gl1-5048 will have to be grown to determine if this mutant ever reverts to its Gl allele.

A putative bt2 Mu-induced allele (bt2-2626) and the standard bt2 allele were transferred into a y1 wx gl1 background. Once these stocks were produced they were either self-pollinated or sib-crossed and the resulting seeds were scored for white waxy non-brittle seeds. These putative reversion seeds were seedling tested to determine their glossy constitution. The results of these tests are given in Table 2. It is obvious that the bt2-2626 allele is very unstable compared to the

Table 2. Reversion frequency of a putative Mu-induced bt2 allele (bt2-2626) and the standard bt2 allele.

	wh bt	wh wx Bt gl	wh wx Bt Gl	wh wx Bt No germination	Total Seeds	Total Frequencies
bt2-2626	9501	55	2	22	9580 x2	
				Total Gametes	19,160	
Frequencies		2.9×10^{-3}		1.1×10^{-3}		3.0×10^{-3}*
Frequency minus one ear segregating 1:1 wh wx Bt : wh wx bt		41 2.1×10^{-3}		16 0.8×10^{-3}	19,080	2.9×10^{-3}*
Standard bt2	11036	0	0	0	11036 x2	
				Total Gametes	22,072	

*These values assume all seeds that did not germinate would have been reversions.

standard allele. The reversion frequency of this allele is between 2-3 x 10^{-3}. One selfed ear segregated 20 white, brittle:20 white, waxy, nonbrittle seeds. Since a white, waxy brittle seed was planted, the only way a 1:1 ratio would result is if the bt2-2626 allele reverted in a progenitor cell that gave rise to

all of the germinal tissue of the ear or the tassel. Thus the gametes of one inflorescence would be heterozygous for the Bt revertant allele and the other would be homozygous for the mutant bt2-2626 allele. All other ears with Bt seeds had only one or two or, at the most, 3 Bt seeds. Because selfs and sibs were involved and also because seed set was irregular on most ears, sectors could not be recognized. The high frequency of single reversions and possible small sectors with reversions suggest that reversion (back mutation?) occurs late in somatic development or possibly in meiosis. This is the same time that Mutator seems to be active in producing forward mutations. There also have been some indications that Mu may also be active in early developmental stages. Thus, it would appear that both forward and reverse mutations occur at the same time in ontogeny in Mutator stocks.

The information obtained to date on the stability of Mu-induced mutants suggests that they are variable in this regard. Some appear to be similar to the standard allele, while others revert at a much higher rate than their standard counterparts, and still others appear to be extremely stable. About 40 percent of all Mu-induced mutants are known to be somatically unstable (i.e., mutable). It could be that these also are germinally unstable. This relationship between somatic and germinal instability, if any, as yet has not been established for Mu. Somatic instability in the endosperm is expressed as very small round dots. Such a pattern would not be discernible in the brittle endosperm. It would also be difficult to recognize instability in glossy plants because of the small size of the revertant areas normally seen in unstable Mu-induced seedling mutants. This past summer we tested several Mu-induced a1 mutable mutants to determine if there is a relationship between the rate of somatic reversions and germinal reversions. The results are yet to be analyzed. The stable gl1-5048 allele could possibly be a deletion mutant. Such a mutant might be expected if the Mu1 DNA insert transposed into, or near, the gl1 locus and then transposed again, taking with it some or all of the structural gene DNA and producing a deletion. We will have to wait on the molecular geneticist for answers to some of these problems with respect to the stability of Mu-induced mutants.

Donald S. Robertson

A recessive brown aleurone mutant (brn)

A self-pollinated ear from a Mu outcross population was found to be segregating for dark brown seeds in an approximate three to one ratio. The parent stock of this mutant is c c r r. Soaked brown seeds were dissected and the aleurone was found to be dark brown. The corneous endosperm frequently showed a slight tinge of brown pigmentation. The scutellum was also brown.

Yellow and brown seeds from the original ear were sown, and the plants were self-pollinated and outcrossed to my yellow standard line (c c r r). The germination of the brown seeds was very poor, and the few green plants that came up were very weak and died soon after emerging. Thus the trait appears to be a lethal. Pooled results from the selfs of several heterozygous ears gave 485 yellow:146 brown seeds. Of the 34 plants from the yellow seeds that were tested, 21 were heterozygous for brn. Thus, the limited data we have suggest it is indeed a lethal recessive aleurone mutant. I would be happy to supply anyone interested in this mutant with seeds of outcrosses made from heterozygous plants last summer.

Donald S. Robertson

A lethal a1 mutation

In a large test in 1981 in which a purple aleurone Mutator line was used as a pollen parent and a1 sh2 as the ear parent, several mutable and stable mutants were obtained. These new mutants were planted and self-pollinated. These selfed ears segregated for the new (Mutator-induced?) mutants and a1 sh2 seeds. One of the selfed ears segregated for dark, medium and light pale purple seeds among the Sh2 seeds. There was no sharp demarcation between these classes. Thus it was difficult to determine their actual frequencies. This mutant was given the symbol a1-SR03 (stable a1 mutant #3 produced by Robertson).

This mutant appears to be a leaky a1 allele. The different intensities of pigmentation could be due to the number of doses of a1-SR03 present (i.e., dark pale purple, a1-SR03 a1-SR03 a1-SR03; medium pale purple a1-SR03 a1-SR03 a1; light pale purple a1-SR03 a1 a1). To test this hypothesis, seeds of each class were planted and, where possible, the resulting plants were selfed and crossed to a1 a1 sh2 sh2 plants or they were reciprocally crossed to a1 a1 sh2 sh2. If the hypothesis is correct, the plants from dark pale purple seeds should be homozygous for the new allele and Sh2 while plants from the other two classes should be heterozygous (i.e., a1-SR03 Sh2/a1 sh2).

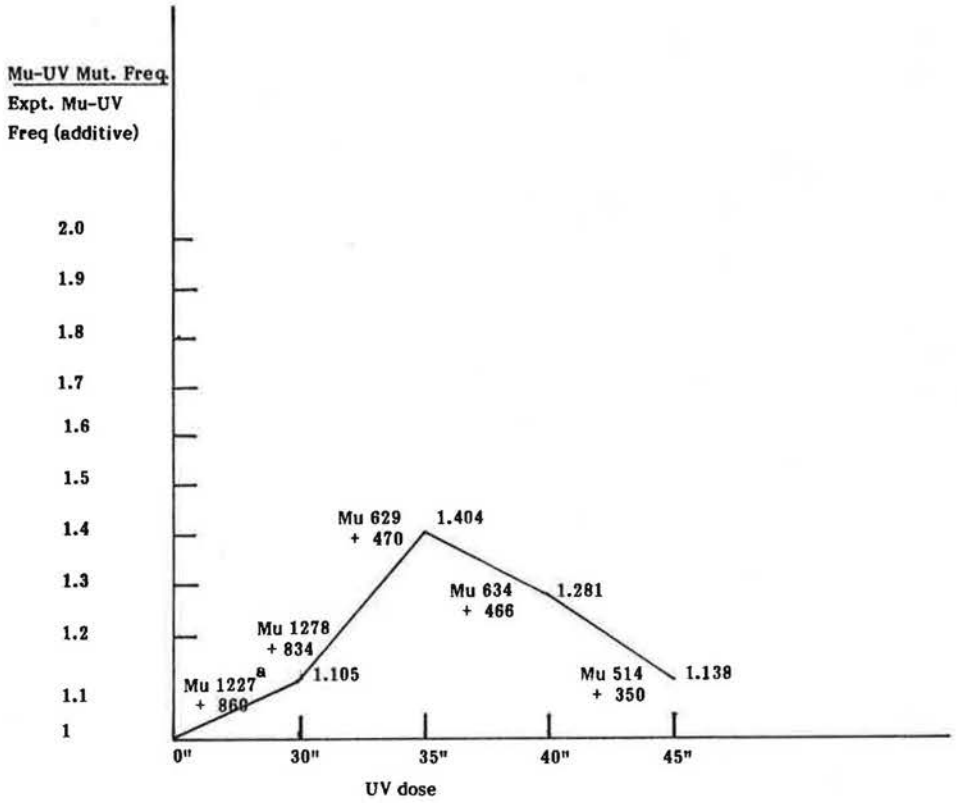
The medium and light pale purple seeds were, indeed, all heterozygous. However, most of the plants from the 30 dark purple seeds planted died under field conditions. Unfortunately I was confined to bed with a back problem during the early growth stages. By the time I was able to get to the field there were only two very weak runts surviving. Thus, I was not able to determine when and how those plants that died succumbed. Seedlings from dark pale purple seeds germinated in the sand bench give green, rather weak looking seedlings.

Thus, this possible Mutator-induced mutant at the a1 locus appears to be a lethal or semi-lethal mutant.

Donald S. Robertson

The effect of ultraviolet light on Mutator activity continued

In 1982 (MNL 56:2-4) we described a possible synergistic effect between Mu and ultraviolet light (uv) in pollen. In 1982 we planted the remaining seeds of the crosses described in the 1982 report. This provided larger populations from which a more accurate estimate of the synergistic response could be obtained. The results are given in Figure 1. The new curve substantiates the synergistic effect reported in the 1982 report. It now appears that the synergistic response increases in a linear fashion to a peak at 35 seconds and then decreases linearly to 45". This pattern suggests that Mu and uv interact to give a greater than additive effect. Since it is not known whether or not Mu alone acts in the pollen to induce mutants, there are two possible explanations for the synergistic effect. 1) If Mu is active in pollen, synergesis could be due to the enhancement of Mu activity when it is irradiated. 2) If Mu has no mutagenic activity in the pollen, it could be activated when pollen is irradiated. No matter what the explanation this synergistic response falls off at higher doses than 35 seconds. Why? Is it possible that at doses higher than 35 seconds uv inactivates Mu? If the trend continues, there should be no synergistic effect observed at 50 seconds or higher doses. Tests of the effect of doses above 45 seconds were made this year, but the results are yet to be analyzed. If high doses of uv do inactivate Mu and the



a. Population size for Mu and + plants.

Figure 1

inactivation is permanent, all plants resulting from irradiated Mu pollen at doses of 50 seconds or higher (if synergesis is indeed not observed at higher doses) should have lost Mutator activity.

Donald S. Robertson

Possible insertion of Mu DNA in the chloroplast of a variegated mutant

In the summer of 1982, a plant was found with green and yellow-green striping. This plant occurred in an outcross progeny in which the male parent was a Mu stock and the female parent was a Q60 standard line. The plant was reciprocally crossed to a B70 standard. Fifty seeds of each of the outcrosses were planted in the field in 1983. By the time D.S.R. returned from the GSA meetings, there were in the female outcross progeny 17 dead or dying plants, one severely striped plant, 16 moderately striped plants, and 16 green plants with no stripes. The viable tissue that could be observed in the dying plants was yellow-green. It seems reasonable that the plants in the "dead or dying" class were wholly yellow-green or very heavily striped. The progeny from the reciprocal cross (i.e., using the striped plant as a male) consisted of only green plants. Thus, it seems that the yellow-green condition is due to the maternal inheritance of defective chloroplasts.

Chloroplast DNA was isolated from the variegated mutant (line 3366), as well as its maternal parent (standard Q60), Q67 and Q66 (the female and male inbred parents of the Q60 standard, respectively), and the B70 standard. The chloroplast

DNA's were digested with several restriction endonucleases and blotted onto nitrocellulose using the Southern technique. The recombinant plasmid, pMJ9 (kindly provided by M. Freeling, Berkeley) containing the Mu sequence from the Adh1 locus of maize, was nick-translated and used in the hybridization reaction to probe for homologous Mu sequences in the chloroplast DNA's. Hybridization to line 3366 DNA was very intense, while there was slight hybridization to both Q60 and Q67 (the female parent of the Q60 standard), and no detectable hybridization to B70 or Q66. These data suggest that Mu DNA has inserted in the chloroplast genome in the mutant line 3366, resulting in the mutant phenotype. We are presently cloning chloroplast DNA from lines 3366 and Q67 for molecular characterization of the Mu-homologous sequence in the mutant line, and for comparison between the corresponding clones.

M. J. Skogen-Hagenson, Donald S. Robertson and David W. Morris

AMES, IOWA
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Twin-ear expression

Two independent sources of the twin-ear expression were identified in 1973. The sources of the progenies that showed the twin-ear expression were an S2 progeny of BS10(FR)C2-2388-10 and an S5 progeny of BSAAo2, an opaque-2 strain of BSAA. Twin-eared plants were selfed to advance to the next generation to verify its expression. Within the selfed progenies, some of the plants had twin ears and were selfed. The selfed progenies were grown in 1975, and the two sources of twin ear were crossed, BS10(FR)C2-2388-10 x BSAAo2. In addition, BS10(FR)C2-2388-10 was crossed to B37o2 and Oh43o2. In 1976, the BS10(FR)C2-2388-10 line and its crosses to BSAAo2S5, B37o2, and Oh43o2 were grown in progeny rows: the frequency of twin-ear expression was 67% (4 of 6 plants) for BS10(FR)C2-2388-10, 5.4% (3 of 56 plants) for its crosses with BSAAo2, 18.2% (2 of 11 plants) for the cross with B37o2, and none (0 of 10 plants) for the cross with Oh43o2. Except for the BS10(FR)C2-2388-10 progeny, the frequency of twin-ear expression was very low, even for the cross between the two cultures that had a frequent expression of twin ears.

The plants that had the twin-ear expression in 1976 were self-pollinated, but they were not grown until 1981. In 1981, 34 self-pollinated ears were planted ear-to-row and those plants that had twin-ear expression were self-pollinated. Twenty-one ears were saved that had the twin-ear expression, and these were planted ear-to-row in 1982. Plants within each progeny row were classified in 1982 to determine the frequency of twin-ear expression: 55 of the 327 plants (16.8%) showed the twin-ear expression. Among the 21 ear-row progenies, the frequency of twin-ear expression ranged from zero (23 plants with no plants having twin ears) to 42.1% (19 plants with 8 plants having twin ears). Fifty-six ears were harvested in 1982 for planting in 1983: 48 ears that were self-pollinated and eight open-pollinated ears were saved. The 56 ears were planted ear-to-row and the plants counted for frequency of twin ears. Of the 889 plants scored, 337 (37.9%) showed the twin-ear expression. Additionally, another 1% (9 of 889) also had the twin ear expression for the ear below the top ear node. The range among progenies was from zero to one progeny in which 17 of 20 plants (85%) showed the twin ear expression. There was not a strong association among families among years for the frequency of twin-ear expression. For example, families 2170, 2177, and 2180 had 9.1, 16.0, and 42.1% twin ears in 1982, whereas their progenies had 35.5% (27 of 76 plants), 36.1% (22 of 61 plants), and 36.8% (21 of 57 plants) twin

ears in 1983. For the eight progenies obtained by open pollination, 16.9% (25 of 148 plants) had twin ears.

The twin ears have two separate ears with two separate shanks attached to the same node. It seems the occurrence of the twin ears must depend on some specific timing during the ontogeny of the plant. The occurrence of twin ears does not seem to be predictable, and its expressivity varies among progenies and plants within progenies. In 1983, some plants were observed to have twin ears at the second node below the top ear node, which had one ear. Other plants had twin ears at the top ear node and one ear at the node below the top ear. Additional studies are planned. Multiple pollinations were made on several plants in 1983. Because the twin ears are two separate ears, one ear was selfed and the other crossed with either another twin- or one-eared plant. The self and half-sib progenies will be studied to obtain further information on the twin-ear expression for these two sources.

Arnel Hallauer

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Xenia effect in modified endosperm texture opaque-2 maize

Seven non-modified endosperm texture opaque-2 inbreds (ROh43o2, RA619o2, RMo17o2, B57o2, B45o2, B46o2, and RN28o2) and four modified endosperm texture opaque-2 inbreds (RM14mod.o2, ROh45mod.o2, RVa35mod.o2 and RSyn-B-3mod.o2) were selfed and crossed reciprocally to study xenia effects of endosperm modification and other traits. The traits measured were endosperm modification, kernel density, protein content, lysine/protein ratio (L/P), zein protein and free amino acids. The degree of endosperm modification was measured by the proportion of modified kernels in a 100K sample. An eleven class scale was used (class 0 = 5% or less modified kernels, class .5 = 5-10% modified to class 5 = 90-100% modified). The selfed seeds of all the non-modified and modified opaque-2 inbreds showed major differences for all traits except kernel density (Table 1). The

Table 1. Means for seven non-modified opaque-2 and four modified opaque-2 inbreds and their F_1 seed for six traits.

Inbreds selfed	Kernel density[a]	Degree endosperm modification	% Protein	L/P[b]	Zein[c]	Free[d] amino acids %
Opaque-2	1.04	.01	9.6	5.3	4.1	18.8
Mod. opaque-2	1.22	4.13	10.7	4.0	5.2	4.8
F_1 Seed (♀/♂)						
o2 x mod. o2	1.12	1.53	10.0	4.5	4.6	10.0
Mod. o2 x o2	1.17	3.48	10.7	4.0	5.2	5.9

a 100 K wt/100 K volume
b g lysine/100 g protein
c g zein/100 g dry wt.
d g L-leucine/100 g protein

reciprocal F1 seeds showed differences in degree of modification, zein and free amino acids (Table 1). These differences could be the result of dosage, maternal effects, or xenia effects. The xenia effects were measured by comparing the differences between selfed and F1 seed average over the different crosses and selfs. These differences are presented in Table 2.

Table 2. The mean differences between selfed and F_1 seed of six non-modified and four modified inbreds for several traits.

Comparisons	Kernel density	Degree endosperm modification	% Protein	L/P	% Zein	Free amino acids %
(o2 ⊗) - (o2 x mod. o2)	-.08**	-1.52**	-.46	.76**	-.71**	8.72**
(o2 ⊗) - (mod. o2 x o2)	-.13**	-3.47**	-1.13**	1.26**	-1.24**	12.94**
(mod. o2 ⊗) - (o2 x mod. o2)	-.10**	2.60**	.64*	-.51*	.55*	-5.25
(mod. o2 ⊗) - (mod. o2 x o2)	.05*	.65	-.03	-.01	.02	-1.03
(o2 x mod. o2) - (mod. o2 x o2)	-.05**	-1.95**	-.67**	.50**	-.53**	4.22**

* and ** Significant at .05 and .01 probability levels.

The reciprocal F1 seed expressed significant differences in all traits except when the modified endosperm texture opaque-2 inbred was used as the female parent. When the modified endosperm opaque-2 inbreds were used as pollen parents, the F1 seed showed differences from the non-modified female parent endosperm for all traits. These results indicate that in addition to dosage effects, xenia effects can also exist in modified endosperm texture opaque-2 materials.

T. M. Sung

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Effect of anaerobiosis on the expression of two forms of fructose-1,6-diphosphate aldolase

The anaerobic response of the maize primary root has been a well characterized phenomenon (Sachs et al. Cell 20:761-769, 1980). Phillip Kelley in our lab has recently identified the functions of 3 more anaerobic proteins besides the already known alcohol dehydrogenase and pyruvate decarboxylase. All of them turned out to be related to glycolysis. The 55,000 dalton anaerobic protein (ANP55) corresponded to glucose phosphate isomerase (JBC, 1984, in press). And both the 35,500 and 33,000 dalton anaerobic proteins (ANP35.5 & ANP33) were recognized by the antiserum raised against fructose-1,6-diphosphate aldolase (submitted to JBC). We have further observed that the two forms of aldolase were not only serologically related, but the balance of their expression was controlled by the oxygen tension of the environment. Pulse label experiments showed that ANP35.5 was the predominant form of the two aldolases under aerobic condition. However, when roots were transferred into anaerobic environment, ANP33 became the major form accounting for more than 70% of the S^{35}-methionine incorporation into aldolase. The mechanism and biological significance of this subtly regulated phenomenon remain to be determined. The two forms of aldolase are not likely to be the result of transcription from two different genes, since only one restriction fragment can be detected in genomic Southern blots probed with the cDNA clone of aldolase (Sarah Hake, unpubl. hybrid select translation data). At this point, we favor the hypothesis of post-translational modification or breakdown of one form into the other. Preliminary evidence indicated that, in the presence of protease inhibitor (PMSF) during extraction, only the higher molecular weight form of aldolase could be detected (Kelley and Freeling, ms. submitted). This being true, we have indirect evidence for the anaerobic "induction" of a protease that clips aldolase.

Che-Hong Chen and Michael Freeling

Change of state of Robertson's Mutator-induced mutants

One of the most striking properties of transposable elements is their ability to undergo reversible changes of state. A change of state is defined as an alteration in the somatic reversion frequency or in the timing of reversion (or both) of a transposable element-induced mutant allele. Such a change can occur in either the autonomous or nonautonomous member of a two-element system, or in the single member of a one-element system. There is no evidence that Robertson's Mu is a two-element system.

I have recently identified two cases of changes of state in Robertson's Mu-induced mutants. One case involves a mutant of the Bz1 locus which was recovered last winter from a Mutator stock; the progenitor derives from a full color line from M. G. Neuffer. This allele, bz1-N1032, is very unstable in the aleurone. Reversion events are frequent and occur late in development. A change of state was demonstrated by a single kernel of the cross: bz1/bz1 X bz1/bz1-N1032 where both have a Mutator background. See schematic in Figure 1. Bz revertant sectors

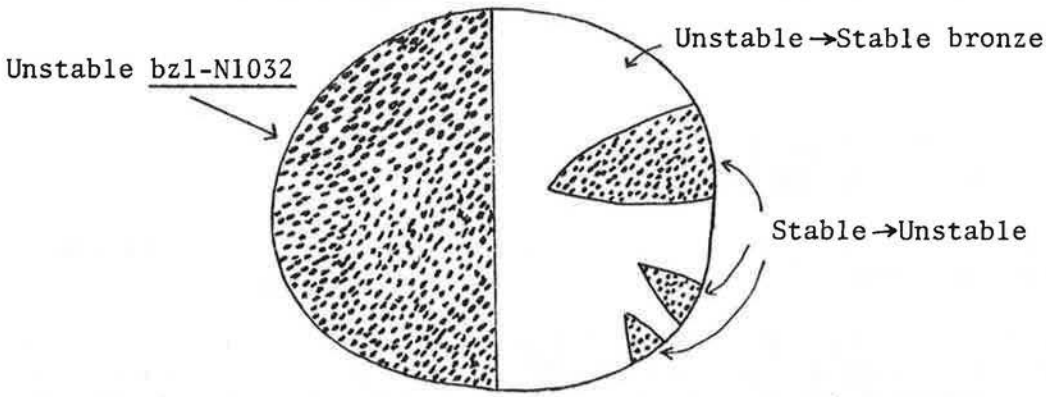


Figure 1. Crude schematic of kernel of the genotype bz1/bz1-N1032. Dotted portions represent tissue in which bz1-N1032 is in an unstable state. Clear portions represent a stable state of the allele.

in the aleurone of this kernel suggest the following sequence of events: 1) At the two cell stage in the development of the endosperm, bz1-N1032 changed to a stable state in one of the two cells. 2) Subsequently, the stable form of the allele changed back to the unstable state in three separate cells. These cells, each genetically unstable bz1-N1032, produced lineages which were surrounded by stable bz1-N1032 tissue. Two changes of state are indicated. The stable to unstable transition appears to have restored the original state of bz1-N1032. All four sectors of unstable bz1-N1032 are the same with respect to rate and timing of reversion. In each sector revertant tissue is approximately one tenth as frequent as non-revertant tissue. The timing of reversion results in single cell revertant sectors about twice as often as two to five cell revertant sectors.

A Robertson's Mu-induced mutant Adh1 allele, Adh1-S3034v, gave a similar result for two kernels whose aleurone had been stained for ADH enzyme activity.

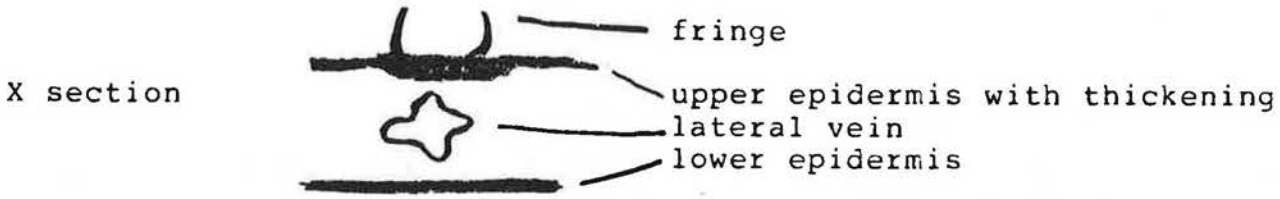
Assuming that individual Mu elements are autonomous requires that the mutation encoding a change of state resides within the Mu element at Bz1 and Adh1, or in the genes themselves. However, trans-acting modifiers have not been excluded. Change of state may be a general property of maize transposable elements.

Mary Alleman

Genetics of the ligule alteration component of the Knotted phenotype

The original Knotted mutation (Bryan and Sass, 1941, J. Hered. 32:343), Kn1-O, specifies a constellation of dominant phenotypes depending on the genetic background: mild dwarfism, outpocketings of tissue at foci along lateral veins of the blade (knots), twisting of the midrib and stem, and a condition that has been called "ligule displacement" (Gelinas, Postlethwait and Nelson, 1969, Amer. J. Bot. 56:671), where the normal ligule is disrupted and bits of epidermal "fringe" grow from thickened tracts above lateral veins. Other Kn mutants specify different phenotypes.

The ligule is a dispensable organ. In suitable genetic backgrounds, a Kn1-O mutant gene specifies a paired fringe structure that we have diagrammed below.



The fringe appears to be like the inside structure of the ligule. To test for this homology directly, we constructed double mutants of liguleless-2 and Knotted. Kn1-O/+, lg2/lg2 plants showed no such fringes whereas siblings with ligules showed both ligule disruption and fringes spread up the blade. Knots were unaffected. We conclude that the fringe is homologous to ligule and that fringes and knots above the same vein are separable. Among about 100 Kn1-O heterozygotes, we found two leaves that showed a particularly informative morphology: the ligule was perfect as was the lower leaf blade, but an area of distorted tissue occurred about one-third up from the ligule. Distal to this area were copious bits of fringe material. Clearly, these ligule bits originated de novo rather than as displaced cells of the original ligule.

In order to test whether de novo ligule formation is a cell autonomous effect of Kn1-O, we have used x-rays to disconnect the long arm of chromosome 1 carrying Kn, leaving a segmental monosomic carrying kn marked by a white stripe (lw, 1.5 mu from Kn1). De novo ligule formation and epidermal thickening above veins is cell autonomous. Our six clones were white in all layers and extended to the periphery of the leaf. Methods--500-1000 rads 225 Kv x-ray through 0.35 mm Cu at 150 rads/min acute onto 4 day seedlings--were adapted from S. Poethig (1982, MNL 56:53); sectors 1/8-1/32 of a leaf width occurred. We do not yet have definitive fate maps for knots, or other components of Knotted phenotype.

Our most informative experiments involved constructing sibling plants that differed in the dose of kn (kn = + = normal) gene in a background containing one dose of Kn1-O. We used the TB-1La translocation to add or remove + as a part of the entire arm; we used a pair of reciprocal 1-3 translocations to add an extra + on an 18 mu segment. Stocks were marked with Adh1 allozymes (Adh1 is less than 0.1 mu from Kn) by J. Birchler. All details will be reported elsewhere. Our results were a double surprise.

Surprise 1. Kn/+/+ had less and developmentally later de novo ligule formation than Kn/+ sibs. However, total degree of knotting and twisting did not differ significantly between these groups. This result was repeated using the TB-1La translocation and once more using the segmental addition method.

Surprise 2. Kn1-O/deletion heterozygotes had no Knotted phenotype at all. Surprise 1 seems to exclude an "overproducer" hypothesis. We are left with a possible case of transvection.

With respect to ligule disruptions, Kn1-O apparently specifies a wrong product, but a product similar to normal kn product, that leads to de novo epidermal thickenings and ligule formation. Other Kn mutations seem to behave differently from Kn1-O in most of these tests. When we obtain a Kn clone, we should be able to derive meaning from DNA sequence and sequence arrangements.

M. G. Neuffer and M. Zuber have given us new Kn mutants that are proving extremely valuable. If you have any mutant that encodes knots, rough sheath, wiggly veins, liguleless, or the like, we would very much appreciate hearing about it. Thank you.

Michael Freeling and Sarah Hake

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Indiana University

Immunological studies of alcohol dehydrogenase

Immunogenetic analyses of several EMS induced alcohol dehydrogenase variants have led toward the elucidation of the mechanism of enzyme activity. Antisera raised against various allelic forms of the enzyme can precipitate any of the ADH allozymes; the precipitin formed retains enzyme activity. The single exception to this is anti U725, which precipitates antigen as well as the other antisera, but renders the ADH precipitin inactive. The enzyme can be protected from this inactivation by preincubation with its NAD cofactor, or with NADP, which cannot act as a coenzyme. This works both in double diffusion Ouchterlony when NAD or NADP is added to the agar, and in solution mixtures. Ethanol cannot perform such a protective role. These results suggest that the mechanism of enzyme activity involves first the binding of NAD, which causes a conformational change necessary for the actual transfer of protons, in a manner analogous to that of horse liver alcohol dehydrogenase (Biochemistry 9:4655, 1970). The prebinding of anti U725 may simply sterically prevent subsequent binding of NAD to ADH, or it may induce a conformational change in the enzyme so that it could not then bind NAD, or it may allow binding of NAD but not allow the conformational transition to the active state.

A number of Adh CRM+ nulls which have no detectable enzyme activity have also been studied immunologically. Simply precipitating these nulls by antisera raised against active forms of ADH does not restore enzyme activity. However, if the null extracts are preincubated with high levels of NAD or NADP, then immunoprecipitated, activity can be restored. Binding studies using the affinity chromatography medium Blue Sepharose have shown that all seven of the CRM+ nulls are unable to bind NAD. One hundred-fold excess of NAD alone is not sufficient to restore spectrophotometrically detectable activity; it is the interaction among enzyme, high concentrations of coenzyme, and immunoglobulin that presumably molds and holds the enzyme in an active conformation.

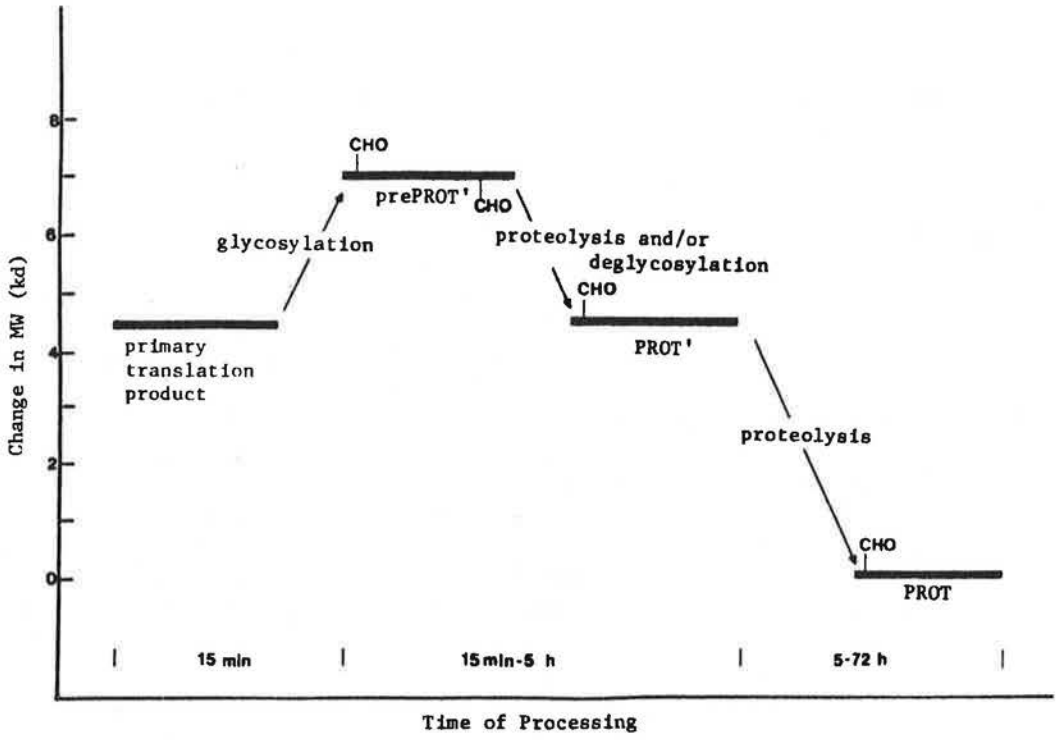
Erin Irish and Drew Schwartz

Synthesis of maize embryo globulins

We reported in last year's Newsletter that the protein products of the Prot locus, PROT' and PROT, account for the major portion of the globulin fraction in maize embryos. Analysis of Prot protein synthesis in vivo and in vitro indicates PROT is derived from PROT', and PROT' is derived from a relatively short-lived precursor, which we have termed prePROT'.

Prot protein synthesis in developing embryos was examined by pulse-chase radiolabeling methods. Intact immature embryos (23-28 days post-pollination) were excised from surface-sterilized kernels, collected under aseptic conditions, and placed on agar culture medium in petri dishes. The basal medium was that of Miller (in Moderne Meth. Pflanzenanal. v.6, Springer-Verlag, 1963), to which abscisic acid was added to 10^{-5} M; the inclusion of abscisic acid in the culture medium was necessary in order to prevent germination. Embryos which had been conditioned to the medium for two days were pulse-labeled with radioactive amino acids by transferring the embryos to a small petri dish to which either ^{3}H-leucine or ^{3}H- or ^{14}C-labeled amino acid mixture was added in the liquid culture medium. Generally, 80-100 microcuries of label in 1 ml of solution were used per 15-18 embryos. The sealed dish containing the embryos was gently agitated during the pulse-labeling period (15-60 minutes). Pulsed embryos were transferred to culture plates and incubated (chased) for 1-72 hours. All incubations were performed at 24 C. The chase period was terminated by either freezing the embryos in liquid nitrogen or dry ice, or by macerating the embryos in a SDS sample buffer and boiling for two to five minutes. Frozen embryos were generally used for immunoprecipitation analysis. Individual embryo extracts were examined by SDS-polyacrylamide gel electrophoresis and fluorography. In vitro translation of immature embryo RNA was performed using both the rabbit reticulocyte lysate and the wheat germ extract systems (BRL).

The results obtained from the pulse-chase and the in vitro translation experiments are presented schematically in the accompanying figure. The primary



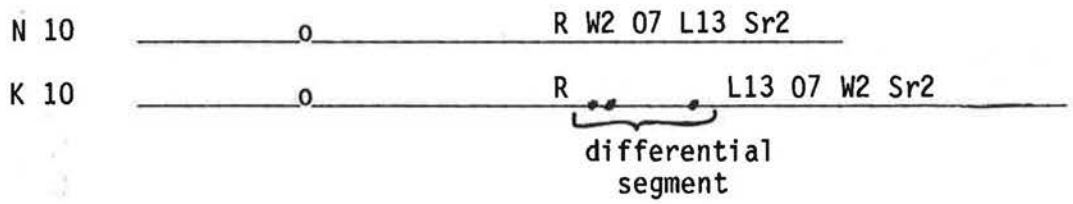
translation product, as determined by in vitro translation of embryo RNA, is approximately 2.5 kilodaltons smaller than prePROT', the initial Prot-specific polypeptide detectable in in vivo pulse-chase experiments. This indicates that the primary translation product is modified co- and/or post-translationally in vivo to yield prePROT'. Although the nature of this modification is not known, glycosylation is suggested as a probable alternative, as preliminary evidence indicates the Prot proteins are glycoproteins, and other seed globulins are

glycosylated in vivo. The processing of prePROT' to form PROT' may involve deglycosylation as well as proteolysis; this suggestion is based on comparison of prePROT' and PROT' with respect to isoelectric points and cyanogen bromide cleavage patterns. The conversion of PROT' to PROT, which is regulated by the unlinked locus Mep, probably involves proteolysis alone, since 1) both PROT' and PROT appear to be glycosylated to the same extent; and 2) cyanogen bromide cleavage of ^{3}H-leucine-labeled PROT' and PROT suggests a leucine-rich amino acid tract is removed from PROT' in the formation of PROT.

Alan L. Kriz and Drew Schwartz

On the genetic constitution of abnormal chromosome 10

In a series of reports in the Maize News Letter, we demonstrated by cytogenetic methodology that the distal portion of the long arm of abnormal chromosome 10, which is found in certain races of maize grown in Arizona, New Mexico, Mexico and the Central American countries, differs drastically from normal 10 in its architecture, amount of chromatin, and linear order of genes. Our conclusions are diagrammatically summarized below:



In both kinds of chromosome 10 the W2 O7 L13 segment is situated between the R and Sr2 loci, but in abnormal 10 this segment has undergone excision and transposition distally in an inverted order with respect to the flanking R and Sr2 genes. A piece of chromatin of unknown origin possessing three small knobs, designated as the differential segment, lies interposed between the R and L13 loci. A second piece of chromatin, again of unknown origin, composed of a large heterochromatic knob with a short euchromatic tail, terminates the long arm. We suggested that a minimum of three breaks occurred in the origin of abnormal 10 from a normal 10. One break was between R and W2, a second between L13 and Sr2, and the third between Sr2 and the telomere. One end of the differential segment, carrying no marker genes, was inserted next to the R locus with the other end joined to the L13 end of the W2 O7 L13 segment. The order of loci in this segment is inverted relative to their orientation to R and Sr2. Finally, a second piece of chromatin, origin unknown, comprised of a large heterochromatic knob and a short euchromatic tail, was inserted subterminally just distal to Sr2, giving the structure of abnormal 10 shown above. The fate of the normal telomere is uncertain; it may be present as the telomere of the long arm of K10 or it may have been eliminated.

Before accepting this complex, even bizarre, scenario we should critically examine the various links in the chain of argument to see if some are of questionable validity. For example, the statement that the L13 O7 W2 block of genes is inverted in the transposed location in abnormal 10 assumes that the order in this segment in normal 10 is R W2 O7 L13 Sr2. This is based on the order determined from the collated linkage data given on page 191B of the 1983 Maize News Letter, where the order and map positions of loci distal to R are as follows:

R	l	Lc	Mst	w2	o7	l13	sr2
61	63	65	67	77	87	91	95

As is well known, backcross data provide more precise recombination values than do F2 data in coupling, and much greater precision than do F2 data in repulsion. Homozygotes for either w2 or 113 are lethal, so backcross data cannot be obtained and F2 data must be resorted to in locating these two genes. Furthermore, homozygous 113 or w2 seedlings cannot be classified for the sr2 phenotype, and w2 is epistatic to 113, thereby increasing the difficulty of determining map positions from F2 data. An additional uncertainty is the variation in recombination values for the same interval in different strains of maize. Since the location of w2 was based on F2 data, and since our argument was so dependent upon ascertaining the correct linear order in the distal portion of the long arm of both normal and abnormal chromosome 10, we decided to undertake the arduous task of genotyping every plant in a population segregating for R, w2 and sr2. This is a laborious process since every tested plant gives the genotype of only one gamete but the data are equivalent to backcross data. Mascia (1978 MNL) employed this procedure in accurately placing the 113 locus four map units proximal to sr2. The following cross was made in order to determine the crossover distance between R and W2 (region 1) and between W2 and Sr2 (region 2) in the heterozygous male parent:

$$\frac{r\ \ W2\ \ sr2}{r\ \ W2\ \ sr2} \quad X \quad \overset{(1)\quad(2)}{\frac{R\ \ W2\ \ sr2}{r\ \ w2\ \ Sr2}}$$

The above mating is a testcross for the R and Sr2 loci, while determination of which W2 allele is present in a gamete comes by pollinating all plants with a strain that would reveal the presence or absence of the w2 allele. This permitted identification of the eight genotypic classes listed below along with their frequencies. The chromosome contributed by the pollen parent is given in the denominator.

I. (0) $\frac{r\ \ W2\ \ sr2}{R\ \ W2\ \ sr2}$
No. = 333
Phenotype: R, striate

II. (0) $\frac{r\ \ W2\ \ sr2}{r\ \ w2\ \ Sr2}$
No. = 380
Phenotype: r, green

III. (1) $\frac{r\ \ W2\ \ sr2}{R\ \ w2\ \ Sr2}$
No. = 103
Phenotype: R, green

IV. (1) $\frac{r\ \ W2\ \ sr2}{r\ \ W2\ \ sr2}$
No. = 94
Phenotype: r, striate

V. (2) $\frac{r\ \ W2\ \ sr2}{R\ \ W2\ \ Sr2}$
No. = 118
Phenotype: R, green

VI. (2) $\frac{r\ \ W2\ \ sr2}{r\ \ w2\ \ sr2}$
No. = 141
Phenotype: r, striate

VII. (1-2) $\frac{r\ \ W2\ \ sr2}{R\ \ w2\ \ sr2}$
No. = 13
Phenotype: R, striate

VIII. (1-2) $\frac{r\ \ W2\ \ sr2}{r\ \ W2\ \ Sr2}$
No. = 22
Phenotype: r, green

Total = 1204 R-Sr2 recombination = 37.9% coincidence = 0.62
R-W2 " = 19.3%
W2-Sr2 " = 24.4%

These data clearly demonstrate that w2 lies between R and Sr2, and give an accurate determination of the w2-sr2 interval. Our data, together with those obtained by Mascia for the R 113 sr2 region, demonstrate that the linear order for 10L given in the 1983 MNL is correct and any misgivings we held about the map positions of these loci were laid to rest. The reverse order of L13 07 W2 in the abnormal 10 chromosome was unequivocally established by our series of simple terminal deficiencies, since in two independent cases, DfK10(F) and DfK10(I), the W2 locus was deleted but the L13 locus was still present. The data confirm our conclusion that the W2 07 L13 segment of N10 was both transposed and inverted in the origination of abnormal 10.

M. M. Rhoades and E. Dempsey

Further studies with the bz-mut, Mut system

During the past year, we have accumulated considerably more data from testcrosses involving ws3 lg and gl2, which confirm the location of Mut between lg and gl2 on chromosome 2. Plants of Ws3 Lg Mut Gl2/ws3 lg mut gl2; bz-mut/bz-mut genotype were crossed by ws3 lg mut gl2; bz-mut pollen parents. The kernels were scored for mutability (due to interaction of bz-mut and Mut), and the ws3, lg and gl2 traits were classified in the seedling bench. The composite data are presented below:

Crossover region	Phenotype	No. of individuals	
0	Ws Lg Mut Gl	1137	Recombination:
0	ws lg mut gl	1197	
1	Ws lg mut gl	142	Ws3-Lg = 9.1%
1	ws Lg Mut Gl	144	
2	Ws Lg mut gl	214	Lg-Mut = 13.7%
2	ws lg Mut Gl	199	
3	Ws Lg Mut gl	105	Mut-Gl2 = 6.9%
3	ws lg mut Gl	84	
1-2	Ws lg Mut Gl	1	
1-2	ws Lg mut gl	1	
1-3	Ws lg mut Gl	2	
1-3	ws Lg Mut gl	3	
2-3	Ws Lg mut Gl	18	
2-3	ws lg Mut gl	8	
1-2-3	Ws lg Mut gl	3	
1-2-3	ws Lg mut Gl	2	

Total = 3260

Certain classes of seedlings were transplanted to the field for further tests for the presence of Mut. Doubles (2-3) and triples (1-2-3) were of special interest because they are likely candidates for Mut transposition. The expected frequency of doubles and triples within a region of 30 map units is so low that a considerable number of the apparent multiple crossovers are probably due to transposition of Mut to another chromosome, followed by segregation away from the original chromosome at anaphase I. Such an event, combined with an absence of crossing over in the ws3 lg gl2 region of chromosome 2, gives ws3-lg-gl2 (+ Mut) and Ws3-Lg-Gl2 (- Mut) phenotypes. The former plants may then be selected for further testing to reveal the new location of Mut.

Due to the poor growing conditions in the field in the summer of 1983, only 4 of the (2-3) and (1-2-3) crossovers germinated, survived to maturity, and produced

ears. Two of these do not carry Mut and cannot be studied further. Two, however, are potential transpositions; one is ws3 lg (Mut) gl2 and the other is Ws3 lg (Mut) gl2. They have been crossed with Ws3 Lg Gl2; bz-mut stocks and the F1 will be testcrossed in the greenhouse to determine whether Mut is still in chromosome 2.

In our previous report, the phenotype of Mut, bz-mut kernels was not fully characterized. The Bz dots on a bronze background are usually small, comparable in size to the A dots produced in Dt kernels. However, a single kernel may show considerable variation in size of dots and at least two intermediate pigment levels occur along with the full colored Bz. One aspect that remained unanswered was whether or not Mut shows a dosage effect, similar to Dt and Ac2, which give an exponential increase in dots with extra doses, or to Ac where both time and frequency of mutations are affected. Exact reciprocal crosses of related strains were made:

bz-mut/bz-mut; Mut/Mut X bz-mut/bz-mut; mut/mut

All resulting kernels have three bz-mut responding alleles but one cross produces kernels with two doses of Mut, while the reciprocal cross gives kernels with one dose of Mut. The number of dots on 20 randomly selected kernels from each cross was determined. To ensure accuracy of counts, the kernels were soaked overnight in water and the pericarp was removed. No difference was found in the time of mutation; the size of dots, though somewhat variable, was similar in Mut Mut mut and Mut mut mut kernels. To our surprise, the number of dots also did not differ greatly. An average of 32.6 dots was found on kernels with one dose of Mut, while kernels with two Mut had an average of 27.4 dots per kernel. Closely related kernels from the Mut/Mut; bz-mut/bz-mut stock possessing three doses of Mut averaged 34.6 dots per kernel. This value was based on 30 kernels from three sibbed ears. These data, though preliminary, clearly indicate that Mut is unlike many regulators in that no dosage effect is observed.

M. M. Rhoades and E. Dempsey

On the nature of the w2 mutation

Lindstrom (1924) reported that the w2 mutation not only produced recessive white seedlings, but also adversely affected endosperm development. The two genetic effects were tightly linked, suggesting pleiotropy or mutation of two adjacent loci. Discrimination between these two mechanisms would be possible if the two traits were separable by crossing over, but in Lindstrom's time the necessary flanking markers were not available. Even if they had been, the lethality of the white seedlings and the existence of modifying genes affecting endosperm development would have made it difficult to discriminate between the two alternatives. However, some recent observations throw some light on the nature of the w2 mutation.

Our DfK10(F) chromosome is a modified abnormal 10 deficient for the W2 and Sr2 loci as well as the large K10 knob and the euchromatic tail. It is female and male transmissible albeit with a somewhat reduced male frequency. Homozygotes for DfK10(F) have defective endosperm development and produce white seedlings--i.e., the homozygous deficiency has the same phenotype as the w2 mutation. The DfK10(F)/w2 compound behaves exactly the same as the DfK10(F)/DfK10(F) and the w2/w2 homozygotes. Since the known deficiency in 10L and the w2 mutation give identical phenotypes, the possibility that the original w2 mutation is a deficiency including more than one gene should be considered.

Homozygous DfK10(F), homozygous w2, and heterozygous DfK10(F)/w2 kernels all exhibit a third mutant trait. In kernels possessing only dominant alleles for the

complementary loci concerned with aleurone color, the aleurone layer is not uniformly pigmented but has a spotted appearance resulting from the interspersion of numerous islands of colorless cells among pigmented sectors. The colorless areas or spots coincide with the depressed regions of defective endosperm, while the normally developed sectors are colored. The spotted phenotype, first observed in DfK10(F) R -/N10 r w2 heterozygotes, simulated a mutable R system, but this explanation became untenable when we found that the spotted phenotype also occurred in R w2 and DfK10(F) R - homozygotes. Aleurone cells were present in the defective colorless regions of the endosperm so the lack of pigment cannot be ascribed to a missing aleurone layer. We think it likely that the failure to form anthocyanin in the depressed areas of the endosperm is caused by pigment inhibition resulting from the loss or mutation of a gene in the w2 region. Suppression of pigment synthesis in DfK10(F) or w2 homozygotes is comparable to the action of the recessive vp mutation, which likewise inhibits anthocyanin production in cells possessing all of the genes necessary for aleurone color.

In summary, the three apparently unrelated phenotypic effects produced by homozygosity or hemizygosity for w2 are found when the DfK10(F) R - chromosome, known to be deficient for the W2 gene, is homozygous. These observations suggest that three closely linked but separate loci were lost when the w2 mutation arose, but pleiotropy of a single gene cannot be excluded and remains a viable alternative. Since some small deficiencies, such as Stadler's a-x mutations, are known to reduce crossing over in flanking regions, we thought it might be informative to determine the amount of recombination between R and Sr2 in plants heterozygous for w2 and in sib plants homozygous for the W2 allele, since these markers flank the w2 locus. In a population of 935 individuals heterozygous for w2, we found 32.8% recombination between R and Sr2, and 11.3% for the proximal G R region. Among 481 W2/W2 control plants, the R Sr2 recombination was 28.7% and the G R recombination 11.6%. Admittedly, the data are not extensive but should reveal a marked reduction in crossing over if the w2 mutation is a segmental deficiency. Since no reduction was found for the critical R sr2 region, which includes the w2 locus, it can only be concluded that the recombination data offer no support for the hypothesis that the w2 mutation is associated with a deficiency.

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Genotypes at enzyme loci in 363 U.S. inbred lines of maize

In 1982 and 1983 Stuber and Goodman (MNL 56:127-132, 1982; USDA-ARS, ARS-5-16:1-29, 1983) presented genotypes for 21 enzyme loci in 406 U.S., Canadian, and European inbred lines of corn. Since 1980, we have also routinely characterized public inbred lines of maize for their genotypes at polymorphic enzyme loci. Assays have been restricted to loci which could be read unambiguously with electrophoretic conditions used in our laboratory (Crop Sci. 23:572-576). All genotypic information included in this report comes from inbred lines obtained from other stations. The inbreds are being maintained and used in population genetics and population improvement research at the Northern Grain Insects Research Laboratory at Brookings. Genotypes of these inbreds are given to complement the earlier lists provided by Stuber and Goodman.

Electrophoretic methods have been reported for all loci assayed (Cardy et al., Inst. of Stat. Mimeo. Series No. 1317, N.C. State Univ., 1980; Kahler, Crop Sci. 23:572-576, 1983). Enzyme loci, laboratory isozyme mobilities from the gel origin and laboratory allozyme numbers are given for convenience in Table 1.

Table 1. Summary of enzyme loci, laboratory isozyme mobilities, and laboratory allozyme numbers.

Enzyme locus	Isozyme position (cm)	Allozyme numbers[1]	
		lab 1	lab 2
Adh1	3.4	1	6
	4.3	2	4
Idh2	3.5	1	6
	4.0	2	4
Got1	1.7	1	6
	2.8	2	4
Mdh2	Null	0	0
	3.5	1	3.5
	4.5	2	6
	4.1	3	4.5
	3.3	4	3.0
Acp1	1.3	1	4
	1.8	2	3
	2.3	3	2
	1.1	4	6
Acp4	4.5	1	--
	4.75	2	--
	5.0	3	--
	5.25	4	--
	5.5	5	--
	5.75	6	--
Prx1	Null	0	--
	0.8	1	--
	1.8	2	--
	3.0	3	--
Est1	1.1	1	--
	1.7	2	--
	2.2	3	--
Est4	Null	0	--
	2.1	1	--
	3.0	2	--
	3.6	3	--
	4.3	4	--
	4.6	5	--
	1.3	6	--
Glu1	Null	0	0
	2.0	1	6
	3.0	2	2
	3.6	3	1
	1.4	4	9 or 10
	1.8	5	7
	2.8	6	3
Pgd1	5.3	1	3.8
	6.2	2	2

[Ed. note: The locus designated Acp1 is presumed to be the same as that documented by Efron (Genetics 65:575, 1970), designated Ap1, and the same as Acph1 (see Zealand, per Goodman & Stuber, 1983). Est1 is the same locus as E1 of Schwartz (Proc. NAS 46:1210, 1960), and Est4 as E4 of Harris (Genetics 60:186, 1968). Prx1 is presumed to be the same as Px1 of Hamill (MNL 42:36, 1968). Enp1 has replaced Ep1 per Vodkin and Scandalios (Biochemistry 19:4660, 1980).

These equivalences are given in order to avoid ambiguous specifications of genetic units. Pending consideration and concurrence on changes in symbolization, the symbol applied in the original, documented demonstration of the locus should be used. Defining equivalence (experimentally if necessary) is the responsibility of subsequent investigator(s) to the extent possible. Changes based upon scientific information that alters the basis for symbolization may of course be proposed by an individual in publications, and the acceptance of the changes by others must then depend upon their evaluation of the evidence. Changes that are proposed for clarity or systematization should only override the "priority" criterion if there is general agreement among appropriate cooperators, accompanied by published documentation to that effect. The editor would appreciate any responses to this measure of symbolization standards as they might be applied in this News Letter and in the generation of compilations in particular].

Table 2. Genotypes at 13 enzyme loci for 363 inbred lines of maize.[1]

Inbred	Locus												
Line	Adh1	Idh2	Got1	Mdh2	Acp1	Acp4	Prx1	Est1	Est4	Glu1	Pgd1	Pgd2	Enp1
A34	2	1	2	2	1	5	1	2	5	5	1	1	3
A70-12	2	2	1	2	3	1	2	2	3	3	1	1	3
A71-22	1	2	2	1	3	6	2	1	2	1	2	1	3
A73	2	2	2	1	1	1	2,23, 3	1	3,23, 2	1,2, 12	1,12, 2	1	3
A78-3	2	1	2	2	1	2	1	3	2	4	1	1	3
A78-8	2	1	2	2	1	3	2	3	5	5	1	2	3
A78-11	2	2	2	4	1	3	2	3	2	5	1	2	3
A188[2]	2	1	2	1	1	1	2	3	5	5	1	1	1
A238[2]	2	2	2	2	2	1	2	1	3	2	2	1	3
A239[2]	2	2	2	2	2	1	3	3	3	2	2	1	3
A257[2]	2	2	2	4	1	1	2	1	5	2	2	1	3
A265[2]	2	2	2	2	1	6	2	1	3	1	2	1	3
A295[2]	2	1	2	4	3	1	1	1	2	1	1	1	3
A427[2]	2	2	1	2	3	1	2	2	2	3	1	1	3
A509[2]	2	1	2	1	3	1	2	1	4	5	1	1	3
A554[2]	2	1	1	4	3	5	2	2	2	5	1	1	3
A556[2]	2	2	2	2	1	2	2	1	3	1	2	1	3
A570[2]	2	2	2	1	3	6	2	1	5	1	1	1	3
A619[2]	2	1	2	4	1	2	2	3	2	1	1	1	3
A624	2	1	2	4	3	1	1	3	2	2	1	1	3
A628	2	2	2	1	3	2	1	1	2	1	2	1	3
A629[2]	2	1	1	4	3	1	3	3	2	1	1	1	3
A632[2]	2	1	2	2	1	2	2	3	2	5	1	2	3
A634[2]	2	1	2	2	1	2	2	3	3	5	1	1	3
A635[2]	2	1	2	2	1	2	2	3	2	5	1	2	3
A635Ht	2	1	2	2	1	2	2	3	2	5	1	2	3
A639[2]	2	1	2	4	1	6	2	3	5	2	1	1	3
A641[2]	2	2	2	2	1	5	2	3	2	1	1	1	3
A646	2	1	2	2	1	2	2	1	3	5	2	1	3
A648[2]	2	1	2	4	2	2	1	1	4	5	1	1	3
A654[2]	2	1	2	1	1	2	2	2	2	2	1	1	3
A656[2]	2	1	2	4	1	6	2	3	5	2	1	1	3
A657[2]	2	1	2	2	1	1	2	1	2	5	2	2	3
A659[2]	1	1	2	2	1	2	3	1	5	2	2	1	3
A660[2]	2	2	2	1	1	1	2	1	2	1	2	1	3
A661[2]	2	2	2	2	3	1	2	3	2	1	1	1	3
A663[2]	2	2	1	2	3	1	2	2	3	3	1	1	3
A665[2]	2	1	2	2	1	5	2	3	2	1	1	2	3
A670	2	1	2	4	1	2	2	3	2	1	1	1	3
B2[2]	2	1	2	2	3	6	2	2	2	5	2	1	3
B8[2]	2	2	2	3	3	5	2	2	3	5	1	1	3
B14[2]	2	1	2	2	1	2	2	3	2	5	1	2	3
B14A[2]	2	1	2	2	1	2	2	3	2	5	1	2	3
B37[2]	2	1	2	2	3	5	2	3	1	5	2	1	3
B38	2	2	2	1	1	6	2	3	2	1	2	1	3
B39	1	2	2	2	3	1	3	3	1	1	2	1	3
B42	2	2	2	1	1	2	1	1	2	5	1	1	3
B44	2	2	2	2	3	1	3	1	2	5	1	1	3
B46	2	2	2	1	1	6	2	1	3	5	2	1	3
B48	1	2	2	2	1	1	2	1	2	5	2	1	3
B50[2]	2	2	2	4	3	2	1	1	2	1	2	1	3
B52[2]	2	1	2	1	1	4	2	3	4	5	1	1	3
B53	2	2	2	2	3	1	2	2	2	1	2	1	3
B54	2	2	2	4	1	5	2	2	2	2	1	1	3
B55[2]	2	2	2	2	1	2	3	1	4	2	1	1	3
B57[2]	2	1	2	4	3	1	3	2	5	5	1	1	3
B59	2	1	2	2	1	2	2	3	2	5	1	2	3
B66	2	1	2	2	1	2	2	2	5	1	1	1	3
B67[2]	2	1	2	2	1	1	3	3	2	5	1	1	3
B68[2]	2	1	2	2	2	2	2	3	2	5	2	1	3
B68Ht	2	1	2	2	2	2	2	3	2	5	2	1	3
B69	2	1	2	2	1	1	2	3	2	5	2	2	3
B70[2]	2	2	2	2	1	1	1	1	2	1	1	1	3
B73[2]	2	2	2	1	3	5	2	1	2	5	1	1	3
B75[2]	2	2	2	1	2	3	2	3	4	2	2	1	3
B76[2]	2	1	2	1	3	5	2	3	1	5	2	1	3
B77[2]	2	1	2	2	1	2	2	3	2	2	1	2	3
B79[2]	2	1	2	2	1	1	2	3	2	5	1	1	3
B84[2]	2	2	2	1	1	2	3	3	2	5	2	1	3
B85	2	1	2	4	1	2	2	2	2	5	1	1	3
B86	2	1	2	1	1	2	2	3	4	5	1	1	3
B87	2	2	2	2	1	2	2	2	3	2	0	1	3
B217[2]	2	2	2	4	1	6	2	3	2	1	2	1	3
C103[2]	2	2	2	2	2	3	2	1	2	1	1	1	3
C123[2]	2	2	2	2	1	3	1	1	2	2	1	1	3
C123Ht	2	2	2	2	1	3	1	1	2	2	1	1	3
CG2[2]	2	2	2	1	1	1	3	3	2	5	1	1	3
CG3[2,3]	2	1	2	2	1	2	3	1	4	5	1	1	1
CG6[2]	1	1	2	1	1	2	2	2	3	6	1	1	3
CG7[2]	2	1	2	2	1	1	2	2	2	2	1	2	3
CG9[2]	2	2	2	2	2	1	2	3	5	5	1	1	3
CG10[2]	2	2	2	1	2	2	2	2	0	2	1	1	3
CG11[2]	2	1	2	4	1	3	3,23	2	2	5	1	1	3
CG12[2]	2	1	2	4	1	2	3	2	2	5	1	1	3
CG13[2]	2	2	2	1	3	3	2	1	2	5	1,2	1	3
CG14[2]	2	1	2	2	3	2	2	2	2	5	1	1	3
CH591-36[2]	2	1	2	1	1	2	3	3	2	5	1	2	3
CH593-9[2]	2	2	2	1	3	1,12	2	1	5	2	1	1	3
CH611-10[2]	2	1	2	1	1	2	3	3	2	1	1	1	3
CH663-8[2]	2	1	2	4	1	2	2	2	5	2	1	1	3
CH665-1[2]	2	1	2	2	1	2	2	3	2	1	2	1	3

Table 2. Continued.

Inbred Line	Locus												
	Adh1	Idh2	Got1	Mdh2	Acp1	Acp4	Prx1	Est1	Est4	Glu1	Pgd1	Pgd2	Enp1
CH671-28	2	1	2	1	1	1	2	3	3	1	1	1	3
CH701-30	2	1	2	1	1	1	2	1	2	0	2	1	3
CH753-4	2	1	2	2	1	2	2	3	2	5	1	2	3
CI44	2	2	2	4	1,2	1	2	2	2	4	1	1	3
CI64	2,3	1	2	4	3	2	3	1	5	5	1	1	2
CI66	2	1	2	2	1	2	3	2	4	5	1	1	4
CI187	2	1	2	2	3	5	2	1	2	1	1	1	3
CI540	2	2	2	2	1	1	2	3	4	5	1	2	3
CK22	1	1	2	4	1	2	3	2	5	1	1	1	3
CK25	2	2	2	1	1	2	3	2	2	2	2	1	3
CK26	2	1	2	4	3	5	2	1	3	2	1	1	3
CK27	1	1	2	4	3	5	2	2	5	5	1	1	3
CK36	2,3	1	2	4	2	3	3	1	3	2	1	1	3
CK43	2	1	2	4	3	5	3	1	3	5	1	1	3
CK48	2	1	2	2	3	1	2	3	5	5	1	1	3
CK52	2	1	2	4	1	5	2	1	2	5	1	1	3
CK54	2	2	2	2	1	2	2	2	2	5	1	1	3
CK55	2	1	2	4	3	3	2	1	2	5	1	1	3
CK63	1	2	2	2	1	2	3	3	3	5	2	1	3
CK64	2	1	2	4	3	5	2	2	3	2	1	1	3
CK71	1	1	2	4	3	5	2	3	5	2	1	1	3
CM105	2	1	2	2	1	5	3	1	2	5	1	2	3
DE811	2	1	2	2	2	3	1	3	2	1	1	1	3
DE813	2	1,2	2	2	3,13	2,5	1	3	2	5	2	1	3
DE824	2	1	2	2	1	5	1	1	2	1	1	1	3
F502	2	2	2	4	1	3	2	1	4	5	1	1	3
F522	1	1	2	2	1	2	2	3	3	2	1	1	3
F542	2	2	2	2	1	2	2	3	2	5	1	2	3
F564	2	2	2	4	1	2	2	2	2	5	1	1	3
F572	2	2	2	2	1	1	3	3	2	1	1	2	3
FR07	2	2	2	2	1	1	2	2	4	3	1	2	3
FR9	2	1	1	1	3	1	3	3	2	1	2	1	3
FR14A	2	1	2	2	1	2	1	3	2	5	1	2	3
FR37	2	1	2	2	3	2	1	3	1	5	2	1	3
FR43	2	1	2	2	1	2	1	3	2	1	1	1	3
FR49	2	2	1	4	2	1	3	3	2	5	2	1	3
FR51A	2	1	2	2	1	5	1	2	2	5	1	1	3
FR64A	2	1	2	1	3	1	1	3	2	1	2	1	3
FR103D	2	2	2	2	2	3	1	1	2	1	1	1	3
FR123	2	2	2	2	1	3	1	1	2	2	1	1	3
FR153R	2	1	2	2	4	1	2	3	3	1	1	1	3
FR619	2	1	2	4	1	2	2	3	2	2	1	1	3
FR632	2	1	2	2	1	2	2	3	2	5	1	2	3
FR632 cms	2	1	2	2	1	2	1	3	2	5	1	2	3
FR802W	2	2	2	2	2	2	2	1	4	1	1	1	3
FRC121E	2	2	2	1	1	1	2	1	2	5	1	1	3
FRM01W cms	1	2	1	4	3	1	3	3	2	5	2	1	3
FRM017 cms	2	2	2	2	3	5	2	1	2	1	1	1	3
FRN2B	1,2	2,12	2	2	1,3	5	2,3	2,3	2	5	1,2	1	3
FRVa26	2	1	2	2	1	2	2	3	2	1	1	1	3
FR4C082	2	1	2	0	2	5	2	2	3	5	1	1	3
FR38-11	2	2	2	1	3	1	2	1	4	5	1	1	3
GA209	2	2	2	4	3	2	2	2	2	2	1	1	3
Gt112	1	2	2	4	2	1	2	2	2	5	2	1	3
H25	1	1	2	4	2	1	3	2	2	2	1	1	3
H30	2	2	2	4	3	1	2	2	4	5	1	1	3
H49	2	2	1	4	2	1	3	3	2	1	2	1	3
H51	2	1	1	1	3	2	1	3	2	1	2	1	3
H55	1	2	2	1	3	2	0	1	2	1	1	1	3
H60	2	2	2	4	2	2	2	2	2	5	1	1	3
H71	2	1	2	4	2	2	1	1	3	5	1	1	3
H73	2	1	2	2	1	5	2	2	4	5	1	1	3
H84	2	2	2	2	3	2	1	3	1	1	2	1	3
H88	2	2	2	1	1	2	2	3	2	0	1	1	3
H91	2	1	2	2	1	2	1	3	2	5	1	2	3
H92	2	1	2	2	1	2	2	3	2	5	1	2	3
H93	2	1	2	2	3	5	2	3	1	5	2	1	3
H94	2	1	2	2	3	5	1	3	1	5	2	1	3
H95	2	1	2	2	1	2	2	3	2	0	1	1	3
H96	1	2	2	4	3	2	2	1	2	1	1	1	3
H98	2	2	2	1	3	1	2	1	2	1	1	1	3
H99	2	1	2	4	3	5	1	1	2	1	0	1	3
H100	2	1	2	2	3	2	3	3	2	5	1	2	3
H101	2	1	2	2	1	2	2	3	2	0	1	1	3
H107	2	1	2	1	3	5	2	1	2	1	1	1	3
H108	2	2	2	2	3	5	2	1	2	1	0	1	3
H5505	1	2	1	2	2	1	1	2	2	5	1	1	2
HO2271	2	1	2	2	1	1	3	3	2	5	1	1	3
HI25	2	1	2	2	1	2	1	3	2	5	1	2	3
HI30	2	2	2	4	3	3,5	2	1	2	5	2	1	3
HY	1	2	2	1	3	1	2	1	2	1	1	1	3
I159	1	2	2	2	1	1	2	3	2	5	2	1	3
I205	1	1	2	1	1	2	2	1	2	1	2	1	3
Ia65:1271	2	1	2	4	1	5	2	3	4	0	2	1	3
Ind-A-H83	2	2	2	4	3	1	2	1	4	5	1	1	3
IPP(94)	2	2	2	2	1	2	2	1	4	1	2	1	3
ITS701	1	2	2	2	1	1	2	3	4	5	1	2	3
K6	2	1	2	2	3	5	2	2	3	5	2	1	2
K55	2	1	2	2	1	5	3	2	4	5	1	1	3
K64	2	1	2	2	2	2	1	2	5	5	1	1	3
K64Ht	2	1	2	2	2	3	2	2	5	5	1	1	3
K148	2	1	2	4	3	1	2	2	5	5	0	1	3

Table 2. Continued.

Inbred Line	Locus												
	Adh1	Idh2	Got1	Mdh2	Acp1	Acp4	Prx1	Est1	Est4	Glu1	Pgd1	Pgd2	Enp1
Ky21[2]	2	1	2	0	2	1	2	1	2	5	1	1	3
Ky201[2]	2	1	2	4	2	1	2	2	2	5	1	1	4
Ky216[2]	2	1	1	1	1	3	2	2	5	5	1	1	2
Ky226[2]	2	2	1	1	2	1	1	1	2	1	1	1	3
KY228[2]	2	1	2	2	3	5	1	1	2	2	2	1	3
L289[2]	2	2	2	4	1	2	2	2	2	5	1	1	3
L312[2]	2	2	2	2	1	1	1	1	2	5	1	1	3
M14[2]	2	2	2	1	1	1	1	3	2	5	1	1	3
ME2Rt[2]	1	2	2	2	1	2	2	2	2	5	1	1	3
MICH37W[2]	2	2	2	2	1	2	2	1	2	5	1	1	3
MICH37Y[2]	2	2	2	2	1	2	2	1	2	5	1	1	3
MICH37-6[2,3]	2	2	2	2	3	2	2	3	2	2	1	1	3
MO1W[2]	1	2	1	4	3	1	3	3	2	1	2	1	3
MO5[2]	2	2	2	4	1	5	3	2	2	5	1	1	3
MO6[2]	2	1	2	2	2	5	2	1	2	1	1	1	3
MO12[2]	2	2	1	4	3	4	3	2	2	1	2	1	3
MO13[2]	2	2	2	2	1	5	2	2	4	1	1	1	3
MO14W[2]	2	2	2	4	3	3	3	2	6	2	2	1	3
MO17[2]	2	2	2	2	3	5	2	1	2	1	1	1	3
MO17Ht	2	2	2	2	3	5	2	1	2	1	1	1	3
MO17 cms BC5	2	2	2	2	3	5	1,12	1	2	1	1	1	3
MO18W[2]	1	2	2	4	3	5	3	2	2	1	2	1	3
MO20W[2]	1	2	2	4	3	5	3	2	2	3	2	1	3
MO24W[2]	2	1	2	2	1	2	2	2	4	5	2	1	3
MO40[2]	2	2	2	2	3	3	2	1	2	1	1	1	3
MO41[2]	2	2	2	2	2	2	2	1	2	1	1	1	3
MO42[2]	2	2	2	2	3	5	2	1	2	1	1	1	3
MO43[2]	2	2	2	2	3	3	2	1	2	1	1	1	3
MP339[2]	2	2	2	4	3	2	2	2	2	5	1	1	3
MP444[2]	2	2	2	4	2	2	2	2	4	3	2	1	3
MS57	2	2	2	2	1	5	3	1	2	5	1	1	3
MS68	2	2	2	2	1	5	3	3	2	0	1	1	3
MS71[2]	2	2	2	4	1	1	3	1	5	2	1	2	3
MS80[2]	2	1	2	4	1	1	2	3	2	2	1	1	3
MS92[2]	2	1	2	2,12	1	5	3	3	2	5	1	1	3
MS106	2	1	2	2	1	1	0	3	2	5	1	1	3
MS107	2	2	2	4	1	2	3	1	4	2	1	1	3
MS132	2	1	2	4	1	2	2	3	2	1	1	1	3
MS142[2]	2	1	2	2	1	5	3	3	2	5	2	1	3
MS153[2]	2	2	2	4	1	2	2	3	4	5	1	1	3
MS213	2	1	2	1	1	2	1	3	2	5	1	1	3
MS214	2	2	2	1	3	2	1	1	3	5	1	1	3
MS1334[2]	2	1	2	4	1	5	2	2	5	5	2	1	3
N6G	1,12,2	2	2	4	3	5	3	3	2	3	1	1	3
N7A[2]	2	1	2	2	1	1	3	2	4	3	2	2	3
N8B	1	1	2	2	1	2	23,2	1	2	5	1	1	3
N20	2	2	2	1	1	2	3	1	2	5	2	1	3
N22A	2	2	2	4	3	2	2	1	2	1	2	1	3
N28[2]	2	1	2	2	3	6	3	3	2	5	2	1	3
N31	2	1	2	2	2	1	3,23	3	2	5	2	1	3
N38A	2	2	2	4	3	1	2	1	2	5	1	1	3
N103	2	1	2	2	2	3	3	3	3	5	1	1	3
N104	2	1	2	2	2	2,5	3,23,2	3	2,23	5	1,12,2	1	3
N115[2]	2	1	2	2	1,13	5,35	3,23	1	2	5	2,12	1	3
N132[2]	1,2	2	2	4	3	1	2	2	2	3	2	1	3
N139	2	1	2	4	3	1	2,23	1	4	3	2	1	3
N142	2	2	2	1	3	1	2	1	2	5	1	1	3
N143[2]	1	2	2	2	1	5	2	1	2	3	1	1	3
N152[2]	2	2	2	2	3	1	2	3	4	3	1	1	3
NC7[2]	2	2	2	4	1	3,5	0	2	2	3	1	1	3
ND79-9	1	1	2	2	1	2	1	3	2	3	2	1	3
ND79-12	2	1	2	2	3	6	2	3	5	5	1	1	3
ND79-14	2	2	2	4	4	2	2	2	5	2,5	2	1	3
ND80-2	2	2	2	2	3	2	2	2	2	2	2	1	3
ND80-3	2	2,12	2,12	1,2	1	6	2	2	3	1	1	1	3
ND80-14	2	1	2	4	3	1	2	2	2	2	1	1	3
ND203[2]	2	2	2	2	3	5	2	2	2	1	1	1	3
ND405	2	1	2	1	1	2	2	2	5	5	1	1	3
Oh05[2]	2	1	2	2	3	5	2	3	2	3	1	1	3
Oh07[2,3]	2	2	2	2	2	1	2	1	3	2	2	1	3
Oh07B[2]	2	2	2	2	1	1	2	2	4	3	1	2	3
Oh07Bht	2	2	2	2	1	1	1	2	4	3	1	2	3
Oh40B[2]	2	2	2	4	1	2	3	1	2	5	1	1	3
Oh41[2]	2	2	2	2	1	1	1	1	2	5	1	1	3
Oh43[2]	2	1	2	2	1	3	2	3	2	1	1	1	3
Oh45[2]	2	2	2	2	1	3	3	1	2	1	1	1	3
Oh51A[2]	2	1	2	2	1	5	1	2	2	5	1	1	3
Oh507	2	1	2	1	3	3	2	1	2	1	1	1	3
Oh508	2	1	2	2	1	3	2	3	2	5	1	2	3
Oh509	2	2	2	2	3	1	2	2	4	5	2	2	3
Oh509A	2	2	2	2	3	1	2	2	4	5	2	1	3
Oh510	2	1	2	2	3	5	2	2,3	4,1	5	2,12	1,2,12	3
Oh512	2	2	2	4	3	1,3	2	1	4	5	1	1	3
Oh517[2]	2	2	2	2	2	3	2	1	2	1	1	1	3
Oh545[2]	2	2	2	2	1	2	1	1	2	1	1	1	3
Oh551[2]	2	1	2	2	1	5	1	2	5	5	1	1	3
Oh561	2	1	2	2	3	5	1	2	2	0	2	1	3
Oh562	2	1	2	2	3	5	1	3	1	5	1	1	3
Oh563	2	1	2	2	3	5	1	3	1	5	1	1	3

Table 2. Continued.

Inbred Line	Locus												
	Adh1	Idh2	Got1	Mdh2	Acp1	Acp4	Prx1	Est1	Est4	Glu1	Pgd1	Pgd2	Enp1
Oh572	2	2	2	2	1	1	2	2	2	3	1	1	3
Oh1606	2	2	2	4	1	3	2	3	5	2	2	1	3
Oh1646	2	2	2	2	1	2	2	2	2	1	1	1	3
Oh1693	2	2	2	2	3	5	2	2	2,4	2	1	1	3
Oh8710	2	2	2	2	3	2	2	2	3	3	1	1	3
Oh8712	2	2	2	2	3	2	2	3,23	2	3	1	1	3
Oh916§3	2	2	2	4	3	1	2	1	6	2	1	1	3
OS420[2]	2	2	2	4	1	1	2	3	5	5	1	1	3
P309	2	2	2	1	2	2	3	1	3	5	2	1	3
P317[2]	2	2	2	2	1	1	2	1	2	1	1	1	4
Pa32[2]	2	2	1,2	4	3	2	2	2	2,5	2	2	2,12	3
Pa76-20	2	1	2	1	3	1	3	1	2	1	1	1	3
Pa76-32	2	2	2	4	3	5	2	3	2	1	1	1	3
Pa76-30	2	2	2	4	1	6	2	1	2	1	2	1	3
Pa77-48	2	2	2	2	2	2	2	1	3	5	1	1	3
Pa77-50	2	1	2	1	3,13	6	2	1	2	1	2	1	3
Pa77-63	2	1	2	4	1	1	3	1	2	2	2	1	3
Pa77-66	2	2	2	4	3	1	2	2	4	2	1	1	3
Pa77$_2$70	2	1	2	2	3	1	2	2	3	3	1	1	3
Pa91[2]	2	2	2	1	1	2	3	1	2	5	2	1	3
Pa405	2	1	2	2	1	5	2	3	5	2	1	1	3
Pa409$_2$	2	2	2	2	3	1	2	1	3	5	2	1	3
Pa762[2]	2	2	2	4	1	2	2	3	2	1	1	1	3
Pa864P	2	2	2	4	1	2	1	1,3	5	2	1	1	3
Pa872	2	1	2	2	1	2	2	1	3	1	1	1	3
R61	1	2	2	2	1	1	1	1	2	5	1	1	3
R168[2]	2	1	2	1	1	1	3	2	2	5	1	2	3
R168Ht	2	1	2	1	1	1	1	2	2	5	1	2	3
R177	2	1	2	2	1	3	3	1	2	5	1	1	3
R177HtA	2	1	2	2	1	3	3	1	2	5	1	1	3
R181HtB	2	1	2	2	3	1	2	2	2	5	1	1	3
R182	2	1	1	2	1	5	1	2	2	1,2	2	1	3
R222$_2$	2	1	1,12	1	3	1	3	3	2	1	2	1	3
SA24[2]	2	2	2	1	3	1	1	2	3	5	1	1	3
SC55[2]	2	2	2	4	3	2	2	1	1	2	1	1	3
SC213R[2]	2	2	2	4	3	2	2	1,2	2	5	2	1	3
SC344W	2	2	2	4	1	1	3	2	1,2	1	1	1	1
SD8	2	1	2	1	3	1	2	1	2	5	2	1	3
SD10[2]	2	2	2	2	3	5	3	2	3	1	1	1	3
SD12A	2	2	2	2	3	5	2	2	5	1	1	1	3
SD15[2]	2	1	2	1	3	1	3	1	2	1	2	2	3
SD23	2	1	2	2	1	2	2	3	2	2	2	1	3
SD30	2	1	2	1	3	1	3	1	2	1	2	2	3
SD33	2	2	1	4	1	1	3	3	2	5	2	1	3
SDp2A	2	1	2	2	1	1	2	3	2	5	2	1	3
SDp309	2,12	2	2	1	2	2	2	1	2	5	2	1	3
SDp310	2	2	2	1	2	2	3	1	3	5	2	1	3
SDp311	2	2	2	1	2	2	3	1	3	5	2	1	3
SDp312	2	2	1	1	1	2	3	1	3	5	2	1	3
T8[2]	2	2	2	2	1	3	2	2	2	1	1	1	3
T111$_2$	2	2	2	2	3	2	3	2	2	1	2	1	4
T115[2]	2	2	2	4	3	2	3	1	2	5	1	1	3
T220[2]	2	2	2	4	3	2	2	2	4	5	2	1	3
T220A[2]	2	2	2	4	3	2	2	2	4	5	2	1	3
T226[2]	2	1	1	1	3	1	3	1	2	1	2	1	3
T232[2]	2	2	2	2	1	2	0	3	4	2	1	2	3
Tx303[2]	2	1	2	4	1	2	2	2	2	5	2	1	3
Tx325[2]	1	1	2	4	1	1	2	2	2	5	2	1	3
Tx625[2]	2	2	2	2	1	1	2	3	2	1	2	1	3
Va17[2]	2	1	2	1	1	1	2	3	2	1	2	1	3
Va22$_2$	2	1	2	1	1	1	2	3	2	5	2	1	3
Va26[2]	2	1	2	2	1	1	2	3	2	1	1	1	3
Va31$_2$	2	1	1	1	3	1	3	3	2	5	2	1	3
Va35[2]	2	2	2	2	1	4	2	1	2	1	1	1	3
Va36[2]	2	2	2	2	2	3	2	1	2	1	1	1	3
Va50[2]	1	2	2	1	1	2	2	1	2	1	1	1	3
Va58[2]	2,12	2	2,12	2	1	3	2	1,12	2	5	1	1	3
Va59[2]	2	2	2	2	1	6	2	1	2	1	1	1	3
Va60[2]	2	2	2	2	1	2	2	2	2	1	1	1	3
Va94[2]	2	1	2	2	1	2	1	2	2	1	1	1	3
W22Ht	2	2	2	1	2	1	1	1,3	3	5	1	1	3
W59M	2	1	2	2	3	6	2	2	5	5	1	1	3
W64A	2	1	1	2	3	1	3	3	2	1	2	1	3
W117	2	1	2	4	3	1	2	1	3	5	2	1	3
W117Ht[2]	2	1	2	4	3	1	2	1	3	5	2	1	3
W153R	2	1	2	2	4	1	2	3	2	5	1	1	3
W182	2	1	2	4	3	2	1	3	3	4	1	1	3
W182B[2]	2	1	2	4	3	2	1	3	3	4	1	1	3
W182E	2	1	2	4	2	2	1	3	3	5	1	1	3
W202$_2$	2	1	2	4	1	5	2	3	5	1	2	1	3
W462[2]	2	2	2	4	2	1	1	1	3	5	1	1	3
W538[2]	2	2	2	1	2	1	2	2	2	5	1	1	3
W540[2]	2	2	2	2	1	4	2	1	2	1	1	1	3
W544[2]	2	1	2	2	3	1	2	3	4	5	1	2	3
W546	2	1	1	1	1	1	3	3	2	5	2	2	3
W7290	1	2	2	1	2	2	1	2	3	3	1	1	3
W845[2]	2	1	2	2	3	5	2	2	2	5	2	1	3
WF9[2]	2	1	1	1	3	1	3	3	2	1	2	1	3
WJ[2]	2	2	2	2	2	2	2	1	2	3	2	1	3
4C082[2,3]	2	1	2	0	2	5	2	2	3	5	1	1	3
38-11[2,3]	2	1	2	2	1	1	2	3	3	5	1	1	3

[1]Seed source of the inbred lines will be made available upon request.
[2]Designates inbred lines also assayed by Stuber and Goodman.
[3]Inbred lines for which genotypes were observed to be different at some loci between lab 1 and lab 2. Inbred Pa76-30 had a unique PGD phenotype. Allelism tests have not been completed so the phenotype was not included in the table.

Corresponding laboratory allozyme numbers of Stuber's laboratory are also given in Table 1 for all cases where the correspondence has been determined.

Table 2 (see preceding pages) presents genotypes at 13 enzyme loci (Adh1, Idh2, Got1, Mdh2, Acp1, Acp4, Prx1, Est1, Est4, Glu1, Pgd1, Pgd2, Enp1) for 363 U.S. inbred lines of maize. One hundred seventy five of these lines are common to the earlier lists, although the source of common lines may be different. These 175 lines are included because genotypes at loci Acp4, Prx1, Est1 and Est4 were not included in earlier lists. Genotypes are usually designated with a single number in the table because most lines are homozygous and homogeneous. For example, genotype 2 at locus Adh1 symbolizes the Adh1-2/Adh1-2 homozygote. Heterozygotes are designated with two numbers representing the allelic state of the hybrid. Thus genotype 12 at locus Adh1 symbolizes the Adh1-1/Adh1-2 heterozygote. When a line was heterogeneous all observed genotypes were presented with the most frequent genotype first, followed by the next most frequent genotype and so on. A minimum of two, and as many as 40, individuals were assayed per inbred line. Because of the relatively small sample sizes for some of the inbreds, rare variants probably were not detected. Therefore, these data should be considered preliminary. It is possible that some of the "uniform" inbreds are polymorphic due to contamination from seed mixing or outcrossing. In addition, genotypes for some inbreds and loci differ from earlier reported genotypes. In most cases this reflects different seed sources. Seed sources for inbred lines included in Table 2 will be made available upon request.

Among the 594 inbred lines that have been characterized for genotypes at enzyme loci, it is now possible, by inference, to determine expected single-locus and multilocus genotypes of 176,121 possible single-cross hybrids.

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Genetics of horizontal resistance to pests, kernel row number, carbohydrate synthesis, and overlapping super-genes

The authors (MNL 56:30-32 and 57:20-24) reported results on horizontal resistance with the standard 9 wx translocations in IAC Maya lte1 crossed to Zapalote Chico (ZC) and from bibliographic research. Now we report results with the same markers crossed to Cateto Palha Roxa (Purple Husk Cateto). This is a backyard collection picked in the Paraiba Valley between Sao Paulo and Rio near the city of Pindamonhangaba. It is a pure Cateto maintained by a conservative farmer family for generations and is reported to be resistant to weevils. The cross was "back-crossed" to IAC Maya wx y. The field plots consisted of one row 10m long planted in hills at 0.4m within the row. The hills were planted with three seeds each, alternating hills with normal and waxy endosperm of the same ear within the row. Six reps were used. Every fifth row was planted with IAC Maya wx y as pollinator, and the experiment was detasseled. The dry ears were harvested indiscriminately, unhusked. They were classified for presence vs. absence of exit holes of corn earworm, and for degree of damage in the cob by an adaptation of N. W. Widstrom's scale (J. Econ. Entomol. 60(3):791-794, 1967), lumping the 0 and 1 class in one and from two forward in the other, or without visible damage and damaged in the cob. Besides normal and semi-sterile ears, and Wx and wx, since the Cateto is P-WR and B-W and has a high frequency of Plp (Purple restricted to the cob), these factors were also classified, and the kernel row number was counted. A preliminary analysis was done of the two methods of measuring attack of earworm, unholed and holed husks versus undamaged and with visible damage in

the cob. The second method gave lower X^2 values and was henceforward dropped from further analysis. Data from the first reading are given in Table 1. A X^2 analysis in the usual way is in Table 2. In Table 3 are the values of p calculated by additive method, always in the direction of linkage. It is evident in the linkages and X^2 values between wx and translocation that there are quite disturbed segregations. Consequently a second X^2 analysis was done using the contrast (a+d-b-c)/n, squared (Table 4). The values obtained compared with the first method show that this method is also quite indicative of linkages. So a second calculation of p was done using the product moment method for backcross, equating $(1-p)^2/p^2 = (axd)/(bxc)$ which resolves into the quadratic $(axd-bxc)p^2+2bcp-bxc$ (Table 5). The standard error is the same as by maximum likelihood, which is equal to the additive method in this particular case. In general the product moment method was more efficient, giving tighter values of p when the translocations were involved. All p values between the translocations and wx were tighter. For further analysis it was considered the stronger value between the two methods. With these values we applied the theorem: the real distance in centimorgans between two factors with heritability less than one is the difference between the distances of them converted to centimorgans with a third factor outside their span. As illustrated by cross number 5 (2 and 3 were quite abnormal):

	p	cM		p	cM	difference T wx
T Krn	37.2	46.5	wx Krn	44.7	65.0	18.5
T Flt	38.1	48.2	wx Flt	39.8	51.9	3.7
T U	40.2	52.6	wx U	41.3	55.1	2.5
T P-WR	47.4	77.8	wx P-WR	45.9	69.7	8.1
	Mean value of wx T by difference in centimorgans:					8.2

In the following table are the results for T wx with all crosses, comparing both methods, direct, and by difference. In the first line are direct p recombination values between wx and T; in the second line, these values are transformed to cM; in the third line, values are those obtained by differences based on the theorem. The abnormal results for crosses 2 and 3 were also quite improved, cross 2 from 46.7 to 3.0, and cross 3 from 28.9 to 10.4.

	5	6	7	11	12	13	14	15	16	18	20	Mean
p	7.5	10.2	11.8	16.3	6.2	15.8	14.5	15.8	14.1	12.0	10.0	12.2
cM	7.7	10.5	12.3	18.4	6.3	16.8	15.3	16.8	14.8	12.5	10.3	12.9
cM	8.2	7.1	4.6	11.4	8.8	17.1	3.4	21.7	12.1	7.9	3.7	9.6

The first analyses were done without taking readings for the flint (Flt) character. On the stored material a second reading of the data was done for flint x dent dividing visually in two classes (Table 6). For the appropriate chromosomes a reading was done of Flt with P-WR, B-W, and Plp. Since Cateto is Bn and IAC Maya wx y has a high frequency of bn, this factor was also read accordingly. From these last readings only the p values are presented. With the method above illustrated by linkages between translocations and waxy, all combinations of linkages between factors were calculated among all factors. With these values were mounted the tentative linkage groups listed after Table 6, with positions and distances indicated.

We have also taken into consideration the Cytogenetic Working Map in MNL 52:129-145. For linkage of Flt8 in chromosome eight we had unpublished data in crosses of Cateto with 8L.09;9S.16 and 8L.35;9S.31, showing more linkage with the latter in a more distal position of the long arm.

Table 1. First reading of the experiment, without flint x dent (Flt x flt) reading. Abbreviations: U=Unholed husks, H=holed husks; B=colored husks, b=uncolored husks; Wx=normal endosperm, wx=waxy endosperm; N=non-sterile ears, S=semi-sterile ears; Plp=purple cob, P-WR=red cob color, P-WW=white cobs. Results measuring attack of corn earworm, Heliothis zea (Boddie), as given by holed husks. The last sub-division is kernel row number.

	U/H	B/b	Wx/wx	N/S	Plp / P-WR / P-WW	9S.7;9L.9 16	14	12	10	8		1S.48;9L.22 16	14	12	10		1L.74;9L.13 16	14	12	10		2S.18;9L.22 16	14	12	10	8		3L.09;9L.12 18	16	14	12	10		5L.69;9S.17 18	16	14	12	10	8		5S.07;9L.10 18	16	14	12	10	8	
1	+	+	+	+	+			1			1			2	1	3					0		1				1					0						0						0
2	+	+	+	+	0						0					0		1			1		1				1			2	2		4				3			3			2			2
3	+	+	+	+	-						0					0			1		1		2				2			3	5		8							0			1			1
4	+	+	+	-	+			1			1			1		1					0						0						0							0			2			2
5	+	+	+	-	0			1	1		2					0			1		1						0						0	1						1						0
6	+	+	+	-	-						0					0			1		1						0			2			2				1			1						0
7	+	+	-	+	+			1	1		2					0					0						0						0							0						0
8	+	+	-	+	0			1			1			1		1					0		1				1						0							0						0
9	+	+	-	+	-		1	1			2					0					0						0						0							0						0
10	+	+	-	-	+			1			1			2		2				2	2						0			1	1		2			1	1	1		3			2			2
11	+	+	-	-	0			1			1		1			1					0			3			3			2	2		4			1	1	1		3			1	1		2
12	+	+	-	-	-						0			2	1	3		1	5	2	8		2	1	1		4				6		6				1	1		2				1	1		2
13	+	-	+	+	+			1			1					0			1	1	2		1				1						0							0			1			1
14	+	-	+	+	0			1	1		2					0			3		3		1	4	1		6		1	2	5		8			2	2	2		6			4	8		12
15	+	-	+	+	-		3	2	2		7		3	7	4	14		3	7	2	12	1	6	2	1		10			2	2	1	5				1	1		2		1	4	7	1	13
16	+	-	+	-	+						0					0					0			1			1						0							0						0
17	+	-	+	-	0		1			1	2					0				1	1			1	1		2	1		1			2			1	1	4		6			1			1
18	+	-	+	-	-		1	7			8				2	2				3	3						0			2			2			2	2	2		6				1		1
19	+	-	-	+	+						0					0					0						0						0							0						0
20	+	-	-	+	0		1		1		2			1		1					0		1	1			2			1	1		2			1	1			2						0
21	+	-	-	+	-		2	9			11	1		3	2	6					0						0				1		1							0						0
22	+	-	-	-	+						0					0				1	1		1				1						0					1		1						0
23	+	-	-	-	0		1	2			3				1	1			7	6	13			9	4		13			6	6	1	13			2	9	6		17			3	9	2	14
24	+	-	-	-	-			2	5	1	8				3	3	1	3	11	3	18		6	10	4		20			3	15	3	21			3	7	8	1	19			5	18	13	36
25	-	+	+	+	+		1	1	1	2	5			1		1			1		1			1	2		3	1		1	1		3				1			1			1			1
26	-	+	+	+	0			1			1					0			3	2	5	1	1	2	1		5			1	1		2				5			5			1			1
27	-	+	+	+	-		1				1				1	1			2	1	3			1			1			2	3	1	6					2		2						0
28	-	+	+	-	+						0					0					0			2			2						0		1		1			2						0
29	-	+	+	-	0			1			1					0					0			1			1				2	1	3				4			4			1	1		2
30	-	+	+	-	-					1	1			1		1		1	2		3				2		2				2	2	4				2	1		3						0
31	-	+	-	+	+			2			2					0					0		2				2						0							0						0
32	-	+	-	+	0	1					1					0					0						0						0							0						0
33	-	+	-	+	-						0					0					0		1				1						0							0						0
34	-	+	-	-	+			1	1	1	3			1	2	3			1	2	3				1		1				1		1				2	1		3			2	3		5
35	-	+	-	-	0			1	1		2				3	3			3		3			1			1			5	2	7		1	1	8			10				2		2
36	-	+	-	-	-			2	3		5			2	2	4		2	6	2	10		1	1			2			2	7		9				1	1		2				3		3
37	-	-	+	+	+			2			2					0			1		1		2	10			12						0				1			1	1	1	6	13	1	22
38	-	-	+	+	0	2	8	12	2		24					0			4	1	5		7	8	4	1	20		1	5	16	2	24			7	11	5	2	25		1	12	18	3	34
39	-	-	+	+	-	5	14	20	4		43		9	16	6	31	1	11	38	4	54	5	18	15	4		42	2	3	10			15	1	3	6	21			31						0
40	-	-	+	-	+						0					0					0						0						0							0			1	4	3	8
41	-	-	+	-	0	1	1	3			5			1		1					0		1	2	2		5				3		3			1	3	2		6				6	4	10
42	-	-	+	-	-	5	5	2	1		13			1	1	2		1	5	6	12			3	1		4			2			2				3	1	1	5						0
43	-	-	-	+	+						0					0					0						0						0							0			1			1
44	-	-	-	+	0	1	3	4			8		4	10	1	15					0		2				2				1		1			1	1	2		4			1			1
45	-	-	-	+	-	1	9	10	6		26		4	6		10			2	1	3		1	1			2		1	1	1		3	1	1	2		1		5			1			1
46	-	-	-	-	+						0					0					0						0						0				1			1						0
47	-	-	-	-	0		1	4		1	6			1		1		1	4	4	9		4	12	8		24			8	16	2	26			1	1	20	4	1	27			3	13	3	1	20
48	-	-	-	-	-		8	21	1		30			12	8	20		10	25	7	42		12	29	6		47			7	8	7	22			1	3	27	10		41			4	25	9		38
											234					128					221						247						211							250						237

Table 1. Continued.

5L.14;9L.10								6S.79;9L.40								6L.10;9S.37						7L.63;9S.07						8L.09;9S.16							9S.13.10S.40							Total						
18	16	14	12	10	8		18	16	14	12	10	8		16	14	12	10		16	14	12	10		16	14	12	10	8		16	14	12	10	8		18	16	14	12	10	8	
	1					1			3	4			7					0					0		3				3		1	2			3		1	8	9	1		19
						0							0					0					0						0			1			1			4	8			12
1						1							0		1Y			1					0						0		1				1	1		7	6	1		15
						0							0		1Y			1			1	1	2			2			2						0			1	6	2		9
		1				1							0					0			1		1						0						0		1	1	3	1		6
			1			1				1			1					0					0			1			1						0				7			7
						0							0					0					0						0						0				1	1		2
						0							0					0					0						0						0			1	2			3
						0							0					0					0						0						0			1	1			2
			1	1		2							0					0			5	1	6						0		1	6			7			2	19	6		27
						0							0			1Y		1			1		1			2		1	3		2		1		3			6	12	3	1	22
						0							0			1Y		1		1	3		4			5	1		6		2	3	2		7			6	27	9	1	43
						0							0					0					0		1		1		2						0			3	2	2		7
		1				1			3	2	1		6					0		2	2	3	7		2	1			3			4	2		6		1	17	32	10		60
1		6	5	1		13	1	3	3	8			15					0		2	5	2	9	3	6	13	3		25		2	11	2		15	2	9	40	70	20		140
						0							0					0					0						0						0				1			1
						0							1					0		1	3		4			3			3		1	2	1		4		1	4	12	7	1	25
			1			1			1	1	1		3					0	1		3		4		1				1			3	1		4		1	5	19	10		35
						0							0					0					0						0						0							0
						0							0					0			1		1			2			2						0			4	8	1		13
		1				1		1		3			3					0					0		2				2		2	1	1		4		2	7	17	3		29
						0							0					0			1		1						0						0				2	2		4
				1		1			4	4	2		10		1y	1y		2			4	2	6			4	1	1	6		2	8	3	1	14			19	63	30	1	113
		3	4	2		9		5					5	2y		3y		5		2	3	2	7		1	14	2		17		7	13	5		25		8	33	100	50	2	193
						0			5	5			10	1Y				1		1	2	1	4		1	1			2						0	1	1	10	14	4	2	32
						0			1	2			3					0		1			1		1				1		1	1			2		1	7	15	3		26
						0		2		3			5					0		1	1		2			2			2	1		1			2		3	4	12	6		25
		1				1							0		1Y	1Y		2			2	2	4			1		1	2		1				1		1	3	7	2	1	14
						0							0					0		1			1		1	1			2						0			3	10	1		14
						0							0					0		1			1						0			1			1			2	8	5	1	16
						0		1					1					0		1			1						0						0		1	3	2			6
						0							0					0			1		1						0			2			2		1		3			4
						0			1				1					0					0						0						0			2				2
		1				1			2	1	1		4					0		1	4	2	7			5			5			2			2			6	21	10	1	38
						0			2	1	1		4					0		1	1	2	4			4	2		6	1	2	4			7		2	6	30	3		46
						0				2	1		3					0		1	3	1	5			4	6	1	11		2	2			4			8	33	16	1	58
						0			1				1			1Y		1		2	3		5		1	2			3		1	1			2			7	21			28
	1	3	1			5		1	4	2			7		1Y			1		6	7	2	15		3	9			12	2	7	11	1		21	1	8	57	94	18	3	181
	5	14	9			28		3	12	13	3		31		6Y			6	1	20	17	5	43	4	10	17	2		33	1	3	14	6		24	3	32	145	198	37		415
						0							0					0					0						0						0							0
						0			1	2	1		4					0			1		1			4			4		1	3	1		5		1	6	26	9		42
		2	1			3			2	3			5		1Y			1		1			1			8			8						8		5	14	34	16	1	70
						0				1			1			1Y		1					0						0						0				2			2
			1			1			1				1					0		1	3		4						0						0		1	12	21	3		37
1	2	1	4	2		10	1		2	5			8		1Y	1Y		2		2	2	1	5	1		3			4						0	3	6	23	36	11		79
						0							0					0					0			1			1						0				2	1		3
		3	5	1		9		2	4	9			15					0		6	8	2	16			9	5		14		4	3	3		10		3	35	104	32	3	177
1		4	17	5		27		2	23	25			50		4y	7y	1y	12		11	25	5	41		1	30	15	1	47		11	20	10		41	1	3	99	270	84	1	458
						117							206					38					215						233						223	3	23	169	427	159	6	787
																																				9	69	452	963	266	14	1773

Table 2. Usual X^2 analysis of contingency tables. When kernel row number was involved a nx2 table was constructed. Underlined, our identification number. Marker involved in the cross (Cateto Palha Roxa x IAC Maya latente with marker) x IAC Maya wx, y. T= translocation; Wx non waxy ; U= unholed by earworm; Krn= kernel row number; Flt= flint endosperm type; B= booster gene (as seen in the husks); P-WR= red cob colour; Plp= purple cob colour. Last column degrees of freedom for kernel row number also applicable to Krn x Flt in the line below.

	Marker	TxWx	TxU TxKrn TxFlt	WxxU WxxKrn WxxFlt	UxFlt KrnxU KrnxFlt	BxU BxKrn BxFlt	P-WRxU P-WRxKrn P-WRxFlt	PlpxU PlpxKrn PlpxFlt	D.F.
2	9S.7;9L.9	14.41**	1.91	1.68	6.27*	2.06	0.60	1.02	1
			5.96	9.56*	11.06	19.24**	4.04	8.59	4
			2.37	2.21	2.14§	3.29			1§
3	1S.48;9L.22	23.93**	0.02	0.02	1.13	6.20*	0.84	5.20*	
			17.68**	2.44	6.47	3.18	4.37	5.65	4
			5.84*	6.50*	3.77	0.07	0.69		
5	1L.74;9L.13	138.95**	6.52*	5.55**	0.11	0.22	7.05**	0.04	
			8.86**	1.81	4.41	1.73	8.41*	2.48	3
			10.98**	9.32**	11.80**	0.39	0.74		
6	2S.18;9L.22	166.16**	4.45*	7.04**	2.19	1.49	1.02	2.30	
			28.38**	11.45	1.18	1.76	10.11*	2.31	4
			17.02**	9.33**	20.17**	0.34			
7	3L.09;9L.12	122.19**	0.81	1.48	1.55	0.81	1.81	0.00	
			20.43**	12.18*	5.09	5.86	4.40	3.60	4
			28.38**	32.93**	8.65	0.15			
12	5S.07;9L.12	148.39**	1.50	5.45*	0.02	0.49	0.14	0.41	
			28.90**	16.08**	2.54	4.50	6.86	4.19	5
			4.60*	5.50*	11.63*	3.31			
13	5L.14;9L.10	54.81**	0.02	2.34	0.03	9.81**	0.95	3.81	
			17.54*	21.29**	3.40	3.03	1.52	1.62	4
			0.07	0.69	2.40	2.12			
11	5L.68;9S.17	101.80**	12.50**	3.53	0.01	0.00	1.32	0.01	
			14.05*	4.67	16.38**	8.17	2.96	2.57	
			10.20**	5.63*	8.59	3.44			
14	6S.79;9L.40	103.59**	5.41*	5.77*	0.05	0.68	2.73	0.13	
			3.13	0.29	7.26	1.19	3.82	0.60	4
			28.96**	25.41**	5.87	0.50		0.37	
15	6L.10.9S.37	13.64**	4.34*	2.32	0.26	3.32	4.07*	3.40	
			5.24	10.87*	3.86	0.69	0.47	0.83	3
			6.74**	8.13**	5.40	3.07		1.00	
16	7L.63;9S.07	109.16**	5.17*	0.12	1.08	1.28	3.34	0.03	
			7.15	6.73	8.63	3.43	1.82	2.01	3
			27.04**	15.45**	9.80*	35.83**			
18	8L.09;9S.16	126.51**	3.26	1.55	5.14*	0.00	0.14	0.15	
			59.75**	45.39**	5.52	7.59	4.89	4.88	4
			39.15**	22.12**	22.77**	0.57			
20	9S.13;10S.40	133.32**	1.96	3.14	1.57	1.77	0.51	3.43	
			6.42	5.85	5.54	6.28	3.68	7.63	4
			31.88**	45.77**	1.79	2.31			
	Sum U	1256.86**	47.87**	39.99**	19.41	28.13**	24.52**	19.93	
	Total	1155.69**	17.23**	7.83**		10.75**	2.78	0.77	
	Interaction	101.17**	30.64**	32.16**		17.38	21.74**	19.16	
	Sum Krn		223.49**	148.61**	81.34**	66.64**	57.35**	46.96**	
	Total		140.47**	55.54**	12.71**	14.84**	13.44**	2.38	
	Interaction		83.02**	93.07**	68.63**	51.80**	43.91**	44.58**	
	Sum Flt		213.23**	188.99**	114.78**	55.39**			
	Total		181.72**	140.83**					
	Interaction		31.51**	48.16**					
	Number of significant items		6*	4*	2*	2*	2*	1*	17
			8*	6*	1*	1*	2*	0*	18
			11*	11*	5*	1*	0	0	28
			25	21	8	4	4	1	63 212

* Significant at P< 0.05.
** Significant at P< 0.01.
§ KrnxFlt has the degrees of freedom of the Krn line

Table 3. Linkages calculated by the additive method. Underlined our identification number. Marker involved in the cross (Cateto Palha Roxa x IAC Maya latente with marker) x IAC Maya wx, y. T= translocation; Wx non waxy; U= unholed by earworm; Krn= kernel row number; Flt= flint endosperm type; B= booster gene; P-WR= red cob colour; Plp= purple cob colour. Last column, s.e.= standard error for 50% recombination, no linkage, valid for the first five columns of parameters. For the last two n (the population) differs. TxU means (Normal-Sterile) x (Unholed-Holed) in the others always first the sign shown. Higher row number also first.

	Marker	Txwx	TxU TxKrn TxFlt	WxxU WxxKrn WxxFlt	UxFlt KrnxU KrnxFlt	BxU BxKrn BxFlt	P-WRxU P-WRxKrn P-WRxFlt	PlpxU PlpxKrn PlpxFlt	s.e. p=50,0
2	9S.7;9L.9	37.6	-40.6 +48.3 -49.3	-45.7 +43.2 -45.4	-31.6 +47.9 -42.0	+28.2 +43.6 -28.1	+38.6 +44.2	+32.1 +47.4	3.2
3	1S.48;9L.22	31.5	-44.5 -30.5 -42.7	-44.5 +43.0 -41.8	-46.7 +39.1 -47.3	+28.9 +32.0 +48.9	+38.1 -41.5 +47.5	+25.8 -25.8	4.4
5	1L.74;9L.13	10.9	-45.7 +46.6 -38.1	-43.0 +46.6 -40.3	-47.6 +38.5 -46.3	+36.7 -32.1 -43.3	+30.3 -27.5 -48.4	+47.1 -35.3	3.4
6	2S.13;9L.22	10.9	-45.7 +34.4 -36.7	-42.9 +42.9 -40.3	-44.3 +41.7 -38.7	+31.2 +35.6 -46.4	-42.2 -45.7	+43.6 -39.1	3.2
7	3L.09;9L.12	11.8	-49.8 +35.1 -35.3	-47.4 +40.3 -32.3	50.0 +49.3 -47.0	+42.2 +48.8 +45.3	-45.8 -47.8	+35.2 +32.4	3.4
12	5S.07;9L.10	11.0	-49.4 +31.6 -39.2	-43.5 +39.2 -41.8	-46.0 -38.8 -33.3	+38.8 +23.3 -39.2	+48.2 -48.7	+37.8 -23.6	3.2
13	5L.14;9L.10	16.2	-49.6 +33.3 -49.6	+42.7 +30.8 +46.3	-49.6 +47.9 -45.4	+23.9 +44.4 +49.6	+36.6 +45.5	+21.7 +47.8	4.6
11	5L.68;9S.17	18.0	-46.8 +33.2 -35.7	-47.2 +42.4 -41.4	+43.8 +36.0 -36.1	+36.4 +31.6 -28.7	+46.6 +49.6	+35.1 -28.2	3.2
14	6S.79;9L.40	14.6	+42.7 +49.0 -31.3	+41.7 50.0 -32.6	-47.0 +49.0 -48.3	+36.9 -45.6 -43.0	+34.3 +49.2	+45.3 +46.7 -44.3	3.4
15	6L.10;9S.37	18.4	+42.1 +34.2 -31.7	-44.7 +26.3 -29.3	+43.9 -44.7 -31.7	+26.3 50.0 -41.5	+25.0 -46.9	-20.0 50.0 -11.1	8.1
16	7L.63;9S.07	14.4	-45.6 +40.0 -31.6	+48.4 +42.3 -36.2	-45.4 +47.9 -37.2	+32.6 +43.3 -40.2	+36.2 +47.6	+44.1 +47.3	3.4
18	8L.09;9S.16	13.3	+41.2 +25.8 -27.5	+45.1 +33.0 -34.3	-36.7 +34.3 -27.1	+39.5 -27.9 -40.3	+42.7 -36.1	+39.5 +26.3	3.3
20	9S.13;10S.40	11.2	-47.5 +49.8 -35.0	-45.3 +48.9 -30.4	-47.4 -49.8 +46.2	+41.7 -35.4 +41.3	+49.8 +41.1	+37.4 +37.4	3.3
Means		16.9	45.5 37.8 37.2	44.8 40.7 37.9	44.6 43.5 40.6	34.1 38.4 41.8	39.6 44.0	35.7 37.9	
Number of signs in the most frequent direction			7- 12+ 13-	9- 13+ 12-	11- 10+ 12-	13+ 9+ 9-	11+ 7-	12+ 8+	

Table 4. X^2 analysis, $X^2=(a+d-b-c)^2 \div n$. When kernel row number was involved it was reduced to 2x2 at the point where observed minus expected changed sign. Underlined, our identification number. Marker involved in the cross (Cateto Palha Roxa x IAC Maya latente with marker) x IAC Maya wx , y. T= translocation; Wx non waxy; U= unholed by earworm; Krn= kernel row number; Flt= flint endosperm type; B= booster gene (as seen in the husks); P-WR= red cob colour; Plp= purple cob colour

	Marker	TxWx	TxU TxKrn TxFlt	WxxU WxxKrn WxxFlt	UxFlt KrnxU KrnxFlt	BxU BxKrn BxFlt	P-WRxU P-WRxKrn P-WRxFlt	PlpxU PlpxKrn PlpxFlt	
2	9S.7;9L.9	14.38**	8.27** 0.27 0.04	1.71 4.38* 1.91	31.28** 0.52 5.93*	44.46** 3.85* 44.16**	11.17** 2.91	10.05** 0.21	
3	1S.48;9L.22	18.00**	1.53 19.53** 5.13*	1.53 2.53 6.36*	0.94 6.12* 0.71	22.78** 16.53** 0.10	6.64** 3.39 0.56	7.26** 7.26**	
5	1L.74;9L.13	135.43**	1.63 1.02 13.09**	4.35* 1.02 8.97**	0.52 4.35 1.25	15.75** 28.24** 4.16*	31.09** 42.77** 0.22	0.18 4.41*	
6	2S.18;9L.22	150.81**	1.79 19.83** 17.56**	4.96* 4.96* 9.29**	3.16 6.81** 12.65**	35.02** 6.81** 1.31	5.49* 1.62	1.78 5.24*	
7	3L.09;9L.12	122.85**	0.00 18.81** 19.93**	0.57 7.97** 28.98**	0.00 0.04 0.84	5.16* 0.12 2.09	1.41 0.40	9.15** 20.15**	
12	5S.07;9L.10	144.41**	0.04 31.94** 10.97**	4.05* 10.97** 6.42*	1.52 11.85** 25.02**	11.85** 44.76** 10.97**	0.28 0.16	5.88* 18.00**	
13	5L.14;9L.10	53.34**	0.01 13.00** 0.01	2.47 17.31** 0.67	0.01 0.21 1.00	31.80** 1.44 0.01	8.04** 0.89	7.34** 0.04	
11	5L.68;9S.17	102.40**	1.02 28.22** 20.08**	0.78 5.48* 7.23**	3.69 19.60** 18.95**	18.50** 33.86** 16.79**	1.08 0.02	11.61** 24.80**	
14	6S.79;9L.40	103.48**	4.37* 0.08 32.16**	5.61* 0.00 27.83**	0.85 0.08 0.28	14.16** 0.31 4.45**	17.95** 0.05	0.65 0.33 0.91	
15	6L.10;9S.37	15.16**	0.95 3.79 5.49*	0.42 8.53** 7.05**	0.61 0.42 5.49*	8.53** 0.00 1.20	8.00** 0.12	3.60 0.27 9.00**	
16	7L.63;9S.07	108.88**	1.68 8.60* 29.36**	0.23 5.07* 16.51**	1.83 0.38 14.38**	26.16** 3.91* 0.29	14.06** 0.44	1.30 0.27	
18	8L.09;9S.16	125.50**	7.21** 54.80** 47.61**	2.27 26.79** 23.20**	17.36** 22.87** 49.42**	10.30** 45.53** 8.91**	4.51* 16.34**	3.31 17.05**	
20	9S.13;10S.40	134.21**	0.54 0.00 24.06**	1.98 0.11 40.66**	0.74 0.00 1.50	6.14* 18.95** 7.95**	0.00 6.61*	5.81* 5.81*	
	Sum U	1228.85**	29.04**	30.93**	62.51**	250.61**	109.72**	67.98**	
	Total	1161.01**	2.76	1.81		224.44**	62.92**	49.29**	
	Interaction	67.84**	26.28**	29.12**		26.17**	46.80**	18.69 n.s	
	Sum Krn		199.89**	95.12**	73.25**	204.31**	75.72**	103.54**	
	Total		135.06**	52.90**	39.01**	153.08**	29.43**	80.00**	
	Interaction		64.83**	42.22**	34.24**	51.23**	46.29**	23.54**	
	Sum Flt		225.49**	184.88**	137.42**	102.45**			
	Total		181.72**	148.58**					
	Interaction		43.77**	36.30**					
	Number of significant items		3* 8* 11*	4* 9* 11*	2* 5* 7*	13* 9* 7*	9* 3* 0	3* 8* 1*	38 42 37
			22	24	14	29	12	16	117 / 212

* Significant at P< 0.05
** Significant at P< 0.01

Table 5. Linkages calculated by the product method. Underlined our identification number. Marker involved in the cross (Cateto Palha Roxa x IAC Maya latente with marker) x IAC Maya wx, y. T= translocation; wx non waxy; U= unholed by earworm; Krn= kernel row number; Flt= flint endosperm type; B= booster gene; P-WR= red cob colour; Plp= purple cob colour. Last column, s.e= standard error for 50% recombination, no linkage, valid for the first five columns of parameters. For the last two n(the population) differs. TxU means (Normal-Sterile) x (Unholed-Holed) in the others always first the sign shown. Higher row number also first. Values of p in script, tighter values than by additive method presented in other table.

	Marker	Txwx	TxU TxKrn TxFlt	WxxU WxxKrn WxxFlt	UxFlt KrnxU KrnxFlt	BxU BxKrn BxFlt	P-WRxU P-WRxKrn P-WRxFlt	PlpxU PlpxKrn PlpxFlt	s.e p=50,0
2	9S.7;9L.9	37.3	-44.7 +43.5 -43.8	-45.0 +41.7 -44.3	-40.0 +38.0 -47.3	+42.8 -33.0 -41.1	-48.9 -48.8	+42.7 -25.5	3.2
3	1S.48;9L.22	25.8	-49.3 +33.4 -41.3	-49.3 +43.9 -41.7	-46.4 +44.6 -47.2	+35.6 -31.2 -48.8	-43.2 +32.7 +47.0	+28.4 -28.4	4.4
5	1L.74;9L.13	7.5	-40.2 +37.2 -38.9	-41.3 +44.7 -39.8	+48.8 +44.0 -42.4	+47.8 -47.4 -47.3	+33.5 -39.6 +46.3	+48.2 -37.0	3.4
6	2S.18;9L.22	10.6	-42.3 +49.2 -36.9	-40.4 +42.3 -40.3	-44.7 +49.2 -38.5	+43.8 +48.7 -47.3	+46.2 -41.3	-39.2 +39.2	3.2
7	3L.09;9L.12	11.0	-46.7 +37.1 -30.8	-45.5 +41.3 -29.9	-45.5 -45.9 -40.3	+46.5 -41.4 -48.5	+45.2 +46.9	50.0 +28.4	3.4
12	5S.07;9L.10	6.3	-45.7 +33.3 -42.5	-42.1 +36.8 -41.9	+49.4 -44.2 -40.1	+46.3 -43.4 +37.8	-48.7 +46.1	+44.8 +42.7	3.2
13	5L.14;9L.10	15.8	+49.3 +33.0 -48.7	+42.1 +30.8 +46.2	-47.0 +49.5 -44.9	+24.4 +41.2 -36.1	+42.0 +47.3	+26.7 +44.9	4.6
11	5L.68;9S.17	16.3	-35.8 +37.7 -38.9	-43.3 +43.6 -42.1	+49.6 -48.8 -40.7	+49.9 -44.3 -42.1	-45.9 -49.4	+41.1 +44.5	3.2
14	6S.79;9L.40	14.5	+40.6 +49.1 -32.0	+40.4 -49.9 -33.2	+49.2 -49.4 +47.9	-45.5 -46.3 -46.8	+42.6 -49.1	-47.6 +47.5 -46.0	3.4
15	6L.10;9S.37	19.3	-26.1 +30.9 -28.2	-34.5 +22.4 -26.4	+45.7 -44.9 -31.9	+31.9 +47.2 -32.6	+25.0 -48.2	-20.5 50.0 -20.0	3.1
16	7L.63;9S.07	14.1	-40.7 +40.3 -31.6	+48.6 +41.6 -36.1	+45.6 -37.4 -41.3	+44.8 -42.6 +30.0	+42.0 -46.7	-49.0 +40.5	3.4
18	8L.09;9S.16	12.0	+43.7 +17.3 -28.3	+45.7 +22.2 -33.3	-41.7 +41.8 -32.2	50.0 -42.9 +46.8	+47.7 +48.5	+47.4 -47.3	3.3
20	9S.13;10S.40	10.0	-45.1 50.0 -31.4	-43.9 -47.2 -28.3	-46.1 -45.4 -49.3	+44.4 +34.2 +44.2	-47.3 +46.8	+37.4 -47.3	3.3
Means		15.4	42.3 37.8 36.4	43.2 39.1 37.2	46.2 44.9 41.9	42.5 42.3 42.2	43.3 45.5 46.6	40.2 40.2 33.0	
Number of signs in the most frequent direction			10- 13+ 13-	9- 11+ 12-	8- 7- 12-	12+ 9- 9-	13+ 7- 2+	9+ 8+ 2-	

Table 6. Second reading of the experiment now including the Flint (Flt) x dent character. Only combinations with Wx and wx an normal versus semi-sterile ears are shown. The data differ slightly from the first reading in Table 1.

	Wx/wx + -	N S + -	Flt/flt + -	2	3	5	6	7	11	12	13	14	15	16	18	20	Totals
1	+	+	+	56	38	42	41	14	36	51	23	28	2	40	40	16	427
2	+	+	-	25	58	52	66	76	34	36	26	53	11	45	44	78	604
3	+	-	+	25	6	15	13	6	25	16	7	10	1	12	19	7	162
4	+	-	-	8	7	9	4	14	15	8	5	7	3	4	5	19	108
5	-	+	+	43	38	2	3	3	0	2	2	6	1	5	3	6	114
6	-	+	-	14	37	1	3	4	4	0	5	6	2	7	5	6	94
7	-	-	+	50	36	75	75	64	94	92	23	89	14	85	103	82	882
8	-	-	-	10	19	35	43	51	36	32	30	31	7	20	17	52	383
				231	239	231	248	232	244	237	121	230	41	218	236	226	2774

Positions and distances in centimorgans of factors studied

2	9S.7;9L.9				T	6	wx	8	Krn9		6	Flt9	7	Ger4								
3	1S.48;9L.22						Zer4	16	Flt1		15	Krn1	10	P-WR	17	wx	10	T				
5	1L.74;9L.13	P-WR	16	Zer4	13	Flt1		21	Krn1	22	wx	8	T									
6	2S.18;9L.22	B-W	14	Zer3	15	Flt2		10	Krn2	19	T	7	wx									
7	3L.09;9L.12			wx	5	T	12	Flt3		27	Krn3	23	Mer2									
12	5S.07;9L.10			wx	9	T	20	Krn5		15	Flt5	14	Ger1									
13	5L.14;9L.10			T	17	wx	27	Krn5		2	Flt5	28	Ger1									
11	5L.69;9S.17					Ger1	2	Flt5		8	Krn5	8	T	11	wx							
14	6S.79;9L.40	wx	3	T	19	Plp	6	Flt6		12	Zer2	24	Krn6									
15	6L.10;9S.37	T	11	wx	5	y	8	Flt6		5	Krn6	24	Plp	25	Zer2							
16	7L.63;9S.07			wx	12	T	18	Flt7	14	Bn20	Krn7	22	Ger2									
18	8L.09;9S.16			wx	9	T	7	Krn8		9	Flt8	22	Ger3									
20	9S.13;10S.40					T	10	wx		32	Flt10											

Since for earworm resistance we had used symbols Zer4 and Mer2, we continued to identify them with glucoside earworm resistance symbol (Ger) starting from one.

With the possible exception of chromosome 10, which had only an Flt factor, all the others have factors for Flt, Krn, and Zer, Mer or Ger.

As seen by the results, with the possible exception of chromosome 6, the Krn and Flt factors are always adjacent to one another, with Zer, Mer or Ger outside their span. Also these groups are near chlorophyll and endosperm characters, meaning that the inheritance of horizontal resistance, kernel row number, and finally carbohydrate synthesis, are governed by overlapping super-genes.

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Genetics of environmental resistance and super-genes: Latente aluminum tolerance

In MNL 56:30-32 the authors suggested that environmental resistance, in Levitt's sense and in general, followed R1 and B, the two major super-genes that

control anthocyanin color distribution in plant parts. Supporting data were presented for heat tolerance in seeds. Now we present some data for aluminum tolerance. The same flint Cateto line, C1, and the same original MNL marker stock, g R sr2, were used. The F2 seeds of the cross were germinated in moist rolled paper towels, and four-day-old seedlings were transferred to acrylic plates and grown in a modified nutrient solution of P.R. Furlani and R. B. Clark (Agron. J. 73:587-594) containing 3.0, 3.5, 4.0 or 4.5 mg Al per liter. After a week the plants were evaluated for Al resistance by visual ratings of Al toxicity symptoms on roots. Each unreplicated experiment, since the segregation was 0.5625 R:0.4375 r, consisted of 28 R colored seeds and 22 r uncolored seeds. With n=187 there were 46 Lte R, 54 Lte r, 55 lte R and 32 lte r seedlings. The deviation from 9 Lte:7 lte is X^2=0.58 with 0.5>P>0.3, non significant, with the X^2 for interaction Lte x R=5.55*, significant at P<0.05. The value of p for Lte R estimated by maximum likelihood is negative, indicating a linkage value greater than possible by reality. After the roots were classified for resistance and susceptibility to aluminum, the seedlings were transplanted to a field nursery in four separate rows. These seedlings transplanted to the field nursery reduced to n=154 at flowering time. At flowering time they were further classified for g and sr2. Since the marker is b and the C1 line is B-b, a classification for this factor was also done. This last reading has a lower number than the former because many plants did not emit tassels and could not be classified for this character. This reading gave 58 Lte B-b, 24 Lte b, 36 lte B-b and 16 lte b. The X^2 deviation from 9 Lte to 7 lte is 1.38, from 3 B-b:1 b 1.68 and the interaction Lte x B-b 0.03, all non-significant values. But the value p=28+10.0 calculated by maximum likelihood is significant. The data with chromosome 10 markers are presented in Table 1. Since no detectable difference was seen among aluminum

Table 1. Factorial presentation of F2 plant data from g R sr2 crossed with the Cateto line C1 classified with aluminum solutions from 3.0-4.5 ppm for Lte and transplanted to a field nursery to complete classification.

	G		g			Interaction	x^2
	Sr	sr	Sr	sr			
R Lte	31	2	6	2	41	(G-g)x(Lte-lte)	13.01**
R lte	21	5	9	3	38	(R-r)x(Lte-lte)	2.31
r Lte	41	5	2	0	48		
r lte	16	0	11	0	27	(G-g)x(R-r)	1.46
	109	12	28	5	154		

** Significant at P<0.01

levels, only totals are presented. The ratio of resistant to susceptible plants gave an almost perfect fit to 9:7. The interaction X^2 value for Lte x g was 13.01**, indicating linkage, while the R Lte and g R interactions were not significant. Thus, one of the Lte genes is between g and R1, the other being near B. Since sr2 had a high mortality of its own, it is useless for the analysis. As shown in Table 2, all p values were calculated by maximum likelihood and by the product moment method. The three tightest values were transformed to centimorgans, and these latter values utilized to get more precise values by differences, as illustrated in the preceding article. These values were adjusted to the known distance between g and R. The most probable position of latente-2 is two units from R.

Transforming the p value obtained for B-b Lte1 to centimorgans leads to 33.0 cM and multiplying by the correction factor from Latente-2 (14 divided by 24) leads

Table 2. Resume of relevant data. C=coupling, R=repulsion, p estimated by the methods indicated, transformation to centimorgans, cm. Estimation of distances by differences in cm and conversion of total calculated cm distance, 24, to the known distance 14 between g and R.

	maximum likelihood	product moment	best estimate	cm
C 3:1 & 9:7 g Lte	35.7± 9.7	30.4± 6.0	30.4	35.7
R 9:7 & 9:7 Lte R	18.4±37.8	19.2±36.0	18.4	19.9
R 3:1 & 9:7 g R	33.3±27.3	49.0± 9.4	33.3	39.8

g Lte = g R - Lte R = 39.8-19.9	=19.9	19.9x(14÷24)=11.6
Lte R = g R - g Lte = 39.8-35.7	= 4.1	4.1x(14÷24)= 2.4
g R =	24.0	24.0x(14÷24)=14.0

finally to 19.2 units from B-b. H. G. Nass and P. L. Crane (Crop Sci. 10:139-140), studying nine endosperm types, concluded that fl1 may have an advantage under stress conditions. It could well be that it was to a factor linked to fl1. Also in one of our Tuxpeno materials in which fl1 was transferred by several backcrosses to a fl1 marker originally from Pennsylvania, USA, we found an unusual tolerance to heat by our stronger rapid aging seed test. So the most probable position of Lte1 is 19 units from B and in the direction of fl1, that is in position 68. It must be between B and fl1.

In the next article we present data obtained with tolerance to heat which will permit a much greater precision of the estimates of p with Latente-2.

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Genetics of environmental resistance and super-genes: Latente heat tolerance

In the preceding article, the authors presented data on linkages of latente with aluminum tolerance. The data fit the hypothesis of two pairs of complementary dominant genes, linked to R and B, responsible for aluminum tolerance. Now we will try to fit heat tolerance data to the hypothesis. In MNL 56:28-30 in Table 1 there are such data. Considering live plants as Lte and dead as lte Table 1 can be built. For the Cateto line C1 there was a perfect fit both

Table 1. Data obtained from Table 1 in MNL 56:28-30. Counts of live Lte2 and dead lte2 in F2 from cross of C1 flint Cateto line with g R sr2 measured by germination after pre-conditioning at 48 C and 100% R.U. from 96 to 120 hours.

	R	r		Segregation	x^2
Lte	499	464	963	9:7	0.46
lte	477	297	774	9:7	0.00
	976	761	1737	9:7&9:7	16.78**

** Significant at P<0.01

for Lte and R to a 9:7. The interaction was highly significant, X^2=16.78** with P<0.01 indicating strong linkage. For C2 the results were much worse, with

significant deviations from the expectations tested and non-significant interaction. The more quantitative effects of the classification for R and r were much worse, and C2 was dropped out from further analysis. The seedlings were transplanted to a field nursery in two separate groups, R and r, and at flowering time classified for the factors g and sr2. A reading of B was not done. These readings are presented in Table 2. Since in the work with aluminum sr2 had a very high mortality of its own, it was eliminated from further analysis.

Table 2. Data obtained in the experiment with C1 partially presented in Table 1. The seedlings were transplanted to the field and at flowering a total of 383 Lte plants were obtained. Totals are the calculated expected, and lte column was obtained by differences.

	Lte	lte	Totals	χ^2	
G R	149	509	658	g Lte	24.06**
G r	175	470	645	Lte R	17.08**
g R	31	288	319	g R	70.00**
g r	28	87	115		
Totals	383	1354	1737		

In Table 3, the values of p with their standard errors are presented for the segregations indicated. For the segregations involving seeds and seedlings, n was

Table 3. Resume of relevant data. C=coupling, R=repulsion, values of p estimated by the methods indicated, transformation to centimorgans, cm. Estimation of distance by differences in cm and conversion of total calculated cm distance, 47.1, to the known distance 14 between g and R.

	maximum likelihood	product moment	product method cm	n
C 3:1 & 9:7 g Lte	indeterminate	34.1±3.0	41.1	$(383 \times 1737)^{\frac{1}{2}}$
R 9:7 & 9:7 Lte R	26.9±7.7	27.8±7.5	31.6	1737
R 3:1 & 9:7 g R	30.0±9.3	43.1±9.1	59.9	383
g Lte = g R - Lte R	59.9 - 31.6 = 28.3	28.3 x (14 ÷47.1) = 8.4		
Lte R = g R - g Lte	59.9 - 41.1 = 18.8	18.8 x (14 ÷47.1) = 5.6		
	47.1			14.0

taken as the geometric mean of n for seeds and n for seedlings. Both maximum likelihood and the product moment method were used. For g Lte linkage, due to high differential mortality in b and c cells, the use of the first method had no real solution. Turning to the results of the second method, it is clear that Lte2 must be between g and R. As indicated in the preceding article, we transformed their values to cM and by differences between these values adjusted to the known distance between g and R, giving the results g Lte 8 units and Lte R 6 units. So the most probable position of Lte2 is 55 in chromosome ten.

Since it seems that heat tolerance and aluminum tolerance are pleiotropic effects of the same effective factor, the data from both experiments were pooled in one joint analysis. The results are shown in Table 4. With an Lte R distance

Table 4. Joint analysis of data for tolerance to heat and tolerance to aluminum.

	maximum likelihood	product moment	product method cm	n
C 3:1 & 9:7 g Lte	indeterminate	33.4±2.8	39.9	970
R 9:7 & 9:7 Lte R	25.8±7.7	25.8±7.7	28.9	1924
R 3:1 & 9:7 g R	31.0±7.6	40.7±5.9	53.7	537
g Lte = g R - Lte R = 53.7-28.9 = 24.8		24.8 x (14 + 38.6) =		9.0
Lte R = g R - g Lte = 53.7-39.9 = 13.8		13.8 x (14 + 38.6) =		5.0
g R =	38.6			14.0

of 5 units, and between g and R, Lte2 must be around position 56 in chromosome ten.

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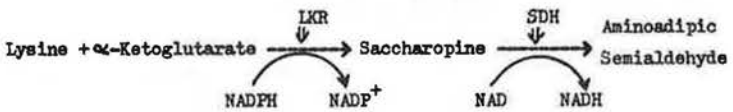
Degradation of lysine in maize: Possible pathway and genetic control

The pathway of lysine degradation via aminoadipic acid has been investigated in maize endosperm. The first two enzymes, lysine-ketoglutarate reductase and saccharopine dehydrogenase, catalyze the following reactions:



LKR = Lysine-ketoglutarate reductase

SDH = Saccharopine dehydrogenase

The enzymes were extracted from a number of inbreds and hybrids, according to the procedure of Arruda et al. (Plant Physiol. 69:988-989,1982). The endosperms were isolated from immature seeds and ground in a chilled mortar with 100 mM potassium phosphate buffer, pH 7.0, containing 1mM EDTA and 1 mM 2-mercaptoethanol. The homogenate was centrifuged at 12,000g for 25 min and the supernatant treated with $(NH_4)_2SO_4$ to 70% saturation. The precipitate obtained was collected and taken up in 3 ml of extraction buffer. The extract was desalted on a Sephadex G25 column (1 x 9 cm) and the protein fraction was assayed for lysine-ketoglutarate reductase and saccharopine dehydrogenase. For LKR the assay mixture contained potassium phosphate pH 7.0 (100 umol), L-lysine (18 umol), alpha-ketoglutarate (9 umol), NADPH (100 umol) and various concentrations of protein in a total volume of 1 ml. For saccharopine dehydrogenase the assay mixture consisted of saccharopine (1 umol), NAD (1 umol), Tris-HCl pH 8.6 (100 umol) and various concentrations of protein.

For both enzymes the activity was linear with time and proportional to the amount of extract added to the assay. The apparent Km values of LKR for lysine and alpha-ketoglutarate were 5.2 and 1.8 mM respectively (Table 1). For SDH the

Table 1 - Kinetic properties of lysine-ketoglutarate reductase and saccharopine dehydrogenase extracted from immature endosperms 30 days after pollination.

Enzyme	pH	Cofactor	Temp.(°C)	Km			
				Lys	Sac	α-KG	Cofactor
LKR	7.0	NADPH	30	5.2	-	1.8	-
SDH	8.6	NAD	30	-	0.05	-	0.2

Km values for saccharopine and NAD were 0.05 and 0.2 respectively (Table 1). The enzymes show differences with respect to cofactor and pH. LKR is specific for NADPH and has a pH optimum close to 7.0. SDH utilizes NAD as cofactor and shows a pH optimum close to 8.6.

The activity of LKR and SDH of some hybrids is shown in Table 2. LKR activity increases with the onset of seed development, reaches a peak at middle stage, and

Table 2 - Lysine-ketoglutarate reductase and saccharopine dehydrogenase activity of immature endosperms of maize hybrids.

Enzyme	Material	Days after pollination	Activity (nmol/endosperm/min.)
			NADPH oxidation
LKR	ML649 x ML202	30	9.4
	ML649 x ML674	15	16.2
		20	18.9
		25	15.0
		40	5.0
			NAD reduction
SDH	ML649 x ML674	30	7.2

decreases at seed maturity. Saccharopine dehydrogenase was analyzed only at 30 days after pollination, when it showed an activity of 7.2 nmol of NAD reduced per endosperm per min.

We investigated the activity of LKR and SDH in some opaque-2 strains. For this purpose we used two inbred lines, ML649 and L438 and a tropical maize variety named Maya, each containing homozygous normal and opaque-2 strains. The LKR activity of L438 opaque-2 endosperm was three times less than the activity observed for L438 normal. In the ML649 inbred line, enzyme activity of opaque-2 endosperm was practically absent as compared with normal endosperm (Table 3). The decrease in LKR activity of opaque-2 endosperm cannot be attributed to the presence of an enzyme inhibitor in the mutant endosperm. Assays conducted with mixtures of enzyme extracts from normal and opaque-2 endosperms showed an activity equal to the sum of the individual activities.

The activity of SDH was not changed by the opaque-2 gene. The activity of this enzyme for normal and opaque-2 versions of the Maya variety was basically the same (Table 3).

The results found here show for the first time that the degradation of lysine is under the control of the opaque-2 gene. Opaque-2, by decreasing the activity of LKR, probably decreases the rate of lysine degradation in the endosperm. This is in good agreement with the decreased extent of lysine breakdown observed in the opaque-2 endosperm by Sodek and Wilson (Arch. Biochem. Biophys., 140:29-36, 1970). The opaque-2 gene, however, affects only the first enzyme in the lysine

Table 3 - Lysine-ketoglutarate reductase and saccharopine dehydrogenase activity of some opaque-2 strains.

Material	Genotype	Days after pollination	Activity (nmol/endosperm/min.) LKR NADPH oxidation	SDH NAD reduction
I438	normal	15	2.0	-
	opaque-2	15	0.8	-
	normal	20	8.2	-
	opaque-2	20	1.2	-
	normal	25	14.3	-
	opaque-2	25	3.1	-
	normal	30	9.2	-
	opaque-2	30	4.8	-
ML649	normal	15	14.9	-
	opaque-2	15	0.8	-
	normal	20	31.2	-
	opaque-2	20	0.9	-
	normal	25	26.2	-
	opaque-2	25	0.7	-
	normal	30	10.2	-
	opaque-2	30	0.5	-
v Maya	normal	25	-	8.6
	opaque-2	25	-	9.4

degradation pathway but does not affect the second enzyme. This finding points out that lysine degradation is an important means by which opaque-2 increases lysine content in the endosperm.

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Two lines of "Flint" maize regulated by a balanced lethal system

Two lines of "Flint" maize showed a segregation of more than 37.5% of chlorophyll lethals (white mutants) in the first self generation. These lines were identified as "74" and "394." Both lines were selfed for 7 and 6 generations, respectively, and both maintained the frequency of chlorophyll lethals in each generation of selfing. The chi-square was significant for 1:1 and 9:7 segregation in both lines considering 7 and 6 generations, respectively, excepting the 1:1 segregation of line "74." Although these lines present a high frequency of chlorophyll lethals, they can persist in nature because they are regulated by a balanced lethal system linked in repulsion.

Juan C. Salerno

Performance of two maize populations selected for presence or absence of chlorophyll lethals

The persistence of lethal genes in maize populations due to natural selection was reported (Salerno et al., IAEA, Austria, 1981). Two maize populations were selected for presence or absence of chlorophyll lethals. Then they were maintained during 4 generations by open pollination. At the same time, these populations were taken to the yield trial, using the original populations as a

check. The grain yield during the 4 generations revealed that the populations selected for presence of chlorophyll lethals had a higher yield than either the populations selected for absence of chlorophyll lethals or the original populations. Preliminary results would suggest the usefulness of these compounds as a potential alternative in breeding procedures.

Juan C. Salerno

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Various expressions and dominance relations of perennialism

Earlier experiments have shown that the perennialism of Zea diploperennis as expressed by the occurrence of rhizomes is more nearly recessive than dominant. In F1 hybrids grown in three localities in Argentina no true rhizomes occurred, but "bulbils," highly-condensed rhizomes, were found. In a selfed population of 64 plants resulting from backcrossing this F1 hybrid twice to corn (7/8 corn), no rhizomes occurred but two plants had tillers arising from below ground level, presumably from bulbils. Thus one expression of perennialism, the occurrence of rhizomes, is definitely recessive. In this respect, our results agree with those of Shaver (J. Hered. 1967), who found perennialism derived from the tetraploid perennial teosinte to be recessive and to be expressed only in the presence of two other recessive genes, id for indeterminate growth and gt for grassy tillers.

The perennialism derived from Zea diploperennis has additional expressions: evergreen stalks (Galinat MNL, 1981), stiff stalks, and robust root systems including adventitious roots. Whether these represent various expressions of the same gene or those of a cluster of closely linked genes has not been determined.

Shaver assigned the symbol pe to the recessive gene isolated from Z. perennis. We would like to assign the symbol Pe to the perennial gene isolated from Z. diploperennis. At the suggestion of Dr. Coe we are using the designation Pe*-d to distinguish it from the gene isolated from perennis.

The combination of evergreen stalks, stiff stalks and robust root systems is strongly, but not completely, dominant since plants homozygous for the gene can be distinguished from the heterozygotes in our cultures. In this population the homozygotes are almost completely sterile.

Paul C. Mangelsdorf and Mary Eubanks Dunn

Linkage relations of Pe*-d

Galinat found that evergreen stalks, one expression of perennialism, is linked with Su on chromosome 4. This finding was verified by an F2 population in a test cross of evergreen stalks with Su G13. We planted only seedlings grown from starchy seeds that were also non-glossy. In this population of 64 plants, almost all were either homozygous or heterozygous for evergreen stalks. Only a few plants could be definitely identified as annuals. These data leave no doubt that Pe*-d is located on chromosome 4.

In a backcross population grown in Major Goodman's experimental field in Raleigh, the results were as shown in the following table.

Genes	Number of Plants					Crossovers	
	XY	Xy	xY	xy	TOTAL	No.	Percent
Su Pe*-d	44	5	30	16	95	35	36.8
G13 Pe*-d	40	5	34	16	95	39	41.1
Su G13	30	19	15	31	95	34	35.8

Though these results are by no means conclusive, they best fit a sequence of Pe*-d Su G13 rather than the other two possible sequences. Galinat had similar results in a population of 200 plants from the same test cross. In both his population and ours there was an excess of evergreen stalks. This may involve the gene Ga on chromosome 4 carried by Palomero Toluqueno, one of the parents of the original hybrid.

If the sequence of genes on chromosome 4 is Pe*-d Su G13 and the crossing over between Su and Pe*-d is of the order of 36.8 percent as our data suggest, then Pe*-d must be on the long, not the short arm of chromosome 4.

In this connection it may be recalled that the most "potent" of the polygene segments introduced into an inbred strain of corn from four varieties of annual teosinte all showed linkage with Su on chromosome 4 (PCM, 1974). Whether Pe*-d is part of that gene assemblage in Z. diploperennis or only linked with it has not yet been determined. It seems reasonably certain, however, that the annual teosintes have inherited their polygene segments on chromosome 4 from their diploid perennial ancestor.

Paul C. Mangelsdorf and Mary Eubanks Dunn

Robust root systems may impart drought resistance

Evergreen stalks are associated with robust root systems. These are the product, at least in part, of auxiliary root systems provided by the development of adventitious roots, commonly called "brace" or "prop" roots. We washed out the root systems of several plants with prominent adventitious roots. One of these had fourteen such roots, all of which had penetrated the soil to a considerable depth. All of these auxiliary roots comprised a single long fleshy root, similar to the tap root of some species, plus a number of lateral fibrous roots. We estimated that the total auxiliary root system was at least three times that of the remaining roots, the seminal and permanent.

Evergreen plants with auxiliary root systems are more resistant to drought than plants lacking such systems. Whether they contribute to higher yields is not certain. On this point we have only cursory observations. Of the 95 plants grown in our 3-point-test population in Raleigh, 74 were heterozygous perennial and 21 were annual. The heterozygotes consistently bore better developed ears than the annuals. It also remains to be determined whether plants with evergreen stalks will be practically useful. Grown in the South they might prolong the growing season and increase production for silage.

One more interesting observation: In its stiff stalks and prominent adventitious roots, our heterozygous perennials are quite similar to the widely used commercial inbred B73. Could these traits in B73 have been derived from Zea diploperennis? Major Goodman tells us that hybrids involving B73, although usually highly productive, are notoriously drought susceptible. Is this actually true or do they only exhibit wilting, another trait derived from Zea diploperennis? Wilting in some species, the aggressive vine kudzu for example, is a device imparting drought resistance rather than a symptom of drought susceptibility.

Although the practical possibilities of breeding perennial corns may have been greatly overstated, it may be that certain traits derived from Zea diploperennis can be usefully employed in corn breeding.

Paul C. Mangelsdorf and Mary Eubanks Dunn

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Meiotic chromosome behavior in anther-derived pollen plants of maize

Microsporocytes of five pollen-plants via anther culture of maize were examined by conventional aceto-carmine squash techniques. It was found that one was dihaploid (2n = 2x = 20), which was probably brought about by spontaneous chromosome doubling. Among the other four plants, one was aneuploid (2n = 2x = 18); the other three were haploid (2n = x = 10).

During meiosis, the dihaploid plant consistently demonstrated regular chromosome behavior, such as 10 closely synapsed bivalents throughout pachynema and metaphase I. Neither univalents nor end-to-end associated bivalents were observed. The pollen grains produced by this plant were almost totally fertile and the seed-sets were about 100 percent. In the aneuploid, meiosis was extremely irregular. At pachynema, asynapses and foldback type non-homologous associations were persistently present. At anaphase I, fusions of all the chromosomes into a large chromatin body and laggards were frequently found. Furthermore, precocious splitting of univalents and irregular distribution of dyads also appeared at this stage. The pollen-grains of this plant were almost completely sterile, and only one well-developed kernel was obtained after repeated self-fertilizations.

For the three haploid plants (2n = x = 10), irregular meiosis was consistently observed. At pachynema, non-homologous associations of chromosomes by folding back were commonly present. Occasionally fusions of all chromosomes into a chromatin mass were also found. At diplonema and diakinesis, 10 clearly separate univalents were common configurations, even though on several occasions one or two secondary associations of heterologous chromosomes were seen. At anaphase I, distribution of chromosomes varied a great deal--distributions such as 3 to 7, 2 to 8, 9 to 1 were all observed. A 5 to 5 distribution of chromosomes was sometimes found. However, at the same stage, 10 chromosomes grouped into three or four masses of chromatin were also present. At anaphase II, variations in chromosome distributions were ubiquitous--distributions such as 5 to 5, 4 to 4, 3 to 7 were all found. In addition, laggards, chromosome bridges, fusion of chromosomes into chromatin masses were also present. Another irregular division, with one cell having one chromosome, the second having two chromosomes and the third having seven chromosomes of a presumptive quartet, was also observed.

One (83-P-5) of the three haploid plants had about five percent of the microsporocytes with doubled chromosome number (2n = 2x = 20) at pachynema. Whether this change of chromosome number is due to natural doubling or due to fusion of microsporocytes is unclear.

Y.C. Ting and Margaret Yu

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Twin stalks in maize

It was recently noted that the old French INRA inbred line F1254 shows 4.5% of plants with two stalks. The duplication affects the mesocotyl, the coleoptile, and the primary seminal root. In the dormant seeds, the double plumules cannot be detected.

The appearance of such a trait is more regular than the phenomenon of "siamensis" reported by Sangueneti (1940, Anal. Inst. Fitotecn. Santa Catalina, Llavallol, Argentina, 17-134).

As far as the determination of the nature of this trait is concerned (true polyembryony or early fasciation taking place during embryogenesis), some heterozygous plants for the liguleless-1 gene were selfed. Among 37 plants with double stems, all couples were concordant either for liguleless leaves (8 plants) or normal leaves. The hypothesis of true polyembryony is then discarded, a minimum of 6 plants would have been enough at the P=0.05 level to find a seed with discordant stems. This trait with low incomplete penetrance is probably due to oligogenic control. After a cross with another donor line for a special gene (lgl, sul, ...), a minimum of 4 backcrosses is needed to find again such a trait in the F1254 background.

M. Pollacsek

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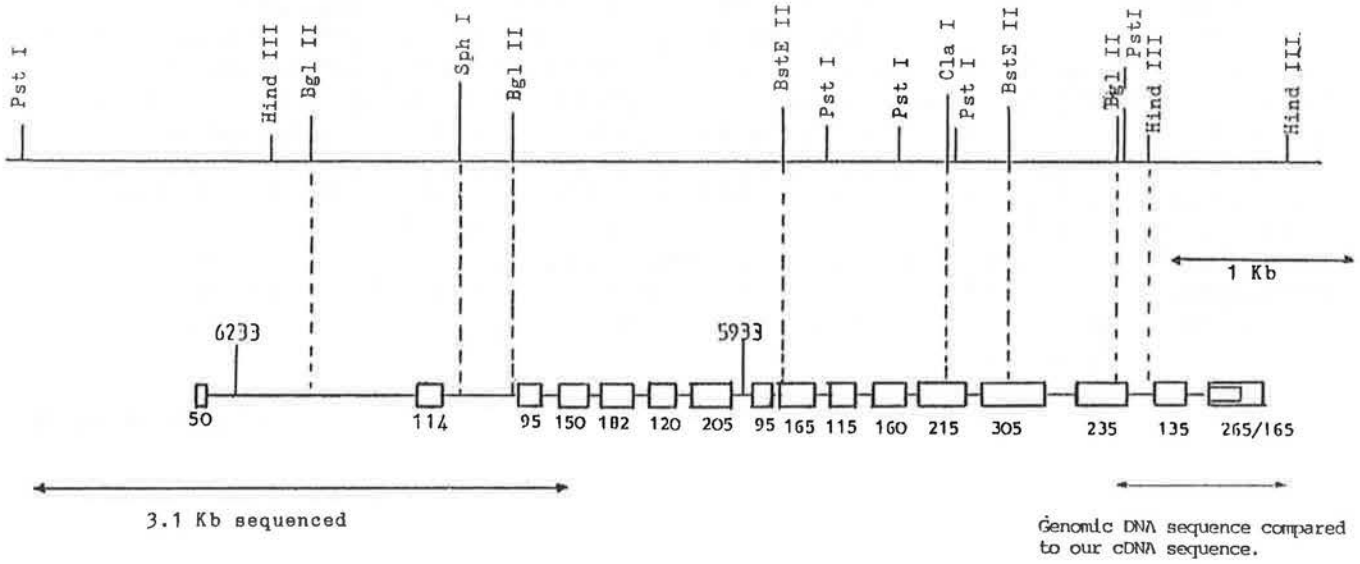
University of Cologne

Structure of the shrunken locus on chromosome 9

We have continued the study of our genomic clone of the Shrunken gene by both S1 mapping experiments with RNA-DNA hybrids and partial sequencing of the 5'- and 3'-ends of the gene. The structure of the gene is shown in the figure. Two



introns are found at the 3'-end by comparing the genomic DNA sequence with the cDNA sequence. The rest of the gene structure was examined by digestion of RNA-

DNA hybrids with endonuclease S1. Another 13 introns were detected by this method and positioned within genomic restriction fragments. The first exon is approximately 50 bp long and has been located between the left-most HindIII site and an XbaI site further 5'. Because the third 95 bp exon contains a PvuII site, it was possible to locate its 5'-intron-exon junction within our sequence. By using synthetic oligonucleotides complementary to a sequence derived from the third exon for priming a reverse transcriptase reaction in the presence of dideoxynucleotides, we have obtained some of the sequence of the 5'-end of the mRNA. This allowed us to position the first two exons within our genomic sequence data and tells us that we are looking at the 5'-end of the mRNA. The transcription start was precisely mapped by S1 endonuclease digestion of RNA-DNA hybrids. There is a TATA box-like sequence (TCTATTTAT) 25-30 bp in front of the multiple start sites, and also a sequence resembling a CCAAT box (CCATCT) around position -90. The first ATG is located within the second exon, the following open reading frame is continued in exon 3 and exon 4 as far as the latter is sequenced.

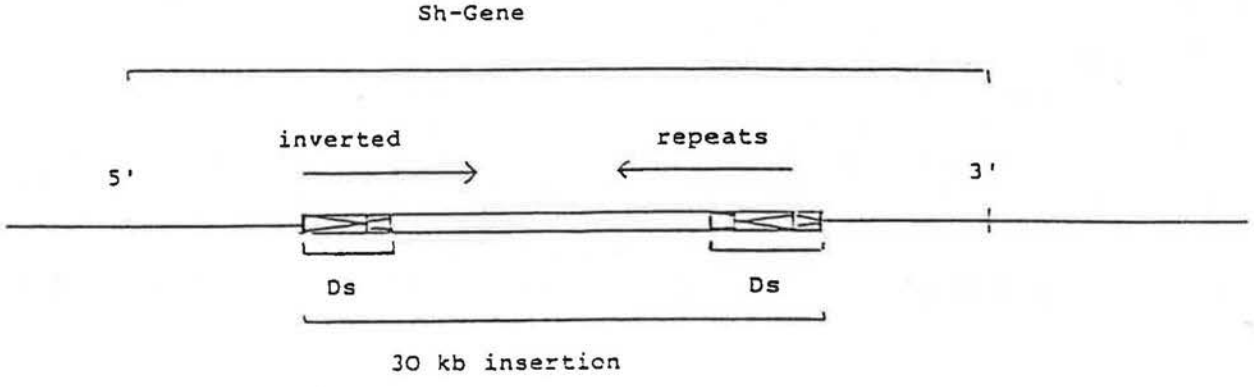
All introns so far sequenced start with GT and stop with AG, as in nearly all animal and plant genes examined up to now. The transcribed region extends 5.5 kb at the genomic level, while the length of the mRNA is 2.7 kb. The gene is interspersed with 15 introns which are larger at the 5'-end than at the 3'-end.

W. Werr, W. B. Frommer and P. Starlinger

The structure of the sucrose synthase gene in mutant sh-m5933 and in several revertants

We have analyzed the structure of the shrunken (Sh) locus, encoding endosperm sucrose synthase, in a strain containing an unstable recessive mutation (sh-m5933) caused by Ds. In addition, we have analyzed the structure of the Sh locus in nine spontaneous Sh revertants of sh-m5933.

The sh-m5933 allele contains a ca. 30 kb insertion in the Sh locus, of which more than 15 kb are present in our genomic clone. That part of the 30 kb inserted DNA not contained in our clone was analyzed by restriction mapping using the blotting technique and probes derived from the 5'-end of the sucrose synthase gene. The restriction pattern of the first 7 kb of uncloned insertion DNA is similar to that of the cloned part of the insertion. The patterns can be explained by the following: (a) the 30 kb insertion terminates in long inverted repeats and (b) the first 1 kb segment adjacent to the 3'-end of the sucrose synthase gene is missing from the same position of the DNA adjacent to the 5'-end of the sucrose synthase gene. It will be shown below (see next report) that most of the DNA inserted into the sucrose synthase gene is not the element Ds.



In addition to the long insertion in the sucrose synthase gene, another aberration is present in sh-m5933. Part of the 30 kb insert DNA, including the uncloned junction with the 5'-end of the sucrose synthase gene and a large segment of DNA adjacent to it, are duplicated in sh-m5933. This duplication must be located on the same chromosome, as it does not segregate from the mutation upon crossing.

In nine spontaneous sh revertants, the 30 kb insertion within the sucrose synthase gene is excised and the structure of the wild type sucrose synthase gene is restored. The large duplicated segment that includes part of the insertion and part of the sucrose synthase gene is, however, retained. In one of the nine revertants, an alteration in the duplication has occurred. The data show that a 2 kb segment of insertion DNA near the junction with the sucrose synthase gene has been deleted.

The retention of the long duplication in the revertants explains the observation that all of the revertants show phenotypes associated with the presence of Ds in the vicinity of the sucrose synthase gene. Only in one of the revertants is a different pattern obtained. This is the same revertant that has suffered a deletion within the duplicated DNA segment at the junction of the 5'-part of the sucrose synthase gene and part of the insertion. The correlation between an altered pattern of chromosome breakage and the loss of DNA adjacent to the junction is an indication that this DNA is a part of Ds responsible for the chromosome breakage pattern. This work is detailed in Courage-Tebbe et al., Cell 34:383, 1983.

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The sequence of a 2040 bp Ds element

The 30 kb insertion mentioned in the preceding report is larger than most known transposable elements in other organisms. We considered the possibility that the 30 kb insertion is a transposon-like structure with two transposable elements flanking some other DNA. In order to investigate this hypothesis, we sequenced 4.3 kb of DNA at the junction between the insertion and the 3'-end of the sucrose synthase gene. The structure of this 4 kb segment was known to contain duplicated sequences from previous hybridization studies. The DNA sequence confirmed this: A 2040 bp sequence is present twice at the junction. One copy is inserted into the middle of the second, and the two copies are inverted relative to each other. The 2040 bp sequences share properties with known transposable elements: They are terminated by 11 bp inverted repeats, as is common for transposons. The 8 bp sequences flanking the central copy of the 2040 bp structure are identical. This same 8 bp sequence is present within the central copy only once. This is the only difference between the outer and the inner copy of the 2040 bp structure, and is explained by assuming that the inner sequence is an insertion into the outer one, creating a short duplication of 8 bp at the site of insertion. This is a second property common to transposable elements.

The sequence similarities between the 2040 bp structure and other known transposons do not prove that it, and not the large 30 kb structure present in the sh-m5933 sucrose synthase gene, is a Ds element. To evaluate what constitutes a Ds sequence, we have examined other Ds-induced mutations for similarities with the 2040 bp structure. We have shown that the 2040 bp sequence hybridizes to a 1.3 kb insert found in mutant Adh1-2F11, a Ds-induced mutation (see a subsequent report). This sequence also hybridizes to the ends, but not the middle, of an Ac element we

have isolated from the wx locus (see a subsequent report). This strongly supports our contention that the 2040 bp structure is Ds and is derived from an internal deletion of an active Ac element. This work is in press in Nature.

H. P. Doring, E. Tillmann and P. Starlinger

A 4 kb insertion in sh-m6233 is a double Ds

Mutant sh-m6233 belongs to the series of mutable shrunken alleles caused by the insertion of Ds that were isolated and characterized by McClintock. We have studied the DNA of the mutant by the blotting technique and found that a 4 kb DNA insertion is present in the sucrose synthase gene. The preliminary restriction map of the inserted DNA is indistinguishable from the restriction map of the "double Ds" described in the two preceding reports. However, the orientation of the sh-m6233 Ds structure is opposite to that of the 4 kb Ds sequenced from sh-m5933.

We have not yet cloned the whole foreign insert of sh-m6233, but have isolated a clone carrying part of the insertion and part of the shrunken gene. This allowed us to determine the exact point of insertion. It is located 2.5 kb towards the transcription start relative to the insertion site in sh-m5933. The sequence adjacent to the insertion site begins with the same 11 bp that form inverted repeats at the termini of the double Ds structure in sh-m5933.

These results do not support the assumption that the Ds elements causing mutations sh-m5933 and sh-m6233 are non-transposable, as was originally suggested by B. McClintock. The assumption that mutable sh alleles are caused by a "non-transposable" Ds was partly derived from the observation that phenotypic revertants to Sh wild type are unstable themselves and give rise to new mutable sh alleles at considerable frequency. It will be interesting to see whether the phenotypic revertants of sh-m6233 arise by transposition of Ds to a nearby site. Short range transposition has been described by Brink for transposable element Mp (which is functionally identical to Ac) at the P locus.

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Adh1-2F11: A Ds insertion in the alcohol dehydrogenase gene

Using the allyl alcohol selection technique, we have isolated a mutant in the Adh1 gene. The mutant has a null phenotype. In the pollen it reverts to wild type ADH activity at a frequency of between 10^{-4} and 10^{-3} in the presence of Ac. In the absence of Ac it is stable. The mutant differs phenotypically from another Ds-induced mutant, Adh1-Fm335, isolated by Osterman and Schwartz, which is much more unstable and quite leaky.

Restriction maps of the wild-type Adh1-F and the mutant Adh1-2F11 allele were constructed by analyzing genomic blots with an Adh1 probe isolated by Bennetzen and Freeling. The mutant allele contains a 1.3 kb insert in the Adh1 gene.

Northern blots with RNA isolated from wild-type and mutant strains were probed with the Adh1 clone. In the wild type, one band of approximately 1650 bp length is seen. In the mutant, two bands are seen. One is identical in size to the wild type band, the other band is approximately 1.3 kb larger and may be the product of cotranscription of the insert. This latter band also hybridizes to a Ds probe derived from the cloned sh-m5933 insert.

These observations show that the transposable element Ds can exist in different forms which differ at least in size. We will show below that Ds can be an internal deletion of Ac. The finding, however, of a second Bam site in the Adh insertion but not present in the 2.04 kb Ds of sh-m5933 or in Ac (see below), raises the interesting possibility that different copies of Ds may not only differ

from Ac by internal deletions but also by other mutations, or even the addition of sequences not present in Ac. We have in the meantime cloned the Adh1-2F11 allele and are studying it at the DNA level. This work is in press in Mol. Gen. Genet.

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Cloning of controlling element Ac from waxy-m7

Unlike Ds, Ac elements are capable of autonomous transposition. Ac-induced mutations are not available at the Sh or Adh1 loci. There are, however, two Ac-induced mutations at the waxy locus. One of these, wx-m9, had been previously cloned by Fedoroff et al. The other mutant, wx-m7, differs from wx-m9 in several physiological respects. In homozygous condition, wx-m7 reverts to wild type earlier and more frequently than wx-m9. Other differences in the Ac action exhibited by these alleles are also observed (B. McClintock, Carnegie Inst. Wash. Yearbook 1963, 1964, 1965). We have cloned the Ac element from wx-m7 to examine these changes in phase at a molecular level.

A genomic library of wx-m7 was constructed in the lambda vector EMBL4. It was screened using a probe of the wild type locus. The clone contains a 4.3 kb insertion in the wx gene. The insertion is located about 2.5 kb towards the transcription start of the waxy gene relative to the insertion site of Ac in wx-m9. The Ac insertions in wx-m7 and wx-m9 are indistinguishable by restriction analysis and occur in the same orientation in both alleles. The waxy DNA outside of Ac shows some restriction polymorphism with the wild type allele. The double Ds structure hybridizes to both ends of the Ac element but not to internal fragments, suggesting that Ds is formed from an internal deletion of Ac.

Heteroduplex studies (kindly performed by I. Bertram of the Max-Planck-Institut fur Zuchtungsforschung, Koln-Vogelsang) indicate a 4.3 kb insertion relative to the wild type Wx gene as predicted by the restriction analysis. The insertion loop of Ac consistently shows a small stem structure of around 150 bp at its base. As the 11 bp inverted repeats found at the termini of Ds (see above) are insufficient to form such a stem structure, it will be interesting to see whether Ac from wx-m7 differs from Ds in sh-m5933 by sequence at the termini.

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Confirmation of chromosome arm locations for seedling and kernel mutants

In past newsletters (MNL:45:144, 46:131, 47:148 and 48:117) we have reported the arm location for many of the EMS-induced mutants on the basis of a single test. Experience in our laboratory and information from several collaborators, to whom we sent groups of mutants, indicates that some of the locations were incorrect. To validate our earlier work, we crossed 108 presumably located seedling mutants by the appropriate B-A stock. Of the 108, a total of 84 received an adequate test. Forty-eight were confirmed in their original location and 36 were in doubt. Among the 36 unlocated seedling mutants, 7 had clear mutant

phenotypes and the other 29 mutant phenotypes were not clear. Locations on 2S were not tested because the wrong B-A stock was used.

Table 1: Listing of seedling mutants confirmed to chromosome arm.

Chromosome Arm	# of Mutants	Mutant classification
1S	7	2 pg, 1 blh, 2 v, 1 sr, 1 lls
1L	7	1 sr, 1 cb, 2 wl, 1 v, 1 yg, 1 ad
2L	3	1 w, 2 v
3S	3	1 gl, 2 d
3L	3	2 w, 1 gl
4S	1	1 pg
4L	1	1 wl
5L	7	2 w, 1 sml, 1 gl, 1 ppg, 1 nec, 1 wsr
6L	3	1 l, 2 wl
7L	4	2 gl, 2 w
8L	1	1 v
9S	2	1 v, 1 w
10S	1	1 l
10L	5	3 l, 1 ppg, 1 wlv

Total = 48

Abbreviations: ad(adherent), blh(bleached), cb(crossband), d(dwarf), gl(glossy), l(luteus), lls(lethal leaf spot), nec(necrotic), pg(pale green), ppg(pale pale green), sml(small), sr(striate), v(virescent), w(white), wl(white luteus), wlv(white luteus virescent), wsr(white striate), yg(yellow green).

Table 2: Listing of defective kernel mutants located to chromosome arm. Entries are included on the basis of one positive test. Those that are underlined have been confirmed by a subsquent test. Those marked with an apostrophe (') are allelic to like marked mutants on the same arm.

1S	1L	2S	2L	3S	3L	4S	4L	5S	5L	6S	6L	7L	8L	9S	9L	10S	10L
628A	833A	660E	660C	874A'	330D	211C'	1005A'	925A	742	1307A	924	76B	pro-1'	873	744	1009	889
792'	871	890A	999A	948B	627D	740	1104	931A	863A		933	788	-342'	1054	1309A	1435	1330
923	918A	901A	1024A	961	802	912'	1105	1036A	1160		1294	874B'	1058'	1089	1381		1427A
928A'	948A	974A	1078A	1112	932	1167A	1130		1275A		1296A	980	1121'	1421	1391		
971'	1113A	1078B	1162	1283	936A	1225A	1156A'		1299		1320B	1241'	1154A		1409		
991	1172A	1122A	1175	1339A'	1060B	1228	1176A		1328A		1384	1312	1285				
1057A	1303	1233A	1287	1341	1126	1318	1243		1331			1332	1383				
1136B	1315A	1289	1316A		1166	1519B	1520B		1365				1386A				
1142A		1313	1414		1311B				1369				1387				
1176B		1333B			1322C				1380A				1388				
1202A		1404			1379A				1385				1392A				
1208A		1424							1390B				1405A				
1295		1436A							1393A				1429				
1308A									1534				1528				
1322A													1533'				
1338																	
1348'																	
1390A																	
1390C																	
1394'																	
1401'																	
1484																	
1519C																	

The defective kernel mutants have been given a more exhaustive test, and many have been tested at least twice. The results of these tests are listed in Table 2.

M-T. Chang, M. G. Neuffer and W. F. Sheridan

Characterization of TB-8Lc hypoploid plants and location of dek mutants proximal to the breakpoint

Several dek mutants in our collection have not been uncovered by any of the B-A translocations tested. These mutants may lie between the breakpoints for those chromosomes with B-A translocations on both sides of the centromere, or on an arm not uncovered by a B-A translocation. Chromosome 8 is of the latter type. In order to locate mutants potentially on the short arm of chromosome 8, 17 previously unlocated dek mutants were tested this past summer. All of these mutants had been adequately tested by the full B-A set, including TB-8Lc, and were not located. For each mutant, 100-seed samples from 3 normal ears crossed by TB-8Lc were planted. Based on morphology and pollen sterility, 15-20 plants in each family were classified as hypoploids. Compared to normal sibs, the hypoploids were short plants with dark green, glossy, broad crinkled leaves. Part of the leaf tissue was burned by sunshine, indicating a sensitivity to heat or light. These plants had more internodes and shorter internode length. Tassels came out one week later and pollen shed was good. Anthers were smaller in size. The table lists the plant height, 5th leaf length and width, and internode length for normal and hypoploid plants. Data were collected from five plants of each type at

Table 1: Comparison of plant height, leaf length and width, internode length and number of leaves, which is based on normal vs. hypoploid plants.

	normal		hypoploid	
	cm	s.d.	cm	s.d.
plant height	217	12.8	162	13.7
leaf length	81	3.2	78	7.4
leaf width	6.9	0.5	8.2	0.5
internode length				
1	5	0.7	2.8	1.1
2	8.8	1.9	6.2	2.6
3	13.6	1.5	11	2.5
4	17	1.4	13	3.7
5	17.6	2.5	12.6	2.1
6	18	2.5	10.6	2.7
7	17.2	1.3	6.2	2.4
8	13.6	1.5	3.2	1.1
9	10.8	1.5	1.5	0.5
10	7.2	1.5	1.0	0.6
11	4.4	1.4	0.7	0.3
12	3	0.7	0.4	0.1
13	2	-	0.3	0.1
14			0.2	0.1
15			0.13	0.05
16			0.1	-
sum of internode length	138.2		69.93	
number of leaves	13 - 14		15 - 17	

random. Good selfs segregating for mutant kernels were obtained from hypoploids of each of the 17 mutants. Fourteen of the 17 mutant ears had 3:1 segregating ratio, and 3 gave ears with a majority of mutant kernels, indicating that the mutant was probably located proximal to the 8Lc breakpoint, and possibly in the short arm of chromosome 8. These three mutants (E1089, E1092, E1168) will be incorporated into 3 point linkage analysis of chromosome 8.

M-T. Chang and M. G. Neuffer

Characterization of hyperploid B-A translocations

Beckett's set of B-A translocations has been used in Neuffer's laboratory for locating recessive seedling mutants and kernel mutants to the proper chromosome arm. In general, the right B-A translocation plants can be identified by having 25% or greater pollen sterility. Proper gene markers also helped to identify the desired plants, but homozygous B-A translocations are the best. The genetic markers used included genes for anthocyanin formation, carotenoid synthesis and inviable defective kernel mutants. By having homozygous R-scm and a recessive anthocyanin gene marker in the stock, kernels that had colorless endosperm with a colored scutellum were the hyperploid B-A translocations. Genes for carotenoid formation gave white/opaque endosperm which were distinct from the yellow endosperm, and in some cases had a lethal seedling phenotype, such as vp9 (lethal embryo) or o2 (luteus seedlings), that helped to identify the hyperploids. The same discriminations were applied to all the defective kernel markers as well. In addition, most hyperploid B-A translocations have distinguishable characters, and those characters repeat again and again under different enviromental conditions. It is feasible to use those morphological characters as an indicator to identify the right hyperploids in the field. Table 1 lists the morphological characters of

Table 1: Morphological characters of the B-A translocations

TB-1La:	large nodes, zig-zag internodes, large clumped tassel with a brittle rachis and the pollen shedding frequently poor.
TB-2L-1S:	flaccid tassel, small anthers, poor pollen shed and pollen clumps at high temperatures.
TB-3Sb:	thin leaves, large anthers and early pollen shed.
TB-3La:	wrinkled leaves.
TB-5Sc:	small plant, short broad leaves, pollen shed before complete tassel emerged from flag leaf, poor pollen shed and pollen clumped easily.
TB-5La:	thin, weak tassel stalk.
TB-6Sa:	tall plant (over 10 feet), tassel and top young leaves frequently dried, anthers slow to exert and poor shedding, usually the anthers won't shed at high temperatures.
TB-6Lc:	bented tassel and late pollen shed (about 15-30 minutes), poor pollen shedding at high temperatures.
TB-7Lb:	short, erected, dark green leaves, erect tassel branches.
TB-10L20:	long and thin light green leaves, large tassel with long tassel branches, bented tassel and usually very good pollen shed.

Table 2: Comparison of plant height, anther length, flag leaf and penultimate leaf length/width, tassel length, tassel branches, length of top sheath to branches and number of tillers of the hyperploids.

BA	plant ht.		anther length	flag leaf length		flag leaf width		penultimate leaf length		penultimate leaf width	
	cm	s.d.	mm	cm	s.d.	cm	s.d.	cm	s.d.	cm	s.d.
1Sb	188	17	6	44	3	6.3	0.6	57	5	8.6	0.4
1La	225	7	6	25	10	6.1	1.6	50	9	9.8	1.5
2L-1S	250	12	4	43	5	6.7	0.4	61	8	8.6	1.0
3Sb	280	14	7.5	34	9	3.8	0.6	55	5	5.1	0.7
3La	259	24	6	56	4	6.6	0.7	73	4	8.6	0.4
4Sa	249	22	6	32	11	3.3	1.9	67	8	7.0	1.1
4L4933	268	18	6	44	11	6.0	1.0	60	11	7.8	1.3
5Sc	191	3	4.5	25	8	5.6	2.1	44	7	9.8	0.8
5La	251	10	6	34	11	5.6	0.9	55	13	6.9	0.8
6Sa	334	17	6	53	9	5.4	1.3	68	9	6.3	1.6
6Lc	236	10	6	28	7	4.5	0.5	43	6	6.0	0.8
7S	228	7	5	38	11	6.1	1.3	61	8	8.8	1.4
7Lb	229	11	7	24	3	5.0	0.8	38	7	6.9	1.3
8Lc	221	13	5	33	8	5.0	0.6	52	8	7.7	1.0
9Sb	285	22	7	36	5	7.4	1.4	62	7	9.2	1.3
9Lc	181	6	5	40	4	6.0	0.7	55	4	8.0	1.2
10Sc	245	8	7	42	8	6.8	0.9	65	4	9.7	0.9
10L20	289	12	6	54	8	6.1	0.7	72	9	7.2	0.8

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BA	tassel length cm	s.d.	tassel branches #	s.d.	top sheath to branches cm	s.d.	tillers #
1Sb	37	3	13	2	11	2.3	0
1La	41	2	24	3	3	1.9	0
2S-3L	40	10	20	2	11	4.0	0-2
2L-1S	43	3	22	6	6	6.8	0-2
3Sb	36	2	29	7	9	3.1	0
3La	41	2	25	7	7	1.8	0-2
4Sa	42	4	17	5	9	3.0	0-2
4L4933	44	4	24	4	12	3.5	0-2
5Sc	31	4	13	9	8	1.7	0-2
5La	37	2	28	3	8	5.3	0-3
6Sa	47	2	25	5	4	2.3	0
6Lc	32	3	18	4	3	2.9	0
7S	37	2	15	3	9	3.4	0
7Lb	31	3	21	2	7	1.9	0
8Lc	35	2	16	4	9	2.9	0
9Sb	41	4	28	9	11	2.7	0-1
9Lc	34	2	8	3	10	1.9	0
10Sc	43	5	17	3	8	1.3	0-3
10L20	48	2	25	3	2	2.3	0-2

the B-A translocations. Genetic markers plus morphological identification insured that the right hyperploids were used in each group. Table 2 lists comparisons of certain characters of the hyperploid translocations.

M-T. Chang

Linkage of hcf mutations to mapped markers: a progress report

Many of the hcf mutations isolated by C. D. Miles and coworkers have been tentatively assigned to chromosome arms by B-A translocation analysis. Some preliminary linkage data to mapped markers are given below. The hcf stocks employed were obtained from Miles and used directly, or were subjected to one or more successive crosses with the inbred Mo17. Stocks with multiple markers were obtained from the MGC Stock Center.

hcf*-9 (allelic to hcf*-3): F2 progenies that segregated hcf, sr and zb4 in a given progeny were obtained by crossing +/hcf*-9 individuals with pollen from a + + P-WW/sr zb4 P-WW plant. Data for one progeny are given in Table 1, and show hcf*-9 is linked to zb4.

TABLE 1. Linkage of hcf*-9 to sr and zb_4.

Phenotype	+++	++hcf	sr zb_4+	sr++	+zb_4+	sr+hcf	+zb_4 hcf	sr zb_4 hcf	TOTAL
No. of Individuals	147	76	31	34	28	17	6	4	343

see text for further explanation

All traits did not segregate at 25% in progenies where they were observed, preventing estimation of recombination distances. Segregation for the progeny described in Table 1 was as follows: zb4, 19.9%; sr, 24.9%; hcf, 29.8%. Of all sr individuals referred to in Table 1, 24.4% were also hcf, and of the zb4 individuals, 14% were hcf. The low number of members of two recombinant classes with hcf prevent establishment of a gene order.

Comments on expressions: 1. Night temperatures of 70 F did not give good expression of zb4. After emergence of the first leaf, placing seedling flats overnight at 50 F produced a fine but distinct yellow green horizontal band on leaves after further growth. Similar banding could not be produced for progenies segregating hcf*-9 only. The number of fine bands on each leaf usually corresponded to the number of nights material was kept at 50 F. 2. The hcf trait was scored by counting lethals, many of which had been confirmed to be hcf. The high number of "hcf's" may thus be due to lethality for other reasons, e.g. poor

vigor of sr individuals with large amounts of albino tissue. In the future, hcf individuals will be scored by fluorescence induction.

hcf*-6: F2 progenies segregating hcf*-6, sr and zb4 are being analyzed.

hcf*-18, hcf*-43: Summer 1982 crosses indicated that hcf*-18 and hcf*-43 are allelic. The low number of pr hcf individuals that segregate in the F2 progenies a cross between +/hcf*-18 and +++/bm pr ys is evidence that hcf*-18 is linked to pr (see Table 2).

TABLE 2. Linkage of hcf*-18 to pr.

Progeny No.	Total	pr+	pr hcf	+hcf	++	colored kernals	not colored kernals
1	388	98	2	50	238	283	105
2	127	17	0	19	91	87	40

see text for further explanation

Two problems with this test prevent establishment of gene order of hcf*-18 relative to pr, ys, and bm: (1) the hcf phenotype only occurred in 13-15% of the individuals for several F2 progenies examined (as assessed both by lethality of 2-3 week-old seedlings and by induction of fluorescence). Not all progenies segregating hcf*-18 or hcf*-43 have this property. Future linkage tests will be repeated with several +/hcf*-18 or hcf*-43 individuals from progenies which segregate 25% hcf seedlings. (2) ys could not be scored unambiguously in unfertilized seedling flats, and bm expression required seedlings where at least 4 leaves had fully expanded. The hcf seedlings generally died sometime before the third leaf had expanded. The F3 generation is being examined for segregation of hcf in ears that are ys/ys, or bm/bm or pr/pr or combinations thereof.

Other hcf markers being tested for linkage, but where F2 progenies have not yet been generated, are (the numbers in parentheses are tentative chromosome arm assignments provided by C. D. Miles): hcf*-2 (1L), hcf*-4 (1L), hcf*-12 (1L), hcf*-13 (1L), hcf*-41 (1L), hcf*-44 (1L), hcf*-50 (1L), hcf*-21 (5L) and hcf*-42 (9L). All of these hcf mutations have either very distinct effects on photosynthesis or have been shown to be nonallelic.

Mary Polacco

Aneuploidy in the inbred line W23 is probably due to nondisjunction at the second division of the microspore

In 1980, I reported (MNL 54:26) that small kernels on W23 ears often produced aneuploid plants. This year reciprocal crosses between W23 and Mangelsdorf tester and between C R-sc W23 and M. G. Neuffer's "down tester" (bz2 a1 c2 a2 pr c1 bz1 wx r) were attempted. Pollen, but not ears, was obtained from Mangelsdorf tester, and (Mangelsdorf tester X normal) crossed by W23 set only scattered kernels, so that part of the experiment may be a failure. Reciprocal crosses of R-sc W23 and down tester were successful, however, and good seed set was often obtained. When R-sc W23 was pollinated by down tester, all 12 ears had colored kernels. From the reciprocal cross, most of the 14 ears obtained had colored kernels with one to five (or perhaps six) colorless kernels, many of which had colored scutella.

If aneuploid eggs and polar nuclei were produced by R-sc W23, then colorless kernels should have appeared on the R-sc ears pollinated by down tester. Because colorless kernels appeared only on the reciprocal cross, the events leading to the production of the colorless kernels must have occurred on the male side. And because the colorless kernels have colored scutella, it is probable that nondisjunction occurred at the second division of the microspore, producing pollen grains with one sperm carrying an extra chromosome and one sperm lacking it

entirely. Then, when the deficient sperm fertilizes the polar nuclei, a kernel with colorless endosperm and trisomic embryo should be produced. If the deficient sperm fertilizes the embryo, the endosperm should receive both chromosomes carrying the color factor involved. Several kernels were observed to be unusually dark in color, so these may represent the class that will produce monosomic seedlings.

Kernels from all classes of seed will be analyzed by taking root tip counts. Although it seems most likely that nondisjunction at the second pollen division is involved, the possibility of heterofertilization has not been ruled out.

J. B. Beckett

Evidence for frequent random disjunction of the A and AB elements of certain B-A translocations

It has been generally assumed that the A and A^B chromosomes of a B-A translocation heterozygote usually pair at meiosis and go to opposite poles. Recent results indicate that disjunction sometimes may be more nearly at random. Known translocation heterozygotes of TB-9La, TB-9Lc, and TB-9Sd were crossed as female by a wx tester stock. Root tip counts were taken and the 21-chromosome plants were grown to maturity. Pollen samples of all plants were taken and each plant was crossed to an appropriate female tester. Each plant was crossed as female by a wx tester. This generated a set of TB's in which the wx allele is on the A chromosome, and Wx is on the B^A of TB-9Sd and on the A^B of TB-9La and TB-9Lc. A portion of the preliminary data is presented below:

	Pollen observations			Critical crosses	
	Wx	wx	Aborted	onto c sh wx	X wx
TB-9Sd					
11031-3	2072	1426	587	seg. c sh wx	seg. wx
11031-4	3046	2162	340	seg. c sh wx	seg. wx
11031-6	4642	4683	230	seg. c sh wx	seg. wx

	Pollen observations			Critical crosses	
	Wx	wx	Aborted	onto wx gl15 Bf	X wx
TB-9La					
11027-1	2228	1843	795	seg. Bf, wx	seg. wx
11027-3	4465	3777	1090	seg. Bf, wx	seg. wx
11027-8	2384	2126	569	seg. Bf, wx	seg. wx
TB-9Lc					
11026-1	4930	4384	952	seg. wx, gl15, Bf	seg. wx
11026-2	16	6035	495	all wx	all wx
11026-4	3062	3377	264	mostly wx, a few Wx	seg. wx
11026-5	2129	1182	812	seg. wx, gl15, Bf	seg. wx
11026-6	2913	2531	970	seg. wx, gl15, Bf	seg. wx

Of the eleven plants listed above, nine uncovered c sh, Bf or gl15 Bf and thus must have been translocation heterozygotes.

The Wx:wx ratio of the TB-9 heterozygotes is significantly skewed in favor of the Wx class. The excess of Wx in the pollen of the TB-9Sd heterozygotes may be explained by the random movement of the B^A to either pole during meiosis. However, the excess of Wx pollen in the TB-9La and TB-9Lc heterozygotes must have a different explanation. If the B^A chromosome of translocation heterozygotes generally moves at random to the poles at meiosis I, as studies have shown, then half of the pollen carrying the A^B chromosome should lack part of 9L and likely abort. Because these grains actually represent a portion of the Wx class, the excess of Wx pollen becomes even more striking.

Of the remaining two plants, 11026-2 appears to be a tertiary trisomic (AAB^A) and 11026-4 appears to be a new type (AAA^B). Plant 11026-4 gave approximately 1Wx:1wx when crossed as female by a wx tester. However, when outcrossed onto a wx tester, it gave very few Wx kernels. It also failed to uncover gl15 and Bf, so it presumably did not possess an A^B and a B^A, both of which are usually required for nondisjunction to occur. If it was a tertiary trisomic (AAB^A), it should have been homozygous wx, so the few Wx kernels found on the testcross onto wx gl15 Bf ear would have been contaminants. However, if the plant was of the constitution AAA^B, then the presence of Wx on the A^B could explain the approximate 1Wx:1wx segregation observed in the pollen. Although confirmation is needed, it appears that the plant was an AAA^B trisomic. The low percentage of Wx transmitted by the pollen is presumably a consequence of poor competitive ability of the aneuploid AA^B pollen grains.

The generation of this AAA^B plant from the TB-9Lc heterozygote may be due to the random disjunction of the A^B to the poles at meiosis. This may also explain the excess of Wx pollen observed in the TB-9La and TB-9Lc heterozygotes. Root tip counts will be taken on the seedlings from the Wx kernels of plant 11026-4 to determine if they truly represent the AAA^B class. Meiotic observations will also be undertaken.

B. Kindiger, J. B. Beckett and C. Curtis

A modified chromosome 6

A "modified" chromosome 6 ("MO 6") has been recovered from Tripsacum dactyloides-introgressed "recovered maize" material originally obtained from J. M. J. deWet, U. of Illinois-Urbana, as (Tr75- X -155) selfed. The modification is in the short arm and appears to be a maize-Tripsacum translocation. From pachytene analysis, "MO 6" appears to have gained chromosomal material as well as a medium sized terminal knob. The break appears to be in the terminal portion of the 6S satellite. When "MO 6" is in the heterozygous state, bivalents are commonly observed at diakinesis. At pachytene, the translocation is not easily detected. "MO 6" is most readily seen in root tip mitosis. A comparison of the "MO 6" to the normal 6, at mitotic metaphase, is sketched below.

"MO 6" Normal 6

"MO 6" is both male and female transmissible and is not accompanied by any detectable sterility. A backcrossing program is presently underway to move the chromosome into B73, MO20W, KYS, W23, and MO17 backgrounds. Further cytological analysis is also being conducted.

B. Kindiger and S. A. Modena

Correction on a root tip squash technique

In last year's News Letter, the note "A modified root tip squash technique" by B. K. Kindiger and J. B. Beckett (MNL 57:32-33, 1983) contains an error in the protocol. The quantity of cycloheximide in step 2 of Procedure should be changed from 7 mg to 70 mg in 100 ml tap water.

B. Kindiger & J. B. Beckett

A mutator-induced photosynthesis mutant

Don Robertson has given us several families of maize mutants which contain the Robertson's Mutator genetic background, and are putative photosynthetic mutants. Of the seven families we have examined, two show a marked increase in chlorophyll fluorescence, which indicates a block in photosynthetic electron transport. Figure 1 shows the fluorescence trace of an excised leaf from one of these mutants, temporarily designated hcf*-Mu-5, compared to a leaf from a wild-type plant of the same family. The wild-type trace was run at an increased sensitivity (0.1 mv/cm compared to 0.25 mv/cm for the mutant) in order to visualize the wild-type curve more easily, but the difference between the two is clear. The mutant exhibits a much higher fluorescence yield, which does not level off sharply, but curves upward to its maximum level. This probably indicates that the plastoquinone pool is functioning normally in accepting electrons from PSII. Maximum fluorescence is maintained when the plastoquinone pool is saturated and can no longer oxidize PSII, nor become oxidized itself due to a subsequent block in the electron transport chain. The shape of the Mu-5 fluorescence curve is consistent with a lesion in the b6/f complex or in photosystem I.

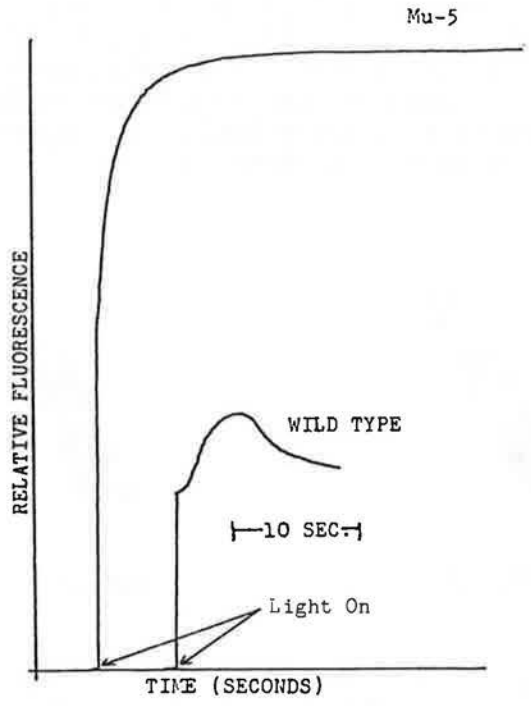


Figure 1.

LDS-polyacrylamide gel electrophoresis indicates that this is probably a photosystem I mutant. It is missing all of the major polypeptides which are associated with PSI (see Figure 2), and TMBZ staining of the gel shows the mutant

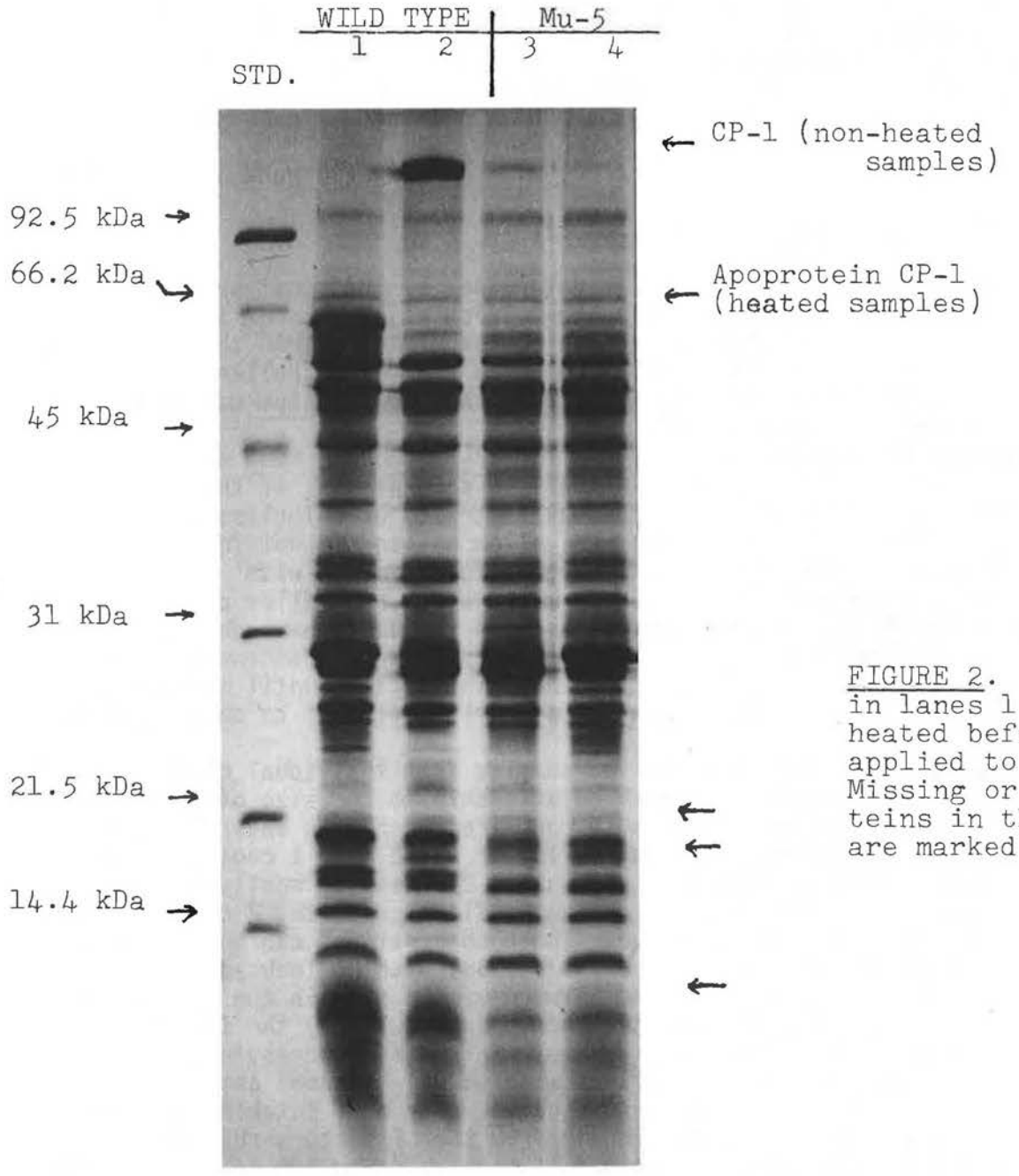


FIGURE 2. The samples in lanes 1 and 4 were heated before being applied to the gel. Missing or reduced proteins in the mutant are marked on the right.

to have a normal complement of cytochromes f and b6. Oxygen electrode studies, which have not yet been done, should provide further information on the exact location of the lesion. The gel profile is nearly identical to that of hcf*-50, an EMS-induced PSI mutant. Allelism tests are being conducted between hcf*-Mu-5 and hcf*-50, hcf*-49, and hcf*-44 (all deficient in PSI function).

A second mutant with increased chlorophyll fluorescence shows changes in four thylakoid polypeptides migrating in the 14-21 kilodalton region. The altered polypeptides have not been identified.

We are currently generating new photosynthetic mutants which are Mutator-induced. Since the Mu element has been cloned (Strommer et al., 1982, Nature 300:542-544) and can be used as a probe, mutants with this insertion may provide a way of locating nuclear genes for which the gene products are unknown, but which are known to play a role in the biosynthesis of the chloroplast membrane.

Marjorie Hunt and Don Miles

Pollen color with whp and mutable alleles of c2

Because yellow vs. white pollen color is determined by the genotype of the sporophyte (presumably of the anther wall), the effect of mutable c2 alleles with whp should be to confer yellow pigmentation on pollen grains within the pollen sac adjacent to C2 wall tissue, resulting in mixed white and yellow pollen. Combinations were derived involving whp with c2-m1 (autonomous, Spm-controlled) and c2-m2 (two-element, Spm-controlled).

Plants homozygous for whp and heterozygous for c2-m1/c2 have been derived; they produce uniformly yellow pollen grains. Anthers on such plants, if they are R-r P1, show well-defined sectors that include sizable tracts of colorless tissue. For c2-m1, the expectation of mixed white and yellow pollen was not found.

Four plants homozygous for whp and heterozygous for c2-m2/c2 with C1/c1-m5.Spm have been derived; each produced mixed white and yellow pollen. Three plants were R-r P1 Bz, in which purple sectors in the anthers were well-defined. The fourth plant was bz bz, and displayed yellow-fluorescent sectors in the anther walls. Presumably c2-m2 is itself null in function until mutations to C2 occur, while c2-m1 is itself functional at a level sufficient to confer yellow pollen color.

Color (ammonia-enhanced yellow) in pollen samples from individual c2-m2 anthers with large purple sectors showed a general relationship to the size of the sector. Among 6 anthers with 75-100% purple walls, 3 contained mostly or entirely yellow pollen, 2 contained mixed yellow and light yellow or white, and 1 contained white pollen. Among 11 anthers with 30-50% purple walls, 2 contained mostly or entirely yellow pollen, 3 contained mixed yellow or light yellow and white, 2 contained light yellow, and 4 contained white pollen. These observations can be related to the tests performed by Rhoades (Genetics 23:377, 1938) with Dt-induced sectors in anthers, in which a relationship was found between sector size in the anther wall and the percentage of A progeny from that anther. Unfortunately the few single-anther pollinations attempted with the c2-m2 material were unsuccessful. A single bulk pollination from one of the three plants with purple-sectored anthers and mixed yellow and white pollen, in a testcross on c2, gave half mutable and half colorless kernels (i.e., there were no progeny from the C2 sectors that were assumed to be present). The expected ratio is 1/4 mutable and 3/4 colorless. Why the testcross ratio might be 1:1 is unclear.

E. H. Coe, Jr.

Some observations on anther-wall fluorescence and pollen color with bz

As discussed in MNL 56:49, the yellow fluorescence of anther walls that is characteristic of bz bz plants is unaffected by the genotype for other color factors, except for the white-pollen genotype, homozygous c2 whp. As reported in the preceding note, a c2-m2 whp bz Spm plant showed anthers with yellow-fluorescent sectors, which is consistent with the other genotypic information. On the other hand, homozygous bz-m4 plants show variable anther fluorescence, according to observations over a period of days on a family of 16 plants in the summer of 1979. Some individuals were found to have strongly yellow-fluorescent anthers, others weakly, and others variable; observations of different samples from the same plants were inconsistent. The bz-m4 allele may be marginal in its expression, depending upon conditions, at the time of anther wall pigment synthesis.

When yellow pollen from bz bz plants is placed on a slide with ammoniacal glycerol and examined under the microscope, the yellow color is found to be localized in one or two small vesicles, usually near the pore, rather than distributed throughout the pollen grain as in Bz pollen. Presumably this localization reflects a difference in the accumulated flavonoids in the two different genotypes.

E. H. Coe, Jr.

Bulk Pollen Method to test allelism of Recessive Lethal Mutants

Allelism tests for recessive lethal mutants (either kernel or seedling) are made difficult by the fact that the homozygotes do not survive to produce gametes for testing. The best individuals that can be obtained for testing are the heterozygotes resulting from the self of a known heterozygote. The normal kernels (in the case of a lethal kernel mutant) or the normal seedlings (in the case of a lethal seedling mutant) from such a heterozygote will be randomly 2/3 right. According to Coe (MNL 51:61) the most efficient method for such a test is to "self a plant in source A and cross onto two plants in source B; test each of the two plants in B by crossing each onto one ear of a sib (set consists of 5 pollinations; failure of heterozygosity in source A is 1/3 and failure of showing heterozygosity in one or both plants of source B is 25/81; three such sets, totalling 15 plants, will have about 75% certainty of including an ideal test; 6 sets will have about 93% certainty)."

However, we have determined that a better way is to bulk the pollen from 3-5 tester plants and cross on 3 ears of the mutant family to be tested. The male plants may be selfed for insurance and may be used on as many target sets as there are mutants to be tested. Our experience with this procedure is that it is practically foolproof in obtaining positive allelism tests, and statistically very reliable as a negative test. The negative test may be made more secure by either selfing a second ear on the target plants or by performing a split double pollination self of the target ears.

M.G. Neuffer and Dan J. England

Progress in screening for agronomically and botanically useful EMS-induced mutants

Early in 1981 EMS-treated pollen of Mo17 was used to pollinate 75 plants of the inbred A632. Many mutations and aberrations have resulted, among which some are quite novel. Phenotypic expression is being followed from kernel to harvested ear. Among the more interesting dominants are Tillered-1590, Golden-1585, Semidwarf-1592 and Hairy-sheath-frayed-1595, but many unusual phenotypes affecting

most organs have been found. "Frayed" describes a leaf margin which has protrusions up to 1 cm long. A dominant very like Hsf*-1595 in the mature stage has just been found to have 1-2 odd little tubes exserted from the coleoptilar node, in one case developing into a tiller. Many of the sectored (MNL 57:30) and whole-plant dominant mutations noted in the M1 (1982) have been outcrossed to wx translocation stocks for location to chromosome arm.

Out of ca. 1140 M2 families examined last summer, over 270 had plants that differed significantly in time to pollination, plant or ear height, or other agronomically interesting characteristics. Outcrosses of these are being selfed, and next summer we will check the heritability of the traits and the form and relative yield of affected plants.

Segregation of mutants in the M2 does not fit standard genetic ratios because of the sectoring of M1 plants. It is not known if most EMS mutants are sectored in the M1, but, judging by the low proportions of many M2 mutant plants, many are. Some low ratios that were found were: bleached 2/38, virescent 5/37, rolled leaf 3/36. One family had 1 out of 17 plants from one side of the ear and 4 of 20 from the other side showing virescent (we had marked many ears above the midrib of the subtending leaf). Another case was 5/20 vs. 0/20 pale luteus. In plantings of 20 kernels we frequently found 1/20 white seedling, luteus, dwarf, etc. This explains why some mutants have not been seen until the M3 generation--rare M2 heterozygotes were selfed. Johri and Coe (MNL 55:31) found that ears generally do not sector cleanly into halves, as we have also observed, contributing to these low ratios.

Because of the many low ratios we cannot be sure that all variants noted are heritable. Ear variants were very common but one type was too frequent--small ears with large cupules, narrow shanks and short husks. Perhaps this was due to smut infestation in plants weakened by some mutation that had no direct effect on the ear. Among the more notable effects are few rows (8,10), many rows (18,20), short ears, short husks, fertile lower florets, long paleas, high cupule wings, secondary ears, branched silkless, large cupules, and many others. Unfortunately we could not self all plants in the field so many of these were found on plants that were open pollinated. It will take a while before we are sure these are heritable. Tassels also varied: male sterile, few branched, tiny anthers, awned paleas, barren branch tip, etc.

While placing the M1 ears in storage, a special effort was made to note any endosperm mutants that were readily distinguishable. Six mutants allelic to su1 were found, the number which one would predict by estimating one mutation per locus per 1000 M1 plants grown, using EMS treatment of pollen (Neuffer 1978, In D. B. Walden ed., Maize Breeding and Genetics).

Another subset out of ca. 4000 unexamined M2 families will be even more carefully examined next summer.

Robert McK. Bird and M. G. Neuffer

Elimination of superscripts in the designation of B-A and A-B elements derived from B-A translocations

Because of numerous difficulties we and others have encountered in using superscripts in designation of the two reciprocal elements of B-A translocations, we propose to keep all characters on-line. For TB-4Sa, for example, the B^4 and 4^B elements (or chromosomes) can be designated the B-4 (or B-4Sa) and the 4-B elements, respectively, without loss of clarity.

These usages are being applied in the current News Letter; if you see problems with this form, or have other suggestions, we would like to hear from you.

J. B. Beckett and E. H. Coe Jr.

Position of loci with respect to B-A translocations on chromosomes 3 and 4

Since my 1975 report on the position of genes with respect to B-A translocations (MNL 49:130), I have accumulated additional data involving new mutant genes and new B-A translocations. In this report, only data for chromosomes 3 and 4 are presented. Older data from MNL 49:130 are included for completeness. The following table lists only genes that have been assigned map positions; inclusion of the gene symbol indicates that the recessive allele is uncovered by (i.e., is distal to) the translocation and "+" indicates that the gene is not uncovered. Data from 8 new B-A translocations (designated TB-3Lf, -3Lg, -3Lh, -3Li, -3Lj, -3Lk, -3Ll, and -3Lm) are included.

TB-3La	gl6	ts4	lg2	ba	na	a3	a1	sh2	et
TB-3Lf	gl6		lg2				a1	sh2	et
TB-3Lg	gl6		lg2				a1	sh2	et
TB-3Ld	+	+	lg2	ba	na		a1	sh2	et
TB-3Lh	+		lg2				a1	sh2	et
TB-3Li	+		lg2				a1	sh2	et
TB-3Lj	+		lg2	ba	na		a1	sh2	et
TB-3Lc	+	+	+	ba	na		a1	sh2	et
TB-3Lk	+		+			a3	a1	sh2	et
TB-3Ll	+		+			a3	a1	sh2	et
TB-3Lm	+		+				a1	sh2	et

The following mutants previously were assigned to chromosome 3 (usually 3L) by others, especially M. G. Neuffer; they have less well defined map positions:

	cp*-330D	**cp*-1379A**	**d*-282**	**de*-932**	**dek6**	**pm**	**rgh*-1060B**	**su*-748A**	**vp1**	**y10**	**yd2**	**yel*-5787**	**ys3**
TB-3La	cp	cp	d		dek	pm	rgh	su	vp	y	yd	yel	ys
TB-3Lf			d	de						y		yel	
TB-3Lg	cp	cp	d					su	vp		yd		
TB-3Ld					dek	+		su	vp	y			
TB-3Lh								su	vp	y	yd		
TB-3Li		cp						su		y	yd		
TB-3Lj		cp						su			yd		
TB-3Lc						+		su		y	yd		
TB-3Ll								su					
TB-3Lm					dek								

All mutants with temporary symbols listed above (except yel*-5787, which came from the Coop Stock Center) were obtained from M. G. Neuffer; they have not been tested for allelism. A few pollinations were made onto gl7 and gl12 stocks obtained from the Coop Stock Center, but results are not given here because both may be allelic to gl6.

The following mapped genes on chromosome 4 are uncovered as indicated; data from four new B-A translocations (designated TB-4Lc, -4Ld, -4Le, and -4Lf) are included.

	st	la	su	bt2	bm3	gl4	gl3	c2	dp
TB-4Sa	st	la	su	bt2	+	+	+	+	+
TB-1La-4L4692			+		+	gl4	gl3	c2	dp
TB-9Sb-4L6222			+			gl4	gl3	c2	dp
TB-9Sb-4L6504			+		+	gl4	gl3	c2	dp
TB-4Lb					+	gl4	gl3	c2	dp
TB-4Lc						gl4	gl3	c2	dp
TB-4Ld					+		gl3	c2	dp
TB-4Le					+		gl3	c2	dp
TB-4Lf					+	gl4	gl3	c2	dp
TB-7Lb-4L4698					+		+	c2	dp

The following mutants are confirmed to be on 4S:

Temporary symbol	Source
d*-156A	M. G. Neuffer
hcf*-23	M. G. Neuffer (K. J. Leto)
pgspt*-1269	M. G. Neuffer
nec*-562	M. G. Neuffer
py*-60-1106	D. S. Robertson
shsu*-211C	M. G. Neuffer
su*-lethal	C. R. Burnham
vit*-X832mut	O. E. Nelson, Jr.
wcb*-719A	M. G. Neuffer
wtcb*-10A	M. G. Neuffer

Tests for allelism need to be made; several are probably allelic.

The following mutants are confirmed to be on 4L:

TB-1La-4L4692			py*-PI177593		wlv*-378A
TB-9Sb-4L6222	ol	ptd*-1130			
TB-9Sb-4L6504	ol				
TB-4Lb	ol				
TB-4Lc	ol		py*-PI177593	v8	
TB-4Ld	ol				
TB-4Le	ol			v8	
TB-4Lf	ol				
TB-7Lb-4L4698	+				

The ol and v8 stocks were obtained from the Coop Stock Center, ptd*-1130 and wlv*-378A were supplied by M. G. Neuffer, and D. S. Robertson supplied py*-PI177593.

Additional tests are in progress. A comprehensive summary of data for all other chromosomes is planned for the next News Letter.

J. B. Beckett

Color factors that affect white corn quality

Breeders who are developing white hybrids for food and milling purposes strive for pure white colors in the kernels, because off-white colors can result in inferior products after processing. While some of these effects have not been studied genetically as far as I am aware (and are low-expression traits such as smoky gray or yellow coloring in the pericarp and endosperm), there are major factors affecting the colors whose phenotypic effects can be defined, and a summary may be useful. Problems arise especially when yellow endosperm-red cob inbreds from elite yellow dents are converged with white endosperm-white cob sources. The accompanying chart is an attempt to systematize the major factors that are involved, more to emphasize what is expected and what we do not yet know than to finalize a key for these expressions. Any comments or inputs from cooperators who have worked with these expressions in ways I may have overlooked would be appreciated.

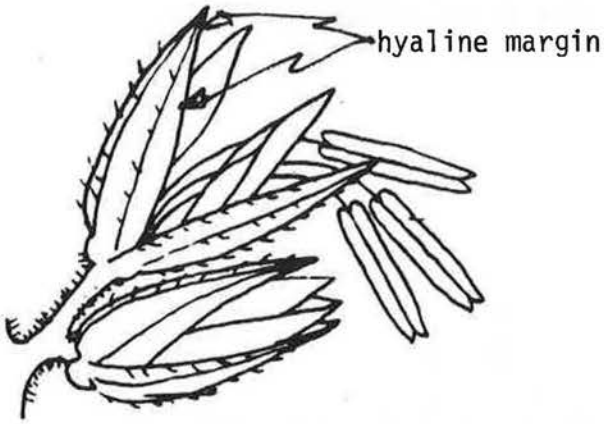
	Tissue											
	Coleoptile; plant base	Leaf tips; tassel & husk tips	Anthers	Silks	Tassel glume faces	Tassel glume bars	Tassel glume edges	Cob	Cob (in light)	Pericarp	Genotype	
	Phenotype											
	R	R	SR	SR	R	SR	B	RB	RB	SR	B-b P-wr r-r(s)	
	M	M	SM	SM	M	SR	B	RB	RB	SR	B-b P-wr r-r(m)	
	M	G	G	G	M	SR	B	RB	RB	SM	B-b P-wr r-g	
	R	R	SR	SR	R	SR	W	W	MR	SR	B-b P-ww r-r(s)	
	M	M	SM	SM	M	SR	W	W	MR	SR	B-b P-ww r-r(m)	
	M	G	G	G	M	SR	W	W	W	SM	B-b P-ww r-g	
	R	R	SR	SR	R	G	B	RB	RB	SR	b P-wr r-r(s)	
	M	M	SM	SM	M	G	B	RB	RB	SR	b P-wr r-r(m)	
	G	G	G	G	G	G	B	RB	RB	MB	b P-wr r-g	
	R	R	SR	SR	R	G	W	W	MR	SR	b P-ww r-r(s)	
	M	M	SM	SM	M	G	W	W	MR	SR	b P-ww r-r(m)	
	G	G	G	G	G	G	W	W	W	C	b P-ww r-g	

Key

B Brown
C Colorless
G Green (not pur/red)
M Medium color
R Red
S Sun-dependent
W White

The assumptions of the chart are that standard U.S. cornbelt genotypes are being used--that is, dominant for A1, A2, etc., but recessive for c1, r, and pl. The constitution c1 r pl determines colorless aleurone and embryo tissue (in the light or in the dark), and defines by pl that pigmentation in any parts of the growing plant (e.g., coleoptiles, plant base, anthers, etc.) will be light-dependent--i.e., sun-red. Pigment in these parts of the plant will be present if an active allele is present either at the B locus (B, the so-called "booster", or B-b, barred glumes) or at the R locus (r-r). Presence of pigment is regularly dominant to absence. At the B locus, barred alleles (represented by B-b in the chart) and null alleles (b) are common; stronger alleles from exotic sources will have the same effects as B-b, magnified in degree. Inbred 33-16 is B-b P-ww r-r pl; A619 is b P-ww r-r(w) pl; B37 is b P-wr r-r pl; K55 is b P-ww r-r pl; Mo17 is b P-wr r-r(w) pl; Mo20W is B-b P-ww r-r(w) pl. Some strains may carry dominant modifiers of plant color such as C2-Idf, which results in dosage-dependent reduction of some or all pigmentation. A very few breeding lines carry Pl, which

will result in pigmentation that is sun-independent and stronger in expression. The effects of the salmon silk factor, sm, are interactive with P-wr and r-r, altering red silk color to salmon or brown depending upon genotype; these interactions are excluded pending clarification of the details. The designations r-r(s) and r-r(m) are intended to designate strong versus medium alleles, strong alleles including the "cherry" types; the expression in the pericarp of a cherry-red "stain" in areas exposed to light is one of the problem pigmentations. As indicated in the chart, reliably colorless pericarp is expected only in the b P-ww r-g constitution (no detectable pigment in any plant parts). Pearly white corns (including inbred K55) nonetheless often carry r-r and show red coleoptiles, leaf tips, anthers, etc.; either the r-r and the background genotype do not result in red "stain" color, or else light exposure never occurs on these ears.



(reproduced from drawing by O. Ceska)

In MNL 57:33-34, my note about expression of P-wr in the tassel has brought inquiries about the exact localization of the faint, yellow brown color in the hyaline margins of the tassel glumes. The accompanying drawing is marked to show the edges where the pigment is recognizable. I would be interested to know whether this pigmentation is found to be reliable as an indicator of cob color.

E. H. Coe, Jr.

Change of designation: Clt*-985 and Bif-1440

The EMS-induced dominant short plant mutant (D*-985) reported earlier (MNL 51:59-60) as being located on chromosome 8 resembles in several aspects the clumped tassel dwarf mutant reported by Gelinas et al., Amer. J. Bot. 53:615. The D*-985 phenotype is a short plant, clumped tassel with normal anthers and pollen, and a smaller but well set ear. The leaves often have "lineate" like flecks of pale green on the leaf blade near the base. Since this description fits that of the original mutant (L. F. Bauman, pers. commun.), our designation should be changed from D*-985 to Clt*-985.

Another EMS-induced dominant mutant (located on chromosome 8) designated thin tassel (Tht*-1440) can be described as having a barren inflorescence. The mutant phenotype is the absence of many florets from both the tassel and the ear. It is therefore better designated as barren inflorescence with the gene symbol Bif1.

M. G. Neuffer

Flavonoid phenotypes in diverse tissues

The accompanying chart was assembled in an attempt to summarize currently available information on genotypes and their tissue-specific effects on flavonoid pigmentation. The complete genotype given in the first row should be understood to be constant on each line except for the factors shown (e.g., a1).

E. H. Coe, Jr.

FLAVONOID PHENOTYPES

Tissue																Key
Aleurone tissue	Scutellum	Plumule	Coleoptile; plant base	Roots; 1st node in dark	Seedling auricle	Leaf tips; tassel & husk tips	Husks; culm; sheaths	Tassel glume faces	Tassel glume bars	Anthers	Pollen	Silks	Pericarp	Cob; tassel glume edges	Leaf blade	B Brown Bn Brown-necrotic C Colorless F Fluoresces yellow G Green (not pur/red) I Intense purple L Lacquer-red M Medium color Mt Mottled color N Navajo crown color P Purple R Red S Sun-red Sm Salmon silks T Tan W White (not yellow) Y Yellow () Determined by alleles at R, B, other loci
P	()	()	R	R	R	R	P	P	P	P	Y	R	L	P	P	A1 A2 A3 B bh Bz1 Bz2 C1 C2 ch In Lc P-rr Pl Pr R-r Sm Vp1 Whp
C	C	C	MB	C	G	G	T	T	T	G	Y		B	B	G	a1 [others as above]
CB	C	C	MB	C	G	G	Bn	B	B	G	Y			B	G	a2
P			M	M	G	R	P	P	B	P	Y					a3 B-b
P	()	()	R	R	G	R	G	R	G	P	Y	R	L	RB	P	b
Mt			R	R	R	R	P	P	P	P	Y	R	L	P	P	Bh c1
B			MB	C	G	MB	Bn	B	B	BF	Y		L	B		bz1
B			MB	C	G	MB	B	B	B	B	Y		L	B		bz2
C	C	C	R	R	R	R	P	P	P	P	Y		L	P	P	c1-n
M	C	C	R	R	R	R	P	P	P	P	Y		L	P	P	c1-p (germ. in light)
C	C	C	M	C	G	G	M	P	P	M	Y			M		c2
M	C	C	M	C	G	G	M	P	P	M	Y			M		c2 in
M	C	C	M	C	G	G	M	P	P	M	Y			M		c2 su
C	C	C	G	C	G	G	G	G	G	G	W	G				c2 whp
C	C	C	G	C	G	G	G	G	G	G	W	G				c2 in whp
P													B	B		b Ch P-ww
IP	()	()	R	R	R	R	P	P	P	P	Y	R	L	P	P	in
IR	()	()	R	R	R	R	P	P	P	PR	Y	R	L	P	P	in pr
P	()	()	R	R	R	R	P	P	P	P	Y	R	L	P	M	lc
P	()	()	R	R	R	G	P	P	P	P	Y	R	L	RB	G	b lc
P	()	()	R	R	R	G	G	P	P	P	Y	R	C	RB	P	b P-wr
P	()	()	R	R	R	G	G	R	G	P	Y	R	C	C	P	b P-ww
C	()	()	R	R	R	G	G	R	G	P	Y	R	C	C		b P-ww r-r
C	C	C	G	G	G	G	G	G	G	G	Y	G	C	C		b P-ww r-g
P	()	()	S	S	R	R	P	P	S	S	Y	R	L	RB		pl
R	()	()	R	R	R	R	P	P	P	PR	Y	R	L	P	P	pr
P			R	R	R	G	P	P	P	G	Y	G	L	P		R-g
C	()	()	R	R	R	G	P	P	P	P	Y	R	L	P		r-r
C	()	()	R	R	R	G	P	P	P	P	Y	G	L	P		r-g
P	()	()	R	R	R	R	P	P	P	P	Y	Sm	L	P	P	sm
Mt																vp1
P			R	R	R	R	P	P	P	P	Y	R	L	P	P	whp
P			G	C	G	G	G	G	G	G	Y					b R-g
P			M	M	G	G	M	M	P	G	Y					B-b R-g
P	P		M	M	G	G	M	M	M	G	Y					B-Peru r-g
P	P		R	R	G	R	G	R	G	R	Y					B R-sc
N	P		R	R	G		G	G	G							b R-nj
C			R	R	G	R	G	R	G	R	Y	R	R			b r-ch
Mt						G	P	P	P	P						B-Ec r-g
C	C	C	R	R	R	R	P	P	P	P	Y	R	L	P	P	C1-I
C	C	C	M	M	G	R	M	M	M	M	Y		M			C2-Idf
M			R	R	G	R	P	P	P	P	Y	R	L	P	P	In-D
P	()	()	S	C	G	R	G	P	G	R	Y		L	R		b pl
C		P				R				R						r-r:Pu
P	()	()										B				P-ww sm

Location of Bif, C1t*-985 and pro on chromosome 8

During the past two years we have attempted to establish the order and map distances on chromosome 8 for two dominant mutants, Bif (barren inflorescence) and

Clt*-985 (clumped tassel, short plants) and also the recessive mutant pro (proline requiring). Bif and Clt had been located on chromosome 8 by use of the T wx series. The backcrosses showed no linkage with 16 of the 17 translocations used but the following data for wx T8-9d (breakpoints 8L.09 9S.16) show good linkage:

	Wx Dom.	Wx +	wx Dom.	wx +	T	% co
Bif	18	2	0	20	40	5
Clt	120	43	65	83	311	35

This evidence, along with some preliminary indication of linkage between Clt and ms8, suggests that Bif is near the T8-9d breakpoint on chromosome 8 and that Clt is some distance away on the long arm.

We attempted to set up the F1 Bif Clt + + + +/+ + pro v16 ms j to be crossed on + + + v16 ms8 j. Because the crosses involving combinations of Bif and Clt were difficult to make and because pro is a recessive lethal requiring selfing (impossible for ms8 plants) to establish genotypes of the backcross progeny, our results have been fragmentary. For these reasons no reliable 3-point data were obtained. It is possible, however, to take fragmentary data and combine it in groups of 2-point tests that will establish working distances for the genes involved.

The following are our data from these tests arranged as 2-point tests following the format of Emerson, Beadle and Fraser:

GENES X Y	LINKAGE PHASE	NUMBER of INDIVIDUALS X Y	X y	x Y	x y	TOTAL	RECOMBINATIONS NUMBER	PERCENT
Bif Clt	CB	3	24	23	11	61	14	33
	RB	62	40	32	82	216	72	23
						277	86	31
Bif Pro	CB	9	14	20	3	46	12	26
	RB	16	9	2	42	69	11	16
						115	23	20
Bif V16	CB	47	29	36	37	149	84	56
	RB	52	29	29	24	134	58	44
						283	142	50
Pro V16	RB	5	13	34	17	69	22	32
	CB	15	8	6	12	41	14	34
						110	36	33
Sht V16	CB	10	28	48	1	87	11	13
	RB	39	9	12	45	105	21	20
						192	32	17
V16 Ms8	CB	202	28	27	146	403	55	14
Ms8 J	CB	204	25	33	140	402	58	14

Three-point data:

F1 GENOTYPE	PARENTAL COMBINATIONS	RECOMBINATIONS REGION 1	REGION 2	REGION 1 & 2	TOTAL
v16 ms8 j	114 177	27 27	32 25	5 7	
+ + +	291	54	57	12	414
		13.0%	13.8%	2.9%	

The map distances for v16, ms8 and j agree with the published map and are in harmony with the listed 3-point data obtained for these markers. Our tentative map is Bif (pro Clt) v16 ms8 j. The parentheses indicate that there is still some doubt about the relative order of pro and Clt.

M. G. Neuffer and Dan J. England

Will the real Ky27 please stand up

The inbred line Ky27--white endosperm and purple plumule--was selected for inclusion in a survey for genetic variation among the polypeptides of the chloroplast thylakoid membrane. Analysis was conducted using a modified Laemmli SDS gradient-density PAGE system. A certain wild type protein band migrating faster than the Bio-Rad 14.4 Kd marker was absent from Ky27.

The source of first seed was from Marcus Zuber (USDA/University of Missouri), who recalls that it was in the breeders' seed collection here when he arrived in the late forties.

A second accession was obtained from E. H. Coe, who obtained it from J. B. Beckett (both USDA/University of Missouri). Beckett pedigrees this line back to 1955 when he obtained it from R. W. Jugenheimer, while at the University of Illinois. It also has white endosperm, purple plumule, and absence of the protein band.

P. L. Pfahler of the University of Florida/Gainesville contributed original seed from the University of Kentucky, marked "Lot 1959-3." The three phenotypic markers are as above.

C. G. Poneleit of the University of Kentucky supplied several lines originally derived from the land variety "Boone County White": Ky21, Ky27, Ky30A, Ky39, Ky56, and Ky122. A few other lines have been lost. Pfahler supplied two lots of Ky49. All lines mentioned in this listing are white endosperm and none, including the Ky27, has purple plumule. The Ky27 (lot 75/2050-54) is not missing a protein band, but Ky122 (lot 72/1016-20) does have that phenotype.

I conclude that the strain Ky27 currently supplied by the University of Kentucky differs from earlier releases, and that the genetic variation affecting expression of a chloroplast membrane protein was probably in the original Boone Co. White population. Though one might most trust seed supplied by the original breeding station, genetic shifts can and do occur!

Stephen A. Modena

New genes affecting polypeptides of the chloroplast thylakoid membrane: Lcs1, Lct1, Lct2

This report concerns variants discerned among the set of polypeptides peripheral or intrinsic to the chloroplast thylakoid membrane of the genus Zea. The phenotypes are detected by means of a system of gradient-density SDS gel electrophoresis, based on modified Laemmli buffers and with the addition of urea to the resolving gel. The density range of the resolving gel, the pH resolving buffer, and the amount of SDS (added to the cathodic buffer) are varied in order to optimize the resolution of the particular migrational variant. Proteins are visualized by staining with Coomassie Brilliant Blue R-250. Apparent molecular weight references are relative to the migratory positions of the six proteins composing the Low Molecular Weight Standards, offered by Bio-Rad Laboratories, and hold true only within the running conditions used to visualize the particular protein of interest. A precise protocol is available from the author (c/o E. H. Coe, 210 Curtis Hall, UMC, Columbia, MO 65211).

For the sake of brevity, detailed accession information and gel photos of variants are omitted from this report. It can be obtained from the dissertation of Stephen Anthony Modena (upon its acceptance) or by direct communication. A gel profile of "wild type" maize is included and labeled for the general orientation of those working with chloroplast proteins.

I have adopted the practice of naming these genes in a trivial manner. I do not know the function of the proteins involved. Their exact relative migratory location on a gel will depend on the interaction of gel running conditions. The

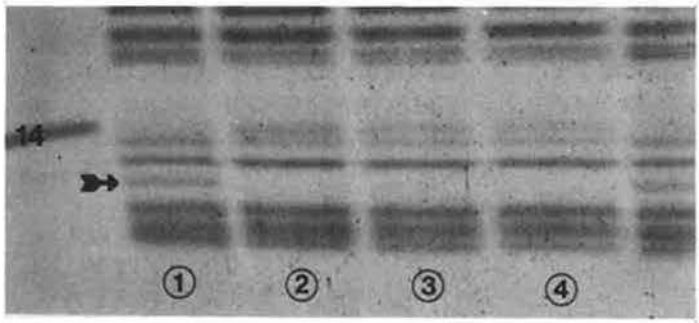


Figure 1

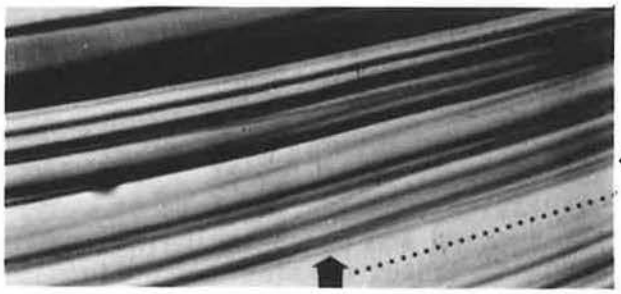


Figure 2

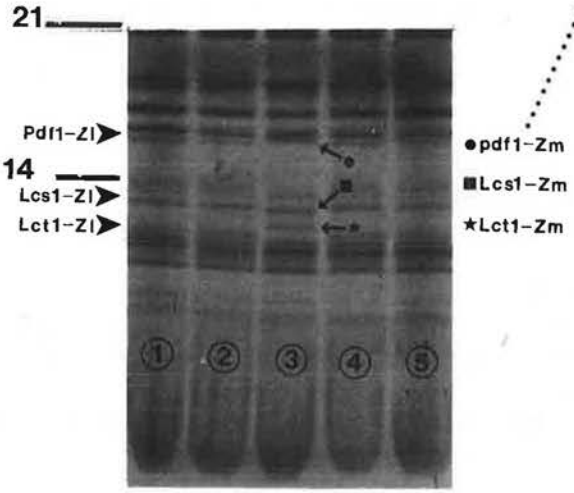
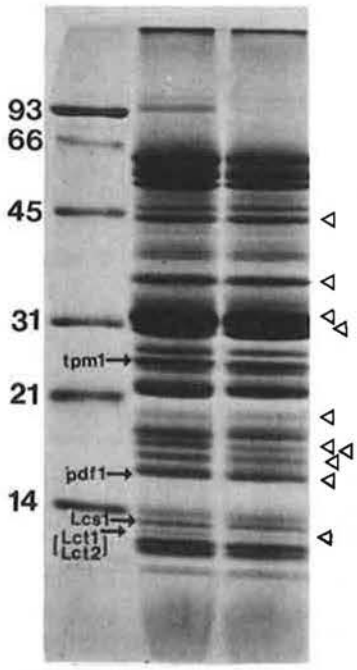


Figure 3



"pink doublet"

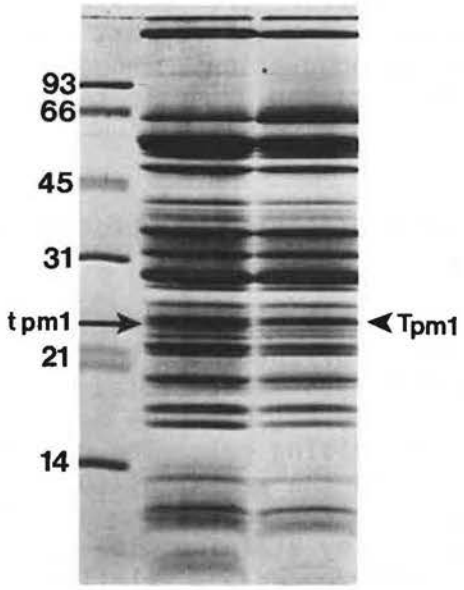


Figure 5

NOTE: Not all gels were run under the same conditions.

Fig. 1: Lane 1 is Lct2/Lct2 (Mo17); lanes 2, 3, 4 are lct2/lct2 (Ky27). **Fig. 2:** Portion of a 0-8M urea horizontal gradient gel showing that protein bands can often be "moved" for viewing or scoring. **Fig. 3:** Lanes 1, 2, 4, 5 are accessions of Zea luxurians; lane 3 is Mo17. **Fig. 4:** Lane 1 is Bio-Rad markers; lanes 2 and 3 are domesticated maize. Arrows are labeled with genes that affect that protein. Open arrowheads point to proteins for which variants have been noted and which are apparently heritable. **Fig. 5:** The phenotypes for Tpm1 and tpm1 homozygotes are shown (reported in MNL 57:38).

electrophoretic conditions used in some laboratories will not even resolve these bands, let alone the variants. Positive identification and further characterization of specific variants will depend on pedigreed stocks. Small quantities of seed, intended for increase first, are available from the author upon request.

I can resolve a cluster of at least seven bands, the slowest migrating of which approximately aligns with the 14.4 Kd. standard. I count from this slowest band.

Lcs1: For the second slowest band of the cluster I have detected three migrational phenotypes: wild-type domesticated maize has the fastest form; Z. luxurians, the slowest; and Z. diploperennis is heterogeneous for an intermediate migrational form and a maize mimic migrational form. Our one accession of Z. perennis is maize-mimic. A limited survey of Corn Belt and exotic germplasm of Meso-America uncovered no other alleles in maize. A cursory survey of Balsas, Chalco, Huehuetenango, and Nobogame indicate that they possess the maize phenotype.

For Z. diploperennis-maize reciprocal crosses, the individuals conform to the phenotype of the Z. diploperennis parent involved. In the progeny of the lineage (((Z. d. x Z. m.) x Z. m.) x Z. m.), in which the Z. diploperennis parent showed the Z. d. slower form as did the first backcross ear parent, the parental phenotypes are segregating. A symbol or allele is not yet assigned because statistically insufficient progeny have been scored.

Data are presented for several crosses involving Z. luxurians:

Cross	"Slow"	"Fast"	Chi-sq 1:1	Chi-sq 9:7	Chi-sq 3:1
B73 x (B84 x Z. l.)[1]	38	32	0.36	0.04	14.93
Mo17 x (B84 x Z. l.)[2]	32	15	5.45	2.22	0.86
(B84 x Z. l.)[3] x Z. l.	23	10	4.97	1.91	0.01
combined data[4]	93	57	8.17	1.79	12.84

[1] individual B-30
[2] individual B-20
[3] combined females B-20 and B-30
[4] data homogeneous for 1:1, 9:7, and 3:1

One gene or two? Or is there a biasing effect here related to the fact that the interspecific F1 plants characteristically are two-thirds pollen aborted? The quality of the Lct1 data (below) is a contrast!

In the belief that at least one nuclear factor is involved, the symbol Lcs1 (last cluster, second down) is assigned and there are two alleles: Lcs1-Zm and Lcs1-Zl. I can not resolve the genotype Lcs1-Zm/Lcs1-Zl into two bands, but there is band broadening uncharacteristic of either of the homozygotes. I favor the idea that this is structural variation.

Lct1 and Lct2: For the third slowest band there are two genes affecting the phenotype: structural variation and a presence-absence conditioning gene.

All Z. luxurians accessions examined possess a faster migrating form of this protein band; all domesticated maize (with two exceptions) and the teosintes (Balsas, Chalco, Huehuetenango, Nobogame, Z. diploperennis, and Z. perennis) examined have the wild type maize form. The single cross, maize x Z. l., has two bands of approximately half intensity; one aligns with maize, while the other aligns with Z. l.

Drawing upon exactly the same progeny as for scoring the Lcs1 gene, segregation data are presented in the following table.

Cross	Single band "slow"	Two bands	Single band "fast"	Chi-sq 1:1	Chi-sq 9:7	Chi-sq 3:1
B73 x (B84 x Z. l.)[1]	35	35	0	0.00	0.87	22.02
Mo17 x (B84 x Z. l.)[2]	26	21	0	0.34	0.00	8.69
(B84 x Z. l.)[3] x Z. l.	0	12	21	1.94	0.46	1.94
combined data[4]	(61+21)	68	(21+61)	1.13	0.10	32.00

footnotes as above

The gene symbol Lct1 (last cluster, third down) is assigned and there are two allelic forms: Lct1-Zm and Lct1-Zl.

The second locus affecting the expression of this band was recovered from the inbred line Ky27 (See accompanying article for a fuller story.) This was readily detected because the band of interest is apparently absent. Though various running conditions have been tried, I have not been able to detect a band at a position away from maize wild type. Nor is there any indication of an adjacent or nearby band intensifying, indicating migrational overlap--not in the F1 or the F2. Because of the apparent dominance-recessive nature of the segregation, this is unlikely to be another version of Lct1. The test cross and F2 data are presented:

Cross	"Present"	"Absent"	Chi-sq 1:1	Chi-sq 3:1
Mo83049.2-41 x Mo83048	37	35	0.01	20.17
Mo83049.1-1 selfed	52	22	11.36	0.65

The gene symbol lct2 is assigned and there are two alleles: Lct2 and lct2-Ky27. The inbred Ky122 has a gel phenotype like Ky27, but an allelism test has not been done.

The "absence" of this protein in lct2 plants raises at least two interesting possibilities: 1) this is a "dispensable" component of the thylakoid membrane or 2) there is another protein in the membrane (at a different migrational position on the gel) which performs a homologous function, affording the Lct1 protein to be absent.

Others wishing to work with these genes are welcome! Better techniques and fresh approaches may resolve some of the above ambiguities. Meanwhile I am converging these genes to some standard maize backgrounds.

Stephen A. Modena

Linkage studies of lesion and necrotic mutants

For the past couple of years I have been mapping a number of dominant and recessive lesion mutants, as well as a number of recessive necrotic mutants. Since several chromosomes are involved in the study, I have divided this summary by chromosome. While much of the data is only preliminary, some may be of value for those interested in particular chromosome arms or in mapping in general. As somewhat of a novice at mapping, I have found it very easy to determine linkage between genes already on the map; however, placement of new genes has been much more difficult. The potential location of new genes distal to the known genes on a chromosome points to the need for mapping of many different types of new genes, as well as perhaps some sort of selection scheme for mapping. Rapid selection of those mutants linked to known, easily scored markers could be done using small progeny sizes. Unlinked mutants, confirmed to arm, could then be scanned with additional TB stocks with breakpoints further out on the arm for potential new distal mutants. In any case, extending the map is not going to be easy.

CHROMOSOME 1S: The two dominant lesion mutants, Les2 and Les*-1449, are both located by T wx linkage studies (Table 1) on chromosome 1S. It is suspected that

TABLE 1. T wx Linkage Data for Dominant Mutants

T wx Stock	BP	# Pl	Mut Wx	N Wx	Mut wx	N wx	% CO	Chi-sqr
Les*-845 (Les2)								
1S-9c	.48 L.22	270	93	50	26	101	28	57.22
1L-9(4995)	.19 S.20	300	84	61	59	96	40	12.48
1L-9(8389)	.74 L.13	252	75	63	51	63	45	2.31
Les*-1449								
1S-9c	.48 L.22	137	41	24	17	55	30	24.50
1L-9(4995)	.19 S.20	131	43	25	14	49	30	24.21
1L-9(8389)	.74 L.13	160	16	61	25	58	54	39.42
Les*-1461								
1S-9c	.48 L.22	37	10	10	8	9	49	.06
1L-9(4995)	.19 S.20	91	21	20	25	25	49	.02
1L-9(8389)	.74 L.13	212	64	46	35	67	38	12.98
Les*-A607								
2S-9b	.18 L.22	162	54	30	27	51	35	14.24
2L-9d	.83 L.27	248	78	46	47	77	38	15.52
Spc1								
3L-9c	.09 L.12	126	46	17	10	53	21	42.7
Les*-F331035142								
7c-9(4363)	.00 .00	57	26	5	4	22	16	26.69

these two mutants are the first case of allelism between the EMS-induced lesion mutants, since both have a similar phenotype, small whitish lesions. Les2 appears linked to sr1 (Table 2) and approximately 20 to 30 map units from l1s1, a

Table 2. 2-Point Linkage Data

GENES X	Y	Linkage phase	Number of individuals X Y	X y	x Y	x y	Total	Recombination Number	Percent
CHROMOSOME 1S									
Les2	l1s1	C B	26	10	23	40	99	33	33.3
Les2	sr1	C B	54	0	2	34	90	2	2.2
l1s1	sr1	R B	6	41	32	11	90	17	18.9
Nec*-495C	sr1	R B	0	25	36	0	61	0	0

recessive lesion mutant. Similar data for Les*-1449 will be available following the summer, 1984 and may provide evidence of allelism with Les2. Several allelism tests were performed with the recessive mutants on 1S. les*-501B, an EMS-induced recessive lesion mutant, is allelic to l1s1. nec*-495C, a necrotic mutant characterized by large white patches on the leaves, is not allelic to zb4 or nec2, but is linked to sr1 (Table 2). It is interesting to speculate that nec*-495C is the recessive counterpart to Les2, since Les2 produces small white lesions and nec*-495C, large white patches.

CHROMOSOME 1L: Les*-1461 is located by T wx linkage data on 1L (Table 1); however, backcross linkage data showed no linkage with either br1, an1, bz2, gs1, or bm2. If the T wx data are correct, then Les*-1461 must be distal to bm2. If so, further mapping of Les*-1461 will require the placement of a new gene distal to bm2.

CHROMOSOME 2: The dominant lesion mutant, Les*-A607, was originally given to Dr. Neuffer by Dr. Kermicle. It is believed to be a spontaneous mutation. T wx

linkage data have placed this mutant on chromosome 2 (Table 1). No linkage with any other T wx stock was observed. Since it is linked to both wx T2-9b (2S.18) and wx T2-9d (2L.83), arm placement is not possible. This mutant has a phenotype resembling that of Les1, and allelism is not known at this time. Since Les1 is linked to sk1 and wt1 on 2S, Les*-A607 is also being mapped relative to these two genes, as well as other standard markers on 2S and 2L.

CHROMOSOME 3L: Spc1, a dominant EMS mutant on 3L, appears linked to lg2 (Table 3). I believe that Spc1 is much closer to lg2 than indicated since in the backcross, no Spc1 lg2 plants were observed. The high number of normal plants could be due to contamination, since the Spc1 plant was not used as the female.

Table 3. 3-Point Linkage Data

F1 Genotype	Parental combinations	Recombinations: Region 1	Recombinations: Region 2	Recombinations: Regions 1 and 2	Total
CHROMOSOME 3L					
Spc1 + +	16 29	0 9	18 20	0 3	
45		9	38	3	95
+ lg2 a1		9.5%	40%	3.2%	
CHROMOSOME 5					
nec*-493 + +	55 35	0 0	4 1	0 0	
90		0	5	0	95
+ a2 bm1		0%	5.3%	0%	

CHROMOSOME 4L: nec*-642A has previously been shown to be uncovered by TB-3La in our lab. Backcross linkage data with su1 gl4 c2 showed nec*-642A to be unlinked to any of these genes. Linkage data with dp1 are still to come. This mutant may be located on the distal tip of the chromosome, similar to Les*-1461, and may require additional mutants to be located before it can be placed accurately.

CHROMOSOME 5: Two recessive necrotic mutants have been located on chromosome 5. Both nec*-493 and nec3 have very similar phenotypes. In fact, these mutants were originally presumed to be allelic. Allelism tests this past summer have proved these two to be non-allelic. In addition to the allelism tests, nec*-493 appears linked to a2 and nec3 linked to bm1 bt1 (Tables 3 and 4). Further linkage data are necessary in order to establish the exact order of these genes; however, for nec*-493, the order is either a2 nec*-493 bm1 or nec*-493 a2 bm1. It is interesting that two very similar mutants should be so close together.

Table 4. 4-Point Linkage Data

F1 Genotype	Parental combinations	Recombinations: Region 1	Recombinations: Region 2	Recombinations: Region 3	Total
CHROMOSOME 5					
nec3 + + +	34 22	0 0	0 0	8 10	
56		0	0	18	74
+ bm1 bt1 pr1		0%	0%	24%	

CHROMOSOME 7: A new EMS-induced dominant lesion mutant, Les*-F331035142, has been tentatively located on chromosome 7 by T wx linkage data (Table 1). This mutant has not shown linkage with seven other T wx stocks, eliminating chromosome 9 as a possible location. Testing with wx T7-9a(7L.63) is still in progress. This mutant is characterized by numerous, small, necrotic lesions appearing in mid-season.

Dave Hoisington

DAEJEON, SOUTH KOREA
Chungnam National University

Influence of cytoplasm on tiller and ear numbers of a Korean local maize line

A Korean local maize line with multiple ears and tillers (MET) was reported (MNL 56:62) and its potential value in silage production was also reported (Agronomy Abstract 1981, p. 57). However, the genetic nature of the line for the tillering and earing habits was lacking. Therefore we attempted to find some of the genetic nature of the line. We made a cross between the MET and unrelated U.S. hybrid, Mo17 x B68. The F1 and F2 plants showed no apparent difference in the characters concerned, probably due to the masking effects (apical dominance) of heterosis on the initiation of tiller and ear. The reciprocal cross was the same. However, very distinct differences were observed in backcross generations. When the MET line was used as recurrent female parent, the number of tillers and ears increased greatly over the backcross in which the MET was used as male parent. The reciprocal effects of the backcross generation show the apparent cytoplasmic effects of the MET line on tiller and ear numbers. Genetic parameter estimates by Mather and Jinks' generation mean analysis also indicated an absence of mono- or digenic-chromosomal genes, additive and dominance gene effects, or interactions of gene effects. Our unreported data also showed that the characters of the MET line are maintained without any change in number for four generations of selfing of the original open-pollinated MET line. Our results suggest that the high tillers and ears of the MET line are effectively transferred by a backcross program using MET as female recurrent parent.

Bong-ho Choe and Chang-deok Han

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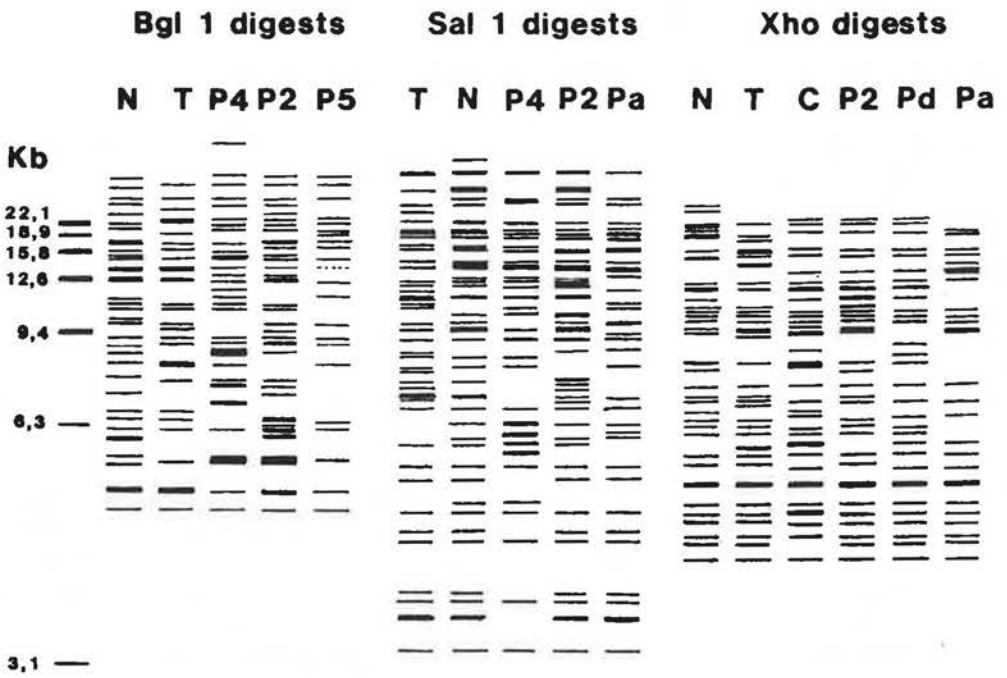
A Peruvian progeny as a source of a new mitochondrial variability

From a population of Peruvian maize, cultivated in greenhouse conditions, one plant was pollinated by the French early line F7. In the offspring of this hybrid, a plant (P2) was recognized as susceptible to methomyl by leaf test bioassay. By backcrossing this material by F7 or F215 inbred (as male recurrent parent), some lines were obtained that were remarkable in several aspects:

1. After three backcrosses by F7, most of the progenies appear male-sterile and susceptible to HmT (Helminthosporium maydis race T) toxin and methomyl. These traits are characteristic of Texas cytoplasmic male-sterility. However, the male fertile plants obtained among the segregants did not restore the male sterility of F7-T line.
2. Part of this material, crossed at least 2 times by F215 (cms-T maintainer), gave different results: most of the plants were male-fertile and segregated for resistance/susceptibility to HmT and methomyl.
3. A mtDNA analysis of P2, P4 and P5 hybrid progenies (Peruvian X F7) and several offspring from P2 has been started, according to the methodology described by A. Berville and M. Paillard (Les Colloques de l'I.N.R.A., 11:125-136, 1982). BglI, SalI and Xho restriction enzymes were used as shown in the figure. Obviously, P4 exhibits a pattern, whatever the enzyme used, never described for maize mtDNA: the two large bands at 9 and 5.5 Kb are typical. P5 looks like T after digestion by SalI (not shown) but appears different after digestion by Xho (see figure); furthermore, P5 is resistant to both methomyl and HmT toxin. In contrast, P2, which is susceptible to both compounds, exhibits a pattern that is



Diagrams of Bgl I Sal I Xho digests of maize mitochondrial DNA - N,T,C,respectively Normal, Texas and charrua cytoplams. P_2 P_4 and P_5 are sister progenies of the Peruvian plant. Pa and Pd are progenies of the P_2 plant. Experimental conditions for digestion agarose gel electrophoresis were as described by Vedel and Quetier (Physiol. Veg., 1978, 16 : 411-425). Mt - DNA extraction were done by chloroform phenol method.

quite different with the 3 restriction enzymes. Moreover, P2 progenies, Pa and Pd, for example, are different from the P2 origin (with Xho and SalI), which in several aspects look like N patterns (BglI and SalI). However, in other progenies showing the same phenotypes, P2 pattern does not appear to be modified.

It is too early to explain why in this population the mtDNA is so unstable. The cross by the French line may have initiated rearrangements or partial amplifications within the mitochondrial genome. The mtDNA analysis of our material from backcrossing by F7 or F215 could permit us to establish whether the male parent is involved in such mtDNA variability. Nevertheless, it seems evident that in this maize population there could coexist several patterns of mtDNA, similar to or different from the T, C, N (see figure) or S (not shown here) types previously described.

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Availability of maize germplasm with high stearic acid composition of oil

Since the first discovery and reporting of high stearic acid composition of germ oil in PI 175334 (Nepal), (J. Agric. Food Chem. 18:365-370, 1970), selfing in

an ear-to-row breeding procedure has resulted in a uniform plant type with various levels of stearic acid. A number of closely related lines derived from PI 175334 were grown in 1982, from which 250 selfed ears were obtained and analyzed for fatty acid composition. The majority of ears (240) had kernels with germ oil ranging from 13 to 19.8% stearic acid composition. Only 10 ears had less than 13% stearic acid composition of oil, and these were discarded. Seed of the 240 ears was combined into seven populations based on stearic acid levels as shown in Table 1. The average fatty acid composition of these populations shows a strong inverse relationship between stearic acid and linoleic acid. An increase in stearic acid resulted in a corresponding decrease in linoleic acid. The overall correlation coefficient was -0.87 between stearic and linoleic acid in these populations. There was a small but consistent increase in palmitic acid and decrease in oleic acid associated with an increase in stearic acid.

Table 1. Average fatty acid composition of oil from various high stearic acid populations.

		Fatty acid composition (%)					
Stearic acid composition %	No. of ears	Palmitic 16:0	Stearic 18:0	Oleic 18:1	Linoleic 18:2	Linolenic 18:3	Arachidic 20:0
19-20	7	14.0	19.3	26.7	37.2	0.8	2.0
18-19	34	13.8	18.4	26.8	38.2	0.9	2.0
17-18	43	13.7	17.5	27.1	38.9	0.9	2.0
16-17	48	13.4	16.5	27.1	40.0	0.9	2.0
15-16	52	13.0	15.5	27.3	41.1	1.0	2.1
14-15	40	13.0	14.5	27.5	42.0	1.0	2.1
13-14	16	12.9	13.6	27.5	42.8	1.0	2.2

Seed from the 34 ears with stearic acid composition between 18 and 19% of total oil was combined and released as GE180. Since GE180 has an 8 to 9-fold increase in stearic acid percentage as compared to most maize germplasm, it was released for use in oil genetic, physiology, and biochemistry studies. Seed from the other populations with 1% increments of stearic acid between 13 and 18% was not released, but will be made available to anyone wishing to study these lines.

M. D. Jellum

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Inheritance of stearic acid composition of maize oil

A limited survey of the fatty acid composition of the germ oil of Plant Introduction germplasm revealed a higher than average stearic acid composition in PI 175334 (Nepal cultivar 'Tusa Rani'). The original PI seed source had kernels with germ oil ranging from 1.5 to 6.3% stearic acid composition (J. Agric. Food Chem. 18:365-370, 1970). Selfing with an ear-to-row breeding procedure resulted in a number of lines with a much higher level of stearic acid than the original seed. After several generations of selfing, a number of lines with stearic acid levels of 9 to 16% of total oil were crossed with inbreds averaging 1 to 3% stearic acid composition of oil. Data from three of these crosses which had the F1 and F2 generation seed along with the backcross to each parental line were recently reported (J. Hered. 74:383-384, 1983). The results showed that a major single gene recessive for high stearic acid was responsible for controlling stearic acid composition of the germ oil in these crosses. However, the distribution pattern of kernels for the germ oil with high stearic acid in the F2

and BC2 generations was relatively broad and indicated the effects of modifying genes. A number of other crosses in which only the F1 and F2 generation seed was available for analyses also indicated the existence of a single gene with dominance for low stearic acid composition of oil.

M. D. Jellum and N. W. Widstrom

Location of genes controlling oleic and linoleic acids in maize oil

As a followup to a study of the inheritance of oleic and linoleic acids in maize oil (Crop Sci. 15:44-46, 1975) inbreds GE82 and X-187 were crossed to a series of waxy-marked chromosome 9 reciprocal translocations to determine chromosomal location of genes controlling these two fatty acids. The results indicated the existence of genes controlling germ oil composition for oleic and linoleic acids on the long arm of chromosome 5 of inbred GE82. The same gene(s) could be responsible for controlling both fatty acids. For inbred X-187, the data indicated the location of a gene controlling linoleic acid on the long arm of chromosome 4. Although not verified by F2 data, the results also indicated the location of gene(s) controlling linoleic acid on the short arm of chromosome 1 of both inbreds. Oleic and linoleic acids generally have a very high negative correlation with each other in corn oil, and the gene(s) show dependent segregation of the two fatty acids. However, the strong association between oleic and linoleic acids was not evident in these inbreds, which may have been due to differences in palmitic acid composition of the parental lines.

M. D. Jellum and N. W. Widstrom

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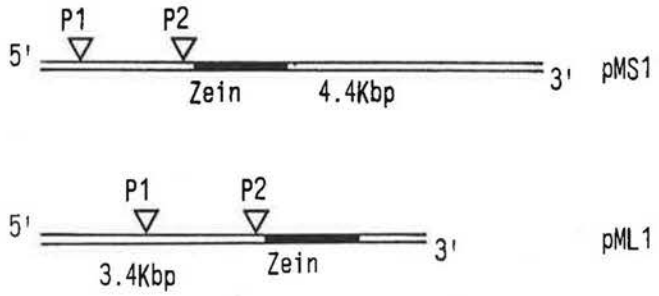
The zein gene promoter system

The two major zein protein size classes of 19,000 and 21,000 daltons are synthesized and stored in approximately equal amounts in maize endosperm. Although the synthesis of proteins of each class appears to be closely coordinated, several important differences have been found in the organization and expression of the two gene classes. The gene clusters for the 19,000 and 21,000 dalton zeins are located on different chromosomes, the cDNAs specific for each class do not cross hybridize, and regulatory loci such as opaque-2 and floury-2 affect the two classes differently.

Since the isolation of maize genomic clones containing intact zein genes and extensive flanking sequences, it has been feasible to analyze some of the differences between the genes coding for the two zein protein classes and their mechanisms of expression. As a representative of the 19,000 dalton protein class, we have analyzed the maize clone pMS1 (with a 4.4 Kb maize insert) and as a representative of the 21,000 dalton protein class the clone pML1 (with a 3.4 Kb maize insert). Both classes contain one zein gene free of intervening sequences (as with all zein genes studied to date), and at least 1.5 Kb to the 5' side of the protein coding sequences. The 5' regions have been sequenced and RNA isolated from maize endosperm has been used to accurately map the zein mRNA start positions. These experiments have been combined with in vitro transcription data to demonstrate that both genes are transcribed from two widely separated promoter regions, as indicated in the diagram below.



The two promoters, termed P1 and P2, are separated by about 1,000 bases with no protein coding function. Furthermore, although the positions of P1 and P2 relative to the zein protein coding sequence are very similar in pML1 and pMS1, the sequences of the entire region are different. The sizes of the mRNAs that are transcribed from each promoter correspond in size to the zein mRNAs isolated from endosperm. It is thought that the larger zein mRNAs (over 2,000 bases long) are transcribed from promoters that lie still further 5' of P1.

In addition to the analysis of the transcription of pMS1 and pML1 zein genes in maize endosperm, we have studied the expression of these genes in vitro and in heterologous in vivo systems. The activity of the P1 and P2 promoters for these two genes is summarized in the table below.

		pMS1		pML1	
		P1	P2	P1	P2
In vitro	Xenopus oozyte germinal vesicle extracts	-	-	+	+
	Hela cell extracts	-	-	+	-
In vivo	Yeast	+	-	-	+
	Acetabularia	active but promoter not known		results inconclusive	
	Maize endosperm	+	+	+	+

The two clones show quite different activities in the different systems. pMS1 was inactive in both in vitro systems but was able to synthesize zein protein, detected by immunofluorescence, in Acetabularia. Conversely, pML1 was active in the in vitro systems but only gave inconclusive results in Acetabularia. Although both clones were actively transcribed in yeast, pMS1 used exclusively P1 whereas pML1 only used P2. The RNA starts used in yeast were identical, however, to the nucleotide, with those used in endosperm.

The ability to transcribe and, as in the case of Acetabularia, to translate the zein genomic clones greatly increases our ability to study the control of expression of these genes. The clear-cut differences in the expression of the two genomic clones, both in terms of absolute activity and with respect to the activity of the two promoters, throw some light on the differences observed in the expression of the two zein protein classes in endosperm. Such differences may, in fact, lie behind differences in action of mutated regulatory loci. With the heterologous systems that we now have in use, we hope to study more closely the signal structures associated with zein gene expression. More definitive studies must, however, await the development of a homologous transcription system.

P. Langridge, J. Brown and G. Feix

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University of Florida and USDA-ARS

Distribution and expression of small circular mitochondrial DNAs

A cell suspension of the Black Mexican Sweet line of maize contains two circular DNA molecules of 1.94 kb and 1.4 kb in addition to a 2.3 linear DNA and the principal mtDNA genome. A previous report showed that under certain conditions the 1.94 kb molecule could replicate preferentially with respect to the other mitochondrial DNAs (Smith, Pring, and Chourey, MNL 57:47-48, 1983). This characteristic of the 1.94 kb DNA led to an investigation of its distribution and transcription.

MtDNA was prepared from several lines, electrophoresed, transferred to nitrocellulose, and hybridized with nick-translated probes. The probes were either a cloned BamH1 fragment of the 1.94 kb DNA or a full-length clone of the 1.4 kb molecule. No homology was found between either of the cloned molecules, the other known linear small mtDNAs (S1, S2, 2.3 kb, 2.1 kb) or the restricted mtDNA of any cytoplasm tested. Many cytoplasms tested for presence of the DNAs carried the 1.94 kb DNA but not the 1.4 kb DNA; no cytoplasm was found to contain the 1.4 kb DNA without the 1.94 kb DNA. The 1.94 kb DNA was present in Black Mexican (cell suspension and plant), Wf9(N), Wf9(T), Wf9(C), Wf9(S), a fertile cytoplasmic revertant of S cytoplasm, S cytoplasm in two additional backgrounds, and in cytoplasms RB, BB, PR, and ES, members of the C group of male-sterile cytoplasms. The 1.4 kb DNA was present in Black Mexican (cell suspension and plant), 38-11(S), A619(C), Wf9(C), and in BB, PR, and ES cytoplasms. The 1.94 kb and 1.4 kb DNAs were undetectable in several cytoplasms tested, including VG, fertile cytoplasmic revertants of VG, ME, CA, LBN, B, and R. The 1.4 kb DNA was absent in Wf9(N), Wf9(S), Wf9(T), and RB cytoplasm. Although mtDNA prepared from 38-11(ME) showed no homology to either the 1.94 to 1.4 kb DNAs, nuclear DNA prepared from this line and restricted with either BamH1 or EcoRI, electrophoresed, and transferred to nitrocellulose, showed homology to the cloned probes. The 1.94 kb molecule hybridized to BamH1 fragments of 6.7 and 9.5 kb, while the 1.4 kb molecule hybridized to a 6.8 kb EcoRI fragment.

To detect transcription of the 1.94 and 1.4 kb DNAs, total mtRNA was extracted from Black Mexican Sweet line cell suspension, glyoxylated, electrophoresed in 10 mM phosphate 1.6% agarose gels, transferred to nitrocellulose, and probed with the DNAs. Two transcripts were detected for each probe. The 1.94 kb DNA hybridized to transcripts of approximately 1,005 and 600 nucleotides. The 1.4 kb DNA hybridized to transcripts of approximately 890 and 460 nucleotides. Both probes also hybridized to contaminating DNAs in the RNA preparations which corresponded to their own molecular size. This hybridization may have obscured the detection of full-length transcripts.

The transcription of these small circular DNAs within the mitochondrion could indicate that these molecules may play a role in mitochondrial function. However, the existence of cytoplasms which apparently lack one or both of these molecules indicates that they are not indispensable to the mitochondrion. The nuclear homology to these molecules may be an indication of transposition of the DNAs between the mitochondrion and the nucleus, and a possible mechanism to compensate for the absence of these DNAs in the former.

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Morphological characterization of five developmental mutants

Among the defective kernel mutants there are two types of developmental mutants: (1) those that are blocked prior to the formation of leaf primordia and (2) those that form leaf primordia but fail to germinate when tested in culture or at kernel maturity (Sheridan and Neuffer, J. Hered. 73:319-329, 1982). We have examined four mutants (cp*-E1113A, rgh*-E1210, fl*-E1253B, and dcr*-E1428) of the first type and one (cp*-E1399A) of the second type. Mutant E1113A is located on 1L; the remaining four have not been located to chromosome arm.

All five developmental mutants were examined at intervals to determine when a mutant phenotype was first apparent, to document the course of development, and to assess the stage specificity of their blocks. Homozygous mutant kernels from segregating self-pollinated ears of Missouri-grown materials were examined by paraffin sectioning at early (13-17 days post-pollination), middle (17-35 days) and late (36-60 days) stages of development. These materials were kindly provided by M. G. Neuffer. North Dakota-grown materials were examined by paraffin sectioning and in fresh dissections at kernel maturity (60 days or later).

Embryos of the five mutants were markedly retarded in their size and developmental stage (Figure 1). This was evident from the time mutant kernels

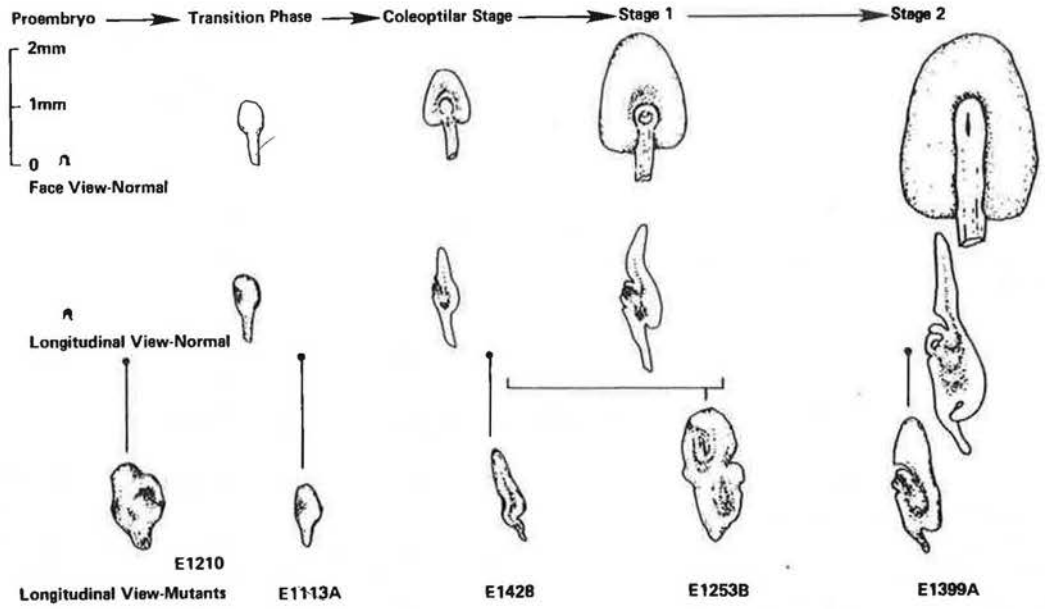


Figure 1. Maize embryo development. Top row: Normal embryos: external aspect, face view based on freshly dissected material. Middle row: Normal embryos, median longitudinal sections. Bottom row: Mutants characterized in this study, median longitudinal sections. Stages are those of Abbe and Stein. (based in part upon Randolph, 1936; Abbe and Stein, 1954; and Sheridan and Neuffer, 1982.)

were first distinguishable. The mutants differed, however, in the course of their development (Table 1). Four of the mutants, E1113A, E1210, E1399A and E1428, were stage specific in their blocks. The fifth, E1253B, was blocked variably from just before to just after formation of the first leaf primordium (Figure 1). The

Table 1. Morphological Characterization of Developmental Mutants

	E1113A	E1210	E1253B	E1428	E1399A
Mature kernel phenotype	collapsed	rough	floury	defective crown	collapsed
Age at which mutant kernels are first distinguished	13 days	15 days	13 days	13 days	13 days
Stage of developmental block*	transition	abnormal proembryo	variable abnormal coleoptilar to stage 1	abnormal coleoptilar	stage 2
Developmental profile:					
Early development	normal	abnormal forms callus-like mass	normal	abnormal fails to develop shoot apex	normal but small
Middle and late development	blocked	proliferates lobes and meristematic regions	abnormal enlargement of embryonic structures	shoot apex area necrotic embryo unhealthy	normal but small
Maturity	blocked	as above with degeneration aleurone proliferates	as above with degeneration aleurone proliferates	embryo unhealthy with widespread necrosis	normal but small non-germinating

* Embryonic stages are as follows: Proembryo = spherical mass of undifferentiated cells; transition stage = embryo proper atop elongate suspensor, epidermis and shoot meristem differentiating, scutellum becoming shield-shaped; coleoptilar stage = shoot-root axis organizing and coleoptilar ring formed. Stages 1, 2, etc. = distinguished by formation of successive leaf primordia. (See E. C. Abbe and O. L. Stein, 1954, Am. J. Bot. 41:285-293)

stage-specificity and the variety of developmental profiles and arrest phenotypes of these five mutants suggest that they represent specific genes controlling diverse processes in embryo development.

Janice K. Clark and William F. Sheridan

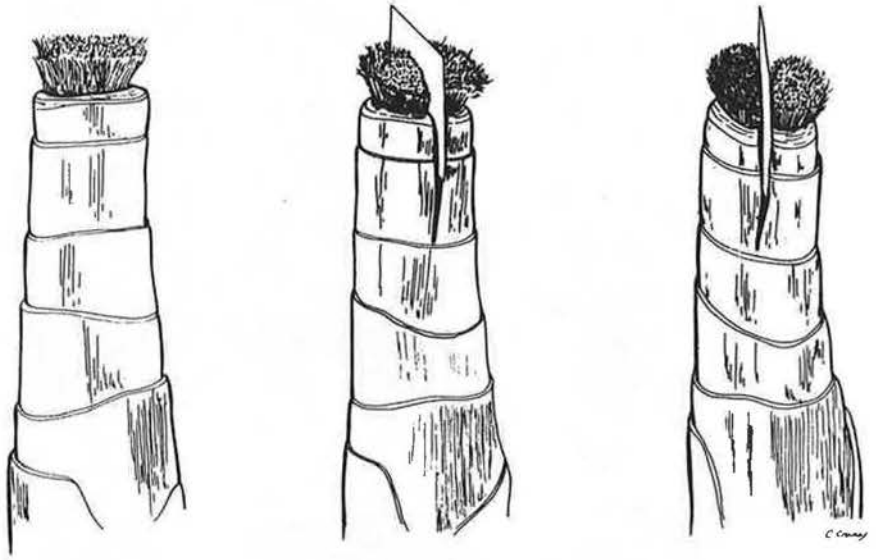
Testing lethals for allelism using a double pollination technique

We have been interested in testing for allelism among a collection of lethal defective kernel (dek) mutants. Because the mutants are lethal in the homozygous condition, it is necessary to cross heterozygous plants to test for allelism. Ideally each plant would be self-pollinated to determine whether or not the plant is heterozygous, and would also be cross-pollinated or would serve as a pollen source for cross-pollination. Since our stocks do not regularly bear two ears on each plant it is advantageous to be able to use a single ear to accomplish both a self-pollination and a cross-pollination.

Two procedures have been described for us where double-pollination (selfing and crossing) of a single ear is accomplished by pollinating a portion of the ear on one day and the remaining part of the ear the following day. The first of these methods used ears that are cut back in the usual manner. The silks visible on the first day of emergence are self-pollinated and the following day the additional silks that have emerged subsequent to the first pollination are cross-pollinated (M. G. Neuffer, pers. commun.). The second procedure involves cutting back an ear in the usual manner and self-pollinating the following morning. However, as soon as the pollination is completed a cut is made across the husks just above the top of the ear so as to sever the silks on one side of the ear. The following day the

cut silks are brushed away and newly grown silks below the severed portions are then cross-pollinated (J. R. Laughnan, pers. commun.). Both of these procedures require returning to the female parent plant on a second day. In order to avoid this repeated visit, we have used a procedure where half of the silks on an ear are self-pollinated and half are cross-pollinated on the same day. This procedure equals or surpasses the results obtained with the above described procedures.

Double-pollination of a single ear is performed as follows: When the silks have completely emerged, they are trimmed back by cutting across the upper portion of the husks just above the tip of the ear in the usual fashion. Then a knife is used to slice downward a short distance into the rim of the husks and just into the ear tip to form a slit. A small paper square 3/4 inch x 3/4 inch (cut from a file card) is inserted into the slit to provide a separator for the silks that will subsequently emerge, dividing them into two equal parts. The slit in the husks and the ear tip holds the paper separator in a secure fashion (see Fig. 1).



Legend for Figure 1.

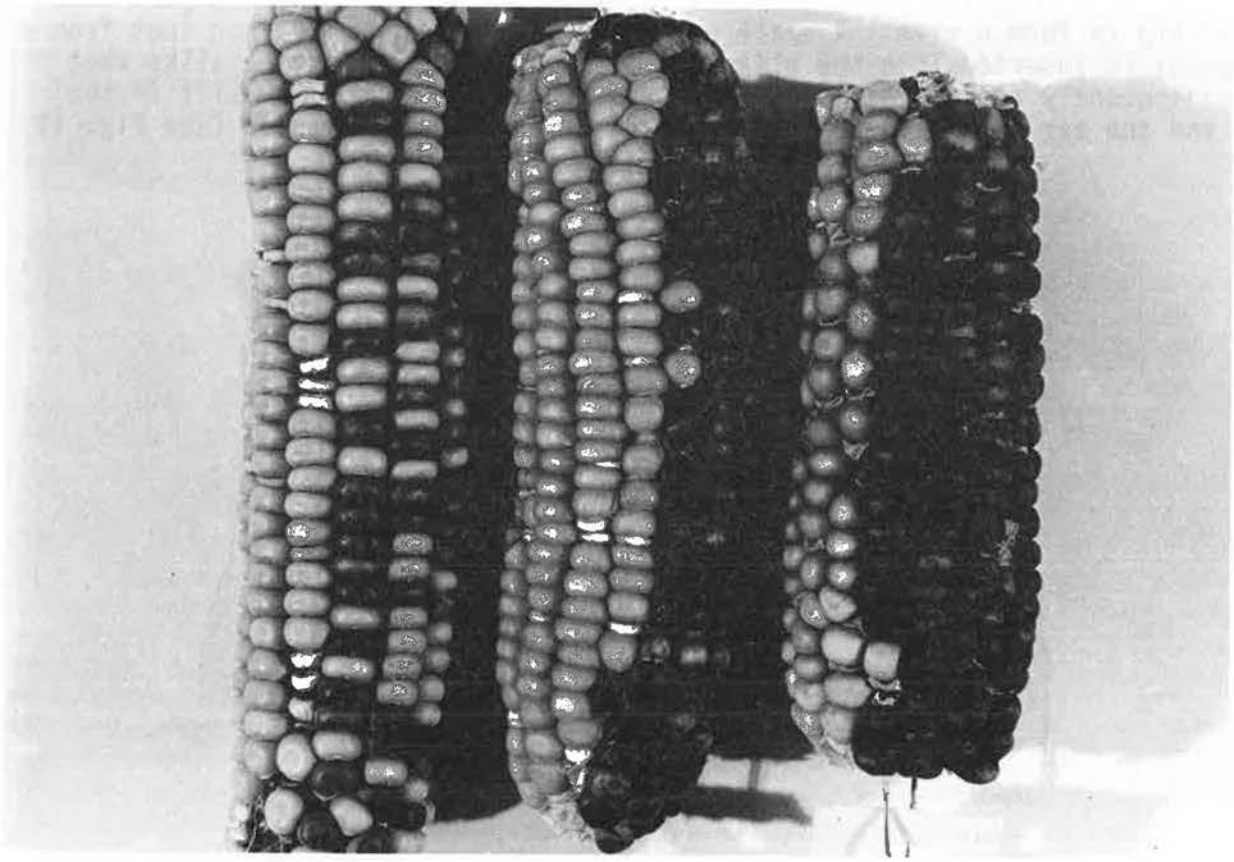
Preparation and pollination of an ear to be double pollinated. Left, newly emerged silks on an ear that was cut back the previous day. Middle, a slit has been cut into the husks and ear tip and a square of paper inserted into the slit so as to separate the silks into two groups. Right, pollen from the pollen parent has been carefully poured onto one group of silks while the other group of silks remains to be pollinated.

Pollen from the cross donor is transferred to glassine bags to facilitate handling, the pollen parent is self-pollinated, and pollen is taken to the plants to be double-pollinated, where the pollen is carefully poured onto the silks on one side of the paper separator. The shoot bag is then replaced. After the cross-pollinations are completed, pollen is collected and used to accomplish self-pollination on the other side (Figure 1).

Discrimination of the cross-pollinated kernels from the self-pollinated kernels on a double-pollinated ear is aided by having the crossed kernels colored (anthocyanin) and the selfed kernels colorless. If the mutants to be tested are available in both colored and colorless stocks, ears can be produced with colored kernels on one half of the ear and colorless kernels on the other half. The colored kernels are the result of the cross-pollination and the colorless kernels are the result of the self-pollination.

An unambiguous test result is obtained when the self-pollinated ear of the pollen parent segregates for a dek mutant and the self-pollinated half of the

female ear also segregates for a dek mutant. In such cases, the two mutants being tested are allelic if the cross-pollinated half of the ear of the female parent bears dek mutant kernels as well as normal appearing kernels, while the presence of only normal kernels on that half of the ear demonstrates that the two mutants are not allelic (see Figure 2).



Legend for Figure 2.

Double pollinated ears. Left: the ear was self pollinated on the left side. This side bears colorless kernels and is segregating for the dek mutant E1391 (note the floury collapsed kernels) located on chromosome arm 9L. The right side was pollinated with pollen from a plant heterozygous for E744 (a dek mutant with defective opaque kernels). Note that the pollen parent plant was heterozygous for one of the anthocyanin factors but that the cross pollinated half of the ear is nonetheless readily distinguishable. The absence of dek mutant kernels on the right side of the ear demonstrates that E1391 is not allelic with E744. Middle: this ear was self pollinated on the left side which is segregating for E627D (note the defective floury kernels) located on chromosome arm 3L. The right side was pollianted with pollen from a plant homozygous for all of the anthocyanin factors, resulting in a clear delineation of the self pollinated colorless kernels and the colored kernels produced by the cross pollination. Since self pollination revealed that the pollen parent plant was not heterozygous for a dek mutant this ear does not allow for any conclusion regarding allelism. Right: this ear was self pollinated on a plant heterozygous for E873 (note the collapsed kernels) located on chromosome arm 9S. The right side of the ear was pollinated with pollen from a plant heterozygous for E1054, (a collapsed dek mutant) and homozygous for the anthocyanin factors. Since both sides of the ear are segregating for a dek mutant, this ear demonstrates that these two mutants located on chromosome arm 9S are allelic.

Since the probability is two-thirds that a particular kernel selected for planting is heterozygous for the gene of interest, the likelihood that any particular pollen parent plant is heterozygous, or that any particular female parent plant is heterozygous, is two-thirds. When a pollen parent plant is self-pollinated and crossed onto a female parent plant that is also self-pollinated (double-pollinated), then the likelihood that both parents are heterozygous is 2/3 x 2/3 = 4/9 or 0.444 (see Coe, MNL 51:61, 1977, for similar considerations). If one pollen parent plant is selfed and crossed onto three different female plants, the probability that the pollen parent and at least one of the female parents crossed by it will be heterozygous is 0.642 (the probability of failing to achieve this situation is 0.358). If three pollen parent plants are selfed and each is crossed onto three different female plants, then the probability of failing to achieve an unambiguous test where both the pollen and female parent are heterozygous is 0.046 (=0.358 x 0.358 x 0.358) and therefore the likelihood of successfully conducting the allelism test is 95.4%. This requires a total of 12 plants, three to be used as pollen parents and nine to be used as female parents for double-pollination. If four different pollen parent plants are used and each is crossed onto two female parents that are double-pollinated, then the probability of a successful test is 97.3%, and this system also requires a total of 12 plants. If six different pollen parent plants are used and each is crossed onto one female parent plant that is double-pollinated, then the probability of a successful test is 97.1%, and this system also requires a total of 12 plants. The choice of which of these schemes to employ (assuming the desire for at least a 95% probability of successfully conducting the test) will depend on the number of plants of both the pollen and the female parent families that are available for pollination.

William F. Sheridan and Janice K. Clark

Isolation of Ds induced dek mutants

It would be desirable to have a collection of Ds induced defective kernel (dek) mutants since such a collection could aid in determining the nature of the defects in this type of mutant. In particular, such mutants would allow for comparison of the stable mutant condition (in the absence of Ac) with the unstable condition (in the presence of Ac), which should result in the production of sectors of both normal and mutant tissue in the endosperm and embryo of developing kernels. An examination of the developmental fate of such kernels when the size of the genetically normal tissue sectors is varied (by varying the dosage of Ac) may aid in understanding whether or not the defective condition can be rescued by cross-feeding from normal cells in the same tissue, and whether or not plants consisting of normal and mutant sectors can be grown from such kernels. In order to pursue this goal, a stock homozygous for bz2-m and a purple kernel stock homozygous for the normal allele at all of the loci controlling anthocyanin synthesis were planted in the spring of 1982. The stocks and advice were kindly provided by M. G. Neuffer. The kernels of the bz2-m stock selected for planting all carried one dose of Ac at an unlocated site on chromosome 9, as well as Ds at the bz2-m locus. The resulting plants bore ears that produced embryo sacs, half of which contained one dose of Ac and the other half lacked Ac. These ears were pollinated with pollen from the purple stock, resulting in the production of fully colored ears. The kernels on these ears were homozygous for the normal allele at each of the loci involving anthocyanin synthesis, except for being heterozygous at the bronze-2 locus (Bz2/bz2-m). From 20 such ears, 5,906 kernels were obtained and planted as 20 families in the spring of 1983. This planting produced 4,944 plants, from which 3,415 self-pollinated ears were obtained. An average of 195 ears was obtained from the 20 families, and the range of ears per family was from

87 to 265 (see Table). Among the 20 families, 8 yielded one or more ears segregating for dek mutants.

bz2-m Plot - Summer 1983 Summary

Family Number	Number Ears Harvested	Number Ears all Cl	Number of dek Ears	Number Striped Plants
1	137	7	0	0
2	143	3	0	0
3	142	42	2	0
4	216	2	1	0
5	249	1	0	1
6	87	5	0	0
7	222	5	0	0
8	190	6	0	0
9	265	9	0	1
10	221	7	74	0
11	204	3	0	0
12	246	12	0	0
13	210	10	0	0
14	43	0	0	0
15	126	1	0	0
16	152	6	38	0
17	167	2	1	0
18	202	6	1	2
19	101	1	1	0
20	92	0	1	0
	3415	128	119	4

In family #10 there were 141 ears not segregating for dek kernels and 74 ears segregating; in family #16 there were 114 non-segregating ears and 38 segregating ears. In both of these cases it is most likely that the normal allele at a dek locus mutated to the recessive state after zygote formation, but early during the development of the embryo that grew into the bz2-m stock plant used as the female parent in 1982. Such an event could give rise to an ear bearing a large sector (approximately half the ear) that would be +/dek, the other portion being +/+. Kernels from the mutant sector would produce an equal proportion of +/dek and +/+ kernels when pollinated by the purple kernel stock. All of the kernels produced on the non-mutant sector would produce +/+ kernels. For family #3 there were two ears out of 142 harvested that were segregating for dek mutant kernels, and in five other families (#4, 17, 18, 19, and 20) there was one ear segregating for a dek mutant among a large number of ears in each family. In these seven cases the mutation events resulting in the production of dek alleles must have occurred late in ear development of the bz2-m plants used as the female parents in 1982. These events most likely occurred after kernel primordia were formed, but not much later than zygote formation. This appears likely because only one (or 2 in the case of family #3) of the resulting kernels produced a plant carrying the mutant dek allele (as revealed by subsequent self-pollination).

It is not known whether any or all of the new dek mutants were produced by the insertion of a Ds element in a dek locus. It is important in evaluating these data to keep in mind that although the kernels planted in the spring of 1983 were heterozygous at the bz2-m locus (barring earlier changes as proposed to explain the results obtained with families #10 and #16), only one-half of such kernels would contain a single dose of Ac while the other half would lack Ac. Consequently, only that half of the total number of plants which carry Ac could be expected to express mutability in a new dek mutant as well as at the bz2 locus. Some transposition events would most likely result in self-pollinated ears with only a sector of dek kernels rather than a fully segregating ear. Although a detailed scrutiny remains to be done, the preliminary examination of all of the 3,415 ears described above has not identified any sectored ears.

Among the 4,944 plants, there were four plants identified as possible dominant mutants. These were found in three families (#5, #9, and #18) and all four have the same phenotype. They had yellow and green striped leaves either on the top three or four leaves of the main stem (two cases), or on the tillers only (two cases). Future tests are planned to determine the mutability of the new dek mutants, their allelism, and whether they are responsive to Ac. The yellow and green striped plants were outcrossed onto early stocks and the resulting progeny will be examined for this trait.

W. F. Sheridan

Availability of Black Mexican sweet corn and several early Northern flints

During the past year, I have received several requests for kernels of Black Mexican sweet corn, as well as inquiries as to where it might be obtained in large quantities. I have not been able to locate a commercial supplier of this variety. Because Black Mexican sweet will grow as a suspension culture as well as for other reasons, a readily available supply of this material is needed.

I plan to grow a good amount of pedigreed Black Mexican sweet in my experimental field this summer. The material will be propagated by hand pollination and pedigrees will be maintained. I will be happy to share this material with other cooperators. Businesses should include with their request a contribution to help defray the costs of propagation, handling, and mailing at the rate of 10 cents per kernel for samples of more than 50 kernels. All requests will be filled in October contingent upon satisfactory growth and harvest conditions.

The Red River valley has good soil and climatic conditions for growing early Northern flints as well as Black Mexican sweet corn. The Northern flints available include such standards as Longfellow, Parkers, Gaspe, and Tama as well as several Canadian flints. Also available are a number of flints grown by the Indians of the Dakotas and northern Minnesota including Mandan yellow, Mandan white, Rhee and Bear Island Chippewa. Other early northern varieties including Ivory King, Shoshoni, Gehu, and others are available. Write for a detailed listing. Requests for the above early materials will be filled on the same basis as for Black Mexican sweet corn.

William F. Sheridan

Morphological studies on five embryo-lethal mutants

Five embryo-lethal mutants of maize were examined from early in kernel development when they first became distinguishable through later stages until maturity using North Dakota grown material. Mutant and normal kernels were fixed and examined by histological techniques. Three of the mutants (E747B, E1130, E1418) did not form leaf primordia during embryo development while one (E1425A) did so rarely. The fifth mutant, E1311C, formed several leaf primordia by kernel maturity but was unable to germinate when cultured or when mature mutant kernels were planted.

All of the mutants were blocked at a specific stage in embryonic development, except E1425A which was variable. The mutant E747B developed normally to the coleoptilar stage, forming a shoot apical meristem and an embryonic axis. The embryo subsequently underwent unorganized proliferation and necrosis. By kernel maturity, no organized structures were apparent.

Embryogenesis in the mutant E1130 began with the formation of a fairly normal transition stage embryo. However, the mutant embryos did not proceed to form either shoot or root apical meristems. At maturity, the embryos still retained a transition stage shape, but were larger than normal with vacuolated cells, and a wrinkled appearance.

The embryos of E1418 began their development normally. However, after the transition stage, they elongated and formed a root apical meristem and in some cases a rudimentary shoot apical meristem. Then the shoot apical meristem stopped developing while the root apical meristem continued development. In one mature mutant embryo, a necrotic area on the face of the embryo marked where the shoot apical meristem would normally be located.

Embryos of E1425A were variable in their development. In some cases, the embryos were small, necrotic and malformed. In other cases, they appeared to be more normal, even to the extent of leaf primordia formation in one case. The

variability was seen in kernels from the same ear as well as between ears. One possible explanation for this phenomenon is the existence of a modifier gene for the expression of E1425A.

The last mutant, E1311C, was normal throughout embryogenesis except that its development was slowed and it was smaller at maturity. In addition, the mature embryo axis was bent at an abnormal angle, resulting in the misalignment of the shoot and root apical meristems.

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The dek mutants - new mutants defective in kernel development

We are continuing the characterization of a large collection of EMS induced defective kernel (dek) mutants. All mutants of this type are defective in both their embryo and endosperm development. Most of these mutants are lethal because of inviability when tested as mature kernels, but a few have a mutant seedling phenotype. The pleiotropic nature of the defective kernel mutants is also expressed, in some cases, by an alteration of the carotenoid and anthocyanin synthesis in the kernels. The culturing of immature mutant embryos has revealed a wide range of mutant seedling phenotypes, as well as the occurrence of normal appearing seedlings in some cases. Our previous note (Neuffer and Sheridan, MNL 55:29-30, 1981) reported on the characteristics of 15 dek mutants and assigned dek numbers to each of them. This list is expanded below to include 21 mutants representing all of the chromosome arms except 7S and 8S. Since our published reports (Neuffer and Sheridan, Genetics 95:929-944, 1980; Sheridan and Neuffer, Genetics 95:945-960, 1980 and J. Heredity 73:318-329, 1982) referred to these mutants by their E number, a laboratory designation, this number is included in the Table.

Symbol	E No.	Chrom. arm	Mature kernel pheno.	Lethality or Seedling Phenotype*	Culture Phenotype*	Carot. synth.**	Antho. synth.
dek1	792	1S	clf	L	(not germ)	B	B
dek2	1315A	1L	dsc	L	gr	N	?
dek3	1289	2S	gm	L	w-gs	N	?
dek4	1024A	2L	cp fl	L	gr-nl	N	N
dek5	874A	3S	sh	w-gs	w-gs	N	N
dek6	627D	3L	o sh	L	n	N	N
dek7	211C	4S	sh su	gs	w-gs	N	N
dek8	1156A	4L	sh	?	gr-sml	N	?
dek9	1365	5L	crp	L	(not germ)	dil	dil
dek10	1176A	4L	cp	L	gr-crl-stb	N	N
dek11	788	7L	et	L	w,gr	N	N
dek12	873	9S	cp	L	gr-nl-crl	N	N
dek13	744	9L	de o	L	pg-gs	N	N
dek14	1435	10S	cp	L	yg	N	N
dek15	1427A	10L	cp fl	L	gr	N	N
dek16	1414	2L	fl	L	n	N	?
dek17	330D	3L	cp	L	(not germ)	N	N
dek18	931A	5S	cp	L	gr-nl	N	N
dek19	1296A	6L	cp o	L	gr	N	?
dek20	1392A	8L	cp	L	gr	N	?
dek21	1330	10L	msc	L	w	dil	mosaic

* w, white; gs, green striped; gr, green; nl, narrow leaf; n, normal; sml, small; crl, curled; stb, stubby; pg, pale green; yg, yellow green.

** B, blocked; N, normal; dil, dilute; ?, undetermined

Efforts are underway or are soon to be initiated for mapping these mutants. The mutants have been transferred from the original genetic stocks in which they were isolated to a large-embryo strain (Alexander's high oil), and some of them have been transferred to Black Mexican sweet corn as well as to an early maturing genetic stock. We will be pleased to share these mutants with other investigators.

William F. Sheridan, Ming Tang Chang and M. Gerald Neuffer

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Grassy tillers, gt, tentatively located on the short arm of chromosome one

A line homozygous for grassy tillers was crossed to the waxy reciprocal translocation series in 1981. The segregating progenies were grown in Grinnell, Iowa in 1983. Zero grassy tiller plants were observed among 30 waxy segregates from the cross gt gt x wx T1-9c (1S.48; 9L.22), thus indicating that gt is located on the short arm of chromosome one. The lowest number of gt gt plants observed in any other progeny was 5 out of 30. This observation is tentative for a number of reasons. Due to unfavorable climatic conditions, fewer than the desired number of plants grew to a readable stage. More importantly, grassy tillers is known to vary widely in penetrance (Tracy and Everett, MNL 56:77-78) and this may be a factor even though wx T1-9c has the same background as most of the other wx translocations used in this experiment. Further testing will be carried out to confirm these results. (Thanks to Paul Sisco for making the original gt gt x wx translocation crosses.)

W. F. Tracy

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Studies on growth pattern in root callus cultures

Callus was initiated from one-week-old seedling roots on Linsmaier and Skoog's medium with 2,4-dichlorophenoxyacetic acid (2,4-D). Growth pattern was compared in haploid and diploid root callus cultures. Haploids were selected from crosses of C x C-I stocks. Growth was assessed by fresh and dry weight analysis after a month. Haploid callus growth was slow (0.5 + 0.05 g) compared to diploid (1.01 + 0.10 g). Rhizogenetic ability was greater in haploid calli as evidenced by profuse rooting, especially in suspension cultures under similar conditions. Diploid root was found to be superior over haploid for callus initiation and growth.

Effects of growth regulators GA3 and abscisic acid (ABA) were also studied using diploid root cultures. Callus was initiated from roots of one-week-old seedlings on LS media with 2,4-D plus different concentrations of either GA3 (10-40 mg/l) or ABA (1-4 mg/l). After one month, the fresh and dry weights of calli were analyzed. With increasing concentrations of GA3 and ABA, a decrease in callus growth was observed compared to control. It may be suggested that these growth regulators influence the growth of root callus cultures.

P. Suprasanna, K. V. Rao and G. M. Reddy

Cross feeding and pigment synthesis in root cultures

In view of the several advantages of somatic cell genetics, a study of genetically defined marker systems with well understood biosynthetic pathways like anthocyanin synthesis in maize may help in understanding the mechanisms of gene action at a cellular level with different explant tissues. Among the different explants used for initiating callus cultures, seedling root was found to be more efficient and was used in the present study. Callus cultures were derived from seedling root of anthocyanin genotypes on LS medium with 2,4-D. After a few subcultures, a few sectors of the calli and the regenerated roots exhibited pigment formation. The callus/root extracts of different genotypes in 1% methanolic HCl gave absorption maxima of 530 nm in the Pr genotype, 520 nm in the pr genotype, and 210 and 260 nm in the c2 genotype, suggesting that the accumulation of pigments may be cyanidin, pelargonidin and cinnamic acid respectively.

To gain insight into the gene-controlled pathway of anthocyanin biosynthesis, cross-feeding studies were conducted with root cultures of certain genotypes. Various substances like cinnamic acid, coumaric acid, caffeic acid, naringenin and quercetin were supplemented to the medium, and seedling roots of different genotypes were callused.

Immature kernels of C-I were allowed to germinate in the presence of caffeic acid supplemented medium. Roots were dissected out from such seedlings and callus was initiated on LS medium with 2,4-D. After a month, root calli were transferred to LS basal medium. Purple pigment was observed in the regenerated roots, suggesting that C-I may utilize caffeic acid in the synthesis of anthocyanin. Further studies with C-I and other mutants are in progress.

K. V. Rao, P. Suprasanna and G. M. Reddy

Maize x sorghum hybridization

Although all reported attempts to date to hybridize maize with sorghum have failed, the results do offer a certain element of encouragement. Any attempt to produce a fertile hybrid that can serve as a bridge to the exchange of germplasm between these two important crop species is worth trying.

Maize and sorghum materials used in the hybridization program are listed in the Table. Maize stocks used as females were planted in isolation from their male counterparts and were detasseled as a measure of reducing contamination from stray pollen. Also, male sterile sorghums were isolated from male fertile sorghums. A total of 923 reciprocal controlled pollinations (480 maize x male fertile sorghum and 443 male sterile sorghum x maize) were made by conventional methods. In general, silks were shortened. Salicylic acid (10^{-2} M) solution was sprayed on male sterile sorghum heads and maize silks.

MAIZE	SORGHUM
SC 410[a]	MALE STERILE
SC 440[a]	CK 60 A[d]
SC 444[a]	2077 A[d]
SC 507[a]	2219 A[d]
SC 6875[a]	MALE FERTILE
Ganga 5[b]	CSH-1[d]
Sweet corn[b]	CSH-5[d]
DHM 101[b]	CSH-6[d]
DHM 103[b]	CSH-9[d]
EH-400175[b]	CK 60 B[d]
EH-428773[b]	2219 B[d]
AA_2Pr[c]	Sorghum halepense[e]
AA_2CRbpl[c]	S.arundinaceum[e]
pr[c]	S.almum[e]
bz_1[c]	S.verticillifolium[e]

a From Pioneer Seed Company Ltd., Hyderabad.
b From Maize Research Station, Hyderabad.
c From Genetics Department Stocks.
d From AICSIP(IARI-RRS), Hyderabad.
e From ICRISAT, Hyderabad.

A total of 67 'potential' hybrid seeds (46 from maize female and 21 from sorghum female) were recovered. Approximately 60% of these seeds have been transplanted into clay pots. With the exception of two, all of those showing near normal growth turned out to be parthenogens or contaminants. The two 'potential' plants (SC 440 female x CSH-5 male) have reached maturity with slow growth and were morphologically more similar to maize than to sorghum. None of them gave viable pollen. Further work on the production of maize x sorghum hybrids with suitable genetic markers is in progress.

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Chromosome linkage study of the Rf4 locus

Restoration of the cms-C group of cytoplasms has most recently been shown to be under the control of a single dominant gene, designated Rf4 (Kheyr-Pour et al., Genetics 98:379-388, 1981), although previous work indicated the presence of three or more Rf factors, designated Rf4, Rf5 and Rf6 (Josephson et al., Proc. Ann. Corn Sorghum Res. Conf. 33:48-59, 1978). The fertility response is sporophytic in nature, with all pollen grains from a heterozygote being fully normal.

A series of crosses was made using two different cms-C fertile lines (A619 and NY821LERf), and a series of wx translocation stocks which had been converted to cms-C sterility to detect any possible linkage between the Rf4 locus and a particular wx translocation.

It was hoped that the restorer locus could be mapped to a single chromosome, further reinforcing the recent evidence that a single gene controls this trait.

The series of wx translocations listed in Table 1 was obtained from the Cooperation seed stock and crossed onto an Oh51A cms-C sterile line. Semi-sterile ears indicating presence of the translocation were planted, selfed and the progeny selected for both semi-sterility and segregation of the wx gene. The wx kernels were planted, and sterile plants were paircrossed onto three different cms-C cytoplasm lines:

1) C x A619 (Rf4 Rf4)
2) C x NY821LERf (Rf4 Rf4)
3) C x NYD410 (rf4 rf4)

Rows of the NYD410 crosses were planted adjacent to the paired A619 or NY821LERf cross as a check on the cms-C sterility of the wx translocation stock. If the NYD410 paircross was fertile, that wx translocation paircross was discarded. The remaining wx translocation paircrosses were selfed, and only the wx kernels from semi-sterile ears were planted. The plants were scored for sterility of the tassels, with the few occurring late breaks included in the sterile category as this is a recurring problem in cms-C steriles. Any cross exhibiting >25% sterility (nonlinkage gives a 3:1 fertile:sterile ratio) was indicative of linkage of the Rf4 gene to that particular translocation since the wx T stocks were rf4 rf4. Table 1 indicates the results of these crosses and the associated P values assuming a 3:1 ratio.

There are four crosses which differ significantly (P = 0.05 and P = 0.01) from a 3:1 ratio. Three of them (Nos. 2, 5 and 9), which exhibit a much higher degree of fertility than expected, can be explained by misclassification of wx kernels or small sample size. The cross of interest is #12, T8-9(6673), which has a much higher proportion of steriles, suggesting that Rf4 is on chromosome 8. The other

TABLE 1. X^2 values for fertility of translocations.

Cross no.	wx translocation	Observed	Expected	X^2	P value
1	1-9c	44[a]	44	0.00	1.00
		15[b]	15		
2	1-9^{8389}	56	48	5.33*	0.02
		8	16		
3	2-9b	51	49	0.33	0.57
		14	16		
4	2-9d	40	46	3.18	0.075
		21	15		
5	3-9c	59	51	5.01*	0.025
		9	17		
6	4-9g	48	48	0.00	1.00
		16	16		
7	4-9^{5657}	34	37	0.99	0.32
		15	12		
8	5-9a	49	46	0.80	0.37
		12	15		
9	6-9b	48	39	8.31**	0.004
		4	13		
10	7-9a	42	43	0.09	0.76
		16	15		
11	8-9d	48	42	3.43	0.06
		8	14		
12	8-9^{6673}	23	46	44.56**	0.001
		39	16		
13	9-10b	45	47	0.34	0.56
		18	16		

[a]number of fertile plants
[b]number of sterile plants
*significant at P=0.05 level
**significant at P=0.01 level

translocation involving chromosome 8, T8-9d, shows no evidence of linkage, so the Rf4 locus must be located some distance away from the breakpoint in T8-9d. The reported breakpoints of the two translocations are:

T8-9d:	8L.09	9S.16
T8-9(6673):	8L.35	9S.31

Therefore, the Rf4 locus appears to be on the long arm of chromosome 8, apparently near the end of the chromosome since there is no linkage to the wx gene evident near the 8L.09 breakpoint translocation, and the number of fertiles in the T8-9(6673) cross precludes a tight linkage at the 8L.35 breakpoint, which would be typified by very few fertile plants.

Additional studies to confirm the location of Rf4 on the long arm of chromosome 8 are needed.

Alice Johnson

Iojap cytoplasmic male steriles

Male sterile plants from iojap crosses were characterized on the basis of agarose gel electrophoresis of mitochondrial DNA, cms restoration patterns, and injection of Helminthosporium maydis Race T toxin. Both cms-S and cms-T male sterile plants were isolated from these crosses. The cms-S sterile line was derived from a single (R181 x ij ij) F2 plant. The cms-T sterile lines were found in the genotype (W182BN x ij ij) x W182BN BC1. Additional crosses were made in

the 1983 nursery between iojap males (pollinators) to the following non-restorers of cms as females: SD10, C0107, R181, Oh51A, RD4501, W182E, MS71, RD5502, and RD6501. The progeny from these crosses will be studied to look for new cms steriles. All steriles will be further characterized by restriction endonuclease analysis and stability of the cms trait under field conditions.

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Rapid monitoring of purity in seed lots of hybrid maize: Modifications of current technologies

The basic methodologies of starch gel electrophoresis of maize isoenzymes have been outlined by Cardy et al. (NCSU mimeo 1317; 1317 rev.). In contrast to the requirements for initial genotyping of lines (relatively few plants run over at least 4 pH systems involving 24 or more isoenzyme loci), the monitoring of hybrid seed lots for contamination with female selfed and other rogue seed necessitates the electrophoretic separation of isozyme extracts from a large number of plants over a reduced number of enzyme loci. Variants showing obvious electrophoretic migration differences between male and female lines of a hybrid can be used to detect rogue seed. Coleoptile segments from 5-day seedlings can be used as the source of enzyme. In addition, embryo samples harvested prior to physiological maturity (2-6 weeks following pollination) can also be used as a source of enzyme extract for many hybrids (Smith, S. in review).

We have made modifications to the methods described by Cardy et al. that allow enzyme extracts from large numbers of plants to be assayed with efficient use of time, economic and labor resources. Modifications involve changes in gel dimensions, reduction in electrode buffer volume, changes in sample grinding procedures, alterations in sample application, use of additional isoenzyme stains, increased flexibility in staining protocol, and use of tissue from developing embryos rather than seedling coleoptile sections when data are required prior to the regular harvest date. These modifications, listed below, allow two full-time technicians to run routinely 2000 individual plants per week (20 samples each of 100 plants) for tests of hybrid versus female selfed seed. Approximately 125 plants can be cut and ground per hour by only one person. These modifications also allow 1200 plants per week to be assayed for other rogue, including outcrossed, genotypes.

Modifications (Gel systems A, B, C, and D are those described by Cardy et al.; slight modifications in pH may be required to obtain best resolution):

1. Gel size i) 23 cm long x 23 cm wide x 1 cm deep; volume 670 ml (B and D gel systems).
   ii) 13 cm long x 23 cm wide x 1 cm deep; volume 550 ml (C gel system). NOTE: C gels cannot be double-stacked as gel shrinkage occurs causing gaps to appear along the upper line of sample application.
2. Electrode tray volume 325 ml (platinum wires are fixed to the tray with banana plug connections to the power cables in order to prevent breakage of soldered connections).
3. Grinding process (leaves not removed unless for GLU1) - multi-plant grinder allows 5 individual plants to be ground simultaneously.
4. Sample application - reduced sample wick size (M. C. Mieth punch #448) and application of samples in 2 parallel rows of 50 to give 100 sample wicks per gel.

5. Additional enzyme systems that can be used, gels (recipes for the pH 7.0 and pH 7.4 systems are given at the end of this script), and tissues:

Diaphorase	C, 7.0	embryo, coleoptile
ACP4	B	embryo (Kahler, J. Hered. 74:239)
LAP	D	embryo
G2DH	pH 7.4	embryo
Aconitase	pH 7.0	coleoptile
Menadione reductase	pH 7.0	coleoptile
Hexokinase	C, D, 7.0	embryo

6. Increased combination of stains that can be used on gels of different pH thereby maximizing the possibility of staining for two or more loci with the minimum number of gels (NOTE: "A" gel needs very rarely to be used):

B	C	D
ACPH	GOT	PGD MDH
GLU	CAT	PHI ADH
MDH	ENP	IDH ACPH
PGD	ADH	CAT PGM
	DIA	

(PHI may have migrating and staining difficulties in some locations)

7. Use of embryo tissue as source of extract. Developing embryos from 2-6 weeks following pollination can be used adopting the techniques described by Cardy et al. with the modifications outlined herein. GLU1 and CAT3 are not active, and ACP1 cannot routinely be clearly resolved from embryo tissue. However, other additional enzyme stains can be used with embryo extracts (see 5 above). More complete details are presented by Smith, S. (in review).

Buffer recipes:

i) pH 7.0 Tris-citrate system - electrode buffer, 16.4 g Trizma base, 9.1 g approx. citric acid (adjust to pH 7.0), 1 liter water; gel buffer, 1:14 dilution of electrode buffer.

ii) pH 7.4 Tris-maleic system - electrode buffer, 12.4 g Trizma base, 4.1 g approx. maleic acid (adjust to pH 7.4), 3.7 g Na_2 EDTA·$2H_2O$, 2.0 g $MgCl_2$·6H2O, 1 liter water; gel buffer, 1:10 dilution of electrode buffer.

Our extensive experience in running electrophoretic tests of seed purity in maize leads us to make the following additional comments. Some constraints apply with regard to the use of some loci in certain circumstances when testing for rogue genotypes. These are the difficulties of detecting migration differences between the alleles Glu1-6 and Glu1-7 and between adjacently migrating variants of the E8 locus. Also the locus Mmm can only be used to detect selfed female plants when the female line is homozygous Mmm-mmm and the male line is homozygous Mmm-MMM, since the heterozygous genotype cannot be distinguished from the homozygous Mmm-MMM. Other constraints are due to alleles encoded by different loci which have an overlapping distribution. Thus, Mdh4-12 obscures the contribution of Mdh5-12, and likewise Mdh2-6 obscures Mdh1-6.

If possible, at least two loci that reveal obvious differences between male and female parents should be used to detect female selfs, thus alleviating most interpretational problems caused by any residual segregation at one or more loci. This rule applies to all hybrids whether they be single, 3-way, 4-way, or modified crosses. In those instances where segregation is seen to cause interpretational problems, 50-100 individuals from parental seed lots should be assayed in order to provide information on levels of segregation that could be expected in parental lots and which might not therefore be considered a problem in hybrid seed purity. Segregation information should be shared with breeders in order to ascertain, in particular, whether the observed levels of segregation are to be expected for each parent in question. Identification of possible outcrosses requires that a greater

number of loci be assayed, since in most instances the genotype of contaminating pollen is unknown. An additional complication in the identification of outcrossed individuals is that the alien pollen may be from segregating genotypes, thereby causing the genotypes of outcrossed female plants to differ one from another. Thus, the combined effects of outcrossing and segregation within parental seed lots can confound any interpretation of the causes of genotypic variation which may be recorded within hybrid seed lots. However, even when outcrossing is suspected, it is usually unnecessary to assay Cat3, Enp1, Got1, Got2, and Adh1 since these loci reveal relatively little variation across Canadian and U.S. commercial maize germplasm (Cardy and Kannenberg, Crop Sci. 22:1016-1020; Stuber and Goodman, USDA-ARR-S-16; Smith, S., Crop Sci. in review). Little or no variation is also revealed by Mdh4, Mdh5, Mmm and Pgm1. However, other loci of these same enzyme systems do reveal considerable variability.

S. Smith and H. Weissinger

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Production of maize pollen embryoids and the influence of some factors on their frequency of induction

Since 1975 there have been several reports on the induction of haploid plants from cultured anthers. It appears from the literature that information on the factors which control induction of microspore callus followed by embryoid formation, and their cytological behavior, is still fragmentary. The present communication represents some of the positive factors which induce and control microspore de-differentiation and cytological aspects of pollen callus culture.

Zea mays cultivars (compositae and adecuba) were grown to maturity under field conditions. Standard anther culture techniques were applied for inoculating anthers to the basal medium. The basal medium used for anther culture was modified N6. Flower sizes containing pollen grains in the mid to late uninucleate stages were correlated by extensive cytological studies of the anthers using 1% acetocarmine in a drop of ferric acetate. Anthers containing mid to late uninucleate stages were dissected and aseptically inoculated in the medium. Different factors for induction of haploidy so far studied are as follows:

1. Effect of sucrose: Sucrose concentration was found to play an important role for increasing induction frequency in maize anther culture. In order to find the optimal sucrose concentration for callus and embryoid production, four levels of sucrose concentrations (3%, 5%, 9% and 12%) were tested. Of these the 12% level was found to be optimal for the induction of pollen embryoids in both of the cultivars. Maximum induction frequency at this concentration reached up to 2.4%.
2. Effect of cold pretreatment: Cold pretreatment of the flower buds, at 0 C to 7 C for one to four days before excision and culture to the nutrient medium, was found to increase induction frequency. Best success was obtained when the flower buds were pretreated at 7 C for 4 days; the induction frequency increased up to ten times higher than the control.
3. Effect of growth regulators: Auxin (2,4-D), cytokinin (kinetin) and organic component (casein hydrolysate) were tested in various combinations and concentrations along with the basal medium. Effects of these growth regulators for increasing induction frequency are shown in Table 1.

Table 1. Comparison of the effect of different growth regulators on the induction frequency of callus or embryoids.

Medium	No. of anthers inoculated		No. of callus or embryoid produced		Induction frequency (%)	
	Adecuba	Compositae	Adecuba	Compositae	Adecuba	Compositae
1. N_6	980	950	-	-	-	-
2. N_6+2,4-D(1 mg/l)	1040	1120	-	-	-	-
3. N_6+2,4-D(2 mg/l)	1200	1180	18	3	1.5	0.25
4. N_6+2,4-D(4 mg/l)	1270	1320	12	21	.94	1.59
5. N_6+2,4-D(2 mg/l)+Kn(1 mg/l)	1210	1050	22	12	1.81	1.14
6. N_6+2,4-D(4 mg/l)+Kn(1 mg/l)	1120	1235	18	22	1.60	1.78
7. N_6+2,4-D(2 mg/l)+Kn(1 mg/l) +CH(500 mg/l).	1130	1240	26	20	2.30	1.61
8. N_6+2,4-D(4 mg/l)+Kn(1 mg/l) +CH(500 mg/l).	1225	1250	23	30	1.87	2.40

Medium 1-8 all contains N_6+12% sucrose.

Cytological status of the anther derived callus tissues was also examined. Cytological observations revealed mixoploid chromosome numbers with cells containing haploid chromosome complements predominant. Callus cells contained an average of 52% haploid, 26% diploid, and 22% mixoploid, containing haploid, diploid and aneuploid chromosome complements.

Nitai Kumar Paul and Parthadeb Ghosh

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Maize: A proposal for a new model experimental system

Successful application of tissue culture technology provides powerful tools to supplement conventional breeding methods in crop improvement. However, such success depends not only upon the ability to regenerate plants in vitro but also upon the ability to grow our plants in vitro as model systems. Cells cultured in vitro as calli are able to regenerate plants in two ways: by a sequential differentiation of shoots and roots which by association with each other form plants, or by somatic (nonzygotic) embryogenesis. In the first case, organogenesis, shoot and root meristems are multicellular in origin. On the other hand, somatic embryogenesis arises apparently from single cells. The establishment of a more or less dedifferentiated tissue culture under defined culture conditions gives rise to a subsequent regeneration of plants which frequently show phenotypic alterations. Plants of multicellular origin cannot always be expected to be genetically uniform. However, plants obtained through somatic embryogenesis are suitable for mutant research, genetic analysis, maintenance of genetic stocks, and breeding (Vasil, I. K., Cell & Tiss. Cult. Tech. for Cereal Crop Improv., 131-144, 1983).

In maize, it is now possible to obtain plants either through organogenesis or from somatic embryogenesis. In our work, the primary genotypes used included several floury-a inbred lines, red flint inbred lines and hybrids between them. The experiments were carried out in the IFSC (Buenos Aires, Argentina) during the spring of 1983. Ears were removed from greenhouse-grown plants at 12 to 18 days after pollination. The kernels were taken from the middle part of the ears and then were sterilized for 20 min in 2.5% sodium hypochlorite solution and rinsed twice (5 min each) with sterile water before culture. Immature embryos were isolated from the kernels and were then placed on the solid culture medium with the plumule-radicle axis in contact with the medium and the scutellar side exposed. The primary cultures were incubated at 28 C in the dark.

Calli were initiated on three different media:

Medium A: Inorganic components of Murashige-Skoog, organic components of Straus, plus 20 g sucrose/liter, 8 g agar/liter and 1 mg 2,4-D/liter.

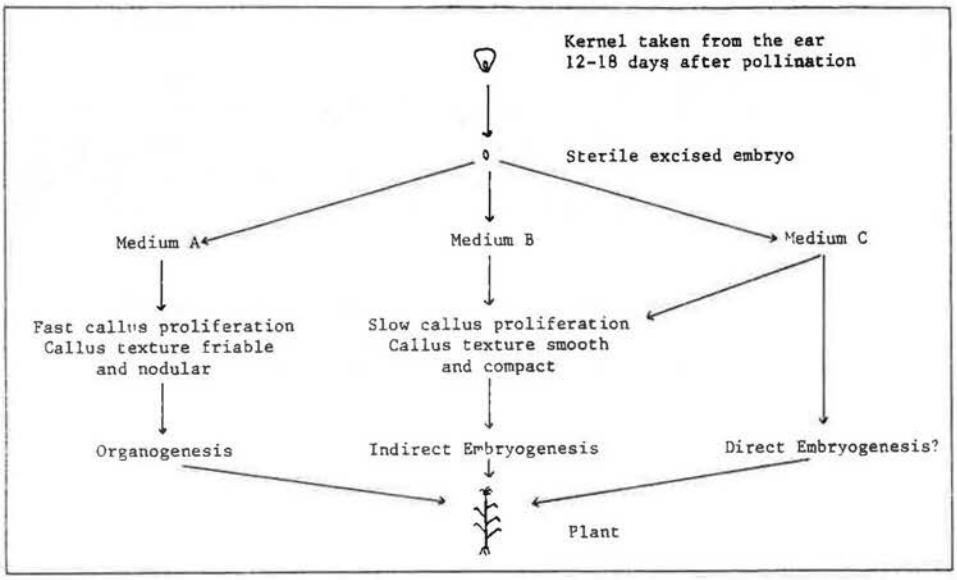
Medium B: Medium A plus 120 g sucrose/liter, 400 mg proline/liter, 8 g agar/liter and 1 mg 2,4-D/liter.

Medium C: Yu-pei medium plus 120 g sucrose/liter, 400 mg proline/liter, 500 mg casein hydrolysate/liter, 8 g agar/liter and 1 mg 2,4-D/liter.

The frequency of positive culture response from immature embryos of all genotypes ranged from 60 to 100%. Calli were white to pale yellow in coloration. However, the texture of the calli obtained in the three media differed markedly. The variation of the texture of the calli among media was markedly higher than the variation among genotypes in each medium.

Medium A is the common medium developed by Green and Phillips and used by several authors to culture maize in vitro. Calli obtained in such medium were friable and nodular. After 30 days, these primary cultures developed 1 to 1.5 cm good callus masses. Medium B is a variation of medium A, with a high osmotic concentration and proline level. Medium C is a medium developed by Chinese scientists to obtain androgenesis in maize. The textures of the calli obtained in medium B and C were highly similar to each other. Culture initiation began with enlargement of the scutellar surface, which resulted in a very prominent dome-shaped, compact and smooth scutellum, with continued growth. However, these scutellum calli obtained in medium B and C proliferated more slowly than calli obtained in medium A.

Figure 1



The morphogenetic events observed in the three media were different (Rapela, this MNL). These facts let us suppose that it is possible in our material to induce both organogenesis and embryogenesis with an adequate change of the medium constituents. Also we will introduce evidence of direct embryogenesis in some cultures obtained in medium C. We suggest for the maize material cultured under our conditions a tissue culture model experimental system as shown in Figure 1.

Miguel Angel Rapela

SEM characterization of an organogenic callus culture of maize

Excised immature embryos placed on tissue culture initiation medium A (see previous article) showed, after one month of culture, an extensive callus proliferation from the scutellum. In previous observations we have noticed that when such cultures were placed on medium with 0.5 mg 2,4-D/liter or without 2,4-D, some morphogenic events occur. In order to examine such cultures, the calli were fixed in 2.5% glutaraldehyde in 0.1 M phosphate buffer and then rinsed with buffer. After acetone dehydration, the samples were critical point dried and coated with gold-palladium, and scanned at 15 KL on a Jeol JSM-U3 Scanning Electron Microscope (SEM).

Although tissue cultures appeared to the naked eye to grow as true callus, SEM examination showed this growth to be a mix of isodiametric and elongated cells (Fig. 1--see opposite page). Both unorganized and organized callus cells appeared to be the first step of culture initiation observed in our material. As Springer et al. (Protoplasma 101:269-281, 1979) and Mott and Cure (Physiol. Plant 43:139-145, 1978) pointed out, maize callus cultures obtained through either immature embryos or excised mature embryos in basal media with 2,4-D are not composed of uniform or undifferentiated cells. We have observed in our cultures variations in cell size, shape and cell wall characteristics (Fig. 2). Extensive regions of the callus surface also showed a cuticular wax-like surface (Fig. 1).

On the 20th day of culture, large regions of callus surface exhibited morphological modifications. The occurrence of specialized growing points appeared to be the second developmental step in our primary cultures. Numerous primordia-like protuberances appeared which could eventually develop into roots or shoots (Fig. 3). Numerous growing points continued to appear at the end of one month of culture.

Random differentiation of xylem occurs in primary cultures (Fig. 4). Also, distinct xylem development and secondary wall growth occur in the primordia-like protuberances (Fig. 5). We have not observed embryogenesis in the callus cultures obtained in medium A. All the morphogenic events observed appeared to be organogenic in origin.

The observations made here indicate a multicellular origin of these phenomena (Fig. 6). The regeneration of plants showing higher somaclonal variation (Larkin and Scowcroft, TAG, 60:197-214, 1981) could be explained in part by a multicellular origin of such plants. This fact would be very important for increasing the genetic variability necessary to any plant breeding enterprise.

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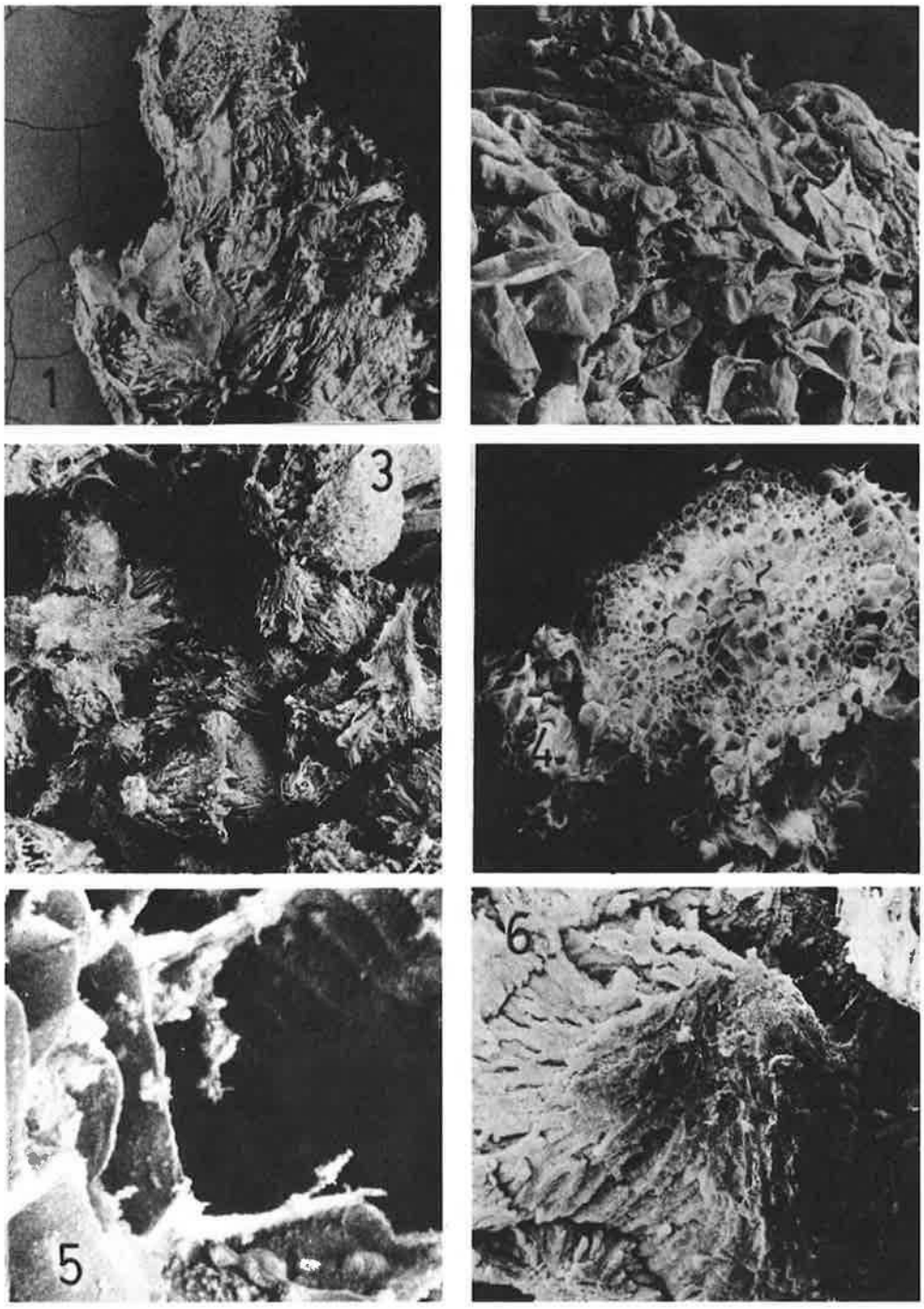


Legends for Figures

Figs. 1 - 6. Organogenic events in maize callus cultures in medium A. Fig. 1. Callus with isodiametric and elongated cells and waxy cuticle (x50). Fig. 2. Appearance of callus surface cells (x250). Fig. 3. Appearance of primordia-like protuberances (x100). Fig. 4. Random differentiation of xylem (x150). Fig. 5. Xylem development and secondary wall growth (x2000). Fig. 6. Multicellular origin of the primordia-like protuberances (x100).

Indirect somatic (nonzygotic) embryogenesis in tissue cultures of maize

Excised immature embryos, placed with the scutellum side exposed to air on tissue culture initiation medium B and medium C (see previous article), one week after the initiation of cultures showed proliferating callus tissue at the periphery of the scutellum. Such tissue could be seen in all of the periphery of the scutellum in contact with the medium, and occasionally on the exposed side of the scutellum. However, good callus proliferation was seen on the underside surface of the scutellum. The callus formed was compact, smooth and white (embryogenic callus tissue). Friable and nodular callus (organogenic callus tissue), as obtained in medium A, was not observed in medium B and medium C. Figures 1-6 show embryogenic events in these studies.

After 10 to 15 days in culture, structures resembling coleoptiles were observed on the underside surface of the scutellum. After 3 weeks, such structures appeared as well organized somatic embryos always showing bilateral symmetry and bipolar structure. As reported by Lu et al. (TAG, 62:109-112, 1982), somatic embryos were formed on the same 2,4-D containing media in which the primary culture was obtained. The level of 2,4-D in the medium did not appear to be involved with the embryoid formation over a large range. Both in media with a high level of 2,4-D (3 mg/liter) and in media with a low level (0.5 mg/liter) we observed the same results. However, both the level of sucrose and the high concentration of proline had a strong effect on embryoid formation and on the callus external aspect. We occasionally observed somatic embryogenesis both in media with a normal level of sucrose (2%) and 400 mg proline/liter and in media with a high level of sucrose (12%) without proline. However, only when high levels of both were put together in the medium did we observe a high frequency of somatic embryogenesis. We do not know the reason for such differential response of the embryos.

Sometimes several embryos germinated. However, germinated embryos also produced embryogenic callus. The embryo germination could be due to the fact that several embryos were larger than 2 mm at the time of culture.

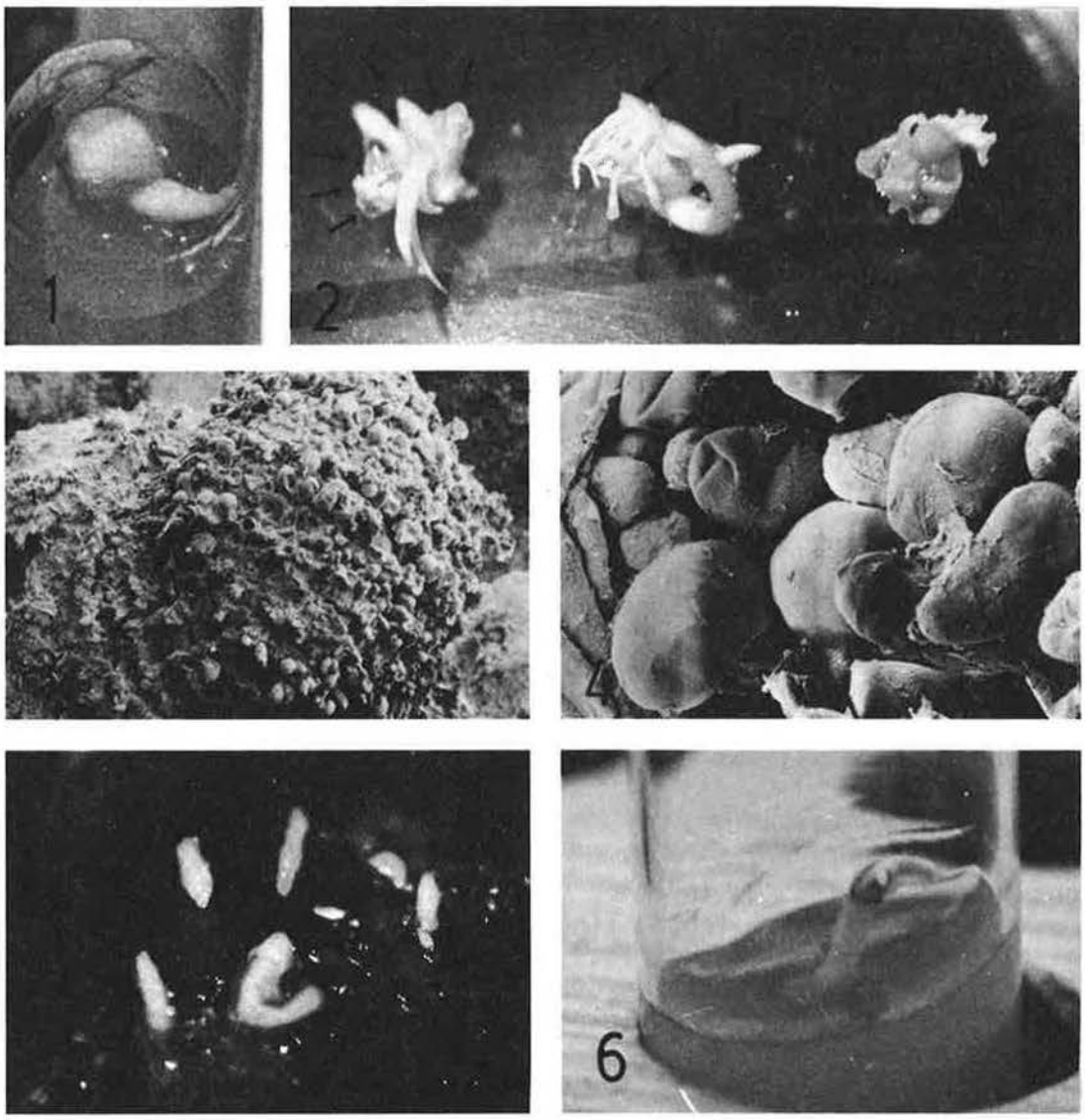
We have detected differences in the percentage of formation of compact and smooth callus tissue and of such callus forming somatic embryos due to the genotype and the effect of the medium (Table 1).

Table 1. 1- Percentage of immature maize embryos forming compact and smooth callus tissue.
2- Percentage of compact and smooth callus tissue forming somatic embryos.

GENOTYPE	MEDIUM B		MEDIUM C	
	1	2	1	2
83-0323 ($f1^a$)	100	50	100	66
83-0324 ($f1^a$)	100	43	100	71
83-0326 ($f1^a$)	83	60	100	80
83-0323 ($f1^a$)x 83-0333 (NRF)*	85	100	100	100
83-0330 (NRF)x 83-0310 ($f1^a$)	80	40	75	66
83-0331 (NRF)x 83-0324 (NRF)	100	25	100	75
83-0345 (NRF)x 83-0337 (NRF)	75	50	100	80

* NRF = Normal Red Flint.

There are great differences in the macro, micro and organic formulation between the MS modified (B) and Yu-pei medium (C). The most important differences are the level of NO_3NH_4, which is 10 times higher in MS than in Yu-pei medium, and the addition of casein hydrolysate to the Yu-pei medium. Only KNO_3, KH_2PO_4,



Legends for Figures

Figs. 1 - 6. Embryogenic events in maize callus cultures in medium B and C. Fig. 1. Immature embryo after 15 days of culture showing smooth and compact callus formation. Fig. 2. Somatic embryos (arrows). Fig. 3. SEM overview of the embryogenic tissue (x100). Fig. 4. SEM close-up of the early stages of somatic embryo formation (x200). Fig. 5. Several somatic embryos excised from the culture. Fig. 6. Germination of a somatic embryo transferred to medium A with 2% sucrose and without 2,4-D. (SEM microphotography in collaboration with Jorge Herkovits).

thiamine-HCl and pyridoxine-HCl are more concentrated in the Yu-pei medium than in the MS modified medium. In addition, MoO_4Na_2, $CoCl_2$, L-asparagine and Ca pantothenate are absent in Yu-pei medium.

As shown in Table 1, we have obtained higher levels of somatic embryogenesis in Yu-pei medium than in MS modified medium. So it would be important to study the effect of some media components on the induction of somatic embryogenesis in maize.

As we have pointed out (Rapela, MNL 57:57-58, 1983), there is also a close relationship between genotype and response to form embryos in culture. The genotype of the donor plant influences both the rate of callus proliferation and somatic embryogenesis induction.

Somatic embryos formed in MS modified medium B and in Yu-pei medium C were transferred to MS medium A without 2,4-D but with 2% sucrose, to induce germination. At the time of writing this communication these embryoids germinated normally.

In our cultures of maize immature embryos on Yu-pei medium C we have observed evidence of direct (nonzygotic) embryogenesis as reported by Conger et al. from mesophyll cells of Dactylis glomerata (Science 221:850-851, 1983). Embryos longer than 2 mm at the time of culture germinated, but such embryos produced embryogenic callus in either medium B or C. Two to 3 weeks before culture initiation the coleoptile of the germinated embryos was 2 to 3 cm long, while the underside surface of the scutellum exhibited embryogenic calli. All the germinated/embryogenic embryos cultured in Yu-pei medium C also showed a probable direct embryo formation on the coleoptile surface. Direct embryo formation began nearer the scutellar node and decreased up to 1 cm from the first point. We have observed between 5 to 15 structures resembling direct embryoids on the surface of each coleoptile that emerged from germinated embryos on medium C. The embryo length ranged from 0.5 to 5 mm and was independent of the position lengthwise on the coleoptile.

Miguel Angel Rapela

Maize anther culture in vitro

In recent years in China, as in other countries, pollen-derived plants of maize have been obtained, from which inbred lines were also developed. After the development of the N6 medium (Chu et al., Sci. Sinica 18:659-668, 1975) for anther culture of rice, and the development of the Yu-pei medium (Ku et al., Proc. Symp. P.T.C., 35-42, 1978) for anther culture of maize, a limited amount of success was obtained for maize anther culture.

For our experiments, we used a medium constituted with the inorganic salts and organic components of Yu-pei medium, complemented with 500 mg casein hydrolysate/liter, 1 g activated charcoal/liter, 250 mg proline/liter, 120 g sucrose/liter, 8 g agar/liter, and 3 mg kinetin/liter. The pH was adjusted to 5.8 with NaOH 0.1 N before autoclaving. With the aim of obtaining embryoids without callus formation, we have not used 2,4-D in the medium.

The detached tassel and surrounding leaves were wrapped in foil and incubated at 5 C for 7 days prior to plating of anthers. After the cold treatment, the anthers were taken from the lower flower of each spikelet located on the middle of the spike. The stage of microspore development was determined using Trypan blue. The anthers were plated at about the middle uninucleate stage on petri dishes and were incubated at 28 C in darkness.

We have examined a narrow range of genotypes for their response to anther culture (Table 1). We used two floury-a and two red flint genotypes, and also anthers derived from Zea diploperennis plants. An embryoid success rate of 3% and 5% were observed in the red flint genotypes.

Table 1

Genotype	% Embryoid formation
83-0310 (floury-a)	0
83-0320 (floury-a)	0
83-0360 (NRF)*	3
83-0363 (NRF)	5
Zea diploperennis	0

* NRF = Normal Red Flint

Our production of embryoids was poor in comparison with the results of other authors. A possible explanation rests in the fact that the anthers used in our experiments were derived from greenhouse plants. Probably, by plating anthers derived from field-grown plants the results would be improved.

Miguel Angel Rapela

Extrachromosomal inheritance

In a summary and bibliographic reference in M.N.L. 52:35-36 (1978), we pointed out the effects in inheritance of a Euchlaena mexicana "prepotent" cytoplasm. This "prepotent" cytoplasm was introduced in the Instituto Fitotecnico de Santa Catalina in 1941, registered under No. 2587 as Euchlaena mexicana, designated as "Florida variety," coming from Huixtla, Mexican city, near the south Guatemala boundary.

Luis B. Mazoti

Genetic instability

Line S+40 multiple dominant A C R Pr B Pl #1877 Randolph (year 1933); produces a low frequency variation: two cases in 30 generations of the gene C to C-Im.

The aforementioned line, crossed by the activator line A C R Pr gl, gives rise to a new line which has increased mutational frequency of C to C-Im and of other characters. This C-Im mutant character segregates with non-mendelian inheritance, in the following generation segregates in a mendelian form, and in the next generation, in some cases, again segregates in a non-mendelian way.

Other heritable variation appears: A to a; R to r; sh extreme; abortive grains, big grains; small grains; crown crinkle grains; aleurone "Cocoa" colour; very intense sunred colour plant. Other confused heritable variations arise: tillering extreme; slender culm; fasciated culm; abnormal ear insertion; filodium in place of leafs and other abnormal developments.

The aforementioned events do not agree with present mathematical genetics and it could be solved with more knowledge of molecular genetics. In spite of the lack of such knowledge, these natural systems may be a useful source of characters in plant breeding.

Luis B. Mazoti

Chromosomal study of F2 hybrid between Zea mays, Zea perennis and Zea diploperennis

In 1982 Zea mays tetraploids (2n=40) were crossed in a greenhouse with the tetraploid hybrid Zea diploperennis x Z. perennis (2n=40) obtaining an annual and fertile hybrid with a chromosome number 2n=40.

F2 hybrid plants already mentioned have a great genetic variability, but in all cases are very prolific and fertile. From chromosomal study of the F2 it has been verified that all the descendants have a chromosome number of 2n=40, and the chromosomes match forming bi- or tetravalents, having practically no mono- or trivalents (Table 1). The average chiasma per cell is 36.94, the average pollen fertility in the population is 95% with a minimum of 93% and a maximum of 99%. In anaphase an equal chromosome number migrates to each pole and there are not any delayed chromosomes or inverted bridges.

Table 1: Meiotic configurations of F_2 the tetraploid hybrid of Z. diploperennis x Z. perennis.

Number of cells	I	II	III	IV	%
39		8		6	25.82
29		4		8	19.20
24		6		7	15.89
24		10		5	15.89
16		2		9	10.59
9		12		4	5.96
4		14		3	2.64
4		16		2	2.64
1	2	15		2	0.66
1	4	14		2	0.66
151	0.03	7.29		6.34	

I: Monovalent; II: bivalent; III: trivalent; IV: tetravalent.

From the F2 hybrid study between Z. mays, Z. diploperennis and Z. perennis it has been verified that, when the hybrid has a chromosome number of 2n=40, there is a high meiotic regularity and fertility in the hybrid and all its descendants.

Maria del Carmen Molina

New hybrid from the species of Zea

From hybrid studies carried out among Zea mays, Z. perennis and Z. diploperennis a hypothesis on fertility and chromosome matching in hybrid species of Zea was reached, that these are determined fundamentally by the chromosome number. When the hybrid has 20 or 40 chromosomes, it will have high fertility and chromosome matching, regardless of the species it is crossed with, whereas when the chromosome number is 2n=30 there remains a high number of unmatched chromosomes, which causes the hybrid to be highly sterile.

In order to demonstrate that if the hybrid has 2n=40 chromosomes it is fertile regardless of the species hybridized, a cross was effected in 1982 between a Z. mays tetraploid (2n=40) which had as a marker the sugary gene, and a hybrid tetraploid of Z. diploperennis x Z. perennis (2n=40), obtaining a new hybrid with the following essentials:

$$\frac{\text{Z. m. tetraploid su su x (Z. d. x Z. p.)}}{\text{F1 = 2n=40 Z. m. t. x (Z. d. x Z. p.)}}$$

The cross was viable in 90% of the cases, all seeds being fertile. The F1 plants are annual and similar morphologically to Z. mays - very prolific, medium tassel, uncovered grain, with large coriaceous glumes.

From the chromosome study it was determined that the chromosomes match as bi- or tetravalents and only exceptionally as mono- or trivalents (Table 1). In anaphase an equal chromosome number migrates to each pole, which results in fertile balanced gametes, and only rarely have inverted bridges been observed. The pollen fertility is 98% and fertile seeds are obtained in a 95% proportion.

Table 1: Meiotic configurations of the tetraploid hybrid of Zea mays x (Z. diploperenni x Z. perennis).

Number of cells	I	II	III	IV	%
41		4		8	31.78
24		6		7	18.60
20		2		9	15.50
15		8		6	11.62
12		10		5	9.30
9		12		4	6.97
2		14		3	1.55
2	2	7		6	1.55
1	2	13		3	0.77
1	2	7		6	0.77
1	2	1		9	0.77
1	1	12	1	3	0.77
129	0.05	5.86	0.007	6.89	

I: monovalent; II: bivalent; III: trivalent; IV: tetravalent.

The results of this work are:

1) The chromosomes of Z. mays, perennis and diploperennis are homologous or homeologous so it could be considered that the three species might have had the same origin, subsequently diverging in some of their genes, which accounts for their morphological and protein differences, but they maintain nevertheless their chromosome homology.

2) Owing to the fact that the chromosomes of the three species of this hybrid have been matched and have a high fertility, it has been demonstrated that if the hybrid has a chromosome number of 2n=40 it is fertile no matter what species of Zea are hybridized.

Maria del Carmen Molina

Cytogenetic study of F2 of tetraploid hybrid of Zea diploperennis by Zea perennis

In 1980 in the Instituto Fitotecnico de Santa Catalina, the species Z. diploperennis (2n=20) was crossed with Z. perennis (2n=40), obtaining two types of perennial hybrids:

1) A triploid hybrid (2n=30), extremely sterile (2% fertile seeds), with an elevated number of monovalent and trivalent chromosomes which caused sterile unbalanced gametes to be produced.

2) A tetraploid hybrid (2n=40) of high fertility and meiotic regularity, which was the product of the matching of an unreduced gamete from Z. diploperennis with a normal gamete of Z. perennis.

From this second hybrid an F2 was obtained, which will be analyzed in this work. The hybrid tetravalent F2 has a chromosome number of 2n=40 in all cases. It is exceedingly tillered, with an average of 75 tillers per plant and a high yield in M.V. and M.S., which makes it a promising material to obtain a new perennial forage.

The F1 is the same as Z. diploperennis and Z. perennis; it has distichous tassels, made up by fruit-bearing capsules which disarticulate when ripe, shedding the fruit. The F2 has separated this character, and a percentage of plants have appeared in which the fruit capsules have soldered, which prevents disarticulation when ripe. In addition a new type of tassel has emerged, double row, the grain being slightly uncovered. This segregation in the F2 indicates that one of the two species has recessive genes which cause the capsules to solder, not loosening when the grain is ripe--a freak due to genetic interaction in both species.

From the chromosome study of the F2, it has been determined that the chromosome number of 2n=40 as well as a high meiotic regularity have been maintained uninterrupted, resulting in a decrease in mono- and trivalents (Table 1). The 151 cells analyzed produced an average of 33.63 chiasmas per cell.

Table 1: Meiotic configuration of F_2 the tetraploid hybrid of Zea mays x (Zea diploperennis x Z. perennis).

Number of cells	I	II	III	IV	%
35		4		8	26.7
28		6		7	21.3
23		2		9	17.5
19		8		6	14.5
18		10		5	13.7
4				10	3.05
2		12		4	1.52
1		16		2	0.76
1		14		3	0.76
131		5.64		7.17	

I:monovalent; II:bivalent; III:trivalent; IV:tetravalent.

In anaphase an equal number of chromosomes migrate to each pole and only rarely were inverted bridges seen. From 40 plants analyzed, the average pollen fertility is 90%, with a maximum of 98.3% and a minimum of 72.3%, the seed fertility being in every case superior to 80%.

From the cytological study of the F1 triploid and tetraploid and the F2 tetraploid, it has been deduced:

a) Fertility and chromosome stability of hybrids are directly influenced by the chromosome number.

b) The chromosomes of Z. diploperennis and Z. perennis are homologous or homeologous since in hybrids they practically match on the whole.

Maria del Carmen Molina

Photosynthetic oxygen evolution of Zea mays leaf slices measured by polarography

A comparative analysis of the photosynthetic activity of Zea mays (flint type) leaf slices was performed using Ullrich's polarographic method (Ullrich et al., Z. Pflanzenphysiol. 79:336-346, 1976) with slight modifications. Maize plants were grown in the field.

After 6 weeks from emergence, a part 35 cm long was excised from the 6th leaf (from the base), delimited by the central vein and a cut made 35 cm from the tip. The bottom of the excised area was put in a glass vial with 2 ml of cold distilled

water and transported to the laboratory in an isolated box kept at low temperature. Rectangles of approximately 1 cm^2, cut 20 cm from the tip, 14-19 mg fresh weight, were briefly washed with distilled water and sliced by hand with razor blades to produce slices of about 1 mm x 10 mm. Slices were submerged for at least 30 min. in 15 ml of cold distilled water (5-10 C) to reach a steady-state respiratory activity. Washing with calcium salts (sulfate, nitrate, chlorhydrate) did not increase O_2 production in light in comparison to control washed with distilled water only. A 400 W (658 u moles $m^{-2}s^{-1}$) Hg-HPL R lamp was used as the light source.

The experimental solution contained 20 m mol Na phosphate (pH 5.6) and 10 m mol $NaHCO_3$ (final pH 6.5). The solution was pre-flushed in the dark with pure N_2 (3 l h^{-1}) before the addition of $NaHCO_3$ until 70% of O_2 saturation was reached. The calibration of YSI polarographic instrument, the final volume of solution and the experimental temperature agreed with the original method. O_2 evolution rates were not corrected for the concurrent rates of respiratory O_2 uptake.

This method was used to analyze apparent photosynthesis in inbred lines and hybrid-derived populations (MNL 58, 1984). In 1982 three inbred lines, selected for further experimentation, showed high (2) and low (1) activity, with values between 250.0 and 168.8 u moles O_2 g $FW^{-1}h^{-1}$ (mean 205.0). In 1983 the extreme values of the same lines were 253.9 and 157.3 (mean 205.3).

Jaime R. Jatimliansky

Relationships between photosynthesis and canopy traits in flint type maize

A study to determine the genetic basis of photosynthesis and its relationship to morphological traits and yield determinants is in progress. For this purpose, lines of the flint type maize grown in Argentina are used. The first crosses were made using three inbred lines developed by the Institute which differed in photosynthetic activity measured by polarography (MNL 58, 1984). These lines, their F1 hybrids, backcrosses and F2 were tested in a trial with four repetitions in which several traits were measured, among them apparent photosynthesis and canopy traits. Apparent photosynthesis was measured by polarography at Hanway's stage 2.5 (AP-1) on the 6th leaf from the plant base, and at stage 5.0 (AP-2) on the 3rd leaf from the top. Leaf orientation values were estimated on the 3rd leaf from the top (LOV3), the leaf above LOVA) and the leaf below (LOVB) the ear; values were calculated by Pepper's formula (Crosbie, Pearce and Mock, Crop Sci. 18:87-90, 1978). The leaf area index (LAI) was calculated considering as estimator for the leaf area, for all the leaves, the product length x maximum width x 0.75.

Phenotypic correlations between these characters for inbred lines (IL) and their F1 are shown in the table.

		LAI	LOV3	LOVA	LOVB
AP-1	IL	-0.88**	-0.58*	-0.27	-0.02
	F_1	0.02	-0.15	-0.03	-0.16
AP-2	IL	0.04	-0.43	-0.68*	-0.69*
	F_1	-0.71**	-0.58*	-0.44	-0.25
LAI	IL	—	0.55	0.36	0.07
	F_1	—	0.69*	0.64*	-0.11

*, ** Significant at the 0.05 and 0.01 level.

The results indicate that: (1) There are negative correlations between apparent photosynthesis measurements and traits related to canopy. Upright leaves and high leaf area index correspond to low photosynthetic activity per fresh weight. (2) LAI shows a positive correlation with LOV measured on leaves placed above the ear. More erect upper leaves are supposed to contribute to more efficient use of light energy, inducing more vegetative growth. (3) It is

observed, with few exceptions, that there is a general correspondence between the phenotypic coefficient values of inbreds and those of their F1. This should be of practical value for prediction of F1 features based on inbred measures.

Jaime R. Jatimliansky, Francisco J. Babinec and Miguel J. Arturi

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Perennial teosinte-Gaspe hybrids: inheritance of pollen grain size

In the Maydeae (Gramineae), maize (Zea mays) is the species which possesses pollen grains of great size. Within the same tribe, the species pertaining to the Tripsacum genus are the ones which have the smallest pollen grains, while the teosintes (Z. mexicana, Z. perennis and Z. diploperennis) have intermediate pollen grains between those of maize and Tripsacum. The variety of size of pollen grains among species is closely related to the length variation of the silks. Our well-known maize, with long silks, possesses large pollen grains, with sufficient reserve to produce long pollen tubes. Contrary to this, the Tripsacum species, which have short silks, have small pollen grains, with a capacity to produce only short pollen tubes.

Teosintes, species closely related to maize, develop silks intermediate in length between those of maize and those of Tripsacum, and similarly their pollen grains are also intermediate to those species. From what has been explained, it may be considered that the size of pollen grains is a specific character which is closely related to the reproductive needs of the species. The object of this report is to present results obtained in the study of the inheritance of this specific character (size of pollen grain) in hybrids between perennial teosinte (Z. perennis) and Gaspe (Z. mays).

The experimental results (size of pollen grain) were obtained on the basis of the individual plant, and originate from the analysis of that character in 10 perennial teosinte plants, 11 Gaspe plants, 110 F1 plants and 605 F2 plants produced by crossing perennial teosinte and Gaspe. The four populations studied were cultivated in the corresponding cycle during 1982/1983. The pollen was harvested on each tassel at the moment of natural dehiscence. Each pollen sample was treated with a mixture of sol. I_2-KI and lactic acid in a ratio 1:1 v/v. Measurements were made on 20 pollen grains of each plant, taken at random, by means of a microscope provided with a micrometric ocular, and a magnification of 400 X. The size of the pollen grain was evaluated upon the biggest diameter. Measurements of the pollen grain size based only on the largest diameter are approximately 8% higher with respect to the average of the largest and smallest diameter.

Table 1: Number of plants (N) and mean pollen grain size (PGZ) in perennial teosinte (Zp), Gaspé (Gs) and its F_1 and F_2 populations.

	N	PGZ (um)(1)
P_1 (Zp)	10	78.1 ± 1.1
P_2 (Gs)	11	96.9 ± 2.3
F_1	110	80.7 ± 3.7
F_2	605	88.1 ± 6.1

(1): major diameter

Table 2: Means and variances of perennial teosinte (Zp), Gaspé (Gs) and its F_1 and F_2 populations.

	Mean PGZ (um)		Variance		
	obs.	m.p.(1)	T(2)	E	G
P_1 (Zp)	78.1	—	1.2	—	—
P_2 (Gs)	96.9	—	5.3	—	—
F_1	80.7	87.5	13.7	—	—
F_2	88.1	87.5	37.2	13.7	23.5

(1): mp.--mid parent value; T--total; E--environmental; G--genetic.

Table 3: Differences between means for PGZ of perennial teosinte (P_1), Gaspé (P_2) and its F_1 and F_2 populations.

Comparison	DF	"f"	sig.(2)
P_1 vs. P_2	19	23.5	***
P_1 vs. F_1	118	2.2	*
P_1 vs. F_2	613	5.2	***
P_2 vs. F_1	119	14.2	***
P_2 vs. F_2	614	4.8	***
F_1 vs. F_2	713	12.3	***
F_1 vs. F_1T (1)	129	8.2	***
F_2 vs. F_2T (1)	624	0,5	NS

(1): F_1T and F_2T--mid parent value; (2): NS--no significant; *--significant at 5% level; ***--significant at 0.1% level.

Perennial teosinte has significantly smaller pollen than Gaspe (see Tables 1 and 3). The average size of the pollen grain for the F1 population is more similar to perennial teosinte than to Gaspe. The average size of the pollen grain for the F2 population approaches the theoretical average among its progenitors (see Tables 1 and 3). From the results obtained in Table 2, heritability was calculated, this being high (0.82).

The frequency distribution of pollen grain size in the four populations studied is presented in Figure 1. The parents are separated in size, and the F1 is more

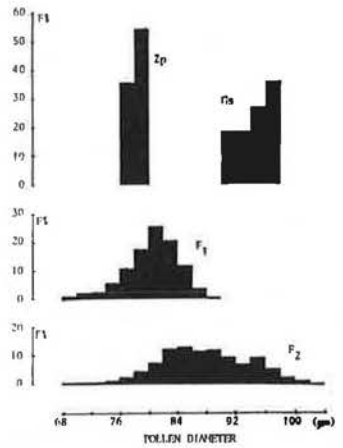

Figure 1: Frequency distributions for PGZ (major diameter in μm) in perennial teosinte (Zp), Gaspé (Gs) and its F_1 and F_2 populations.

variable than perennial teosinte but with a distribution and mean nearer to this species. The F2 presents an ample distribution which exceeds parental extremes.

The data obtained indicate that: (1) the size of pollen grain is inherited quantitatively; (2) the size of F1 pollen grains is closer to teosinte, probably due to the different chromosome contribution of both species (20 teosinte chromosomes, 10 maize chromosomes) and consequently the character is controlled by the mother's genotype; (3) the size of the pollen grains of the F2 does not differ significantly from the theoretical mean, showing that genes with additive effect prevail. The frequency distribution of the F2 is transgressive in both ways, that is to say, there are plants with smaller pollen than perennial teosinte and larger than Gaspe.

Jorge Luis Magoja and Ida Graciela Palacios

Effect of de*-7601 on seed free amino acid content

A spontaneous mutation which produces defective kernels and is a lethal recessive (de*-7601) brings about numerous biochemical alterations, as much in the germ as in the endosperm (MNL 56:108, 1982; MNL 57:71, 1983). We have obtained data on the level of free amino acids in defective and normal kernels. In 21 ears of the red flint line WK-01 segregating defective kernels, and in 10 ears of WK-01 not segregating for this mutation, the protein content and the water soluble free amino acid content (FAA) of the defatted whole kernel was analyzed.

The results obtained are detailed in Table 1. The protein level is not significantly different for the three genotypes studied. In defective kernels, the FAA level is greater than in normal kernels. The defective kernels have an average of three times more FAA than normal ones. The results obtained lead us to deduce that the locus de*-7601 conditions a high FAA level. Nevertheless the high variability for FAA in defective kernels (de/de) and in heterozygous normal ones (+/de) makes us suspect the existence of another variation in the genetic control of FAA content.

Table 1: Protein and free amino acid content (FAA) in normal and defective kernels.

	defatted whole kernel		
	protein	FAA (uMLeu/100 mg prot.)**	
Genotype	(%)	$\bar{X}$ ± SD	range
$+/+$	11.7^{a}(*)	16.4^{a} ± 1.6	12.9 - 18.8
$+/+$: 2^{+}/de	11.5^{a}	17.1^{a} ± 4.5	10.3 - 25.6
de/de	11.4^{a}	48.0^{b} ±26.9	18.1 -116.7

(*) Individual means within a column followed by different letters are significantly different at 0.1%level.

(**) Free amino acid content expressed in micromols of Leucine per 100 mg of protein.

Among the ears segregating for the defective character, there are some whose normal kernels have a higher FAA level than normal (intermediate between normal and defective). On the other hand, there are ears (with low frequency) in which the mutants have a FAA level which is not significantly higher than that of the normal phenotype grains.

For a better interpretation of this variation, in ears having normal kernels with a higher FAA level than normal, the kernels were individually analyzed. In one ear where 60 kernels were analyzed, a variation range for FAA content was obtained between 13.1 and 51.8 uM Leu/100 mg prot., with an average 23.0 uM Leu/100 mg prot.

These results lead us to deduce that the content of FAA segregates in those ears. Other ears where a few grains were analyzed also indicated segregation for FAA content. On the other hand, in other ears the character does not segregate. In ears segregating for FAA content among normal kernels, high levels of FAA were found in a proportion of approximately 25% (1/4 of the grains have a low level, 1/2 an average level and 1/4 a high level). These results have induced us to think of the probable presence of a co-dominant gene which controls the FAA content.

From what has been expressed previously the following may be deduced: (1) high FAA levels seem to be associated with the de*-7601 gene with a very high frequency (in most cases); (2) the FAA content in the material studied appeared to be controlled by a co-dominant gene, presumably linked to the de*-7601 locus--with a low frequency cases exist such as the one presented, where high FAA levels do not associate with the defective character (it segregates independently); (3) the de*-7601 gene does not have an effect per se on FAA level.

It is probable that the defective mutation in the WK-01 line has been produced simultaneously with another, which conditions the high FAA content.

Apparently, the gene which conditions the high FAA level is linked to de*-7601 since both characters do not segregate independently in most cases.

The fact that there is a co-dominant gene involved in the control of FAA content, and that it does not produce any modification in the grain's phenotype (the grains being red flint), has a special and practical meaning. In fact, the high FAA levels in normal grains are associated with a much better protein quality (high levels of lysine and tryptophan).

We are actually working to isolate homozygotes for the high FAA content character and high protein quality, and to effect a more critical test to confirm this variant.

The results presented indicate once more that it is possible to develop maize of as high protein quality and "normal genotype" (without floury mutants) as those obtained from other origins (MNL 57:75, 1982).

A co-dominant gene which will condition high protein quality and does not modify the normal genotype of the grain might be of great importance.

J. L. Magoja, I. G. Palacios and M. E. Streitenberger

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

As we reported last year (MNL 57:75, 1983), we have obtained inbred lines of red flint maize with high-quality protein and normal genotype (having no floury mutant in their genetic background).

The best lines obtained have an average lysine of 3.3 g per 100 of protein in the endosperm (see Table 1). The protein and lysine contents are the average from 5 to 10 ears of each line.

The variability of lysine content in each line is low. Between lines, there exists a significant negative correlation between protein content and protein quality: high levels of lysine are found more frequently in lines with less protein content in the endosperm.

In the selection process used which was reported previously (MNL 57:75, 1983) inbred lines OU and DY which were obtained from the Instituto Fitotecnico de Santa Catalina were used as donors for the red flint feature. These lines have an average lysine content of 2.1 g per 100 g endosperm protein. Based upon the content of lysine of OU and DY lines, the increase of lysine obtained in AL lines was calculated. Notwithstanding the fact that at first sight the average increase does not appear to be spectacular, it must be pointed out that this protein quality is found in red flint maize (hard endosperm). The AL lines are

phenotypically indistinguishable from the red flint lines with normal lysine content.

Table 1: Protein and lysine content of high-quality protein selected inbreds.

Inbred	defatted endosperm		Lysine increase over normal lines % (*)
	Protein %	Lysine(G/100g prot.)	
	Mean ± SD	Mean ± SD	
AL 23	11.0 ± 0.7	3.2 ± 0.2	+ 52
AL 24	10.5 ± 0.4	3.1 ± 0.1	+ 48
AL 27	11.5 ± 0.7	3.1 ± 0.1	+ 48
AL 28	11.1 ± 0.7	3.1 ± 0.2	+ 48
AL 30	11.5 ± 0.5	3.1 ± 0.2	+ 48
AL 33	10.3 ± 0.5	3.2 ± 0.3	+ 52
AL 35	9.8 ± 0.6	3.4 ± 0.1	+ 62
AL 36	9.8 ± 1.2	3.5 ± 0.5	+ 67
AL 37	8.3 ± 0.1	3.7 ± 0.2	+ 76
AL 39	8.6 ± 0.3	3.6 ± 0.2	+ 71
Average	10.3 ± 1.1	3.3 ± 0.2	+ 57

(*):Average lysine content of normal DY and OU lines= 2.1 g/100g prot.

It appears, as has happened with modified opaque-2, that it is difficult to obtain a very high protein quality when trying to preserve totally the normal phenotype (hard endosperm). Difficulties are greater in flint maize than in dent, due to the fact that the former possess a greater proportion of hard endosperm than the latter.

Nevertheless, we believe that the protein quality of AL lines is good, considering the fact that the normal endosperm structure has not been modified and that the high protein quality has been obtained without using floury mutants, which with opaque-2 has been employed massively all over the world.

The AL lines obtained have no outstanding agronomic characteristics to permit their use in the production of hybrids. In selecting lines of high quality protein and normal genotype, the lysine content in endosperm protein has been taken into account. Therefore, the material developed has only the donor's value of high protein quality. We have actually started to use AL lines to improve the protein quality of red flint maize with outstanding agronomic characteristics.

We hope in the near future to produce red flint maize hybrids of high quality protein and normal genotype.

J. L. Magoja, A. A. Nivio and M. E. Streitenberger

Perennial teosinte-Gaspe hybrids: inheritance of tassel branching traits

The new division of the genus Zea proposed by Doebley and Iltis (Amer. J. Bot. 67:982, 1980) is based upon morphological characteristics of the male inflorescence. Among other characteristics, we have studied heredity, in hybrids between perennial teosinte (Z. perennis) and Gaspe (Z. mays), of three of these: (a) tassel branch number, (b) tassel branching axis length and (c) lateral tassel branch internode length. It is our intention to make known here the inheritance of those three quantitative traits of the tassel which are specific, and which distinguish maize from perennial teosinte.

On at least two mature tassels of each plant belonging to perennial teosinte, Gaspe and its progenies F1 and F2, measurements were carried out. The four populations studied were cultivated during spring-summer in the years 1982/83.

(a) Tassel branch number (TBN): The number of tassel branches was evaluated upon 151 perennial teosinte plants, 31 Gaspe plants, 85 F1 plants, and 429 F2 plants. TBN represents all primary and secondary branchings, providing they have 4 or more spikelets. As can be observed in Table 1, perennial teosinte has a low TBN, but Gaspe also has a low TBN for a Z. mays species, although definitely higher than teosinte. In the F1 TBN does not differ significantly from Gaspe (Table 3). The average TBN of F2 is higher than for F1, and an ample variability can be observed for TBN (Figure 1). In F2 there are plants which have no branches in the tassel (TBN=0) up to plants which have 25 branches. Broad sense heritability calculated upon the results presented in Table 2 is high (0.80), and the genetic variance of F2 is great in comparison to environmental variance (5:1).

Table 1: Number of plants (N) and mean tassel branch number (TBN) in perennial teosinte (Zp), Gaspe (Gs) and its F_1 and F_2 populations.

	N	TBN
P_1 (Zp)	151	2.8 ± 1.2
P_2 (Gs)	31	5.9 ± 2.5
F_1	85	5.8 ± 1.9
F_2	429	7.8 ± 4.2

Table 2: Means and variances of perennial teosinte (Zp), Gaspé (Gs) and its F_1 and F_2 populations.

	Mean TBN		Variance		
	obs.	m.p.(1)	T(2)	E	G
P_1 (Zp)	2.8	—	1.4	—	—
P_2 (Gs)	5.9	—	6.3	—	—
F_1	5.8	4.4	3.6	—	—
F_2	7.8	4.4	17.6	3.6	14.0

(1): m.p.--mid parent value; T--total; E-- environmental; G--genetic.

Table 3: Differences between means for TBN of perennial teosinte (P_1), Gaspé (P_2) and its F_1 and F_2 populations.

Comparison	DF	"t"	sig.(2)
P_1 vs. P_2	180	10.6	***
P_1 vs. F_1	234	15.0	***
P_1 vs. F_2	578	14.4	***
P_2 vs. F_1	114	0.2	NS
P_2 vs. F_2	458	2.5	*
F_1 vs. F_2	512	4.3	***
F_1 vs. F_1T(1)	265	5.5	***
F_2 vs. F_2T(1)	609	10.5	***

(1): F_1T and F_2T--mid parent value;(2): NS-- no significant; *--significant at 5% level; ***--significant at 0,1% level.

The results shown in Tables 1, 2 and 3 indicate that: (1) TBN is inherited quantitatively; (2) high TBN (GASpe) is dominant to low TBN (perennial teosinte); (3) the distribution of frequencies for TBN in F2 represents a type of transgressive inheritance, which indicates a significant heterosis effect.

(b) Tassel branching axis length (TBAL): TBAL covers the central axis part of the tassel which presents branching. This characteristic is closely associated with TBN; when TABL is long, there is high TBN and vice versa. TBAL was evaluated

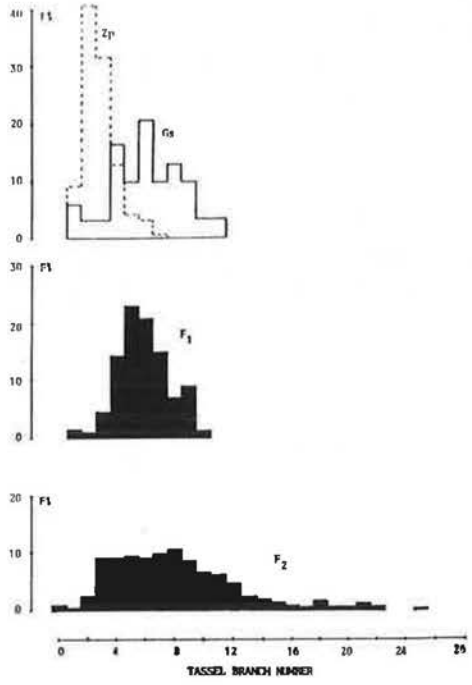

Figure 1: Frequency distributions for TBN in perennial teosinte (Zp), Gaspé (Gs) and its F_1 and F_2 populations.

on 21 perennial teosinte plants, 11 Gaspe plants, 69 F1 plants, and 427 F2 plants. Perennial teosinte has a significantly lower TBAL than Gaspe (see Tables 4 and 6). The TBAL value of the F1 is nearer to Gaspe than to teosinte, and in F2 the average value of TABL is greater than that of F1 (see Tables 4 and 6). In F2 there exists an ample TBAL variability, with plants with TBAL=0 (without branching) up to plants with long TBAL (14 cm) (see Figure 2). From the results shown in Table 5, broad sense heritability was calculated (H=0.77).

Table 4: Number of plants (N) and mean tassel branching axis length (TBAL) in perennial teosinte (Zp), Gaspé(Gs), and its F_1 and F_2 populations.

	N	TBAL (cm)
P_1 (Zp)	21	1.6 ± 0.4
P_2 (Gs)	11	4.0 ± 1.2
F_1	69	3.0 ± 1.1
F_2	427	5.1 ± 2.3

Table 5: Means and variances of perennial teosinte (Zp), Gaspé (Gs) and its F_1 and F_2 populations.

	Mean TBAL (cm)		Variance		
	obs.	m.p.(1)	T(2)	E	G
P_1 (Zp)	1.6	—	0.2	—	—
P_2 (Gs)	4.0	—	1.4	—	—
F_1	3.0	2.8	1.2	—	—
F_2	5.1	2.8	5.3	1.2	4.1

(1): m.p.--mid parent value; T--total; E--environmetal; G--genetic:

Table 6: Differences between means for TBAL of perennial teosinte (P_1), Gaspé (P_2) and its F_1 and F_2 populations.

Comparison	DF	"t"	sig.(2)
P_1 vs. P_2	30	8.6	***
P_1 vs. F_1	88	5.6	***
P_1 vs. F_2	446	7.0	***
P_2 vs. F_1	78	2.8	**
P_2 vs. F_2	436	1.6	NS
F_1 vs. F_2	494	7.5	***
F_1 vs. F_1T(1)	99	0.9	NS
F_2 vs. F_2T(1)	457	5.8	***

(1): F_1T and F_2T--mid parent value; (2):NS--no significant; **--significant at 1% level; ***--significant at 0,1% level.

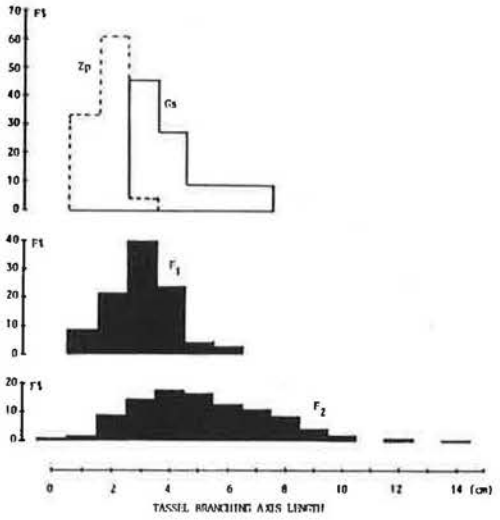

Figure 2: Frequency distributions for TBAL in perennial teosinte (Zp), Gaspé (Gs) and its F_1 and F_2 populations.

The results obtained indicate that: (1) TBAL is inherited quantitatively; (2) TBAL average of the F1 does not differ significantly from the mid parent value; (3) the distribution frequencies for TBAL in F2 is transgressive, especially in the positive direction, so that the obtained mean exceeds the theoretical mean, indicating a significant heterotic effect similar to that found for TBN; (4) in F2, TBAL correlates significantly with TBN (r=0.78).

(c) Lateral tassel branch internode length (LTBIL): LTBIL may vary from short, with male spikelets densely overlapping, to long, slightly superimposed. LTBIL was evaluated in 21 perennial teosinte plants, 10 Gaspe plants, 101 F1 plants and 427 F2 plants. In Table 7 it can be seen that perennial teosinte has a short LTBIL, while in Gaspe it is long. F1 and F2 averages are alike and intermediate with respect to their progenitors. The differences between the four population averages are summarized in Table 9. The distribution of LTBIL frequencies shown in Figure 3 represents an intermediate F1 with less variation than in F2, where LTBIL varies between 2 mm and 7.5 mm. LTBIL heritability is 0.61 and was calculated from the data presented in Table 8.

Table 7: Number of plants (N) and mean lateral tassel branch internode length (LTBIL) in perennial teosinte (Zp), Gaspé (Gs) and its F_1 and F_2 populations.

	N	LTBIL (mm)
P_1 (Zp)	21	3.7 ± 0.3
P_2 (Gs)	10	5.7 ± 0.7
F_1	101	4.3 ± 0.6
F_2	427	4.5 ± 0.9

Table 8: Means and variances of perennial teosinte (Zp), Gaspé (Gs) and its F_1 and F_2 populations.

	Mean LTBIL (mm)		Variance		
	obs.	m.p.(1)	T(2)	E	G
P_1 (Zp)	3.7	—	0.09	—	—
P_2 (Gs)	5.7	—	0.49	—	—
F_1	4.3	4.7	0.36	—	—
F_2	4.5	4.7	0.81	0.36	0.45

(1) m.p.--mid parent value; T--total; E--environmental; G--genetic.

Table 9: Differences between means for LTBIL of perennial teosinte (P_1), Gaspé (P_2) and its F_1 and F_2 populations.

Comparison	DF	"t"	sig.(2)
P_1 vs. P_2	29	11.4	***
P_1 vs. F_1	120	4.5	***
P_1 vs. F_2	446	4.1	***
P_2 vs. F_1	109	7.0	***
P_2 vs. F_2	435	4.2	***
F_1 vs. F_2	526	2.1	*
F_1 vs. F_2T(1)	130	3.2	**
F_2 vs. F_2T(1)	456	1.2	NS

(1): F_1T and F_2T--mid parent value; (2)NS--no significant; *--significant at 5% level; **--significant at 1% level; ***--significant at 0,1% level.

The results obtained indicate that: (1) LTBIL is inherited quantitatively; (2) the F1 has a LTBIL near the mid parent value; (3) the F2 has a LTBIL that does not differ significantly from the theoretical mean, from which it may be deduced that genes with additive effect predominate; (4) the distribution of frequencies in F2 is transgressive in both ways.

Analyzing the results presented with regard to heredity of tassel traits it is deduced that TBN and TBAL are inherited in a similar way and are closely associated, because of their possibly being conditioned by the same genes.

On the other hand, LTBIL is inherited in a distinctly independent manner from TBN and TBAL (LTBIL does not correlate significantly with TBN and TBAL).

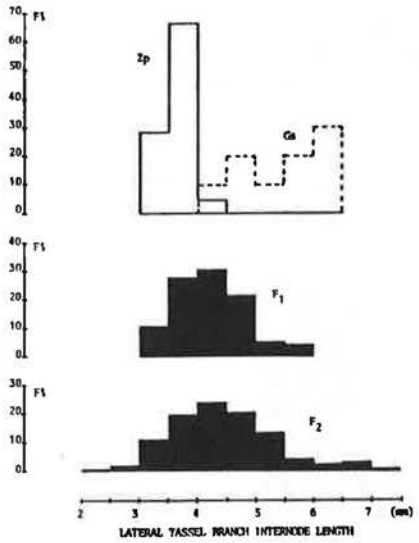

Figure 3: Frequency distributions for LTBIL in perennial teosinte (Zp), Gaspé (Gs) and its F_1 and F_2 populations.

TBN and TBAL are very changeable characteristics in maize, and frequently there is observed, as in the Gaspe case, a low TBN and short TBAL, resembling a perennial teosinte. On the contrary, LTBIL allows a better way to distinguish maize from perennial teosinte and consequently it is the most specific (to our knowledge) of all three characters studied.

Ida Graciela Palacios and Jorge Luis Magoja

Perennial teosinte-Gaspe hybrids: Fertility of F1 and F2 populations

As pointed out in previous articles (MNL 52:37, 1978, Magoja, Com. Direc. Inv. U.N.L.Z. 2 (3):10, 1980), the fertility of hybrids between perennial teosinte and Gaspe is relatively high. The triploid hybrids obtained between perennial teosinte and other maize varieties are generally highly sterile. This phenomenon has been noted by some other authors as well as ourselves. It is well known that triploidy per se conditions high sterility; fertility analyses of different hybrids between perennial teosinte and maize indicate that the grade of genetic affinity of their progenitors has a considerable influence on fertility. There is no doubt, therefore, that the chromosome imbalance of these hybrids does not constitute the only cause of sterility. Consequently, the appropriate selection of maize genotypes, which combined with perennial teosinte will produce hybrids with high fertility, must lead to the attainment of fertile hybrids.

Intending to measure fertility of perennial teosinte-Gaspe hybrids, pollen viability was evaluated in F1 and F2 populations. An F1 population was studied which was comprised of 110 plants originating from the same number of hybrid seeds, obtained by crossing perennial teosinte with Gaspe. The F2 population of 598 plants originated from the seed obtained on F1 plants, by way of free pollination between themselves. The pollen of each individual plant was harvested during the 1982/1983 summer in both populations. The pollen was stained with I_2-KI sol. mixed with an equal volume of lactic acid, and was observed with a microscope. A count was carried out over approximately 200 grains of pollen of each plant. Only the pollen grains that colored totally with the stain were considered fertile.

In Figure 1 the frequency distribution of fertility for F1 and F2 can be seen. Although in F2 the variability observed can be explained on the basis of different genetic combinations that are produced, the high variability of F1 cannot be attributed to environmental causes. It is evident that the variability of fertility of the F1 is due to expression of different F1 genotypes, perhaps brought about by the different gametes contributed by perennial teosinte.

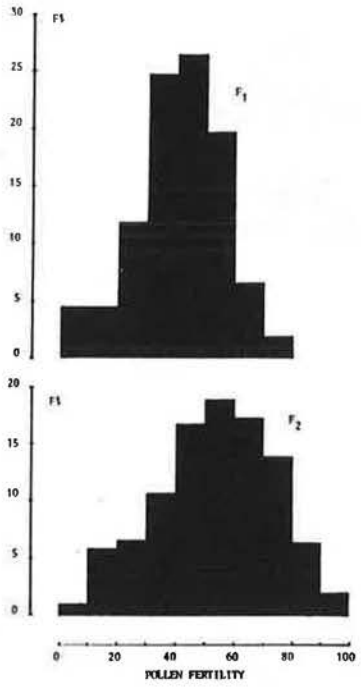

Figure 1: Frequency distributions for pollen fertility (PF %) in F_1 and F_2 populations (perennial teosinte x Gaspé).

In order to have an estimate of the environmental component of the total F1 variability, numerous plants were cloned. The clones of each F1 plant (equal genotype) showed that the variability of conditioned fertility through environment is approximately 33%. In other words, variability of fertility among F1 plants is due in 67% to genetic causes.

The results obtained in F1 and F2 populations are summed up in Table 1.

Table 1: Pollen fertility of perennial teosinte-Gaspé hybrids.

	Fertility (%)			
Population	Mean	range	S^2	N
F_1	41.2[a](*)	0-79.8	231.0	110
F_2	53.4[b]	0-95.0	388.1	598

(*) Individual mean within a column followed by different letters are significantly different at 0.1% level.

It can be noted that the average fertility increases in successive generations of hybrid progenies of perennial teosinte and maize. While we do not present results, fertility increases in the F3. In that the hybrid progenies were obtained without any selection pressure, it is evident that there is a natural preferential selection of better balanced genotypes and chromosomal endowment, which conditions greater fertility.

Comparing the fertility of hybrids between perennial teosinte and Gaspe with other hybrids which we have obtained employing other maize, we were able to deduce that the former are relatively fertile and that perennial teosinte and Gaspe

produce good genetic combinations for fertility. While the fertility of F1 of the hybrids between teosinte and Gaspe is an average of 41.2%, other hybrids obtained do not reach 15%. In F2 progenies, the hybrids between teosinte and Gaspe have 53.4% mean fertility, and other hybrids only 28% average fertility.

The progenies of hybrids between teosinte and Gaspe can yield individuals nearly totally fertile in generation F2. Fertility of hybrids between perennial teosinte and Gaspe approaches the much higher fertility of hybrids between maize and the other teosinte species.

Ida Graciela Palacios and Jorge Luis Magoja

Zein body size of maize and its wild relatives, Zea perennis and Tripsacum dactyloides

The greatest storage protein of endosperm tissue in maize and its wild relatives is zein. This protein accumulates in spheroidal organelles termed protein or zein bodies. With the intention of finding out if there are differences between species as to the size of the protein bodies, measurements were carried out in three maize varieties (one dent and two flint), in perennial teosinte (Z. perennis) and in Tripsacum dactyloides (2n=72).

Microscopic observations were effected upon hard endosperm cuts of mature kernels. The kernels were thin sectioned and destarched with alpha-amylase, and the endosperm tissue was stained with Coomassie Brilliant Blue R 250. Excepting the staining method, the procedure employed for the preparation and observation of the samples is similar to that described by Wolf and Koo (Stain Tech. 45:277, 1970). The size of the zein bodies was evaluated by measuring the largest diameter, and was expressed in micrometers (um). Protein body sizes were measured in three zones: (1) the first to the fourth layer of cells beneath the aleurone layer; (2) from the fifth to the tenth layer of cells; and (3) from the eleventh layer of cells towards the inside of the kernel. In each zone and for each sample approximately 200 zein bodies were measured. The results obtained may be seen in Tables 1 and 2.

Table 1: Protein body size (um) of maize (Z.m), perennial teosinte (Z.p) and *Tripsacum dactyloides* (Td). OU--red flint line; Gs--Gaspé flint; D--Morgan dent.

	Zone					
	1		2		3	
	Mean + SD	range	Mean + SD	range	Mean + SD	range
Z.m. (D)	2.1 ± 0.8	0.6 - 3.9	1.5 ± 0.5	0.5 - 2.4	1.4 ± 0.4	0.3 - 2.8
Z.m. (OU)	1.6 ± 0.3	0.7 - 2.0	1.6 ± 0.3	0.9 - 2.5	1.5 ± 0.3	0.9 - 2.5
Z.m. (Gs)	1.6 ± 0.3	1.2 - 2.6	1.3 ± 0.3	0.5 - 2.0	1.2 ± 0.4	0.6 - 3.0
Z.p.	1.7 ± 0.3	0.8 - 2.5	1.6 ± 0.4	0.7 - 2.9	1.4 ± 0.4	0.7 - 2.0
Td	1.8 ± 0.4	0.8 - 3.5	1.8 ± 0.5	0.7 - 2.8	1.6 ± 0.3	0.7 - 2.3

Table 2: Differences between mean protein body size of maize, perennial teosinte and *Tripsacum*.

	Zone		
	1	2	3
Z. mays (Morgan dent)	$2.1^{a(*)}$	1.5^{b}	1.4^{b}
Z. mays (OU)	1.6^{c}	1.6^{b}	1.5^{ab}
Z. mays (Gaspé)	1.6^{c}	1.3^{c}	1.2^{c}
Z. perennis	1.7^{bc}	1.6^{b}	1.4^{b}
Tripsacum dactyloides	1.8^{b}	1.8^{a}	1.6^{a}

(*) Individual means within a column followed by different letters are significantly different at 5% level.

The high variability which exists with respect to size in the zein bodies within each analyzed zone should be emphasized. The ample variation range is approximately similar in all samples studied, and there are not only size variations within zones but in the same zone. The high number of measurements effected (200 per each zone and 600 for each sample) show that the zein bodies have a very variable size, a character which has not been reported previously.

This is probably due to the fact that the work reported by other authors was based on results carried out with dyes which were not specific (i.e., iodine). Coomassie's high detectability and specificity allows the identification with great precision of the zein bodies embedded in the protein matrix and allows even the smallest (not visible by means of other dyes) to be seen. The dye employed is therefore very appropriate for the staining of the zein bodies.

The first zone of cells, which appears to be the most representative, shows that the dent line possesses larger zein bodies (see Table 2). Flint maize has zein bodies which do not differ significantly from the perennial teosinte, and are of smaller size than the dent maize. In all species there is a general tendency indicating that the size of the zein bodies decreases from zone 1 to zone three, that is to say, from the most superficial layers of cells inward. Although in some cases there exist significant differences (see Table 2), the sizes of the zein bodies are not an appropriate index to distinguish the Maydeae. In sum, one may say that the endosperm structure of the different species is very similar and that the greatest differences are a consequence (as far as proteins) of the different proportion found in the different species, as previously expressed (MNL 56:106, 1982).

Erratum: In the last Newsletter (MNL 57:70, 1983) we committed an error in the magnification factor of the microscopic camera. Consequently, the true values for Tripsacum dactyloides (2n=72) are: the size of the zein bodies vary from 1.2 to 2.4 um in diameter (average 1.8 um); the starch grains vary from 10.2 to 13.5 um in diameter (average 12.0 um).

Luis Maximo Bertoia and Jorge Luis Magoja

Perennial teosinte-Gaspe hybrids: protoandrous vs. protogynous

The Maydeae species are dichogamous, the pollen and silks are not simultaneously produced. While maize is generally protoandrous (pollen is produced before the silks emerge), the wild relatives are protogynous (silks emerge before pollen production).

In maize-perennial teosinte (Zea perennis) hybrids, as well as between maize and diploperennial teosinte (Z. diploperennis), we have invariably observed that the F1 are protogynous plants. By this we have deduced that the teosinte character (protogyny) acts as dominant. Nevertheless, our attention has been attracted to the fact that in the F2 progeny of hybrids between perennial teosinte and Gaspe, the majority of the plants (80%) are protoandrous, and only 20% are protogynous.

The hybrids between perennial teosinte and Gaspe constitute a very particular case, in that Gaspe is a protogynous maize (silks appear three days before pollen production).

The character analysis protoandrous-protogynous was carried out on an F2 population derived from the cross between perennial teosinte and Gaspe. The F2 population, constituted by 567 plants, was cultivated during the 1982/1983 cycle. In each of the F2 plants, the evolutive cycle was measured in days to tassel (T), days to silking (S) and days to pollen (P).

The days to silking less the days to pollen gives a value in days: if they are of a positive sign they are protoandrous, if the sign is negative they are protogynous (see Figure 1 and Table 1). In the protoandrous plants, the pollen is

Table 1: Days of protoandrous and protogynous in F_2 plants.

	Protoandrous	Protogynous
Number of plants	457	110
Mean (days)	16.7	12.9
SD	13.1	13.7

produced at an average 16.7 days before the silks. In the protogynous plants, pollen is produced at an average of 12.9 days after the silks emerge. It can be deduced that the protoandrous and the protogynous plants produce the tassel at the same length of time (there are no significant differences) (see Table 2). Nevertheless, in the protoandrous plants the appearance of the silks takes place later than in the protogynous. Likewise, in the protoandrous plants the pollen is produced before that of the protogynous.

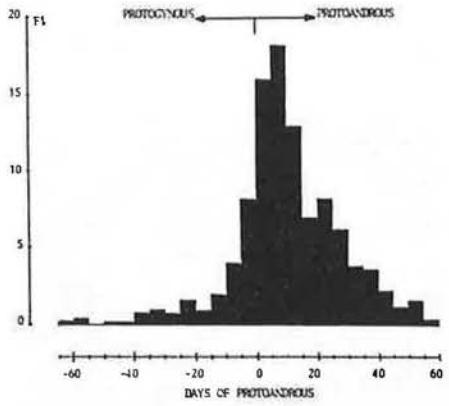

Figure 1: Frequency distributions for days of protoandrous (= days to silking-days to pollen) in perennial teosinte x Gaspé F_2 population.

Table 2: Evolutive cycle of protoandrous and protogynous plants in F_2 population. T--days to tassel; S--days to silking; P--days to pollen.

	T	S	P
Protoandrous	$84.1^{a(*)}$	119.1^{a}	102.4^{a}
Protogynous	81.2^{a}	100.5^{b}	113.4^{b}

(*) Individual means within a column followed by different letters are significantly different at 0.1% level.

To classify plants in two qualitative categories, i.e., protoandrous vs. protogynous, constitutes a simplification of the observed character. As can be observed in Figure 1, the variation is great in protoandrous and in protogynous plants. It seems that protoandry as well as protogyny are two aspects of the same character: the maturity of sexes not being simultaneous constitutes a character which is probably controlled by numerous genes.

The most intriguing aspect of the character is the fact that in a cross between progenitors with protogyny, protoandrous appears as the dominant character. This could be attributed perhaps to the fact that the character results from a genetic interaction between different factors contributed by both species, and subject to a preferential selection in the progeny.

If the wild character (protogyny) results from a greater adaptive value, it should be naturally selected in the hybrid's progeny. We have not detected the association of the protoandrous-protogynous character to other morphological characters studied in the hybrids. Maize specific characters preferably do not associate with protoandrous, and on the other hand, teosinte specifics do not associate with protogynous.

Jorge Luis Magoja and Gabriela Pischedda

Endosperm structure of modified floury-2 flint lines

By inbreeding and selection of hard endosperm, we have obtained after many generations, lines which carry the floury-2 gene with a normal phenotype. Modified floury-2 lines are of the yellow flint type and possess a high proportion of hard endosperm, which makes them indistinguishable from other normal flint lines (see Figure 1). When comparing the protein quality of modified floury-2

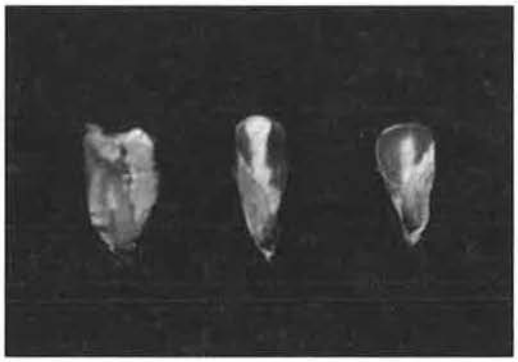

Figure 1: Photograph of longitudinally split kernels: left- *floury-2*; middle--modified *floury-2*; right--extremely modified *floury-2*.

with original floury-2 (floury endosperm), we have not found significant differences for tryptophan content (see Table 1).

Table 1: Protein and tryptophan content of *floury-2* lines

	defatted endosperm	
	protein	tryptophan
Inbred	%	(g/100 g prot.)
AD-02 f12	12.1	0.7
AD-02 f12 modified.	12.1	0.7
AD-02 f12 extr.modif.	13.7	0.7

This fact induced us to investigate the endosperm's sub-cell structure in order to establish if modifications had occurred. Mature kernels of a floury-2 line with floury endosperm (AD-02 f12) and two lines of modified floury-2 were thin-sectioned and prepared for the microscopic observation of the endosperm, in accordance with Wolf and Khoo (Stain Tech. 45:277, 1970). The prepared material was stained with Coomassie Brilliant Blue R250.

The results obtained may be seen in Figure 2. Floury-2 with floury phenotype has no visible zein bodies, as has been established by other authors. Notwithstanding, on the small layer of hard endosperm in the back part of the grain, we have observed zein bodies in line AD-02 f12.

In the modified versions of line AD-02 f12, it can be noticed that the protein matrix is densely populated with zein bodies.

The three lines studied are near isogenic and have the same protein quality, but when the endosperm hardness is modified, zein bodies are visible at optical

microscope level. This has led us to deduce that the presence or absence of zein bodies bears no indication as to protein quality.

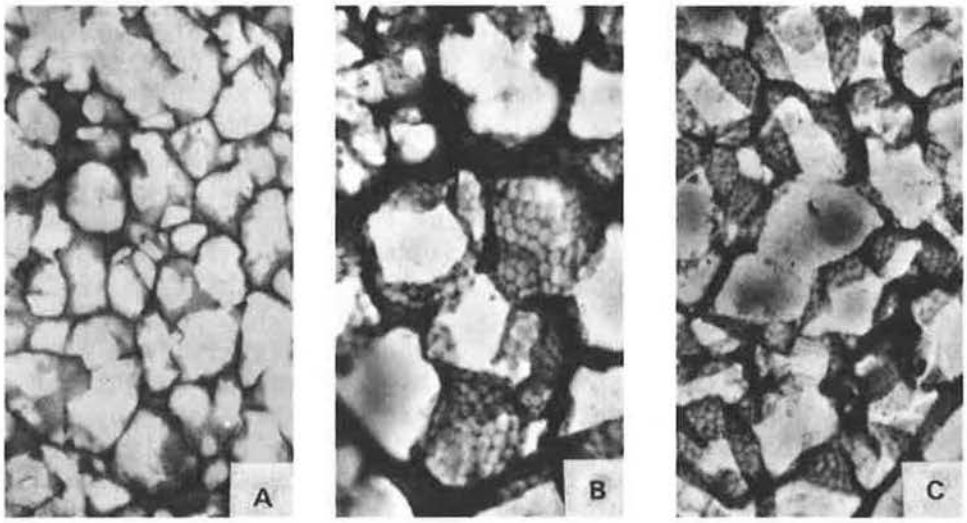

Figure 2: Destarched 3-4 um thick sections of endosperm showing protein bodies. A--*floury-2*; B--modified *floury-2*, C-- extremely modified *floury-2*.

While known mutants repress zein synthesis and consequently the protein quality increases, the microscopic observations of the endosperm structure do not give in every case an idea of the quantity of zein (and consequently its quality).

The results obtained indicate a close association between hard endosperm and presence of zein bodies in this particular case. Nevertheless, the protein quality does not associate with the floury endosperm, so that the material developed may be interesting to improve the protein quality, without modifying the normal phenotype of the grain (flint).

Jorge Luis Magoja and Luis Maximo Bertoia

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Influence of growing temperature on the changes in polypeptide synthetic patterns following temperature shifts and heat shock

We reported in last year's Newsletter (Baszczynski et al., MNL 57:161-162, 1983) that upward and downward temperature shifts over a number of temperature ranges and increments can lead to alterations in polypeptide synthetic patterns. We have extended these studies to examine the impact of normal growing temperatures on the polypeptide synthetic patterns prior to and following 'heat shock' and other temperature shifts. Seedlings of Oh43 were grown at 15, 20, 25, 30 or 35 C. Since temperature influences rate of growth, seedlings used in experiments were always selected at a similar developmental stage (plumules 1-2 cm). The results from 1-D and 2-D gel separations of polypeptides obtained from seedlings grown at the different temperatures indicate that while many similarities exist in the polypeptide synthetic patterns, each growing temperature yields a unique spectrum of newly synthesized products. From plumules grown at

15, 20, 25, 30 or 35 C, subjected to upward temperature shifts, we note that: (a) there is a change in the types of polypeptides synthesized, and (b) the initial growing (pre-shift) temperature influences the polypeptide synthetic patterns obtained following the upward temperature shifts. Most striking is the response to a one-hour 'heat shock' at 42 C. While the same six molecular weight classes of HSPs (Baszczynski et al., 1982, 1983) are observed following a shift to 42 C from each of the growing temperatures, some additional polypeptides, dependent on the initial growing temperature, also exhibit enhanced synthesis following the heat shock. For example, a 31 kD polypeptide is strongly enhanced following a temperature shift from 20 C to 42 C, is less enhanced following a shift from 25 C to 42 C or from 30 C to 42 C, and is not present following a shift from 15 C to 42 C. Similarly, a 23 kD polypeptide intensifies dramatically following a shift from 30 C to 42 C, but shows much less enhancement following a shift to 42 C from 15, 20 or 25 C.

Another observation is that a 10 C upward temperature shift from 15, 20 or 25 C leads to the enhanced synthesis of some of the high molecular weight HSP classes. This strongly suggests that the production of these polypeptides may reflect a change in response to temperature shifts in general. The low molecular weight, 18 kD HSP class, on the other hand, does not exhibit enhanced synthesis following these upward temperature shifts, suggesting that it may represent a 'heat-shock-specific' class of polypeptides. However, when mRNA is isolated from plumules shifted from 15 C to 25 C or from 20 C to 30 C and translated in vitro, all classes of HSPs (including the 18 kD class) are detected. The synthesis of these, relative to the other polypeptides, is comparable to that observed following heat-shock in vivo. This finding indicates that mRNAs for the HSPs are present following these lower temperature shifts, but that the translation of these mRNAs in vivo is under some form of temperature-dependent regulation. Further characterization of the temperature shift response in maize is in progress.

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

Analysis of polysomes and polysomal RNA from control and heat-shocked maize seedlings

Previous studies have shown that 'the heat shock' response in maize involves new or enhanced synthesis of heat shock polypeptides (HSPs) and the increased transcription or availability of mRNAs for these HSPs (Baszczynski et al., 1982, 1983). In order to examine the intracellular distribution of RNAs prior to and following heat shock, polysomes were isolated from maize plumules grown at 25 C or following a shift from 25 C to 42 C. The polymers were separated into two classes: (a) free cytoplasmic, and (b) membrane-associated. From comparisons of sucrose gradient profiles, we estimate the distribution of polysomes in these two classes to be approximately 60-70% in the free cytoplasmic fraction and 30-40% in the membrane-associated fraction. Following heat shock, there is a decrease in the number and sizes of polysome peaks in both polysome classes.

Free and membrane-associated polysome preparations were used to direct the synthesis of polypeptides in a rabbit reticulocyte in vitro translation system (Baszczynski, 1983). A wide range of polysome concentrations was found to be suitable for efficient translation of polypeptides, ranging in size from less than 10 to greater than 100 kD. Fluorographic analysis of electrophoretic separations of the products obtained from translations of total polysomes extracted from heat-shocked seedlings indicates that mRNAs for all molecular weight classes of the HSPs are present on polysomes following heat shock. This includes the 76 kD HSP class, which is not observed in translations of free RNA isolated from this same tissue. When the polysomes from heat-shocked seedlings are separated into free cytoplasmic and membrane associated fractions and translated in vitro

independently, fluorographic analysis of the electrophoretically-separated products reveals that: (a) the high molecular weight HSPs are translated from mRNAs which are present on free as well as membrane-associated polysomes; and (b) the low molecular weight 18 kD class is translated almost exclusively from mRNAs which are present on membrane-associated polysomes. This latter finding suggests some form of cellular discrimination or compartmentalization of the various HSP classes in maize.

We are presently isolating the mRNA from these polysome preparations for analysis on agarose gels, for in vitro translational studies and for subsequent use in the production of cDNA probes.

Chris L. Baszczynski, Carol A. Rees-Farrell and N. Cathy Hogan

Production of polyclonal antibodies to maize heat shock proteins

In an effort to avail ourselves of additional tools for studying the response of maize to thermal shifts and/or stresses, some of the maize heat shock polypeptide (HSP) classes have been isolated and purified to produce polyclonal antisera. This is a laborious procedure since the HSPs in maize are synthesized in low amounts and do not accumulate with time as in Drosophila. To purify the HSPs, polypeptide extracts (labelled with ^{35}S-methionine) were electrophoresed on 1-D gels as described previously (Baszczynski et al., 1982). The regions of the gels corresponding to the HSPs were cut out, and the polypeptides were electro-eluted from the gel slices (in a Tris-acetate buffer containing 0.1% SDS) and simultaneously concentrated using an ISCO sample concentrator. The concentrated samples were made 2 mM with respect to PMSF, an aliquot of each sample was re-electrophoresed and purity was checked by fluorographic analysis of the separated polypeptides. This procedure was repeated until high purity was achieved. Four groups of polypeptides were prepared for use as antigens: (a) the 18 kD HSP class, (b) the 73-76 kD class, (c) the 84-89 kD class, and (d) the 73-89 (high molecular weight) class. Alum precipitates of the purified antigens were made. These were emulsified 1:1 with complete Freund's adjuvant and injected sub-cutaneously into New Zealand White female rabbits. The rabbits were boosted intramuscularly at 6 weeks and at 12 weeks post-injection, and then bled one to two weeks later. The sera were collected, treated at 56 C for 30 minutes to inactivate complement and stored at -20 C until required.

Analysis of the sera by ring tests and Ouchterlony immunodiffusion plates indicated the presence of precipitating antibodies in the sera. Immunoblot analysis of electrophoretically separated polypeptides revealed the presence of antibodies specific to the molecular weight classes of polypeptides against which the antibodies were produced. To screen for the presence of antibodies specific to the maize HSPs (which are detected only by fluorography), total protein extracts from control or heat-shocked seedlings were reacted independently with each of the antisera, the mixtures treated with a cell wall preparation from Staphylococcus aureus to precipitate antigen-IgG complexes, the resulting immunoprecipitates collected and dissolved in lysis buffer, and the samples electrophoresed and analyzed by fluorography.

The results from these studies indicate that: (a) the sera contain antibodies specific to the maize HSPs, (b) there is some cross-reactivity between the various molecular weight classes of HSPs, (c) the high molecular weight HSPs are present in small amounts in non-heat-shocked samples, and (d) there is no detectable synthesis of the low molecular weight 18 kD HSP class in non-heat-shocked material. This suggests that the high molecular weight HSPs exhibit enhanced synthesis following heat shock, while the 18 kD HSP class represent the activation of a set of previously quiescent genes.

The maize HSP antibodies are being used (a) to examine cross-reactivity among different tissues of maize, (b) to compare the immunological similarity in HSPs between different plant and animal species, (c) to purify mRNAs for the HSPs by immunoprecipitation of polysomes from heat-shocked tissues, and (d) for affinity purification of large amounts of HSPs to be used for the production of monoclonal antibodies.

Chris L. Baszczynski

Developmental shifts in the patterns of protein synthesis of maize inbreds and hybrids

Last year we reported preliminary results obtained in a study characterizing shifts in the pattern of protein synthesis occurring as a function of development in some maize inbreds and hybrids (MNL 57:163-164, 1983). The technique employed involved the dissection of embryos from kernels at successive stages following pollination and labelling them with ^{35}S-methionine in Murashige and Skoog medium. When the work was continued this past summer, a different technique was adopted which, it is believed, more closely approximates conditions found in vivo. This procedure was previously described by Kriz (MNL 56:14-15, 1982) and entails the labelling of intact kernels while attached to an ear which has been removed from the plant. ^{35}S-methionine is introduced into the kernel through injection with a Hamilton syringe. For our purpose, optimal incorporation was achieved with 3 ul of undiluted ^{35}S-methionine injected into the embryo side of the kernel, followed by a 2 hr incubation period. Embryos were subsequently dissected from labelled kernels, homogenized in plant extraction buffer (MNL 55:117, 1981) and protein samples subjected to one and two dimensional electrophoresis.

The cultivars employed in this study included Oh43, M14 and W23, as well as some of the reciprocal hybrids produced from crosses of the latter two with Oh43. Results obtained from the previous summer indicated that a minimum of 3-5 days between sampling times is required to observe significant changes in the patterns of protein synthesis. The earliest at which individual embryos could be manipulated was found to be 17 days post-pollination. For these reasons, we selected embryos at 17, 20, 25, 30, 35 and 40 days post-pollination. Samples were arranged on one-dimensional gels to allow analysis of the effects of genotype, developmental stage and period during the growing season over which maturation occurred, on the pattern of protein synthesis. Results indicate that both qualitative and quantitative changes in the synthetic pattern occur during the course of development in all genotypes. Variation between genotypes is also observed, this being mainly quantitative in nature. Hybrids appear to exhibit patterns intermediate to the parental inbreds, but more closely resembling the female parent. In some instances the stage during the growing season at which pollinations were made appeared to affect the rate at which development occurred, as determined by the protein synthetic pattern.

J. Boothe, D. B. Walden and B. G. Atkinson

Chromosome assignment of the genes for the maize heat shock proteins using monosomics

In conjunction with D. F. Weber, we have begun the gene assignment for HSP's using the monosome (generated by the r-X-1 system) strategy. Thus far the data suggest that there is not a structural gene for an HSP on Chromosome 2.

Carol A. Rees-Farrell

Temperature stress to plants at the 6-8 leaf stage produces a response in leaves

Previous reports on temperature stress responses in maize examined the alterations in the pattern of polypeptide synthesis in tissues of 5- to 6-day-old maize seedlings (Baszczynski et al., 1982, 1983). A similar response has now been observed in leaf tissue from entire plants at the 6-8 leaf stage subjected to a brief shift in growing temperature.

Seeds (Oh43) were germinated in flats. Seedlings were transplanted to large pots and grown outside. Leaf samples were taken and labeled at 25 C (100 uCi/ml, ^{35}S-methionine, 2 h). The plants were then placed in an incubator at 45 C. After one hour, leaf samples were labeled at 45 C (100 uCi/ml, ^{35}S-methionine, 2 h). Protein extracts from these leaf samples were subjected to two-dimensional IEF-SDS PAGE followed by fluorography.

Examination of the patterns of polypeptide synthesis revealed enhanced synthesis of seven classes of polypeptides after temperature shift: 108, 89, 84, 76, 73, 23, 18 kilodaltons (kD). Enhanced synthesis of six classes of polypeptides (excluding the 23 KD class) was revealed in plumules and radicles of 5- to 6-day-old seedlings subjected to a one-hour temperature shift (27 C to 41 C) (Baszczynski et al., 1982). Enhanced synthesis of the 23 kD polypeptide class may represent a leaf specific response or a response specific to the temperature shift used in this experiment. Work by others in our laboratory has demonstrated enhanced synthesis of a polypeptide of similar size (23 kD) after a temperature shift from 30 C to 42 C in plumules of 5-day-old maize seedlings (see previous contributions in this Newsletter).

Carol A. Rees-Farrell and D. B. Walden

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University of Wisconsin

Maize endosperm mutants affecting soluble carbohydrate content as potential additives in preparing silage from high protein forages

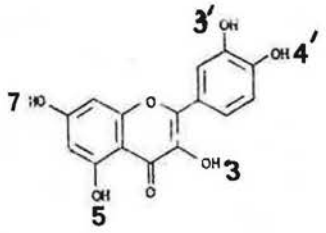
High protein forages, like alfalfa, have long been recognized as marginal with respect to the carbohydrate supply underlying the fermentation process leading to the prompt formation of lactic acid on which preservation, palatability, and nutritional value of high protein silages are dependent. An exploratory study was made of maize endosperm mutants as potential additives to the chopped, high protein forage at ensiling time as a protection against this deficiency. The criterion applied in evaluating mutants for this special purpose, in addition to suitability for production as a farm crop for local use, was content of sugars plus phytoglycogen (a highly branched, water-soluble polysaccharide) in the mature seed. Nine loci were brought under review, namely amylose extender (ae), brittle (bt), brittle-2 (bt2), dull (du), shrunken-2 (sh2), sugary (su), sugary-2 (su2), waxy (wx), and sugary enhancer (se). Attention came to focus on the sugary locus (su), which is well known as the site of the mutant underlying common sweet corn. About one-half the dry matter in mature su kernels is soluble carbohydrate (sugars plus phytoglycogen), a value about 10 times that for dent corn. The su mutant, however, is deemed impractical for use as a high protein forage additive in spite of its very high content of soluble carbohydrates. A basic finding in the present study is that there is a numerically close and inseparable, inverse relationship between soluble carbohydrate content and rate of water loss from the fully developed seed. Because drydown is a major factor affecting suitability of a strain for machine harvesting and subsequent processing of the grain, this adverse relationship imposes an upper limit on the soluble carbohydrate value that would

be generally acceptable in a strain for local use as a high protein forage additive. The su mutant present in common sweet corn clearly exceeds this acceptable limit. A compromise, therefore, between the two opposing variables, high soluble carbohydrate content and drydown, was sought. Experimental data were obtained which indicate that an allele termed sugary-Brawn (su-Bn2) will meet the closely coupled, double requirement. The su-Bn2 mutant yields well, gives an approximately five-fold increase in soluble carbohydrates over dent corn, and markedly increases drydown as compared with su. Field observations suggest that harvestability of su-Bn2 would be made dependable if the mutant were incorporated in strains whose silking dates were about 10 days earlier than characteristic for adapted dent corn. A second intermediate mutant at the su locus, termed sugary crown (su-cr), was found to contain about three times as much soluble carbohydrate as dent corn and is somewhat more rapid in drydown than su-Bn2. These high soluble carbohydrate mutants are visualized for use as additives in the form of ground, dry grain, incorporated in the freshly chopped forage at ensiling time. Silage prepared from snapped ear shoots with green husks and containing mature, high moisture kernels is an optional form of additives that should be further tested.

R. A. Brink

Minor flavonoid glucosyltransferases in maize aleurone and endosperm tissues

The role of the enzyme uridine diphosphate glucose:flavonol 3-O-glucosyltransferase (UFGT) in flavonoid and anthocyanin synthesis has been extensively documented (Larson and Coe, Biochem. Genet 15:153, 1977; Dooner and Nelson, Biochem. Genet. 15:509, 1977). The enzyme acts on the 3 position of flavonoid compounds and is required for anthocyanin stabilization. The purpose of this report is to describe two additional flavonoid-dependent, glucosyltransferases found in extracts of aleurone and endosperm tissues.



Quercetin

Recently we modified the method for isolation and assay of UFGT (Klein and Nelson, PNAS 80:7591). The principal change in our procedure was to replace Tris buffer with Hepes; this improves the stability and yield of UFGT. In the Tris buffer system, no UFGT activity is detectable in preparations from bz1 endosperms (Dooner and Nelson, ibid.; Klein and Nelson, unpublished data). However, with our new protocol, we could now detect low levels of flavonoid-dependent glucosyltransferase activity in bz1 endosperms.

The assay utilizes ^{14}C-labelled UDPglucose and the flavonol quercetin as substrates (Klein and Nelson, ibid.). These minor glucosylated compounds are not resolved from quercetin by paper chromatography in 15% acetic acid; following two dimensional separations two bright yellow, fluorescent spots are visible which are distinct from quercetin, with Rf values as follows:

	15% HOAc	n-Butanol:acetic:water (4:1:5)
Quercetin	0.02	0.68
A	0.10	0.42
B	0.03	0.39
Isoquercitrin (in Bz1 only)	0.39	0.51

Even with two dimensional chromatography, resolution of these compounds is poor; this has hampered attempts to isolate enough of each to identify them by spectral properties, etc. It is clear, however, that these represent glucosylation of the flavonol substrate at some position other than the 3 hydroxyl. The latter compound, isoquercitrin, is well separated under these conditions from the unknown compounds.

In order to assess the possible role(s) of the minor flavonoid glucosyltransferases we surveyed three different genotypes for the distribution of these activities. Dr. Coe generously provided the double mutant bz1 bz2.

	Flavonoid glucosyltransferase activity, mU per endosperm (nmoles substrate incorporated per hour)		
Genotype	A	B	UFGT
bz1 bz2	2.8	1.7	none detected
bz1 Bz2	4.1	2.7	none detected
Bz1 Bz2	none detected	6.7*	438

*Indirectly estimated.

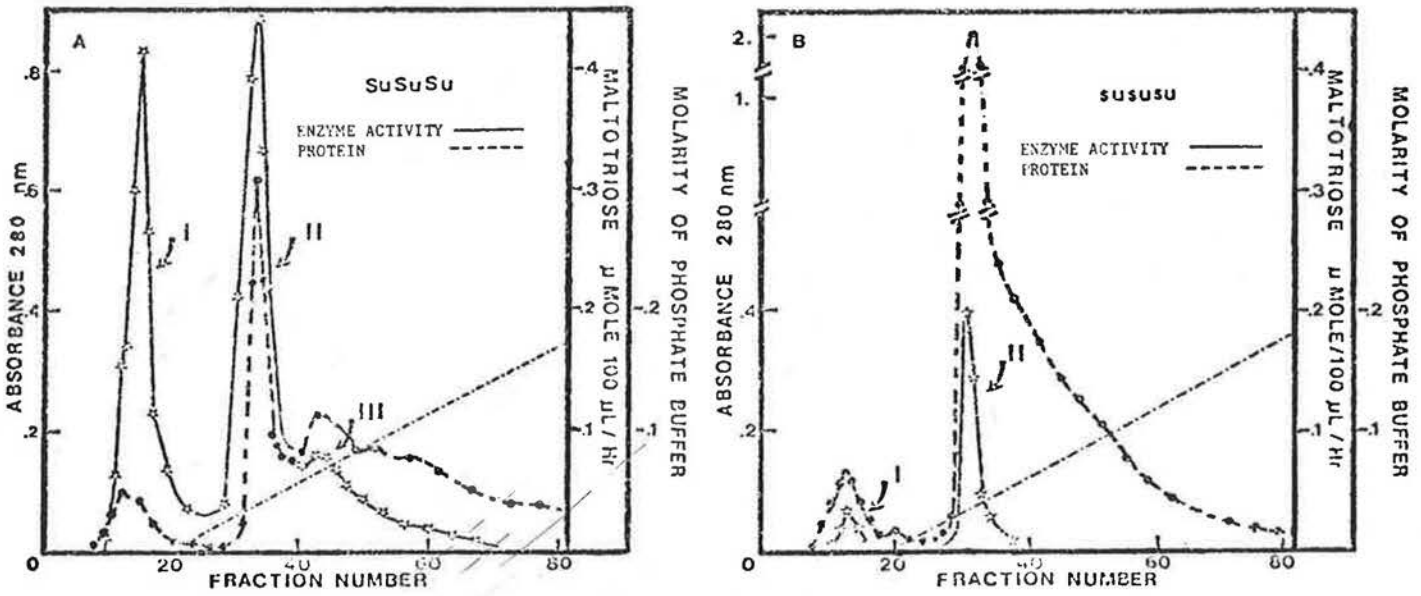
Both activities (A and B) were found at similar levels in the single bz1 and double mutant bz1 bz2. Thus, neither activity appears to be correlated with the bz2 mutation. Only one radioactive compound, other than isoquercitrin, appeared on chromatograms from assays of Bz1 Bz2 extracts; this had a mobility similar to compound B. The high level of UFGT activity in Bz1 Bz2 extracts may compete with activity A for substrate, reducing the level of product A below the level at which it can be detected. Furthermore it is not clear whether any of these glucosides may be modified at more than one position.

Dooner (Phytochem. 18:749, 1979) described three minor flavonoid-dependent glucosyltransferase activities in extracts prepared from bz1 embryos. One activity appeared to be an isozyme for UFGT; the other two activities produced bright yellow, fluorescent compounds which he identified as the 3'- and 7-glucosides of quercetin. He did not detect these activities in extracts from Bz1 or bz1 endosperms (with aleurone). The two endosperm flavonoid-glucosyltransferase activities we described above produce compounds which have somewhat different mobilities than those described by Dooner. The function of these minor flavonoid-glucosyltransferase activities in vivo has not been established. These enzymatic functions may be involved with the synthesis of six diglucosides of quercetin which Styles and Ceska tentatively identified in Bz pollen (Maydica 26:141, 1981). Some of these compounds were glucosylated at the 3', 4' and/or 7 positions as well as the 3 position of the flavonoid rings.

Anita S. Klein and Oliver E. Nelson, Jr.

Localization of debranching enzymes in nonmutant and sugary mutant endosperms

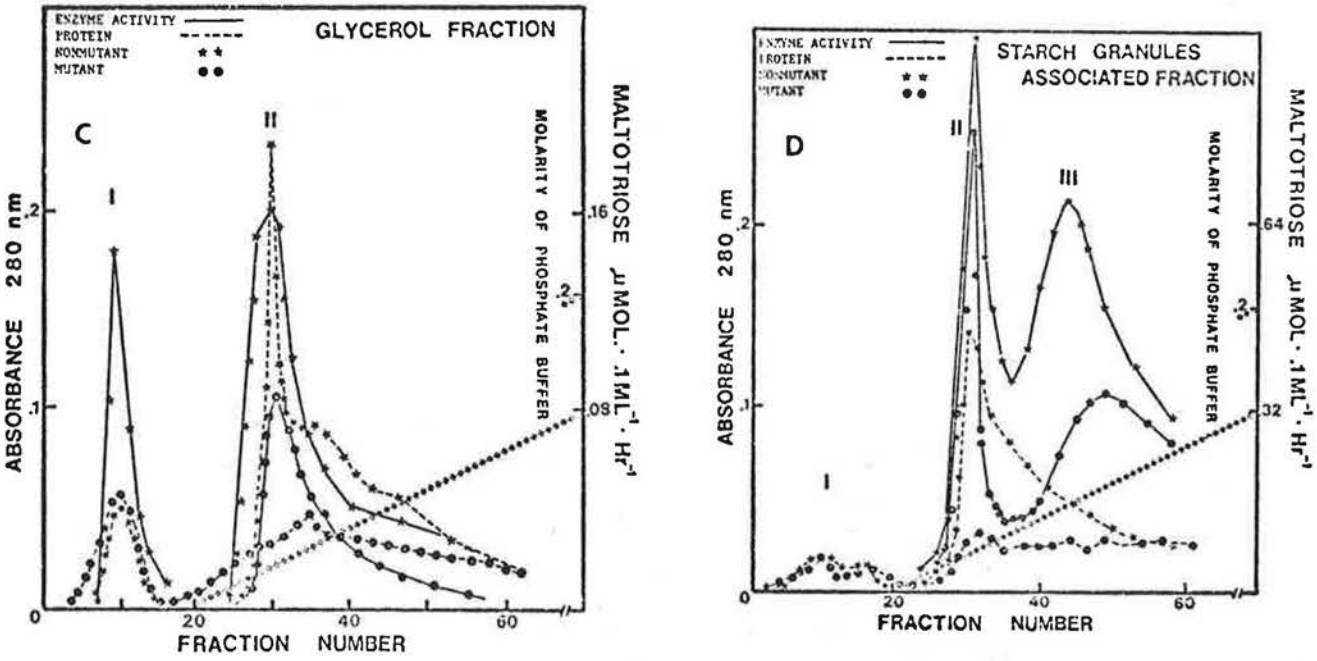
In an earlier study (Plant Physiol., 1984), we have shown that the debranching enzyme (DBE) activity from nonmutant endosperms can be separated into three peaks (I, II, and III) on a hydroxyapatite column (Fig. A). The extract of sugary endosperms lacks one (peak III) of these peaks of activity while the other two (I and II) have much reduced activity, particularly peak I (Fig. B).



A nonaqueous procedure using glycerol as an extracting solvent (Plant Physiol. 67:518-524, 1981) was employed for the isolation of starch granules and the localization of DBE activity from nonmutant and sugary endosperms. The endosperms were initially extracted with anhydrous glycerol in a Virtis blender, followed with three glycerol washes; the glycerol-washed supernatants were combined and considered as glycerol extract (cytosol fraction); the starch granule pellets were finally washed with 0.1 M citrate buffer, pH 6.0 four times. The combined buffer-washed fraction was designated as starch granule-associated fraction (amyloplast enzyme). The extract and buffer-washed fraction from starch granules were further subjected to ammonium sulfate fractionation and DEAEcellulose and hydroxyapatite column chromatography as previously reported. In the nonmutant inbred W64A, two soluble peaks (peak I and II) and two starch granule-associated peaks (peak II and III) of DEB activity were found in the glycerol extract (cytosol enzyme) and the buffer-washed fraction from starch granules (amyloplast enzyme) respectively, indicating that peak I is mainly present in the cytosol compartment whereas peak III enzyme is essentially localized in amyloplasts; peak II is apparently distributed in both compartments. In the sugary mutant, only peak II enzyme showed in the glycerol extract while peak II and III both appeared in the buffer-washed fraction.

A comparison of the level of DBE activity in peaks I, II and III in the glycerol extract and the buffer-washed fraction from starch granules, from nonmutant and sugary mutant endosperms, indicates the deficiency of peak I (cytosol compartment enzyme), and a much reduced activity of peak III (amyloplast enzyme) in the sugary mutant. A detailed analysis of the elution patterns of peaks I and III, which were prepared by two different extraction procedures (aqueous and nonaqueous), on hydroxyapatite columns implies that there is a reciprocal relationship in enzyme activity between peaks I and III in that for Su/Su/Su endosperms column chromatography of buffer extracts shows substantial

peak I and lower peak III activity (Fig. A). However, there is very low peak III activity in the glycerol washings, and the buffer wash of the glycerol-extracted starch has little peak I activity and higher peak III activity than the buffer extracts of the endosperms (Figs. C and D). We can only speculate at the present



time as to the reason for the differences in peaks I and III activity with two different extraction techniques. It is possible that Su and su code for a polypeptide which as a homodimer constitutes peak III only in the absence of cytosol components (nonaqueous extraction), but can combine with the product, present in the cytosol compartment, of another locus to form a heteromeric enzyme (peak I) at the expense of peak III when the endosperms were buffer-extracted. The study on the characteristics of these three peaks is in progress.

David Pan

A d-BASE II program for storing and retrieving information on genetic stocks

The example program is a simplified version of one in use by O. E. Nelson. The program and the format of the data are easily modified to individual needs. There is no need to worry about ordering your files because stock information is seldom retrieved using a unique key that the file can be sorted on. This program retrieves stock information using a genotypic description. The retrieved information is the full genotypic description and the stock number of this plant's seed packet and those of its parents. All records containing the target description are retrieved on the first search. Additional target description information may be specified after each search until the remaining stocks are the handful desired. On each pass you may specify that retrieval is to be done on the basis of two traits. The traits may be specified to include this trait or exclude this trait in seed, pollen, or either seed or pollen. In between operations the program will give you the option of searching again, turning the printer on or off, or starting over with a fresh set of genotypic descriptions.

This program is written in dBASE II (c) Ashton-Tate. That makes it very portable; it will run on any personal computer with a dBASE II interpreter without

```
STRUCTURE FOR FILE:   NELSON DBF
FLD          NAME            TYPE          WIDTH
001        STOCKID             C             012
002        SEED                C             050
003        POLLEN              C             050
004        SEEDID              C             012
005        POLLID              C             012


********************** SEARCH.CMD ************************
*  Cunningham,Jan 1983
*  Modified   Dec 1983,Cunningham
*  This code written for Oliver Nelson, Univ.Wisconin-Madison
*  This procedure searches the data base for traits in corn
*  growing records.  It will run on any DBASE (c) interpreter.
set talk off
save all to memsav
relese all
dele template
dele workfile
use NELSON
eras
copy stru to template
copy to template
copy stru to workfile
use workfile
? 'This is the searching program. All you  need do  is read  the'
? 'messages as  it runs and it  should  be  fairly easy to  use.'
? 'There may be a few places  where after you give it a  command'
? 'it will wait for a carriage return. If it seems to be waiting'
? 'for something try pressing the carriage return.'+chr(10);
+chr(10)+chr(13)
stor 'n' to hardcopy
stor t to running
do while running
stor ' ' to funct
? 'ENTERING A CARRIAGE RETURN FOR THE FIRST TRAIT PASSES;
 THE SEACH MODE'
? 'ENTERING A CARRIAGE RETURN FOR THE SECOND TRAIT SEARCHES ;
ON FIRST TRAIT ONLY'
acce 'ENTER DESIRED GENOTYPIC TRAIT ' to trait1
read
if len(trim(trait1))>1.or.trait1>0
? '  SELECT SEARCH USAGE       A) find in seed;
      C) find in pollen          E) find in seed or pollen'
? '                             B) NOT found in seed;
  D) NOT found in pollen    F) NOT found in seed or pollen'
wait to funct
do case
case (funct='a'.or.funct='A')
stor '('+trim(trait1)+'$seed)' to trait1
case (funct='b'.or.funct='B')
stor '(.not.('+trim(trait1)+'$seed))' to trait1
case (funct='c'.or.funct='C')
stor '('+trim(trait1)+'$pollen)' to trait1
case (funct='d'.or.funct='D')
stor '(.not.('+trim(trait1)+'$pollen))' to trait1
case (funct='e'.or.funct='E')
stor '(('+trim(trait1)+'$seed).or.(';
+trim(trait1)+'$pollen))' to trait1
case (funct='f'.or.funct='F')
stor '(.not.(('+trim(trait1)+'$seed).or.('+trim(trait1);
+'$pollen)))' to trait1
otherwise
? 'UNCLEAR INSTRUCTIONS. PLEASE TRY AGAIN'
loop
endc
acce 'ENTER ANOTHER GENOTYPIC TRAIT ' to trait2
read
if len(trim(trait2))>1.or.(trait2>0)
? '  SELECT SEARCH USAGE       ;
using the same choices as the first trait'
wait to funct
do case (funct='a'.or.funct='A')
stor '(('+trim(trait2)+'$seed).and.'+trim(trait1)+')' to trait1
case (funct='b'.or.funct='B')
stor '((.not.('+trim(trait2)+'$seed)).and.;
'+trim(trait1)+')' to trait1
case (funct='c'.or.funct='C')
stor '(('+trim(trait2)+'$pollen).and.'+trim(trait1)+')' to trait1
case (funct='d'.or.funct='D')
stor '((.not.('+trim(trait2)+'$pollen)).and.;
'+trim(trait1)+')' to trait1
case (funct='e'.or.funct='E')
stor '((('+trim(trait2)+'$seed).or.('+trim(trait2)+'$pollen));
case (funct='f'.or.funct='F')
stor '((.not.(('+trim(trait2)+'$seed).or.('+trim(trait2);
+'$pollen))).and.'+trim(trait1)+')' to trait1
otherwise
eras
? 'UNCLEAR INSTRUCTIONS. PLEASE TRY AGAIN'
```

```
loop
endc
endif
? '              please wait       SEARCHING STOCK RECORDS'
appe from template for &trait1
go top
endif
if (eof)
? 'No finds using specified criteria.  RESETTING for another try'
else
go bott
? 'retrieved',#,' stocks '
go top
stor t to viewing
do whil viewing
? '                                        OPTIONS '+CHR(10)+CHR(13)
? 'N)  research retrived stocks using additional;
 criteria                  V)  to view retrieved stocks'
? 'R)  to reset for a search on all new criteria;
                           P)  to toggle the printer on or off'
? 'S) to jump to a different stock in the work ;
set than the next one      Q)  to exit the program
stor ' ' to operate
wait to operate
do case
case (operate='P'.or.operate='p')
if (hardcopy='N').or.(hardcopy='n')
set prin on
stor 'Y' to hardcopy
else
set prin off
stor 'N' to hardcopy
endif
case (operate='r'.or.operate='R')
? 'RESETTING FOR SEARCH ON NEW SET OF CRITERIA'
stor f to viewing
use NELSON
dele workfile
copy stru to workfile
use workfile
case (operate='n'.or.operate='N')
stor f to viewing
case (operate='q'.or.operate='Q')
rele all
rest from memsav
set talk on
return
case (operate='S'.or.operate='s')
? 'CURRENT RECORD IS ',#
acce 'ENTER RECORD TO MOVE TO ' to jmpval
read
goto &jmpval
case (operate='V'.or.operate='v')
stor 'y' to moreview
do whil moreview='y'.or.moreview='Y'
stor ' ' to moreview
? '                    STOCK NUMBER: '+stockid+chr(13);
+chr(10)+chr(10)+'SEED   GENOTYPE: '+seed
if len(trim(pollen))>1.or(pollen>'0')
? '                    ----------------------------------------'
? 'POLLEN GENOTYPE: '+pollen
endif
? chr(10)+'SEED STOCK NUMBER: '+seedid
if len(trim(pollid))>1.or.(pollid>'0')
? 'POLLEN STOCK NUMBER: '+pollid+chr(13)+chr(10)
endif
? 'ENTER Y TO SEE THE NEXT RECORD'
wait to moveview
if (moreview='y'.or.moreview'Y').and.(.not.(eof))
skip
else
stor ' ' to moreview
endif
endd
endc
endd
dele template
copy stru to template
copy to template
use template
dele workfile
copy stru to workfile
use workfile
endd
```

modification. Data entry is done with the dBASE II append command and is very simple. Data may contain spaces and all printable characters except ";". The stock numbers may contain letters and "-" if you wish. The data files for the stocks are created using the create command and may be edited using the edit command. Data may not be changed inside the program. The dBASE II commands are explained in the manual that comes with the interpreter.

The file the program will use will be called NELSON.DBF. dBASE II will take care of the .DBF part of the name and placing an entry in the disk directory.

Search time on the first pass is about 3 minutes for Dr. Nelson's records from 1970 through 1982; subsequent passes are much faster. We will be happy to supply a complete copy of the version we use at U.W. to anyone who requests one. We use 8 inch floppy disks formatted for a CP/M system.

Randolph Cunningham

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Allegheny College

Heat shock proteins in mitochondria

Hybrid vigor occurs when the F1 is superior in growth, productivity, resistance to disease, or some other measurable attribute. It has been shown that hybrid corn seedlings manifest hybrid vigor in plumule length and dry weight/germinated seed, as well as seedling protoplast poly A+ RNA content and transcriptional response to the phytohormone gibberellic acid (Nebiolo, 1983, Plant Science Letters 28:195, 1982/83). Several investigators have implicated mitochondria in the generation of hybrid vigor (Sarkissian, Genetics 57:843, 1967). In corn, the inheritance of mitochondrial DNA is strictly maternal (Pring, Genetics 89:121, 1978). However, the maternally derived mitochondria may be affected by a hybrid environment. Mitochondria are the primary target to heat shock stimuli (Leenders, Sub-Cell Biochem. 3:119, 1974). A non-dialyzable, heat-labile activity has been isolated from heat shocked mitochondria which, when injected into non-heat shocked salivary gland cells, induces heat shock puffs (Sin, Nature 258:159, 1975). Isolated mitochondria can be subjected to a cell-free translation system to detect proteins synthesized from mitochondrially-expressed mRNA (Forde, PNAS 75:3841, 1978).

Isolated mitochondria of two inbred (W23 and OH43) and two heterotic hybrid lines (W23 X OH43 and OH43 X W23) of maize (generously supplied by David Walden, Western Ontario University, London, Ontario) incorporate ^{35}S-methionine into acid-insoluble material at significant rates for 90 min at 27 C. Heterosis was observed in plumule length of imbibed pedigree seed stock of the four lines for up to 96 hr. Purified mitochondria from 4-day seedlings were obtained by differential centrifugation and banding through a discontinuous sucrose gradient according to Forde. Respiratory activity was determined by measurement of O_2 consumption by a Clarke electrode of mitochondria incubated in an osmotically stable solution containing ADP/succinate as energy source. ^{35}S-methionine incorporation by mitochondria incubated according to Forde was sensitive to 10 ug/ml chloramphenicol and insensitive to 200 ug/ml cyclohexamide. Genotype-specific differences were observed in rate and total amount of incorporation at 90 min, with mitochondria of W23 and W23 X OH43 incorporating 20-30 times the amount of label incorporated by OH43 and OH43 X W23. No effect of hybridity was observed. Pelleted mitochondria were subjected to 15% SDS-PAGE electrophoresis after 30 min of incubation in label mix containing 19 amino acids, ^{35}S-methionine and ADP/succinate. Eighteen to 20 proteins were detectable by fluorography. No

qualitative or quantitative differences among lines were observed. Hybrid vigor is not manifested in mitochondrial protein synthesis.

Utilizing the cell-free mitochondrial translation system developed as outlined above, we characterized the maize mitochondrial response to heat shock. Actively respiring purified mitochondria (200-250 ug protein) were incubated for 10 min at 27 C in label mix without ^{35}S-methionine. After this short equilibration, mitochondria were incubated in water baths at either 27 C or 37 C for 10 min, followed by addition of 5 uCi ^{35}S-methionine, and were returned to the water baths. The 40 min incubation at 37 C, after a 10 min incubation at 27 C, constitutes a heat shock. Heat shock occurs rapidly; transcripts of heat shock genes are observable within 3-5 min. Sin (op. cit.) incubated salivary gland mitochondria for 30 min prior to injection of supernatant and induction of heat shock puffs in non heat-shocked tissues. A significant increase in rate of acid-insoluble ^{35}S-methionine incorporation at 37 C compared to 27 C was observed in this study in all lines.

Mitochondria incubated at both 37 C and 27 C were pelleted (10,000 rpm, 5 min) and dissolved in SDS sample buffer, and an aliquot was taken for measurement of radioactivity and subjected to SDS-PAGE electrophoresis and fluorography. Proteins in the supernatants of pelleted mitochondria were extracted and electrophoresed. Our results include: 1) a significant difference between genotypes in ^{35}S-methionine incorporation at 30 min, similar to acid-insoluble incorporation data discussed above; 2) a significant increase in ^{35}S-methionine incorporation at 37 C compared to 27 C for all lines; 3) a significant amount of ^{35}S-methionine detectable in supernatants of mitochondria incubated at 37 C, not detectable at 27 C; 4) detection of a strongly labelled protein, estimated molecular weight 52 Kdaltons, in fluorographs of electrophoresed mitochondria of all lines incubated at 37 C, not detectable at 27 C; 5) detection of a 52 Kdalton protein in mitochondrial supernatants incubated at 37 C, not detectable at 27 C. This protein is estimated to account for 70-80% of the labelled protein in the supernatant at 37 C, while accounting for only 10-20% of total labelled protein in the intact mitochondria. Synthesis of this protein induced at 37 C is sensitive to chloramphenicol and insensitive to cyclohexamide. This protein may be analogous to a short-lived heat shock protein (50 Kdaltons) observed by Meyer (Plant Phys. 72:26, 1983) in tobacco mesophyll protoplasts. Further investigation may strengthen the implication that this protein is a mitochondrial effector of heat shock gene expression. No qualitative effect of hybridity on proteins synthesized in response to heat shock by mitochondria is evident. Further characterization of this protein, including sensitive quantitation, may provide more information pertaining to the possible differential response of hybrids and inbreds to this stress phenomenon.

Christine M. Nebiolo and Elizabeth M. White

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Extent of haplo-diploid gene expression in maize

In recent years evidence has been collected which suggests that in higher plants a part of the genome is expressed in both phases of the life cycle. However, to establish the biological significance of this phenomenon, it is important to determine what proportion of the genome is shared by the sporophytic and gametophytic generations.

The expression of genes controlling dimeric (or multimeric) enzymes can be detected by comparing the electrophoretic patterns of pollen and sporophyte from

heterozygous F/S plants: three bands are expected in the sporophyte, only two in the gametophyte in the case of haplo-diploid determination; the same pattern if the enzyme is of diploid origin; different banding when different genes are expressed in pollen and in diploid tissues. The possibility that causes other than haploid transcription may be responsible for these results can be ruled out by including pollen with heterozygous gene duplication in the comparison of electrophoretic pattern. This pollen type can be obtained by using B-A translocations (Sari Gorla et al., MNL 57:94-95). This method, tested on ADH-1, allowed us to establish the haplo-diploid expression of GOT-1, while beta-GLU proved to be controlled by different genes in pollen and sporophyte.

Subsequently, PHI was studied by means of this system. It, too, revealed haplo-diploid expression for the gene: two bands for normal pollen from F/S plants, three bands, as in the sporophytic tissues, for hyperploid pollen.

Evidence of the same type of expression has been obtained with regard to GDH, which is probably an esameric enzyme.

MDH expression was studied by means of the IEF technique. In fact in maize at least five forms of this enzyme exist, which form heterodimers between allelic and non-allelic isozymes within the same subcellular location (mitochondria and cytosol). Thus the resulting electrophoretic pattern is quite complex and difficult to analyze by means of the described method. The pollen-sporophyte comparison of F1 plants derived from two inbreds showing different IEF patterns for only one band, revealed three bands in the sporophyte and two in the pollen. This is an indication that at least one MDH form is expressed in the gametophytic phase, and it should be MDH-4 (Goodman classification), since it is a soluble enzyme and the two parental lines showed different electrophoretic variants only for this isozyme.

UDPGpp was considered, also because of its possible significance in pollen biology. We screened a very large number of genotypes (124 inbreds, 8 popcorn, 22 samples from Argentine and ten from Italian populations, about twenty defective kernel mutants in different background) without finding any variant form. It seems that there is no "permissible" variability of the gene coding this enzyme, which is evidently active in both pollen and sporophyte.

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

Effect of exogenous proline and water stress on protein metabolism in normal and pro mutant root tips

The proline requiring mutants (pro) identifiable on the basis of their collapsed endosperm morphology are seedling lethal monogenic recessive mutants. Their growth is resumed if L-proline is added to the medium (Gavazzi et al., Theor. Appl. Genet. 46:338-395, 1975). Free and protein amino acid composition established after feeding root tips with ^{14}C glutamate and ^{14}C ornithine show that proline biosynthesis is not blocked in pro mutants (Dierks Ventling C. and Tonelli C., Plant Physiol. 69:130-134, 1982).

We now present preliminary data on the electrophoretic analysis and protein metabolism of newly synthesized proteins in root tips under normal and mild water stress (-8 BARS) conditions and in the presence or absence of 1mM L-proline. Excised root tips of pro1-1 mutants and normal siblings in W22 inbred line were grown for 24 hours on liquid media and then incubated for 22 hours in (^{3}H) L-amino acids mixture or (^{35}S) L-methionine. The mitochondrial enriched fraction was isolated according to Nagahashi and Hiraike (Plant Physiol. 69:546-548, 1982). SDS PAGE electrophoresis and fluorography of the newly synthesized proteins of the mitochondrial and soluble fractions were carried out according to Bertani et al. (Z. Pflanzenphysiol. 103:37-43, 1981).

The results obtained can be summarized as follows:

1. The presence of proline in the medium induces a change in the polypeptide pattern of soluble and mitochondrial enriched fraction in both mutant and normal siblings.
2. Mutant root tips show a significantly higher uptake and protein incorporation of the labeled amino acids.
3. During water stress the uptake, protein incorporation and efficiency of incorporation are reduced in normal root tips while in mutants they appear unaffected.
4. A comparison of the polypeptide pattern of the mitochondrial and soluble fraction in mutant and normal siblings discloses only minor quantitative changes. No major changes in the polypeptide pattern appear thus associated to the mutational event leading to proline requirement.

Further experiments are underway.

Chiara Tonelli and Alcide Bertani

Opaque endosperm sectors induced by B-chromosomes

Non-disjunction of B chromosome centromeres in the maize male gametophyte has been used to develop powerful methods for chromosome gene location and gene expression studies. The presence of B chromatin in the nucleus may also cause non-disjunctions of the A chromosomes during endosperm development. The event can be detected as sectors on the endosperm when heterozygous combinations for chromosome markers expressed in this tissue are used. A method for chromosome gene location based on this phenomenon can be developed; the advantage is twofold: 1) it can be used to locate genes lying on chromosome segments not covered by the actual TB-A stocks, and 2) comparisons for biochemical traits can be made between endosperm sectors of the same kernel differing only in the chromosome involved in the non-disjunction.

The method has been tested by using two marker genes both located on the short arm of chromosome 7: o2 and Zp20-1, where Zp20-1 is proximal to the centromere at 13 map units from o2. Kernels from the cross between the inbreds 33-16 (o2, zp20-2) and OH-43 (TB-9; O2, Zp20-1) were scored for opaque sectors; the frequency of kernels showing half opaque endosperm phenotype was about 0.001. Four endosperms in which the two phenotypical parts could easily be separated were analyzed using the immunological test to detect the presence of the cytoplasmic protein b-32 (this protein is absent when the locus opaque-2 carries the recessive allele) and the IEF method to reveal the zein polypeptide coded by Zp20-1. While the biochemical opaque-2 phenotypes fitted the expectation (in the opaque sectors the protein b-32 was absent and in the normal sectors it was clearly revealed) the IEF patterns of the sector phenotypes did not differ, i.e. opaque and normal endosperm tissues both showed the zein band 20-1.

These results indicate that the whole chromosome 7 was not involved in the genetical event leading to the formation of the endosperm opaque sectors analyzed, and could be explained on the basis of a chromosomal loss or a mitotic crossing-over.

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Location of pro in relation to Bif

The pro mutant, a proline requiring recessive lethal, has been located on chromosome 8 by means of crosses with a set of B-A translocations (MNL 52:66-67) on a region not uncovered by the TB-8a translocation. The recombination between the mutant and the breakpoint amounts to 27.2% (MNL 53:63). Here we present data on the location of the mutant in relation to a dominant gene mutant affecting the morphology of the male inflorescence. The mutant, known to lie on chromosome 8 but not yet located as to map position, is symbolized Bif (Barren Inflorescence). Females heterozygous for pro were crossed to +/Bif males. Progeny plants pro +/+ Bif in genotype were outcrossed to normal female parents, progeny grown, classified for Bif and selfed to establish their pro constitution. The following results were obtained:

Bif +	+ pro	Bif pro	+ +	n
54	53	17	11	135

They lead to an estimate of 20.7 (20/135 x 100) map units distance between pro and Bif.

G. Gavazzi, G. Todesco and M. G. Neuffer

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Studies on oil quality in maize and Oriental genera of Maydeae

Very few systematic studies have been done on the Asiatic relatives of maize (MNL 57:96-104, 1983). Practically no work has been done on oil quality aspects. The quality of oil is important from the viewpoint of human nutrition as well as the stability of oil during storage. It has been shown that linoleic acid, an essential fatty acid, is hypocholesteremic and palmitic acid is hypercholesteremic, while oleic acid and stearic acids appear to have little effect on blood cholesterol level.

Both the cultivated and wild forms of Coix have traditionally been used by tribal people for human consumption and medicinal purposes. Considering these facts, oil content and fatty acid composition have been studied in maize and Oriental genera of Maydeae, namely, Coix lacryma-jobi L. (wild Coix), Coix lacryma-jobi var. 'ma yuen' Stapf (cultivated Coix), Trilobachne cookei (Stapf), and Chionachne koenigii (Thwaites).

Maize is found to have more oil content than the other genera of Oriental Maydeae studied. But oil content in cultivated Coix and wild Coix is considerably more than in Chionachne and Trilobachne.

Fatty acid composition of maize and its Asiatic relatives revealed that palmitic acid is higher in Trilobachne and lower in both cultivated and wild Coix than in maize. Stearic acid was considerably lower in all the Oriental genera of Maydeae than maize, and oleic acid was the lowest in normal maize. Cultivated Coix has more than two-fold and Trilobachne had 1.7 times the oleic acid content

of maize. Maize had the highest linoleic acid of the genera of Maydeae. Wild Coix and Chionachne had about 0.8 fold the linoleic acid content of maize.

Table. Fatty acid composition and fat content in maize and Oriental genera of Maydeae

Cultivated	Oil %	Palmitic C16:0	Stearic C 16:1	Oleic C 18:1	Linoleic C 18:2	Oleic/Linoleic
Coix	3.45	13.2	1.1	61.5	24.2	2.54
Wild Coix	3.77	16.4	0.6	40.5	42.5	0.95
Chionachne	0.07	14.8	0.6	43.7	41.9	1.04
Trilobachne	1.27	19.0	0.8	51.1	29.1	1.76
Maize	4.81	16.81	3.28	29.18	50.73	0.58

The oleic to linoleic acid ratio, which gives an idea of storage quality of oil, is higher in all the Oriental genera of Maydeae. Maize has the least oleic to linoleic acid ratio, showing its lower stability. Wild Coix, which has a considerably higher amount of oil as well as higher linoleic acid, can be explored for extracting a nutritionally better quality of oil.

N. D. Sharma and J. K. S. Sachan

Modified C-banding procedure for Zea chromosomes

Sachan and Tanaka (Japan J. Genet. 51:139-141, 1976) have described a Giemsa staining procedure to discern constitutive heterochromatin in Zea chromosomes. This modified procedure differs from the original one basically at three steps. (1) the use of monobromonaphthalene in place of 8-hydroxyquinoline for pretreatment, (2) the use of glacial acetic acid for fixation in place of Carnoy's Fluid I, and (3) a relatively higher concentration of barium hydroxide for a longer period of time. The procedure laid down here produces much more distinct bands compared to the earlier one.

Pretreatment: Seeds are germinated at 25 C and excised root tips are pretreated in saturated monobromonaphthalene solution (2 drops per 5 ml of water) for 3-4 h at 10 C.

Fixation: In glacial acetic acid for 24 h at 10 C. Fixed root tips are stored in 70% ethanol till further use.

Maceration: In 5% (1:1 cellulase and Pectinase, pH 6.8) at room temperature for 30-90 min.

Washing: In water for 10-15 min at room temperature.

Squash: Roots were squashed in 45% acetic acid and pressed hard.

Air drying: Coverglass is flipped off by dry ice or after freezing in liquid nitrogen, and slides were air dried for 24-48 h.

Alkali treatment: Slides were placed in 7% barium hydroxide aqueous solution for 10-12 min at 50 C.

Washing: Alkali was thoroughly washed with water for 10-15 min with 3-5 changes.

Saline treatment: Slides were incubated in 2xSSC (0.3M NaCl - 0.03M Sodium Citrate) at 60 C for 75 min.

Washing: Slides were washed in water for 10-15 min with 3-5 changes.

Staining: Washed slides were soaked in phosphate buffer pH 6.8 for 1 min and stained in 1.5% Giemsa (E. Merck) for 10-15 min at room temperature.

Air drying: After brief rinsing in water slides were air dried for 12-24 h.
Mounting: Slides were transferred to xylene for 5-10 min and mounted in Canada balsam.

J. K. S. Sachan and K. R. Sarkar

Origin of knob-heterochromatin in Zea chromosomes

The constitutive heterochromatin (or repetitive DNA sequences) discernible by knob-heterochromatin, C- and Q-bands in Zea has been the subject of speculation for many years. Much variation in shape, size and position of C-heterochromatin is found in both maize and teosinte. The evolution of repeated sequences by unequal crossing over has been indicated by Smith (Science 191:528-538, 1976), showing the possibility of increase or decrease of tandem repeats of repetitive DNA.

We have studied the distribution of C-heterochromatin in different races of maize and teosinte. Our observation of Imbricado, which has subterminal bands, and Confite Morocho, which has terminal bands on the 6th and 7th chromosomes (though both are from the high land of the Andes), suggests a model of crossing over between large terminal banded types and bandless types (Fig. 1) giving rise to two resultant chromosome types, one type having fine subterminal and the other having fine terminal C-heterochromatin.

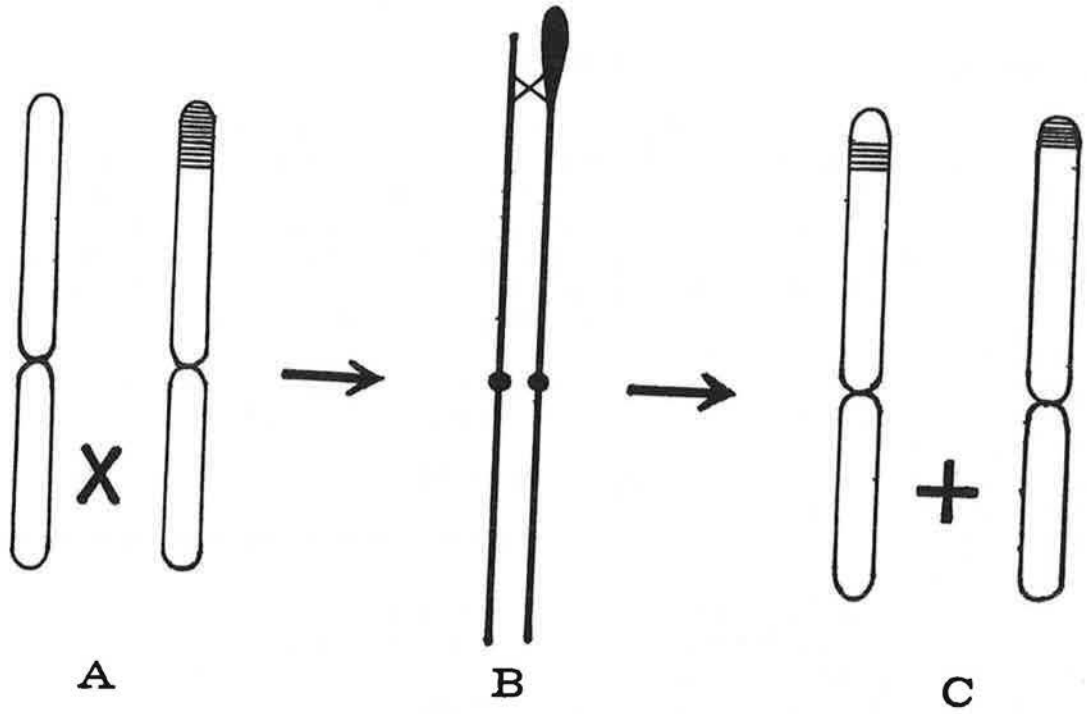

Fig. 1. Proposed Model of Origin of Knobs

A. Pairing between terminally knobbed and knobless homologous chromosomes; B. Crossover involving subterminal region between the terminally knobbed and knobless chromosomes; C. Results in formation of fine subterminal and terminal knobs.

Suppose that the proposed bandless type of parent had a short repetition of DNA not manifested as a C-band or knob, homologous to a DNA repetition in the band on

the terminally banded chromosome type. Chance recombination could have occurred in low frequency at this heterochromatic region and after many generations of unequal crossing over at meiosis, given rise to fine subterminal and terminal bands. The structure of condensation of heterochromatin blocks, permitting crossing over only at the distal region of the band, with the main heterochromatic block looping out at the time of pairing, means that fine subterminal and terminal bands could result. This would explain the variable size of bands in different races as products of varying degrees of crossing over of the heterochromatin. Also Miklos and Nankivell's (Chromosoma 56:143-167, 1976) observation could explain why knobs/bands are inherited in Mendelian fashion and recombination does not normally occur within a knob to yield subterminal and terminal knobs in an observable frequency.

Andy Pereira and J. K. S. Sachan

A new approach for increasing grain yield in maize

We feel that the traditional approaches of plant breeding are of empirical types since they have not utilized the knowledge of physiological and morphological characters related to economic yield of crops. One has, therefore, to look forward for some new components which have been ignored so far to achieve higher yield. For example, it has been found in maize that the initial number of ears per plant, number of ovules per ear and actual ear length are usually more than the effective number of ears per plant, number of grains per ear and effective ear length, respectively. If these initial characters are somehow realized in the hybrids, through high conversion capacity (of number of ovules into number of grains, for example) without reducing the 100 grain weight, that would help in increasing the grain yield.

With these objectives in mind we gathered information on thirteen such characters in four inbreds, namely CM 202, CM 111, CM 400 and CM 300 and their two F1 single crosses (CM 202 x CM 111 and CM 400 x CM 300). It was found that the inbreds, as well as the hybrids, possessed significantly fewer effective ears per plant, grains per ear and shorter effective ear length in comparison to the corresponding number of total ears initiated per plant, number of ovules formed per ear and actual ear length, respectively (Table 1). A perusal of the data also

Table 1. Mean performance(of two years) of the parents and their crosses for yield components and grain yield.

Genotypes	Harvest index (%)	No.of initial ears/plant	No.of effective ears/plant	Percentage effective ears/plant (%)	No.of ovules/ ear	No.of grains/ ear	Percent grain conversion/ ear (%)
1	2	3	4	5	6	7	8
CM 202	42.79	1.89	1.06	66.08	585.06	303.23	51.82
CM 111	36.93	1.68	1.08	64.28	675.37	449.70	66.61
CM 202 x CM 111	37.79	2.40	1.20	50.00	773.38	599.96	77.56
CM 400	49.18	2.08	1.09	52.40	520.40	378.76	72.78
CM 300	35.42	2.16	1.21	56.02	539.78	400.48	74.15
CM 400 x CM 300	47.03	2.44	1.34	54.92	586.86	490.40	83.54
C.D.at 5%	3.44	0.86	0.26	6.73	13.65	13.82	6.53

Table 1. Continued.

Available ear length (CM)	Effective ear length* (CM)	Proportion of effective ear length (%)	Ear diameter (CM)	100 grain wt.(g)	Grain yield/ plant (g)
9	10	11	12	13	14
15.94	14.22	89.20	4.53	23.04	68.52
18.20	17.66	97.03	4.41	27.98	114.56
22.90	21.55	94.10	4.98	28.92	172.31
15.12	13.41	88.69	3.85	18.15	80.46
16.68	15.46	92.68	4.13	25.45	99.84
20.04	17.94	89.52	4.47	32.82	175.34
0.94	0.76	6.94	0.14	0.70	16.27

't' values between (i) No.of initial ears per plant and no.of effective ears per plant= 2.21*
(ii) no.of ovules per ear and no.of grains per ear = 5.01**
(iii) actual ear length and effective ear lenth = 4.58**

* effective ear length: the length of ear upto which grains were formed.

indicates that CM 400 possessed a fairly high number of grains per ear because of its high harvest index, fairly high percentage of grain conversion and high proportion of effective ear length. Similarly the cross CM 202 x CM 111, which possesses the highest number of ovules per ear and high percentage grain conversion, could also possess the highest number of grains per ear. It therefore appears that the hybrid still possesses some scope for improvement in other characters like harvest index and percent effective ears per plant. CM 400 x CM 300 also gives a similar picture. Hence, we feel that parents having high performance in any of these components should be selected for the breeding programs to evolve genotypes with fairly high mean values in all these components (along with high 100 grain weight) to increase the present day grain yield of maize.

B. C. Saha and B. K. Mukherjee

Plantlets from Teo-maize anthers in vivo

A very interesting phenomenon has been observed in some of the progenies derived from maize and teosinte crosses (teo-maize). Plantlets were observed to have emerged from the spikelets in place of normal anthesis (Figures A and B). Such a bunchy appearance is confounded with the crazy top disease of maize. These plantlets grow, produce roots and mature on the mother plant itself. Both male and female meiosis is normal (Figure C) and seeds are obtained on the diminutive ears. Each plantlet attains a height ranging from 40-60 cm on the mother plant itself. However, when such plantlets are removed from the tassel and transplanted to the field they grow into an almost normal plant (Figure D) and produce fertile progeny. Further studies on such plantlets emerging from spikelets of the teo-maize tassel will be helpful in maintaining specific genotypes.

H. K. Singh

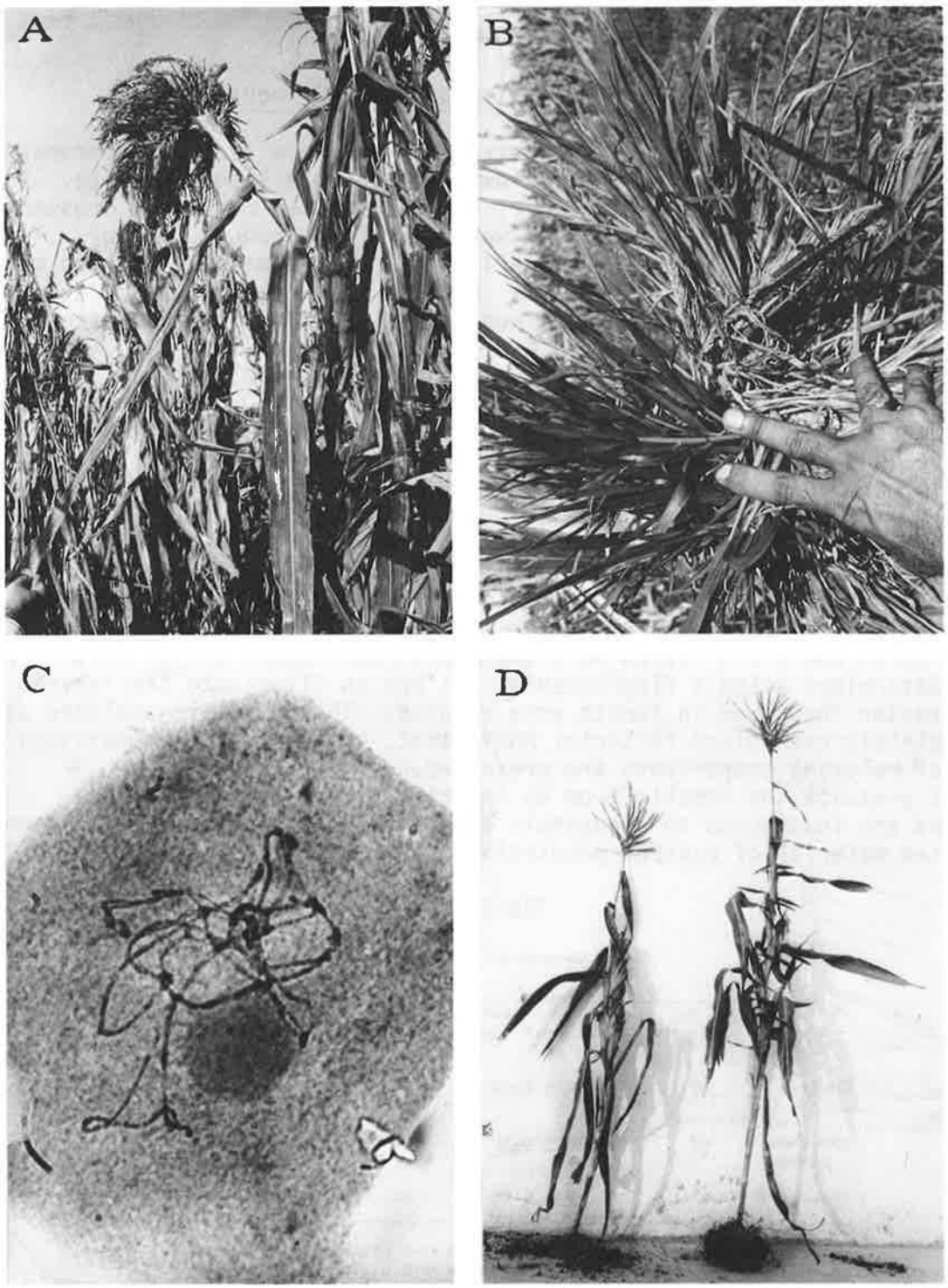

Figure A. Emergence of plantlets from anthers.
B. Close-up of the plantlet coming out of anther.
C. Normal meiosis (pachytene) in PMCs of the plantlets obtained from anthers.
D. Transplanted plantlets showing normal growth.

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Effect of B-chromosomes on induced mutation at the Yg2 locus

This report presents our initial observations on the effect of B chromosomes on induced mutation frequencies at the yellow-green-2 (yg2) locus in maize. Inbred W22 plants (Yg2/Yg2) which contained 0 or 8 B chromosomes were each crossed as female parents by a second inbred which was yg2/yg2 to produce Yg2/yg2 heterozygotes which contained 0 or primarily 4 B chromosomes. These two kernel types will be designated as 0-B and 4-B. Because these two types are the result of a cross between two highly inbred lines, they should be isogenic except for the presence of B chromosomes.

The 0-B and 4-B kernels were mutagenized and grown under rigorously controlled conditions. The kernels were soaked in aerated water at 20 C for 72 h. After this treatment, the primordia of leaves four and five have been estimated to consist of 3,000 and 500 cells, respectively (Plewa et al., MNL 57:147-149, 1983). The kernels were then treated with 10 mM ethylmethane sulfonate (EMS) in a 0.1 M phosphate buffer (pH 7.4) for 8 h at 20 C under aeration, or with 250 rads of 137 gamma irradiation. A concurrent control was included. Following the treatment, the kernels were rinsed in running tap water for 30 min and were placed in soil in 10 cm plastic pots. The pots were coded and placed in a plant growth chamber at 20 C with a 17 h photoperiod. After 20 to 25 days of growth, mature (with ligules present) fourth and fifth leaves were analyzed. The number of yg2 sectors per leaf was determined using a fluorescent light box to illuminate the leaves. Only sectors greater than 1 mm in length were counted. The data were analyzed using a 2 x 3 completely randomized factorial ANOVA test, and pair-wise comparisons (t-test) of relevant comparisons are presented.

Table 1 presents the results from an experiment of this type. These populations are inadequate to accurately estimate spontaneous mutation frequencies in untreated material of control populations. However, the frequency of yg2

TABLE 1

YELLOW-GREEN SECTORS ON LEAF FIVE

	0-B Plants		4-B Plants	
	No. plants	Mean no. sectors per leaf ± SE	No. plants	Mean no. sectors per leaf ± SE
Control	67	0.03 ± 0.01	63	0.03 ± 0.00
250 rads gamma	67	0.09 ± 0.10	65	0.23 ± 0.02*
10 mM EMS	65	0.49 ± 0.08	62	1.19 ± 0.15**

* Different from value in comparable 0-B plants at $P \leq 0.05$.
** Different from value in comparable 0-B plants at $P \leq 0.01$.

YELLOW-GREEN SECTORS ON LEAF FOUR

	0-B Plants		4-B Plants	
	No. plants	Mean no. sectors per leaf ± SE	No. plants	Mean no. sectors per leaf ± SE
Control	69	0.06 ± 0.01	68	0.02 ± 0.00
250 rads gamma	68	0.68 ± 0.10	65	0.85 ± 0.11
10 mM EMS	65	1.62 ± 0.20	63	3.59 ± 0.54**

sectors in EMS-treated plants of the 4-B group was significantly higher ($P \leq 0.01$) than the 0-B group in both leaves four and five. The mutation frequency in gamma-radiated material was significantly higher in leaf five, and higher, but not significantly different in leaf four. These results indicate that the presence of B chromosomes in the maize genome makes the yg2 locus more susceptible to induced mutation by EMS, and possibly by ionizing radiation.

The results of a second experiment are presented in Table 2. The male parent crossed onto 0-B and 8-B females in the W22 inbred genetic background to produce this experimental material was of a different origin from the male used to produce the material analyzed in Table 1. Significantly higher ($P \leq 0.025$) frequencies of yg2 sectors were present in the 4-B group than in the 0-B group in both gamma-irradiation and EMS-treated material.

TABLE 2

YELLOW-GREEN SECTORS ON LEAF FOUR

	0-B Plants		4-B Plants	
	No. plants	Mean no. sectors per leaf ± SE	No. plants	Mean no. sectors per leaf ± SE
Control	24	0.04 ± 0.04	28	0.07 ± 0.07
250 rads gamma	12	0.25 ± 0.18	29	1.00 ± 0.22*
10 mM EMS	18	2.83 ± 0.35	29	4.03 ± 0.36*

* Different from value in comparable 0-B plants at $P \leq 0.025$.

B chromosomes in maize are relatively genetically inert because maize plants that contain low numbers of B chromosomes are phenotypically indistinguishable from plants without B chromosomes. However, they are not completely inert because certain processes have been identified which are controlled by factors located on B chromosomes (reviewed by Carlson, Ann. Rev. Genet. 16:2-23, 1978). One known effect of B chromosomes is that recombination is increased in certain segments of the A chromosomes when B chromosomes are present (Ayonoadu and Rees, Genetica 39:75, 1968; Hanson, Genetics 63:601, 1969; and others). We speculate that the B chromosome-mediated increases in recombination may be related to increased mutation rates. It may simply be that the presence of additional chromatin due to the presence of B chromosomes imposes stress on the DNA replicative and/or repair mechanisms, which causes the cells to be more susceptible to mutagens.

These observations are also relevant to the "bodyguard" hypothesis of Hsu (Genetics 79:Suppl. 137, 1975). Hsu proposed that because heterochromatin is located adjacent to the nuclear membrane in interphase, it may function as a bodyguard to absorb environmental insults (mutagens) to protect the centrally-located euchromatin. This hypothesis specifically refers to protection against chemical mutagens and not to ionizing radiation. B chromosomes of maize are largely composed of heterochromatin. Hsu suggested that an excellent material to test the "bodyguard" hypothesis would be maize, and he stated, "if the bodyguard hypothesis is correct, then the mutation rate induced by chemical mutagens should be inversely correlated with the number of B chromosomes." The experimental results in these experiments with EMS are exactly the opposite of what would be expected if the bodyguard hypothesis is valid; thus, the current experiments do not support the "bodyguard hypothesis." The experimental results with gamma irradiation are also in disagreement with this hypothesis because the bodyguard hypothesis predicts that ionizing radiation would efficiently penetrate the cell and not be influenced by the presence of heterochromatin.

David Weber and Michael Plewa

Effect of hypoploidy and hyperploidy of specific segments on embryo and kernel volumes

We have been screening the maize genome for genes which express dosage effects on the content and composition of the oil of the kernel, and we have reported the identification of such factors (Plewa and Weber, Can. J. Genet. Cytol. 15:313; Genetics 81:211; Shadley and Weber, Can. J. Genet. Cytol. 22:11; MNL 55:126). Nearly all of the oil of the kernel is located in the embryo. The percent oil of the kernel can depend upon the amount of oil present in the embryo, the size of the embryo and the percent embryo of the kernel. Because of the negative correlation between percent oil of the kernel and the levels of linoleic acid of the oil (Alexander, Corn and Corn Improvement, 1977) these factors may also affect the composition of the oil. Therefore, we were interested in determining if dosage of regions of the maize genome known to alter kernel oil also affected kernel and embryo traits.

Kernels from tester ears segregating for TB-5La, TB-10L(19), or one of three compound TB-5La-3L translocations (Shadley and Weber, MNL 54:89; 55:124) were analyzed. In each case kernels from an ear were weighed and hydrated for a 24 hr period. The hydration was necessary for measuring kernel and embryo volume using the procedure of Plewa and Weber (Can. J. Genet. Cytol. 15:313). A dissecting microscope equipped with an ocular micrometer and set at 10X total magnification was used. The length and width of each kernel and embryo were measured. A longitudinal section was then made parallel and adjacent to the embryonic axis. The portion of the kernel containing the embryonic axis was used to measure the kernel and embryo depth. All measurements were made at the greatest linear distance. The products of the three measurements were used as an estimate of embryonic and kernel volumes, and will be designated "embryo and kernel volume." The ratio of the embryo volume to the kernel volume was used to calculate the percent embryo. The measured portion of the kernel containing the embryonic axis was germinated. Chromosome counts were obtained from primary root tip meristematic cells using the standard Feulgen squash technique. All measurements made on kernels from a single ear were done during a single sitting. Students' t-tests were used for statistical analysis. One ear of each translocation type was analyzed. Tables 1-5 present the observations. For all B-A translocations analyzed, kernels with hypoploid endosperm and hyperploid embryos had significantly lower kernel weights, kernel volumes and embryo volumes than sibling kernels with hyperploid endosperm and hypoploid embryos ($p = 0.001$ in all cases). Where measured, kernels with hypoploid endosperms and hyperploid embryos also had significantly lower kernel weights, kernel volume, and embryo volumes than those with euploid endosperms and euploid embryos. However, when kernels with hyperploid endosperms and hypoploid embryos were compared to those with euploid endosperms and euploid embryos, no significant differences were found. For all B-A translocations analyzed, the values for percent embryo were not significantly different between any of the kernel types.

The lower weights of the hypoploid endosperm kernels are consistent with the findings reported by others (Birchler, Genet. Res. 36:111; Lin, Genetics 100:475; Beckett, Can. J. Genet. Cytol. 25:346) and are apparently due to a disturbance of the 2:1 maternal to paternal ratio for the chromosome segment whose dosage is being altered. The embryo volume in all cases was reduced in proportion to the kernel volume. The disturbance in the endosperm has an effect on the embryo through the reduction in kernel size. As a result the percent embryo of the kernel remains the same for all kernel types. The data in Table 2 show that there are no significant differences in the traits measured between kernels heterozygous for the translocation (10 10B B10) and those not bearing the translocation (10 10). Therefore, the translocation itself does not affect the traits measured, nor does the presence of the B chromosome.

TABLE 1 TB5La

KERNEL TYPE	NO. ANALYZED	KERNEL WT. (mg)	KERNEL VOL. (μM)	EMBRYO VOL. (μM)	% EMBRYO
ENDOSPERM: 5 5 5B EMBRYO: 5 5B B5 B5	20	187.5 ± 3.2*	31.44 ± 0.59*	11.52 ± 0.34*	36.7 ± 0.9*
ENDOSPERM: 5 5 5B EMBRYO: 5 5B	17	221.7 ± 6.4	39.21 ± 0.14	14.14 ± 0.38	36.6 ± 1.3

*mean plus or minus standard error

TABLE 2 TB10L(19)

KERNEL TYPE	NO. ANALYZED	KERNEL WT. (mg)	KERNEL VOL. (μM)	EMBRYO VOL. (μM)	% EMBRYO
ENDOSPERM: 10 10 10B EMBRYO: 10 10B B10 B10	10	182.9 ± 4.3	30.24 ± 0.62	8.29 ± 0.23	27.5 ± 0.8
ENDOSPERM: 10 10 10B B10 B10 EMBRYO: 10 10B	6	269.7 ± 4.8	44.69 ± 0.86	12.66 ± 0.79	28.3 ± 1.6
ENDOSPERM: 10 10 10B B10 EMBRYO: 10 10B B10	12	272.6 ± 3.4	43.37 ± 1.16	11.81 ± 0.41	27.2 ± 0.7
ENDOSPERM: 10 10 10 EMBRYO: 10 10	10	269.0 ± 2.2	44.56 ± 0.79	12.64 ± 0.22	28.4 ± 0.6

*mean plus or minus standard error

TABLE 3 TB5La (.1-.57) 3L (b)

KERNEL TYPE	NO. ANALYZED	KERNEL WT. (mg)	KERNEL VOL. (μM)	EMBRYO VOL. (μM)	% EMBRYO
ENDOSPERM: 5 5 5B 3 3 3,5 EMBRYO: 5 5B 3,5 3 B5,3 B5,3	15	97.1 ± 1.7	23.76 ± 0.08	7.49 ± 1.14	32.1 ± 0.4
ENDOSPERM: 5 5 5B 3 3 3,5 B5,3 B5,3 EMBRYO: 5 5B 3,5 3	18	11.53 ± 2.5	27.45 ± 0.95	8.55 ± 0.22	31.5 ± 0.8

*mean plus or minus standard error

TABLE 4 TB5La (.1-.48)3L (5521)

KERNEL TYPE	NO ANALYZED	KERNEL WT (mg)	KERNEL VOL. (μM)	EMBRYO VOL(μM)	% EMBRYO
ENDOSPERM: 5 5 5B 3 3 3,5 EMBRYO 5 5B 3 3,5 B5,3 B5,3	15	88.2±3.2	19.93±0.75	7.45±0.29	37.5 ± 1.2
ENDOSPERM: 5 5 5B 3 3 3,5 B5,3 B5,3 EMBRYO: 5 5B 3 3,5	11	133.7±2.6	29.26±1.30	9.76±0.20	34.0 ± 1.5
ENDOSPERM: 5 5 5B 3 3 3,5 B5,3 EMBRYO: 5 5B 3 3,5 B5,3	9	132.8±2.9	29.31±0.80	10.03±0.41	34.2 ± 1.0

*mean plus or minus standard error

TABLE 5 TB5La(.1-.61)3L (7043)

KERNEL TYPE	NO ANALYZED	KERNEL WT (mg)	KERNEL VOL.(μM)	EMBRYO VOL. (μM)	% EMBRYO
ENDOSPERM: 5 5 5B 3 3 3,5 EMBRYO: 5 5B 3 3,5 B5,3 B5,3	15	87.8 ± 3.3	21.47±0.08	7.59±0.20	35.9 ± 0.1
ENDOSPERM: 5 5 5B 3 3 3,5 B5,3 B5,3 EMBRYO: 5 5B 3 3,5	10	11.68± 2.0	27.86±0.14	9.68±0.29	35.5 ± 0.2
ENDOSPERM: 5 5 5B 3 3 3,5 B5,3 EMBRYO: 5 5B 3 3,5 B5,3	10	11.37± 4.4	25.02±0.09	9.54±0.53	38.1 ± 0.2

*mean plus or minus standard error

Plewa (Ph.D. dissertation, 1975, Illinois State University) found no significant difference in percent oil of the kernel between kernel types segregating for TB-5La. Given this and the data in Table 1, the smaller hyperploid embryos must have a lower amount of oil than the other embryo types. The amount of oil they synthesize is proportional to their embryo size. Therefore, there are no gene(s) on this segment of 5L that express dosage effects upon oil concentration.

Plewa also reported in his dissertation that kernels with embryos hyperploid for TB-10L(19) had a significantly higher percent oil than their hypoploid embryo sibs. However, the milligram oil content in these two kernel types was the same. We found that the hyperploid embryos are smaller than the hypoploid embryos, but both make up the same percent of the kernel. The hyperploid embryos do not synthesize oil in an amount proportional to their size. A gene(s) on this segment of 10L which expresses dosage effects on oil concentration may be responsible for this. The hypoploid embryo kernels may not have a lower content of oil compared to the euploid or diploid due to their equivalent embryo size, which may compensate for the missing copy of the gene(s). Using monosomics, Plewa and Weber (Can. J. Genet. Cytol. 15:313) reported that kernels with embryos monosomic for chromosome 10 had significantly lower percent oil than their diploid sibs.

Altered dosages of the regions of the maize genome reported here have an effect upon kernel size. The effect of this upon the percent oil of the kernel and the oil content depends upon the presence of genes in these regions of the genome which express dosage effects upon oil of the kernel. In the case of TB-5La the small kernel yields a small embryo, but, because there are no genes expressing dosage effects on oil in this region of 5L, the size of the embryo determines the amount of oil present. Oil content is affected but not percent oil. In the case of TB-10L(19) a gene(s) expressing dosage effects upon oil overrides the effect of the small embryo. Percent oil is affected but not oil content. The small kernel effect does not prevent the detection of genes acting on oil of the kernel as long as one compares percent oil values.

J. D. Shadley and D. F. Weber

A proposed method to determine the time of occurrence of nondisjunction induced by the r-X1 deficiency

The r-X1 deficiency is an X-ray induced submicroscopic deletion of the R locus on chromosome 10. When deficiency-bearing gametes are fertilized, a large percentage of the resultant embryos are aneuploid. Because there is non-correspondence between embryo and endosperm markers, and because approximately equal numbers of trisomic and monosomic embryos are produced, it appears that the r-X1 deficiency induces chromosomal nondisjunction after meiosis is completed during the embryo sac (megagametophyte) divisions (Weber, in Maize for Biological Research, p. 79, 1982). It is not known if the nondisjunctive event is restricted to one specific post-meiotic division.

The following assumptions are used in this discussion: a) only one nondisjunctional event occurs during the development of the embryo sac, b) the two nuclei move to opposite poles after the first megagametophyte division, c) after the third division any one of the four nuclei at each pole has an equal probability to migrate to the center of the embryo sac to become a polar nucleus, and d) any one of the four nuclei at the micropylar end has an equal probability to form the egg.

If nondisjunction occurs during the first megagametophyte division, all eggs will be aneuploid, one polar nucleus will be nullisomic (n-1), and the other polar nucleus will be disomic (n+1). When the two polar nuclei are fertilized by a haploid pollen grain, euploid endosperm will invariably be produced. If

nondisjunction occurs at the second megagametophyte division, all nuclei at one pole of the embryo sac will be euploid and all nuclei at the other pole will be aneuploid. When nuclei migrate from each pole of the mature embryo sac to form the polar nuclei, one polar nucleus will invariably be aneuploid and the other euploid; thus, endosperm formed from such an embryo sac will invariably be aneuploid. Half of the embryos would be aneuploid. Finally, if nondisjunction occurs at the third division, all the nuclei at one pole will be euploid and half of the nuclei at the other pole will be aneuploid, and half of the kernels formed from such embryo sacs will have aneuploid endosperm. One fourth of the embryos would be aneuploid.

If one selects kernels that are monosomic for a specific chromosome, one can predict the chromosomal constitution of the endosperm assuming that nondisjunction occurred at specific divisions. If nondisjunction occurred at the first division, the endosperm invariably would be euploid. If nondisjunction occurred at the second division, the endosperm invariably would be aneuploid for the monosomic chromosome. Half of the time the endosperm would be hypoploid and half of the time it would be hyperploid. If nondisjunction occurred at the third division, the endosperm would be aneuploid for the monosomic chromosome half of the time and euploid half of the time.

Thus, the division at which nondisjunction occurs can readily be determined if a suitable genetic marker can be utilized which expresses clearly-defined dosage effects in the endosperm. Endosperm of kernels monosomic for this chromosome could be analyzed. A suitable gene for this purpose would be the Y locus on chromosome 6 which controls the level of carotenes in the endosperm of the kernel. Kernels homozygous for a particular recessive allele of this locus, y-pastel-8549, have white endosperm and produce pale-green seedlings when germinated at 37 C (Robertson, MNL 34:73). We have crossed r-X1 bearing plants which were R/r-X1; Y/Y by male parents that were r/r; Y/y-pas. The purple (R/r) kernels will be discarded and the anthocyaninless (r/r-X1) kernels will include kernels with embryos that are monosomic for chromosome 6. Monosomic 6 embryos that are y-pas/- will give rise to pale-green seedlings when germinated at 37 C. Root-tip cells from such seedlings will contain 19 chromosomes and only one satellited chromosome (chromosome 6). Endosperm samples from such kernels obtained prior to germination will be analyzed for their levels of carotenes by high performance liquid chromatography (HPLC). We have analyzed the levels of carotenes in kernels that are Y Y Y, Y Y y, Y y y, or y y y with HPLC, and we have found that it is possible to distinguish unambiguously the different kernel types. As expected, we find a strict dosage relationship between the number of dominant alleles and the amount of carotene present. Single kernel analysis by this method appears to be quite feasible. The data obtained will allow the precise determination of the number of Y alleles present (which is the number of chromosome 6's contributed by the maternal parent) in kernels monosomic for chromosome 6.

If nondisjunction of chromosome 6 takes place at the first megagametophyte division, the endosperm of kernels with monosomic 6 embryos will invariably contain two Y alleles. If it takes place at the second division, these kernels will have one or three Y alleles in their endosperm. If it takes place at the third division, half of the kernels will have two, a fourth will have one, and a fourth will have three Y alleles in their endosperm. In this way we hope to precisely define the division at which the r-X1 induced nondisjunctional event takes place.

J. D. Shadley, K. Simcox and D. F. Weber

Placement of sporophyte mutants within the long arm of chromosome 5

We would like to report our preliminary results on the use of compound B-A translocations to localize genes on the long arm of chromosome 5. We are working with ethylmethane sulfonate (EMS)-induced mutants generated by Dr. M. G. Neuffer, who provided us with M2 populations segregating for the various mutants. He utilized TB-5La to place these mutants to the long arm of chromosome 5. To avoid confusion, we will use his allele designations and E numbers. This report will be limited to seedling mutants.

The accompanying table gives a short description of the mutant phenotypes as we see them, allelism, and approximate map locations on the long arm of chromosome 5. We have generated a series of compound B-A translocations involving the long arm of chromosome 5 as described in Shadley and Weber (MNL 55:124, 1981). The compound B-A translocations were derived by crossing TB-5La-containing plants with specific reciprocal translocations which have one of their breakpoints in the long arm of chromosome 5 distal to the breakpoint of TB-5La. Embryos hypoploid for the compound B-A translocations are hypoploid for the segment between the breakpoints, and are not hypoploid for the segment distal to the breakpoint. By utilizing compound B-A translocations with different breakpoints, it is possible to generate plants hypoploid for different portions of the long arm of chromosome 5. We believe that this approach has great merit and could be used in maize to generate segmental aneuploids in much the same way as is being done in Drosophila melanogaster by Lindsley et al. (Genetics 71:157, 1972). Specifically, we have generated and are using the following B-A translocations with the following breakpoints: 1) TB-5La-3L5521 with breakpoints at 0.1 and 0.48, 2) TB-5La-3Lb with breakpoints at 0.1 and 0.57, and 3) TB-5La-3L7043 with breakpoints at 0.1 and 0.61.

MUTANT	PHENOTYPE	ALLELIC TO	MAP LOCATION
w 21	white seedling	w 22	proximal to 0.48
w 22	same as w 21	w 21	"
w sect 206	variegated leaf, varied expression	?	distal to 0.61
wl gr lf tp 1308	majority of leaf white, tip and sometimes margin green	gr wl 199 ? (see text)	"
wlv 308	pale green to virescent-like, more virescent-like, seedlings smaller than normal sibs	?	"
wlv 473	same as wlv 308	pgv 735 (MNL 56)	"
gr wl 199	varied expression, pale green to albino-like	wl gr lf tp 1308 ?	"
gl 166	good glossy	gl 8	proximal to 0.48
pg 296	pale green seedlings small, some die	other pg listed here	distal to 0.61
pg 408	pale green fades as plant matures	"	"
pg 71	good expression, variable in extremes, can be seen in mature plant, some seedlings show zebra-like expression	"	"
pg 735	good expression, not as strong as pg 71, fades as plant matures	"	"
tn 409	pale green bands develop which become necrotic (see text)	tn 493	5S
tn 493	similar to tn 409	tn 409	"
gl 681	glossy plants smaller than normal sibs	?	5S

All four pale-green mutants (pg*-296, pg*-408, pgv*-71, and pgv*-735) were found to be allelic, however, some of the other families supplied by Neuffer also segregated for pale-green seedlings. For example, the line containing wl*-1308 (wl gr lf tp) segregated for pale-green seedlings, and when these were allele-tested with pg*-735 and pg*-71 some of the progeny segregated for pale-green seedlings. For this reason, we believe that it is possible that one of the mutagenized lines contained a pale-green mutant, and the four pale-green mutants may not necessarily each represent independent mutations of this locus. Also, two ears from an allele test between wl*-1308 and wl*-199 (gr wl) segregated for a phenotype similar to that found in the 1308 parent. Because of the limited number of positives from this cross, we are not sure of the allelic relationship between these two mutants, and we cannot preclude a mixup of the mutant families.

A pg*-408/pg*-71, Pr/Pr X Pg/Pg, pr/pr cross was made in 1982, and the F1 generation was selfed in 1983. Analysis of eight ears suggests a map distance of approximately 50 map units between these two loci; thus, the pale-green locus could be near the yg1 locus. Chang (MNL 56:44) reported that pg*-735 is allelic to wlv*-473; thus, these five mutants are apparently allelic, and possibly allelic to yg. Obviously, these allele tests need to be made. Because of their approximate chromosome locations, allele tests between w21 and w22 with v3 and v21 should be done, as well as allele tests between wl*-199, wlv*-308, w*-sect206, and v2.

Three mutants, nec*-409, nec*-493 (tan necrotics) and gl*-681 were not uncovered by TB-5La; however, they were uncovered by compound TB-1La-5S. Thus, these three mutants are on the short arm of chromosome 5. Allele tests between the two tan necrotic mutants produced two types of mutant plants, those which developed necrotic bands early and die as young seedlings and those which develop necrotic bands later and survive to maturity. Crosses can be made with these latter types. Glossy seedlings from gl*-681 are smaller in size than their normal sibs. No allele tests have been made between the gl*-681 and tan necrotic mutants.

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Response in tissue culture of undeveloped ears from young plants

Tissue cultures competent to regenerate plants have previously been initiated from undeveloped ears of mature 18-20 week-old plants of A188 (Molnar, Gordon and Rice, MNL 54:52-53, 1980). Ears from young plants tend to be well suited for this type of study since they can be obtained earlier, as a more physiologically uniform population and with less contamination than ears from mature plants.

Plants of A188 were grown in a controlled environment chamber under a 16-hour photoperiod at 20 C in light and 15 C in darkness. Axillary and dominant ears were harvested from plants at various stages of growth and treated as reported for ears of mature plants. The ears were removed aseptically, sliced transversely and placed onto culture media. The media contained the macronutrient and micronutrient salts of Murashige and Skoog (Physiol. Plant. 15:473, 1962) + 0.25 mg thiamine-HCl/l + 20 or 40 g sucrose/l + 8 g agar/l + 1.0 or 2.0 mg 2,4-D/l and plates were incubated at 25 C in darkness. Plates were monitored regularly and the production of scutellar-like tissue scored as a positive response.

Thirty-nine slices from a total of 20 ears responded positively by producing scutellar-like tissue in a sample of 98 ears tested. The data in Table 1

Table 1. Effect of Ear Length on Tissue Culture Response

Length of ear (mm)	Range of slice widths (mm)	% Ears responding	Avg. no. slices responding per responsive ear
0– 5.0	0.27– 0.83	17 (5/29)	1.0
5.5– 10	0.50– 1.0	23 (6/26)	1.7
10.5– 20	0.50– 1.08	35 (8/23)	2.9
20.5– 30	0.96– 1.56	11 (1/9)	1.0
>30.5	1.0 – 2.59	<9 (0/11)	0

demonstrate that both the fraction of ears that responded and the average number of positive slices per responding ear are a function of ear length. Both are maximal for ears 10.5-20 mm in length. This dependence on length was expected as a result of the earlier study and closely parallels the results reported for immature tassels of A188 (Rhodes, Green and Phillips, MNL 56:148-149). It has been suggested that the optimal size correlates with a responsive developmental stage. Conversely, the data do not indicate a major effect either of 2,4-D concentration or of the node at which the ear developed (data not shown). The role of these and other parameters are under continued study in this system.

Larry A. Holbrook and Stephen J. Molnar

Evidence of the totipotency of maize roots in vitro

Established procedures for maintaining regenerable maize tissue cultures involve visually selecting scutellar-like tissues for subculture and discarding other more abundant tissue types. The latter tissues, if tested on growth regulator-free medium, usually exhibit rhizogenesis and are unable to form embryos or shoots. It was, therefore, unexpected to observe the formation of a somatic embryo on a well-differentiated root in tissue culture.

The culture was initiated from the apical meristem of a seedling of A188. It was totipotent with characteristic scutellar-like tissues. These tissues were subcultured onto solidified medium (macronutrient and micronutrient salts of Murashige and Skoog + 0.25 mg thiamine-HCl/l + 40 g sucrose/l + 8 g agar/l) containing 1 mg 2,4-D/l and incubated at 25 C in darkness. After 8 months of subculturing, an embryo was detected 19 days after the ninth subculture on one of the numerous aerial roots. This particular root appeared truncated just past a sharp bend. The epidermis was torn open on the outside of the bend and a ball of friable callus extended from the bend to the end of the root. The embryo arose immediately adjacent to this friable callus on the inside of the bend. The embryo enlarged slowly over the following 45 days and developed a cleft top. Crowding by other roots in the original plate necessitated removing the root bearing the embryo and transferring it to a fresh plate. After 13 days the embryo separated from the supporting root tissue when touched gently. Although the supportive root tissue was now senescing, histological studies comparing this tissue with sections of other roots grown under identical conditions revealed similar cellular structure for both samples. The tissue which produced the embryo also remained white although leaves in the same dish turned green, consistent with its identification as a true root. The free embryo was transferred to a culture room with a 16-hour photoperiod at 25 C, where it rapidly germinated. On auxin-free medium a 10-cm-tall seedling was obtained which did not survive transplantation into the greenhouse.

Although apparently a low frequency event, this observation demonstrates that corn roots can express totipotency in vitro.

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Possibilities of using br2 in maize breeding

Brachytic maize, especially br2, has been widely studied with the purpose of developing brachytic hybrids, but they have been significantly lower in yield than normal. In order to determine the possibility of using br2 lines in breeding programs three versions of hybrids were developed: normal, Br2 Br2; heterozygous, Br2 br2; and brachytic, br2 br2. During 1979-1982, 20 hybrids in the three versions were evaluated for 11 morphological, biological and economical characters.

The results indicate that homozygosis for recessive alleles of br2 exerts an effect on some morphological characters (see Table). First of all the plant

Comparative characteristics of brachytic, heterozygous and normal hybrids

Traits	Years	Genotype			$LSD_{0,05}$
		br2	br2/+	norm	
Plant height, cm	1979	163,6	218,9	225,5	6,3
	1980	152,8	222,6	226,9	2,7
	1981	117,6	168,4	176,2	3,7
Ear height, cm	1979	47,4	85,9	93,4	4,1
	1980	42,1	80,7	82,2	3,3
	1981	29,5	64,1	68,7	2,3
Grain yield, g/ha (14% moisture)	1979	84,4	91,5	88,4	2,4
	1980	70,7	80,0	74,5	1,5
	1981	30,4	37,3	34,4	1,1
	1982	65,2	79,8	75,9	2,9
Vegetation period, days	1979	118,1	118,6	115,8	1,4
	1980	144,7	143,1	141,9	0,9
	1981	101,1	99,1	98,2	0,8
	1982	116,0	114,3	113,2	0,4
Lodging, grades from 1 to 5	1979	1,0	1,9	2,8	0,3
	1980	1,4	2,8	3,4	0,3
	1981	1,1	2,1	2,6	0,3
	1982	1,2	1,7	2,7	0,3
Affection by common smut, %	1979	0,9	4,3	6,9	0,7
	1980	0,2	0,8	2,3	0,2
	1981	0,3	1,1	2,0	0,3
	1982	0,6	0,7	1,7	0,2

height and the ear height of br2 br2 are considerably reduced, which has been proven before. Morphological characters of heterozygous versions in general did not differ from normal, though in some hybrids significant differences were observed. Brachytic versions were most resistant to lodging and to common smut

(Ustilago maydis); normal counterparts were less resistant and the heterozygous versions were intermediate. Vegetation periods of br2 versions were longer than those of normal. Brachytic versions yielded significantly lower than normal, which is in accordance with the results of other researchers, but the heterozygous versions considerably surpassed the normal and brachytic counterparts.

The data we have obtained demonstrate the possibility of using brachytic lines and hybrids (homozygous br2) as crossing components with normal forms with the purpose of developing more productive and more lodging-resistant maize hybrids.

V. E. Micu and T. A. Solonenko

Perspectives of using tb in maize breeding and seed production

The mutant teosinte branched (tb) possesses some characters that are necessary for pollinators: a long flowering period of apical and lateral male inflorescences and high pollen productivity. To examine the possibilities of using this mutant in maize breeding and seed production for some elite lines which are used as male parents, versions segregating 1:1 for normal and tb plants were developed. For a few lines versions consisting of only tb plants were obtained. Using the normal and tb plants as male components in hybridization, two versions of the hybrids were developed: heterozygous (Tb tb) and normal (Tb Tb).

During 1978-1982, 44 hybrids were produced and evaluated for the most significant economic and biological characters (see Table). For the main

Comparative characteristics of normal and heterozygous (tb/+) hybrids of maize

Traits	Years	Genotype		$LSD_{0,05}$
		norm	tb/+	
Plant height, cm	1979	244,3	243,4	3,8
	1980	211,7	216,4	5,8
	1982	208,5	206,5	1,6
Ear height, cm	1979	93,6	94,4	3,6
	1980	73,3	76,3	4,3
	1982	63,6	64,0	1,4
Grain yield, g/ha (14% moisture)	1979	98,8	101,6	2,5
	1980	79,5	79,7	1,1
	1981	35,1	36,7	1,3
	1982	69,7	71,8	2,3
Vegetation period, days	1979	118,4	117,3	1,4
	1980	138,5	138,6	0,9
	1981	91,6	91,5	0,9
	1982	110,0	109,8	0,7
Number of ears per 100 plants	1979	107,8	113,1	4,9
	1980	100,3	105,2	3,9
	1981	91,8	96,0	3,1
	1982	93,4	99,6	2,8
Number of tillers per 100 plants	1979	103,4	106,9	0,9
	1980	114,2	127,7	4,1
	1981	118,3	122,2	3,8
	1982	107,5	109,8	1,4

morphological characters no essential differences between the normal and heterozygous counterparts were revealed. There were also no clear cut differences between the counterparts in the vegetation period. Heterozygous hybrids formed more ears and tillers than their normal versions. As a rule average productivity of heterozygous hybrids was equal to normal and only in 1979 did some of the heterozygous versions outyield the normal ones. Thus, it is clear cut that heterozygosis for Tb tb does not affect the main morphological and economical characters of hybrids.

The studies we have carried out give perspective to the possibility of using male tb lines or lines segregating 1:1 as pollinators in maize seed production.

V. E. Micu and T. A. Solonenko

Photosynthetic pigment content and nitrate reductase activity in normal and id maize

Indeterminate growth mutants (id) were found in collections of maize germplasm. Backcrossing was used to incorporate id in some early lines, and id versions which differed from normal counterparts by flowering dates were obtained. These versions served as models for investigation of the specificity effect of id on different biological and economic characters of maize.

Leaves of normal and id versions of four lines were studied in two periods of ontogenesis: emergence of tassel and silking. The content of photosynthetic pigments was determined spectrophotometrically. An in vivo assay was used for measuring nitrate reductase activity. The same genotypes were studied during two seasons, but it is important to note that in 1981 id versions were at the fourth generation of backcrossing and in 1982 at the fifth.

Our results showed that there are biochemical distinctions between lines (see Table). However, the distinctions between normal and id versions inside every line were the same. This fact made it possible to establish and discuss the results of photosynthetic pigment content and nitrate reductase activity in leaves of normal and id plants as average values of all genotypes. In the table we see a decrease of chlorophyll a, chlorophyll b, total carotenoids and nitrate reductase activity in leaves of the id counterpart compared to normal.

Photosynthetic pigments content (mg/g leaf dry wt.) and nitrate reductase activity (nmol NO_2/g leaf fresh wt. in 1 min) leaves id and normal plants. Average value of four lines.

Biochemical parameter	Period of ontogenesis	Season 1981			Season 1982		
		id	normal	d	id	normal	d
Chlorophyll a	tassel emergence	3,29	6,31	-3,02	3,58	6,86	-3,28
	silking	2,13	5,29	-3,16	2,84	7,00	-4,16
Chlorophyll b	tassel emergence	1,24	2,09	-0,85	1,04	1,94	-0,90
	silking	0,75	2,27	-1,52	0,68	1,90	-1,22
Total carotenoides	tassel emergence	1,28	2,22	-0,94	1,52	2,73	-1,21
	silking	0,90	2,16	-1,26	1,37	2,47	-1,10
Nitrate reductase activity	tassel emergence	38,65	57,36	-18,71	31,23	44,92	-13,69
	silking	20,92	58,33	-37,41	28,08	40,56	-12,48

We have made some interesting comparisons with the results reported in the literature on higher sensitivity of photosynthetic pigments and nitrate reductase activity to changes of environment. So we suppose that these biochemical parameters will be used for investigation in the future of the possibility of exogenous regulation of the effect of the gene id.

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The cell lineage of the maize embryo

Last year we reported on the orientation of the initial divisions of the zygote. Since then we have extended our cell lineage analysis to several later stages of development. Developing embryos of the genotype B/b;Pl/pl;Wd/wd were irradiated (500 R) at various times after pollination. At the time of irradiation samples were taken from a few representative ears and were then examined histologically to determine the developmental stage of the embryo. Mature seeds from irradiated ears were planted and the size and extent of white, green and sun-red sectors were recorded as soon as the sectors became visible. White wd sectors could be scored along the entire height of the plant, although only leaf sectors could be scored in the case of the lowermost nodes. Sectors of b and pl are not well expressed in either leaves or underground internodes and therefore could only be scored above the 7th or 8th node. The apparent cell number (ACN) of a node was usually determined by dividing the circumference of an internode by the width of the sector within it. The ACN of the nodes below ground was determined from the width of a sector relative to the width of the leaf blade. This approach is not very satisfactory because the leaf blade is not always derived from all the cells in the circumference of the node, and the extent to which a sector expands varies depending upon its position within the leaf. Unfortunately, it is the only practical method available.

The longitudinal extent of the sectors we observed is illustrated in Figure 1. As we have previously noted, the variability in clonal patterns is quite considerable. Nevertheless, several consistent features emerge. One of the most striking of these is the early separation of the lineages of the first and second nodes from the upper nodes. Four days after pollination many sectors still ran the entire length of the plant; but by the transition stage of development (6-7 DAP), over half of the sectors were restricted to the first node. By the coleoptile stage (8-11 DAP), the second node has also become clonally separated from other nodes. This feature is part of a general process of clonal restriction that proceeds from lower to upper nodes. A similar phenomenon occurs in the shoot apex during germination (Johri and Coe, MNL 54).

Another interesting feature of these data is the frequent appearance of "saddle sectors," that is, sectors that run from one side of the plant to the other (Figure 2). These sectors are generally restricted to one side of the midrib; on a planar projection of the shoot, they run parallel to the midrib line rather than perpendicular to it. This type of sector indicates that cell division is initially oriented perpendicular to the longitudinal axis of the embryo, so as to produce longitudinal files of cells. If cell division were oriented radially, one would expect saddle sectors to be randomly oriented relative to the longitudinal axis of the embryo (as defined by the plane of the midrib). Thus the apical meristem arises in a region of the embryo that has a highly polarized cell

pattern. In this respect, shoot initiation in corn may involve a process similar to that described by Green and Brooks (AJB 65:13-26, 1978) in Graptopetalum.

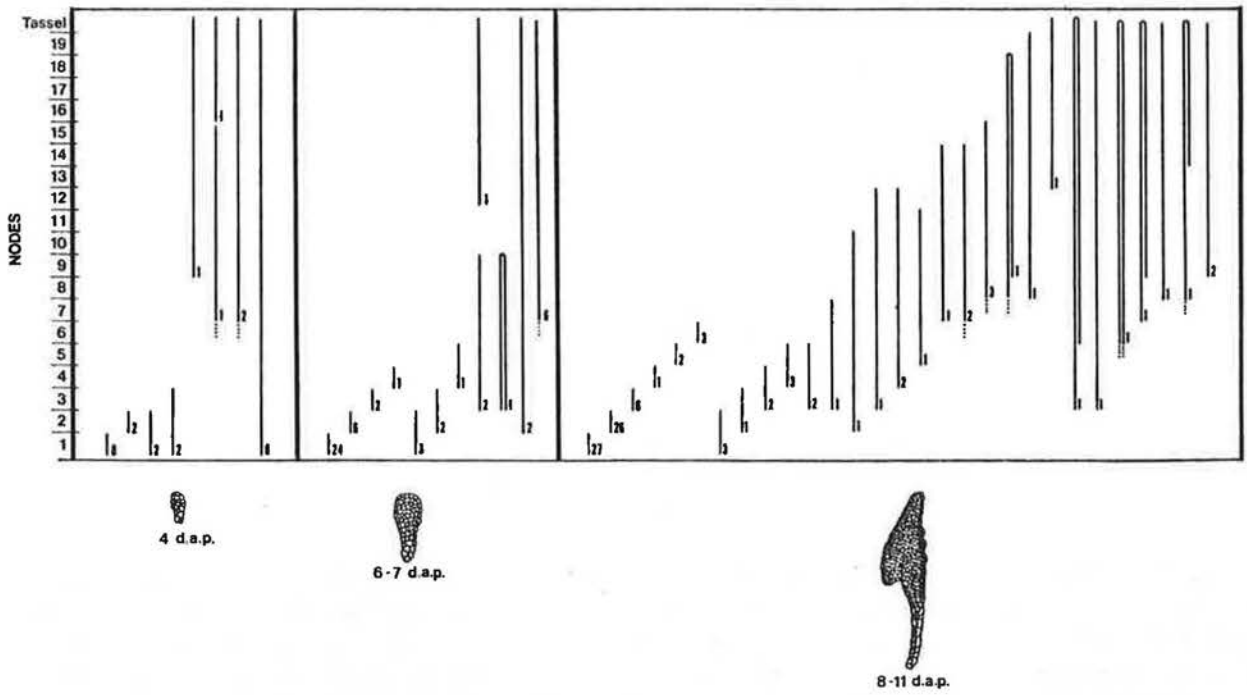

Figure 1. The number and extent of sectors induced at different stages of embryonic development.

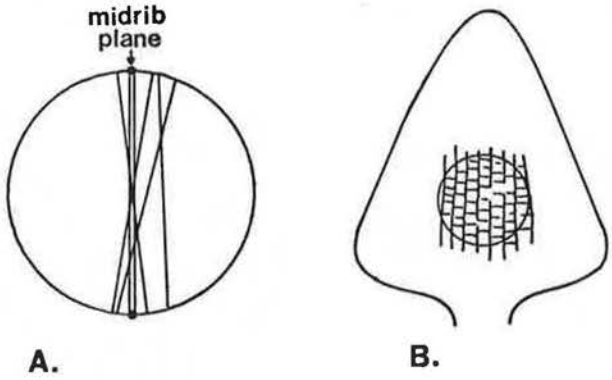

Figure 2. A. The location of "saddle sectors" on a planar representation of the shoot. The tip of the shoot is located in the center of the circle, and lower nodes are located at its periphery.
B. The probable cell pattern in the epidermis of the embryo just prior to meristem initiation.

In order to model the shoot apex at various stages in its development it is important to have an estimate of its ACN. Unfortunately, we observed relatively few sectors--particularly at early stages of development--so our estimates are only rough approximations (Figure 3). Nevertheless, they yield a picture that is consistent with the morphology of the embryo. The apical meristem of the shoot arises from a field of cells on the lateral face of the embryo. Since the meristem is circular in cross-section, it is reasonable to model this field as a

Fig. 3. The apparent cell number of embryonic nodes at various stages of development

NODE	4 DAP	6-7 DAP	8-11 DAP
Tassel			?
T. base		2.1	7.5
18			11.4
17			
16			
15			
14			
13			
12			
11			13.4
10			13.8
9			12.4
8			10.6
7			8.4
6		2.7	9.0
5		4.2	13.3
4		4.4	13.0
3	1.8	5.8	14.6
2	1.9	14.3	21.3
1	3.5	14.4	25.4

circle within the paradermal plane of the embryo. It is clear from the size and extent of clones induced prior to the appearance of the apical meristem (i.e., 4 DAP and 6-7 DAP) that this field encompasses a fairly large group of cells, whose members contribute to a limited and more-or-less specific node or set of nodes. On topological grounds, one would expect the lower nodes of the plant to be derived from cells at the outer periphery of the field, and the upper nodes of the plant to be derived from more central cells. If this is true, then the ACN of the lower nodes should be greater than that of the upper nodes, as indeed it is.

Our interpretation of the data presented in Figures 1 and 3 is illustrated in Figure 4. Four days after pollination 2-5 cells in the L1 layer of the embryo are

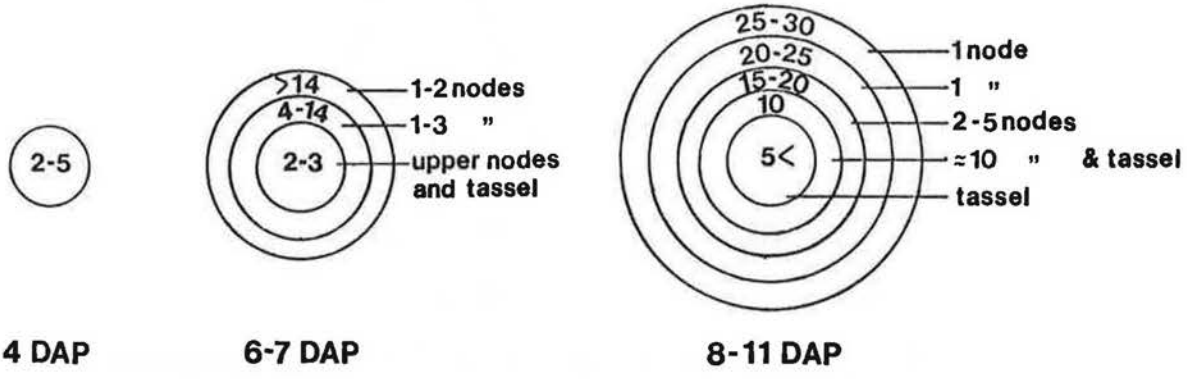

Figure 4. The number and fate of shoot initial cells at different stages of embryonic development.

destined to form the shoot apex. Only one or two of these cells contribute to the upper nodes of the plant; the remainder contribute solely to the lowermost one or two nodes. It should be emphasized that cell fates are not fixed at this stage since many sectors extend the entire height of the plant. Whether or not a cell contributes to upper nodes is probably determined by its position within this group and its subsequent pattern of cell division. In a 6- to 7-day-old embryo the primordium of the apex consists of an outer ring of 14 or more cells destined to form the first one or two nodes, another ring of 4-14 cells destined to form

the second node or a small group (2-3) of more distal nodes, and 2-4 central cells which form all the upper nodes of the plant. It is worth emphasizing this last point: as late as 6-7 days after pollination all the upper nodes of the plant (3 to tassel) may be represented by only 2 cells in the embryo. By the time the shoot meristem is initiated, the population of initial cells has grown even more, and cell fates have become even more restricted. Between 8 and 11 days after pollination this population consists of around 5 rings of cells containing between 50 and 100 cells. The outer 2 rings contribute to the first and second nodes, the third ring forms nodes 3 to 7, the fourth ring, nodes 7 to the base of the tassel, and the central ring forms the tip of the tassel. There are probably 25-30 cells in the circumference of the outermost ring, 20-25 in the second, 15-20 in the third, around 10 in the fourth, and something under 5 in the central-most ring. This last figure is based on the results of clonal analyses of dry seeds, which show the tassel to be derived from around 4 cells. Histological examinations indicate that the meristem of the shoot has about 10 rings of cells in the L1 layer, and about 6 rings in the L2 layer at the coleoptile stage of development. Since most of the sectors we observed were probably in the L2 layer (see Poethig, MNL 57:35), our results appear to accurately represent the size of the meristem at the time of irradiation.

S. Poethig and E. H. Coe, Jr.

The behavior of chromosome B-3Sb

Chromosomes that are regularly lost during mitosis and meiosis are extremely useful tools for genetic and developmental studies. Centric rings, for example, have been used to study the expression of embryo lethal genes in mature tissue, to study the cell autonomy of a mutation, and to obtain pollen that is enriched for a lethal gene. Unfortunately, there are very few centric rings in existence, although there has been a recent effort to generate new ones (Higgins, MNL 57:40). The behavior of tertiary B-A chromosomes is similar to that of centric rings in that they are often mitotically unstable and are poorly transmitted through pollen. It should be possible, therefore, to use these chromosomes in place of rings when the appropriate rings do not exist.

I and several others at the University of Missouri are currently in the process of generating tertiary trisegmental stocks of several B-A translocations. Recently I generated a B-3Sb/3/3 stock of the genotype C1/c1/c1. Because c1 has an albino phenotype (in the absence of C1m), the somatic loss of the B-3Sb chromosome is readily visible. Every leaf on tertiary trisegmental plants had a few albino sectors and in some cases the sectors extended several nodes. All sectors had sharp boundaries, indicating that c1 is cell-autonomous. This feature should make it possible to examine the cell autonomy of several morphological mutations on 3S, and a B-3Sb/c1 d ra2/c1 d ra2 stock is being developed with this in mind.

The B-3Sb chromosome is transmitted via pollen at a frequency of 33% (412/1257) based on the frequency of yellow (C1) kernels resulting from the cross c1/c1;C1m3 X B-3S C1/c1. The results of this cross are shown below.

	C1	c1
PL64-7 X P156-13	39	127
PL64-11 X PL56-13	30	83
PL23-19 X PL56-13	170	298
PL64-9 X 56-21	68	123
PL64-20 X 56-21	105	207
TOTAL	412	838

Only 1 out of 46 C1 kernels examined had 20 chromosomes; the remainder had 21. Thus, there is only 2% recombination between C1 and the translocation breakpoint, which makes C1 an excellent marker for the B-3S chromosome. There was no evidence of a ring chromosome in these preparations. This is an important point because previous studies have indicated that unstable supernumerary B-A chromosomes are often ring derivatives of the original B-A (Carlson, TAG 43:147).

Because of the relatively high rate with which B-3Sb is transmitted through pollen, tetrasegmental individuals are common in selfed progeny of tertiary trisegmental plants. In comparison to diploids, such plants have extremely narrow leaves and do not usually produce an ear. In contrast, hypoploid plants are quite vigorous, and have broader leaves than normal.

Scott Poethig

Mo17 as a suppressor line

Many investigators who have worked with Mo17 have been impressed by the extent to which it suppresses the expression of a wide variety of mutations. Factors whose expression is reduced in this background include seed and plant color factors (A, C, R, B, P1), chlorophyll mutations (g, g2, c1), dominant morphological mutations (Cg, Tp1, Tp2) and disease lesion mutations (Hoisington, pers. commun.). If this trait is indeed general, then it is not only of fundamental interest but is also of considerable practical importance. It is often desirable, for example, to suppress the expression of a mutation in order to maintain or manipulate stocks. This is true in the case of Cg, Tp1 and Tp2, which do not produce pollen in many genetic backgrounds. The fact that the suppressive ability of Mo17 is associated with an extremely high combining ability suggests that this trait may also be useful for breeding purposes. The idea of using suppressive ability as a way of selecting elite inbreds is not new. Zuber tried to use a set of knotted mutations for this purpose some time ago, apparently without much success. Nevertheless, it might be worthwhile to try this approach again using a combination of different types of mutations.

Scott Poethig

Whither Cg?

Cg was originally mapped 5 m.u. proximal to d1 on the basis of 3 point test cross data involving 1g2 and d1 (Galinat, MNL 26). This position is now in doubt because two-point test crosses between Cg and c1, g2 and cr fail to reveal any linkage with these markers.

	Cg +	+ c1	Cg c1	+ +	Total	% Rec.
c1 + x Cg +/Cg c1	100	99	113	108	420	53

	Cg +	+ g2	Cg g2	+ +	Total	% Rec.
Cg +/+ g2 x + g2	59	53	47	39	198	43

	Cg +	+ cr	Cg cr	+ +	Total	% Rec.
Cg +/+ cr x + cr	59	75	102	42	278	52

Information about the linkage between Cg and d1 is still unavailable, but since cl and cr are approximately 20 m.u. on either side of d1, it would be surprising if Cg was linked to this marker. The possibility that Cg is located on 3L is now being tested.

Scott Poethig

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Sequence homology between Zea diploperennis and Zea mays

The complete nucleotide sequence of cytochrome oxidase subunit II in maize mitochondria has been determined (Fox and Leaver, Cell 26:315-323, 1981). This gene, referred to as mox1, is entirely contained within a single 2.4 Kb EcoRI fragment of a total mitochondrial DNA digestion and is interrupted by a 794 base pair intron.

Using the dideoxy nucleotide chain termination sequence analysis of Sanger, a major portion of this same gene in Zea diploperennis has been sequenced. As in maize, the entire gene is contained within a single 2.4 Kb fragment when total mitochondrial DNA is digested with EcoRI. All major restriction endonuclease sites compared within this fragment were also found to be perfectly conserved between the two species. The following segments of mox1 have thus far been sequenced in Z. diploperennis:

Segment sequenced, and %	Nucleotide number
exon 1, 73%	116-432
intron, 100%	433-1227
exon 2, 53%	1228-1303, and 1467-1620

Comparisons of these sequences to those published for mox1 of maize have shown that they are identical. The 115 nucleotides of the 3' flanking region immediately following exon 2 have also been sequenced and compared. These sequences also share 100% homology with those in maize. The results are somewhat surprising considering the great differences between mitochondrial restriction patterns of maize and Z. diploperennis (Timothy et al., Maydica 28:139-144, 1983). DNA used for sequencing was isolated and cloned separately from two different sources of Z. diploperennis. These sources gave two very distinctive Z. diploperennis restriction patterns when digested with BamHI or EcoRI.

The sequence homologies between maize and Z. diploperennis indicate exceptional conservation. This was not wholly unexpected for the exon 1 and exon 2 segments of mox1. Slight departures from absolute homology of the introns and the 3' noncoding tails might be anticipated, but this was not the case. The most reasonable explanations for these results are low mutation rate and/or that these sequences play an important functional role.

The extreme conservation of sequences is indicative of close relationship between the taxa, even though the organization of the mitochondrial genomes may be distinct. This is consistent with our finding (Sederoff et al., PNAS 78:5953-5957, 1981) that while appreciable evolution of the mitochondrial genome in Zea may have occurred by major rearrangement of sequences, there is general sequence conservation.

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Some properties of genetically defined proteins in maize

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SOME PROPERTIES OF GENETICALLY DEFINED PROTEINS IN *ZEA MAYS*

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Protein	Isozymic Forms	Gene Symbol	Chromosomal Location	Alleles[1]	Inheritance[2]	Native M_r[3]	Subunit Structure	Tissue Distribution[4]	Comments	References
Acid Phosphatase	AP_1	*Ap_1*	-	3^a	b	-	Dimer	d,g,i		
	AP_2	*Ap_2*	-	2^a	b	-	-	i	*Acph1* appears to be identical to *$Phos_g$* (11,89,90).	11,27,28 89,90
	AP_3	*Ap_3*	-	2^a	b	-	-	i		
	ACPH-1	*Acph1*	9	2^a	b	-	Dimer	e,f,k		
ADP-Glucose Pyrophosphorylase	B	*Sh_2*	3L	2^{b+}	a	237,000 to 253,000[b,d]	-	b	+ one wild type allele and several mutant, recessive alleles. The two genes (*Sh_2* and *Bt_2*) appear to control the one enzyme activity.	20,39,40 41,94
		Bt_2	4	2^{b-}						
Alcohol Dehydrogenase	ADH-1	*Adh1*	-	2^a	b	80,000 to 90,000[a]	Dimer	b,c,d,e f,g,h,i j,k	Three additional non-genetically defined isozymic forms exist (ADH-3, ADH-4, ADH-5).	5,29,30 32,60,70 72,73,75 81
	ADH-2	*Adh2*	1L	3^a	b	80,000 to 90,000[a]	Dimer	b,c,d,e f,g,h,i j,k		
Aminopeptidase	AMP-1	*Amp1*	1L	3^a	b	92,000[d]	Monomer	a,b,c,d e,f,g		
	AMP-2	*Amp2*	1L	2^a	b	88,000[d]	Monomer	b	The isozymic forms sometimes overlap one another. The genotypes are scored unequivocally since they show differential staining towards different amino acid-naphthylamide substrates.	60,97,98 99
	AMP-3	*Amp3*	5S	4^a	b	83,000[d]	Monomer	a,b,c,d e,f,g		
	AMP-4	*Amp4*	-	2^a	b	61,000[d]	Monomer	a,b,c,d e,f,g		
α-Amylase	AMY-1	*Amy1*	-	2^a	b	43,000 to 45,000[b]	Monomer	b,d,e,f	Other non-genetically defined isozymes exist. Tissue specific differential allelic expression occurs (15,17).	14,15,16 17,69,72 73,76
β-Amylase	AMY-2	*Amy2*	5S	2^a	b	-	Monomer	b,d,e,f		14,15,16 17,69,72 73,76
Branching Enzyme (α-1,4-glucan:α-1,4-glucan-6-glycosyl transferase)	IIb	*ae*	5	2^b	a	70,000 to 90,000[d]	Monomer	b	Two additional non-genetically defined isozymes exist (I,IIa). All three are distinguished by their separation on DEAE or hydrophobic chromatography.	6,7,8 44,63
Catalase	CAT-1	*Cat1*	5S	6^a	b	240,000[a]	Tetramer	a,b,c,d f,g,k	CAT-1 and CAT-2 form intergenic hybrids, while CAT-3 does not. A "null" allele for CAT-2 exists.	13,68,71 74,78
	CAT-2	*Cat2*	1S	3^a	b	240,000[a]	Tetramer	a,d,g		
	CAT-3	*Cat3*	1L	2^a	b	240,000[a]	Tetramer	f,g,k		
Catechol Oxidase	CX	*Cx*	10L	3^a	b	-	-	-	A "null" allele exists.	66
Endopeptidase	ENP	*Enp*	6L	6^a	b	68,000[d]	Monomer	a,b,c,d e,f,g,h k	A "null" allele exists.	53,55,59 60,61,62
Esterase	E_1	*E_1*	7	3^a	b	-	-	b,e	Additional non-genetically defined isozymes exist. E_1 and E_3 are possibly dimers. E_1 is the pH 7.5 esterase (80). E_1 and E_4 have "null" alleles.	11,42,43 45,72,80
	E_2	*E_2*	-	2^a	b	-	-	-		
	E_3	*E_3*	3	2^a	b	-	-	-		
	E_4	*E_4*	3S	5^a	b	-	-	-		
β-Glucosidase	β-GLU1	*β-Glu1*	10	22^a	b	-	Dimer	e,f,k	A "null" allele exists.	64,88,90

Protein	Isozymic Forms	Gene Symbol	Chromosomal Location	Alleles[1]	Inheritance[2]	Native M_r[3]	Subunit Structure	Tissue Distribution[4]	Comments	References
Glutamate Oxaloacetate Transaminase	GOT-5	*Got1*	-	4^a	b	-	Dimer	a,b,d,e f,g,l	Additional non-genetically defined isozymes exist. GOT-1 is localized in the mitochondria; GOT-5 in the glyoxysomes. In the presence of light, GOT-5 decreases in activity in the green leaf, but, GOT-3 increases in activity in green leaves.	35,77,85
	GOT-2	*Got2*	5L	5^a	b	-	Dimer	a,b,d,e f,g,l		
	GOT-1	*Got3*	5S	5^a	b	-	Dimer	a,b,d,e f,g,l		
Glutamic Dehydrogenase	GDH-1	*Gdh1*	1L	2^a	b	-	-	f	Additional loci are believed to be involved in producing GDH isozymes.	35,54,65
Isocitric Dehydrogenase	IDH-1	*Idh1*	8	4^a	b	-	Dimer	f	Intergenic as well as interallelic hybrid isozymes are produced.	35,87
	IDH-2	*Idh2*	6L	4^a	b	-	Dimer	f		
Malate Dehydrogenase	(See Comments)	*mMdh1*	3L	2^a (*mMdh1-m10*, *mMdh1-m8*)	b				Isozymes: Product of mMDH1: Heterodimer of mMDH8/mMDH3 mMDH2: Heterodimer of mMDH8/mMDH5 mMDH3: *mMdh2-m3*, *mMdh4-m3* or Heterodimer of mMDH8/mMDH7 mMDH4: Heterodimer of mMDH3/mMDH5 mMDH5: *mMdh2-m5* or Heterodimer of mMDH3/mMDH7 mMDH6: Heterodimer of mMDH5/mMDH7 mMDH7: *mMdh4-m7* mMDH8: *mMdh1-m8* or *mMdh3-m8* mMDH9: Heterodimer of mMDH8/mMDH10 mMDH10: *mMdh1-m10*	50,51 52,58 101
		mMdh2	6L	2^a (*mMdh2-m3*, *mMdh2-m5*)	b					
		mMdh3	8L	1^a (*mMdh3-m8*)	b	70,000 to 75,000[b,d]	Dimer	a - j		
		mMdh4	8L	2^a (*mMdh4-m3*, *mMdh4-m7*)	b					
	(See Comments)	*sMdh1*	1L	2^a (*sMdh1-s5*, *sMdh1-s1*)	b				sMDH1: *sMdh1-s1* or *sMdh2-s1* sMDH2: Modification of sMDH1 sMDH3: Heterodimer of sMDH1/sMDH4 sMDH4: *sMdh2-s4* sMDH5: *sMdh1-s5* sMDH6: Modification of sMDH5	
		sMdh2	5S	3^a (*sMdh2-s1*, *sMdh2-s4*, *sMdh2-s8*)	b	70,000[b,d]	Dimer	a - j		
Peroxidase	Px1	Px_1	-	4^a	b	33,000 to 50,000[d]	Monomer†	e,f,g,h j,k	† Suggested by genetic evidence. Px1, Px5, Px6 and Px9 have "null" alleles.	9,10,36 37,38,48 72,73
	Px2	Px_2	-	2^a	b	33,000 to 50,000[d]	Monomer†	i		
	Px3	Px_3	-	6^a	b	33,000 to 50,000[d]	Monomer†	f,g,k		
	Px4	Px_4	-	3^a	b	33,000 to 50,000[d]	Monomer†	b,f		
	Px5	Px_5	-	2^a	b	33,000 to 50,000[d]	Monomer†	b,k,l		
	Px6	Px_6	-	2^a	b	33,000 to 50,000[d]	Monomer†	a,b,c,d e,f,i,j k,l		
	Px7	Px_7	-	2^a	b	75,000[d]	Monomer†	e,f,g,h,l		
	Px8	Px_8	-	2^a	b	33,000 to 50,000[d]	Monomer†	e		
	Px9	Px_9	-	2^a	b	33,000 to 50,000[d]	Monomer†	e		
Phosphoglucomutase	PGM-1	*Pgm1*	1L	13^a	b	-	-	f	No intergenic or interallelic hybrid isozyme bands produced.	34,35,84
	PGM-2	*Pgm2*	5S	6^a	b	-	-	f		
6-Phosphogluconate Dehydrogenase	6-PGD1	*Pgd1*	6L	9^a	b	-	Dimer	f	Intergenic and interallelic hybrid isozyme bands produced.	35,86, 100
	6-PGD2	*Pgd2*	-	3^a	b	-	Dimer	f		
Phosphohexose Isomerase	PHI-1	*Phi*	1L	9^a	b	-	Dimer†	f	† Suggested by genetic evidence. Intragenic hybrid isozymes are produced.	34,35

Protein	Isozymic Forms	Gene Symbol	Chromosomal Location	Alleles[1]	Inheritance[2]	Native M_r[3]	Subunit Structure	Tissue Distribution[4]	Comments	References
Starch Granule Bound Nucleoside Diphospho-glucose-Starch Glucosyl Transferase	-	*Wx*	9	$2^{b\dagger}$	a	-	-	b	One wild type allele and several mutant, recessive alleles (some caused by the insertion of a controlling element).	1,26,56 57,92,93
Sucrose Synthase	-	Sh_1	9S	2^{b}	a	365,000[e] (tetramer)	Tetramer, Octamer and Higher Multimers	b		18,19,79 91
Superoxide Dismutase	SOD-1	*Sod1*	-	2^{a}	b	33,500[d]	Dimer	b,c,d,e f,g,h,i j,k	SOD-1 is localized in the chloroplast; SOD-2 and SOD-4 are cytosolic; SOD-3 is located in the mitochondria.	2,3,4
	SOD-2	-	-	-	b	31,000 to 33,000	Dimer	b,c,d,e f,g,h,i j,k	SOD-1 and SOD-3 generate inter-allelic hybrid isozymes; SOD-2 and SOD-4 produce intergenic hybrid isozymes.	
	SOD-3	*Sod3*	-	2^{a}	b	85,000[c]	Tetramer	b,c,d,e f,g,h,i j,k		
	SOD-4	*Sod4*	-	2^{a}	b	31,000 to 33,000[d]	Dimer	b,c,d,e f,g,h,i j,k		
UDP glucose: Flavonol 3-O-glucosyltransferase	-	*Bz*	9S	$2^{b\dagger}$	a	50,000[d]	-	a	† One wild type allele and several mutant, recessive alleles (some arising from the insertion of the Ds controlling element).	22,23,24 25,31,46
Zein		*Zp1* *Zp2* *Zp3* } †	7S	-	-	-	-		† May be a single locus or a gene cluster.	12,21 33,47 49,67 82,83 95,96
				-	-	-	-	b	†† Has a different M_r than the first *Zp2* and is probably a different gene.	
		Zp2 ††	10	-	-	-	-		Zein is composed of 8-15 polypeptides depending on the inbred line used. These are resolved by IEF gels. Six classes of M_r exist, each heterogeneous with respect to pI. The two predominant classes are 22,000 (Z1) and 19,000 (Z2).	
		Zp12	4L	-	-	-	-		The actual number of genes coding for zein subunits is unknown. *In situ* hybridization experiments localize genes on 4L, 5L, 7S and 10L.	
		Zp13	4S	-	-	-	-			

FOOTNOTES

[1] Classification of allelic variants: a) Electrophoretic
b) Activity.

[2] Classification for mode of inheritance: a) Dominance
b) Codominance

[3] Method for determination of Native M_r: a) Based on subunit structure and subunit M_r, b) Sucrose gradient centrifugation, c) Analytical ultracentrifuge, d) Gel filtration, e) Polyacrylamide gel electrophoresis.

[4] Tissue distribution: a) Aleurone, b) Endosperm, c) Embryo, d) Scutellum, e) Root, f) Coleoptile, g) Leaf, h) Husk, i) Pollen, j) Stem, K) Mesocotyl, l) Pericarp.

Note that in each case not every tissue was tested for the presence of each enzyme.

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Allelism tests with 2L mutants

Twenty-four EMS-induced mutants located to 2L were obtained from M. G. Neuffer. These mutants were tested for allelism within phenotypically similar sets. The results are as follows:

opaques:	o*-1195A, ogm*-1488B, and o*-999A are not allelic
flouries:	fl*-1316A and cp-fl*-1024A are not allelic
virescents:	ygv*-72 and wlvpg*-424 are not allelic
pale green spotteds:	pgspt*-278B and pgspt*-464 are not allelic
defectives:	de*-660C and cp*-1225 are alleles; de*-1175, cp*-1225 and dcr*-1428 are not allelic
white seedlings:	w*-332 and w*-77 are not allelic; w*-332 is probably allelic to the known mutant w3

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Classification of zein clones into subfamilies and a suggestion on nomenclature

The various zein cDNA and genomic clones that have been isolated have each been given a name by the laboratory that first isolated the clone. This has resulted in a number of different naming systems. We propose that a new nomenclature based on the concept of zein subfamilies be used. A zein subfamily refers to a subgrouping of closely related zein genes, mRNAs and proteins whose members are more closely related in their nucleotide sequences (and amino acid sequences) to one another than they are to members of another subfamily (Rubenstein, Maize for Biological Research, W. F. Sheridan, pp. 189-195, 1982).

So far we have identified 5 subfamilies and propose now to label these SF1, SF2, SF3, SF4, and SF5 (Messing et al., Genetic Engineering of Plants, T. Kosuge, C. P. Meredith, and A. Hollaender, eds., pp. 211-227, 1983). We have determined the nucleotide sequence of representative cDNA clones for each of these subfamilies; they are A20, A30, B59, B49, and B36, respectively. All of these cDNA clones were obtained from IHP zein mRNA (Burr et al., J. Mol. Biol. 154:33-49, 1982).

We propose that the various clones be renamed so as to indicate their subfamily, origin (inbred line and clone type--cDNA or genomic), and common laboratory name. For example, the cDNA clones A20, A30, B59, B49, and B36 obtained from IHP zein mRNA will be termed SF1(IHP-c)A20, SF2(IHP-c)A30, SF3(IHP-c)B59, SF4(IHP-c)B49, and SF5(IHP-c)B36, respectively. The genomic clones Z4 and Z7 isolated from W22 DNA would be termed SF2(W22-g)Z4 and SF4(W22-g)Z7, respectively since Z4 and A30 are in the same subfamily (SF2) and Z7 and B49 are in subfamily SF4.

COMPILATION ZEIN CDNA AND GENOMIC CLONES

ZEIN SUBFAMILIES	NAMES OF CLONES							
	IHP	W22		W64A		Blk Mex	A619	
	cDNA	cDNA	Genomic	cDNA	Genomic	Genomic	cDNA	Genomic
SF1 Mostly"19kd" -Actual 24kd On 4L,7S,10L	A20	Zg14 Zg15 Zg35						
	B20 B37			pcM6 pcM8				
SF2 Mostly"19kd" -Actual 23kd	A30	Zg31A Zg124 Zg7 Zg19	Z4	pZ19.1	zE19	ZG99		
On chromosmes 4L, 7S	A76 B29 B50 B54 B72			pcMG1			pFW13	MF7
SF3 Mostly"19kd" -Actual 25kd	B59 B41							
SF4 Mostly"23kd" -Actual 27kd On chrom 4L	B49		Z7	Z22.1 Z22.3 pcM1	zA1			
	B14 A28 B60 B69						pFW19	pML1 pML2 pML3 pML4
SF5 Mostly"23kd"	B36							

CLONES	REF
SF1 (IHP-c) A20 SF4 (IHP-c) B49	Geraghty, D., J. Messing, and I. Rubenstein (1982) Sequence analysis and comparison of cDNAs of the zein multigene family. EMBO J. 1: 1329-1335.
SF1 (W22-c) ZG14 SF1 (W22-c) ZG15 SF1 (W22-c) ZG35	Heidecker, G., and J. Messing (unpublished data)
SF2 (IHP-c) A30	Geraghty, D., M.A. Peifer, I. Rubenstein, and J. Messing (1981) The primary structure of a plant storage protein: zein. Nucleic Acids Res. 9: 5163-5174.
SF2 (W22-g) Z4 SF2 (W22-c) ZG31A	Hu, N.-T., M.A. Peiffer, G. Heidecker, J. Messing, and I. Rubenstein (1982) Primary structure of a genomic zein sequence of maize. EMBO J. 1: 1337-1342.
SF2 (W22-c) ZG7 SF2 (W22-c) ZG124 SF2 (W22-c) ZG19	Heidecker, G., and J. Messing (1983) Construction of a maize endosperm cDNA library by a new mRNA cloning technique. Nucleic Acids Res. (In press).
SF2 (W64A-c) pZ19.1 SF2 (Blk Mex TC) ZG99	Pedersen, K., J. Devereux, D.R. Wilson, E. Sheldon, and B.A. Larkins (1982) Cloning and sequence analysis reveal structural variation among related zein genes in maize. Cell 29: 1015-1026.
SF2 (W64A-g) zE19	Spena, A., A. Viotti, and V. Pirrotta (1983) Two adjacent genomic zein sequences: structure, organization and tissue specific restriction pattern. J. Mol. Biol.
SF4 (W64A-c) pZ22.1 SF4 (W64A-c) pZ22.3	Marks, M.D., and B.A. Larkins (1982) Analysis of Sequence microheterogeneity among zein messinger RNAs J. Biol. Chem.
SF4 (W64A-g) zA1	Spena, A., A. Viotti, and V. Pirrotta (1982) A homologous repetitive block structure underlies the heterogeneity of heavy and light chain zein genes. EMBO J. 1: 1589-1982.
SF1 (W64A-c) pcM6 SF1 (W64A-c) pcM8 SF2 (W64A-c) pcMG1 SF4 (W64A-c) pcM1	Viotti, A., D. Abildsten, N. Pogna, E. Sala, and V. Pirrotta (1982) Multiplicity and diversity of cloned zein cDNA sequences and their chromosomal location. EMBO J. 1: 53-58.
SF1 (IHP-c) B20 SF1 (IHP-c) B37 SF2 (IHP-c) A76 SF2 (IHP-c) B29 SF2 (IHP-c) B50 SF2 (IHP-c) B54 SF2 (IHP-c) B72 SF3 (IHP-c) B59 SF3 (IHP-c) B41 SF4 (IHP-c) A28 SF4 (IHP-c) B14 SF4 (IHP-c) B60 SF4 (IHP-c) B69 SF5 (IHP-c) B36	Burr, B., F.A. Burr, T.P. St. John, M. Thomas, and R.W. Davis (1982) Zein storage protein gene family of maize. An assessment of heterogeneity with cloned messinger RNA sequences. J. Mol. Biol. 154: 33-49.

The table represents a compilation of the zein cDNA and genomic clones which have been described in publications from many different laboratories. The rows for the subfamilies are divided by a horizontal line. The clones whose names appear above the line have been sequenced, and hence their membership within a given subfamily has been verified. Those clones whose names appear below the line have been tentatively classified into a given subfamily on the basis of hybridization data. As additional zein subfamilies and zein clones are characterized they can be fitted into this scheme.

Irwin Rubenstein and Daniel E. Geraghty

Transmission of a deficiency for nearly the entire nucleolus organizer region

Last year we reported the transmission of a deficiency thought to include the entire NOR (MNL 57:131-132). The deficiency was initially detected by the presence of polymitotic (po) plants among the progeny of a cross between heterozygous T6-9(067-6) females and heterozygous +/po males. Pachytene analysis of the po plants indicated heterozygosity for a Dp-Df chromosome. Based on the lack of apparent NOR-heterochromatin and the previously reported breakpoint of 6S.39, we believed the break was proximal to the NOR and that a Dp-Df chromosome deficient for the entire NOR had been transmitted. However, later quartet analysis of Dp-Df heterozygotes that were not hemizygous for po (-/+ instead of -/po) revealed a nucleolus in every microspore. We expected 50% to have a diffuse nucleolus if the plants were heterozygous for a complete NOR-deficiency. This observation indicated (1) that the break must be in the NOR and interestingly (2) that even a small portion of the NOR is sufficient to form a microspore nucleolus. Pachytene analysis of the homozygous interchange revealed two chromosomes associated with the nucleolus. The break appears to be in the proximal 1-2% of the NOR-heterochromatin. Thus, these tests indicate the transmission of a deficiency for 98-99% of the NOR and not the entire NOR.

R. L. Phillips, A. S. Wang and W. P. Bullock

A recessive disease lesion mimic

A line segregating for an apparent disease lesion mimic was obtained in a program designed to backcross the polymitotic (po) allele into inbred A632 and simultaneously select for improved seed set on male-sterile (po) plants. This line represented the selfed progeny of a fertile plant derived from the open-pollinated progeny of a po plant. This po plant was from the selfed progeny of a fifth backcross of po to A632. The disease lesion mimic mutation was of apparent spontaneous origin because other lines of similar descent did not segregate for the trait.

Plants first show the lesions at about the 9-leaf stage (6 weeks after planting in 1983). Lesions are concentric necrotic spots with alternating light and dark rings; the lesions may stop at a vein. Watersoaked-appearing areas later encompass the lesions. Eventually the entire leaf becomes necrotic. Ear shoots are not apparent; however, tassels are produced and anthers may dehisce and shed viable pollen. Pollen is variable in size and approximately 50% is partially filled with starch. The entire plant ultimately becomes necrotic and degenerates. By 9 weeks after planting, the lower 2-3 leaves are totally necrotic. By 11 weeks, the flag leaf is one-half necrotic, progressively greater necrosis occurs over the next three leaves, and lower leaves are totally necrotic.

No fungal or bacterial microorganisms are specifically associated with the lesions according to moist-chamber tests with isolated leaf segments (pers. commun., Dr. Thor Kommedahl, Department of Plant Pathology, University of

Minnesota). Unsuccessful attempts were made to spread the disease to normal siblings by grinding affected leaves in water and placing soaked pipe cleaners through the leaves of normal siblings. Dr. Kommedahl described the lesion phenotype as resembling "target spot." A mutant with a target spot phenotype was reported previously in the 1954 Maize Genetics Coop News Letter (28:29); the trait was controlled by a single recessive allele tentatively located in chromosome 9.

Backcrosses of heterozygous normal plants as female to homozygous disease lesion mimic plants as male produced 73 normal and 67 disease lesion mimic plants. In F2's of crosses with several independent waxy-marked translocations, a total of 532 normal and 216 disease lesion mimic plants were produced. Therefore, the trait is probably controlled by a single recessive allele, although the ratio in F2 is somewhat deviant (P = .01-.05). Further tests to map the gene are underway.

R. L. Phillips

Tests for a cytoplasm that restores genetic male sterile-1

Z. diploperennis cytoplasm does not restore ms1. Plants from diploperennis female x +/ms were selfed and also crossed on ms. Testcross progenies that segregated for male sterility came from plants whose self progeny also segregated for male sterility. This does not rule out the possibility that diploperennis cytoplasm might restore one of the other genetic male steriles.

Last year's report, made before plans for 1983 were completed, stated that the A188 and T6-9b "standard normal" lines did not have a restorer cytoplasm for ms1. That may not be correct. Additional self progenies from both lines crossed with +/ms had no male sterile progeny. Plants in those progenies were again selfed and testcrossed on ms1. For the A188 line, the fertile plants in two progenies from A188 x (ms/T2-6) were selfed and testcrossed on ms. The test crosses now growing in Hawaii that segregate for male sterility will identify plants whose self progeny will be tested here in 1984, completing the test for the cytoplasms of the two lines. Note: T2-6 is closely linked to ms. Hence fertile plants from crosses with them should be +/ms heterozygotes.

C. R. Burnham

The su gl4 la stock for 3-point linkage tests

Last year's report stated that seed for the 3-point test was available. I did not grow the material in 1983, but greenhouse tests show no segregation for la in the supposed three-point tests. One su stock is gl and segregates for la. The stocks will be grown in 1984 to make the 3-point backcrosses. Requests that have been made for seed will be filled from those crosses.

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Chromosomal polymorphism in local maize of Kashmir

Meiosis was examined in PMC of 181 plants of 37 varieties of local maize grown in three districts of Kashmir Valley for observing the extent of variation in the chromosome morphology. Besides differences in relative length of chromosomes,

differences were also observed in the presence of B chromosomes, satellites on the short arm of chromosome 6, heterochromatic blocks on the long arm of chromosome 2 and abnormal chromosome 10.

The maize varieties used in the present study were collected from the sub-mountainous areas of the three districts (Anantnag, Pulwama and Baramulla) of the Kashmir Valley. The collections were made during September-November, 1972: Tripachi (4), Badeh (4), Vozij (2), Niver (2), Mishri (1), Kani (1) and Ferozpur (1) - Group I; and 1977 Chiteh-safed (8), Vozij (6), Lidder (4) and Dabher (4) - Group II from the Gujars' fields. However, no two varieties were collected from the same location or the same farmer. A distance of at least one km was maintained from field to field. The list of varieties collected from each district is given in Figure 1.

FIG. 1
MAP OF J&K SHOWING VARIOUS MAIZE VARIETIES COLLECTED FROM EACH DISTRICT WITHIN THE VALLEY OF KASHMIR

Group I and Group II varieties were grown in Srinagar to give 5 plants from each variety during the 1973 and 1978 growing seasons respectively. Young tassels at the proper stage were fixed in 1:3 acetic acid and ethyl alcohol for 48 h and stored in the refrigerator till worked. Squash preparations were made in 2% acetocarmine.

Chromosome morphology was studied at the pachytene stage of meiosis. In order to avoid any discrepancy in the results, which would arise due to differential condensation of the chromosomes, all results pertaining to total chromosome length were transformed into relative lengths (Tables 1 and 2). The cells were also studied at diakinesis for the presence or absence of B chromosomes, and at anaphase and telophase for tracing the course of B chromosomes and for other cytological observations. In the case of Mishri, only one plant could be studied.

No consistent similarity was observed in relative length of certain chromosomes within various varieties in a group, among different groups of varieties or within different districts. However, no differences were observed in arm ratios and hence in the arm lengths. These were in conformity with the results of other workers.

B chromosomes, abnormal chromosome 10, satellite and heterochromatic blocks were found variable from district to district (Table 3) and also within various varieties in a group. However, there was some consistency in these observations on a group basis, e.g., B chromosomes were observed in 100% of Ferozpur, Niver and Mishri groups, while these were observed in 50% of Chiteh-safed and Dabher groups. The frequency was the same in the case of Tripachi and Lidder groups (Table 4).

Except for the Badeh group (75%), the percentage in the remaining two groups ranged from 0 to 8.3.

Table 1.- Mean relative length ± SE of mid-prophase chromosomes of various groups of varieties of maize.

Groups of varieties	CHROMOSOME NUMBER									
	I	II	III	IV	V	VI	VII	VIII	IX	X
Chiteh-safed	16.14 ± 1.46	13.37 ± 0.68	11.73 ± 0.42	11.03 ± 0.39	10.93 ± 0.42	9.23 ± 0.36	7.70 ± 0.34	8.64 ± 0.35	6.65 ± 0.48	5.45 ± 0.53
Vezij	15.59 ± 0.68	13.05 ± 0.40	12.12 ± 0.42	10.29 ± 0.35	11.09 ± 0.36	9.31 ± 0.45	7.61 ± 0.29	8.50 ± 0.28	6.76 ± 0.40	5.52 ± 0.64
Tripachi	15.98 ± 0.95	13.52 ± 0.63	12.32 ± 0.33	9.68 ± 0.28	10.50 ± 0.26	9.30 ± 0.22	7.79 ± 0.22	8.28 ± 0.20	6.81 ± 0.31	5.79 ± 0.50
Badeh	15.38 ± 0.59	13.32 ± 0.34	11.91 ± 0.27	9.93 ± 0.22	10.98 ± 0.24	9.28 ± 0.23	7.83 ± 0.35	8.53 ± 0.25	6.86 ± 0.25	5.81 ± 0.30
Lidder	16.91 ± 1.08	13.93 ± 0.61	12.12 ± 0.50	9.66 ± 0.32	10.83 ± 0.31	8.90 ± 0.31	7.15 ± 0.36	8.07 ± 0.18	6.65 ± 0.40	5.64 ± 1.07
Dabher	15.72 ± 1.05	13.10 ± 0.52	12.08 ± 0.41	9.87 ± 0.35	10.73 ± 0.50	8.97 ± 0.50	7.76 ± 0.41	8.40 ± 0.48	7.05 ± 0.55	6.19 ± 0.65
Niver	15.77 ± 0.82	13.39 ± 0.39	11.79 ± 0.30	9.63 ± 0.28	10.80 ± 0.29	8.98 ± 0.25	7.75 ± 0.26	8.36 ± 0.25	7.02 ± 0.23	6.38 ± 0.17
Mishri	13.30	12.07	11.21	10.29	10.41	10.18	8.40	10.15	7.49	6.47
Kani	17.64 ± 1.56	14.17 ± 0.52	12.17 ± 0.37	9.92 ± 0.38	11.67 ± 0.45	8.83 ± 0.34	7.15 ± 0.45	8.21 ± 0.43	5.79 ± 0.59	3.76 ± 0.59
Ferozpur	17.27 ± 0.34	13.63 ± 0.88	11.16 ± 1.68	9.49 ± 0.05	10.37 ± 0.30	8.91 ± 0.15	7.41 ± 0.44	7.99 ± 0.81	7.05 ± 0.23	6.61 ± 0.17

Table 2.- Mean relative length ± SE of mid-prophase chromosomes of the local maize varieties grouped in three districts.

DISTRICT	CHROMOSOME NUMBER									
	I	II	III	IV	V	VI	VII	VIII	IX	X
Anantnag	16.08 ± 0.97	14.02 ± 0.55	12.12 ± 0.38	10.02 ± 0.37	10.95 ± 0.34	9.07 ± 0.38	7.51 ± 0.29	8.35 ± 0.29	6.72 ± 0.41	5.47 ± 0.41
Pulwama	15.52 ± 0.80	13.05 ± 0.35	11.75 ± 0.28	9.96 ± 0.21	10.23 ± 0.28	9.39 ± 0.22	7.91 ± 0.28	8.79 ± 0.36	7.31 ± 0.31	5.78 ± 0.37
Baramulla	15.90 ± 0.75	13.20 ± 0.47	11.97 ± 0.40	9.78 ± 0.18	10.69 ± 0.33	9.21 ± 0.17	7.76 ± 0.29	8.49 ± 0.24	6.87 ± 0.25	6.07 ± 0.34

Table 3.- Percentage frequency of B-chromosomes, abnormal chromosome 10, satellites and heterochromatic block in the local maize varieties grouped in three districts.

Cytological observation	D I S T R I C T Anantnag	Pulwama	Baramulla
B-chromosomes	31.2	71.4	33.1
Abnormal chromosome 10	68.7	85.5	33.1
Satellites	62.5	28.5	16.5
Heterochromatic block	18.7	57.1	8.3

Table 4.- Percentage frequency of B-chromosomes, abnormal chromosome 10, satellites and heterochromatic block in various maize groups.

Cytological observation	G R O U P S O F V A R I E T I E S Chiteh-safed	Vozij	Tripachi	Badeh	Lidder	Dabher	Niver	Mishri	Kani	Ferozpur
B-chromosomes	50	8.3	25	75	25	50	100	100	0	100
Abnormal chromosome 10	25	75	75	100	50	25	50	0	100	0
Satellite	12.5	50	50	75	50	25	0	0	0	100
Heterochromatic block	0	16.6	25	25	50	75	50	100	0	0

Abnormal chromosome 10 was observed in 100% of Kani and Badeh groups of varieties; Vozij and Tripachi, Lidder and Niver, and Chiteh-safed and Dabher had 75, 50 and 25% respectively. It was totally missing in the remaining two groups.

The satellite was present with a high frequency in the Ferozpur group but was absent in Niver, Mishri and Kani groups. Vozij, Tripachi and Lidder groups had it in 50% of varieties only. However, the frequency was very low in the Chiteh-safed group.

The heterochromatic block was not observed in Chiteh-safed, Kani and Ferozpur groups. In the remaining groups its percentage frequency ranged from 16.6 to 100.

From the above-mentioned observations it was clear that no two varieties, whether having the same varietal name or otherwise, were identical. One of the important factors for this difference seems to be the hybridization between genetically diverse populations, as maize areas in a locality are contiguous and there is a possibility of free exchange of genetic material from one variety to another.

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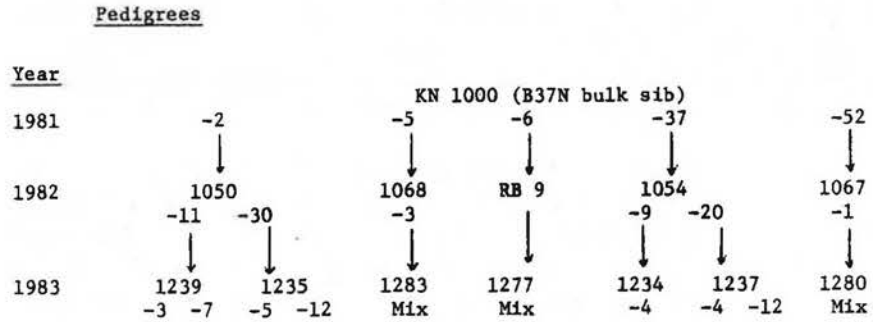
Lack of variation in B37N mitochondrial DNA over three generations

Many studies have now established that there is a great deal of mitochondrial DNA variability detectable by restriction enzyme analysis in a wide range of organisms. In maize, examples of restriction endonuclease fragment differences have been found among the male sterile cytoplasms (Pring and Levings, Genetics 89:121; Borck and Walbot, Genetics 102:109), Mexican races of maize (Kemble et al., TAG 65:120), North American inbred lines (Levings and Pring, J. Hered. 68:350; McNay et al., PMB 2:177), and relatives of maize (Sederoff et al., PNAS 78:5953; Timothy et al., Maydica 28:139). Much of the observed variation is attributable to complex rearrangements rather than to simple base changes leading to loss of particular restriction sites (McNay et al.; Sederoff et al.). Considering the existence of this widespread variability, we wished to examine the relative stability of mitochondrial DNA in individuals from a single inbred line over short time spans: Is the mitochondrial DNA in a particular inbred line homogeneous? Lonsdale et al. (in: Plant Molecular Biology, R. Goldberg, ed., p. 445) have proposed that mitochondrial genomes may exist in multiple forms, which would arise by intramolecular recombination. By self-pollinating maize plants to allow them to sort out any pre-existing variability or to differentially amplify and/or segregate multiple molecular forms, do we begin to see changes within the mitochondrial DNA restriction enzyme fragment patterns? Can we detect rapid changes over short periods of time within a single inbred line? While many generations might be required to sort out pure clones, differences in the stoichiometries of restriction enzyme fragments would be expected to be detectable as differentially staining DNA bands.

The starting material used in our studies was bulk sib seed of the inbred B37N line, kindly provided by Pioneer. In 1981, plants grown from several kernels (probably derived from different ears) were self-pollinated and kernels from 6 of these ears, chosen at random, were grown in 1982 and again selfed. In 1983, mitochondrial DNA was isolated from 7 individual third generation plants and 3 sets of pooled third generation plants (see diagram), as well as from a mixture of



the bulk B37N plants. These samples were subjected to restriction endonuclease digests using 5 enzymes: BamHI (5'GGATCC), XhoI (5'CTCGAG), PstI (5'CTGCAG), HindIII (5'AAGCTT, an AT-rich cutter) and SmaI (5'CCCGGG, a GC-rich cutter). After electrophoresis on 0.7% agarose gels, the DNA bands were stained with ethidium bromide and photographed. A total of 199 fragments were resolved from each individual; this represents a test of about 0.2% of the approximately 600 kb maize mitochondrial genome for the presence of a particular nucleotide sequence. Upon visual inspection, no mobility differences in restriction fragments could be detected either between individuals with the same maternal parent or among all of

the individuals examined. The stained gels gave good resolution of the fragments between 26 and 1.5 kb. Based on standard curve measurements, a change of 75 base pairs would have been detectable in the 1.5 kb range, and changes of 500 bases would have been distinguishable in the 26 kb region. Therefore, if rearrangements, large deletions/insertions or recombinational changes were occurring, they would have been detected in this test. Yet no change was seen. In addition, no differences in band stoichiometries were seen. The mitochondrial genome of the inbred B37N line appears to be very stable.

Thus, despite the variability observed in mtDNA between lines with normal cytoplasms, we have detected no variability between individuals of a single inbred line after three generations of separating cytoplasmic lineages. If there were any mtDNA polymorphisms, three generations may not have allowed sufficient time to sort them out. However, in developing stable inbred lines, breeders probably selected for stability of mitochondrial genomes and also for nuclear/cytoplasmic compatibility. Our results do suggest that, if mtDNA assumes multiple molecular forms, mechanisms must exist to ensure similar distributions during cell divisions. We suggest that mtDNA changes in maize, such as those seen between inbred lines (which appear to result from genomic rearrangements), are rare and are possibly mediated by nuclear genes.

Tony Oro and Kathleen Newton

Speculation about B37N mtDNA evolution

In animals studied to date, the maternally inherited mitochondrial DNA (mtDNA) evolves about 5-10 times faster than the nuclear genome (Brown et al., PNAS 76:1967), based on detection of changes in restriction endonuclease patterns. These differences, indicative of base changes and deletions/insertions in mtDNA, can be readily used to distinguish species. In higher animals, the mtDNA contains a hypervariable region near the origin of replication; analysis of restriction fragment and sequence changes in this region can be used to distinguish individuals from each other. In one study of bovine mtDNA inheritance, a single Holstein had granddaughters differing at one restriction site (Hauswirth and Lapis, PNAS 79:4686).

In the preceding note Oro and Newton showed that no variations in restriction fragment mobility or band stoichiometries were detectable in individuals of a single inbred line after three generations of selfing. In evaluating these data, one problem is that the time-frame for the variation found in the maize mtDNA organization is unclear; that is, we do not know the time of divergence of N and the male sterile cytoplasms nor the age of existing lines of maize, so no calculation of the rate of change is possible.

However, these data can also be used to set an upper limit to the generation of new restriction site polymorphisms arising as the base changes in the sites examined in the B37N mtDNA, using the model of Nei and Li (PNAS 76:5269) for estimating DNA divergence in a population. This model assumes random base-pair substitutions and estimates the time-frame of variability given the number of fragment changes in a population over time. Although we saw no changes, for the sake of calculations we assume 1 fragment change out of the 199 fragments detected in 5 restriction digests of a particular individual over three generations. Using the formula $(N_{xy}/N_0)^2 = e^{-2\lambda rt}$ where N_{xy} = number of fragments in common at time t, N_0 = number of fragments in common at time 0, $2\lambda t$ = rate of nucleotide substitution per nucleotide per unit time, and r = number of bases recognized by the restriction endonucleases used, we calculate a rate ($2\lambda t$) of 8.4×10^{-4} (variance 0.5×10^{-4}) base changes per nucleotide in 3 generations. Assuming a 600 kb genome size for maize mtDNA, we calculate that this rate of substitution represents a change of less than 504 nucleotides (variance 30 bases) per three

generations. Because we have, in fact, detected no changes thus far, this rate of substitution is an upper limit to the variation in maize mtDNA over time. The upper limit of variation in maize mtDNA over several generations is low in comparison with the rate of actual base substitution in animal mtDNA. For example, in a survey of populations of higher primates the rate of nucleotide substitution in mtDNA was 3.2×10^{-2} per year (Brown et al., PNAS 76:1960), forty-fold higher than the upper limit in maize.

Rates of substitution per year in animals are calculated from surveys of existing differences within the whole species or among subpopulations, and from knowledge of the time-frame of speciation from the fossil record. This type of data cannot be obtained for maize because of the paucity of fossils by which to date the emergence of species in higher plants.

We speculate that the changes observed among N, T, C and S mtDNAs and among inbred lines are most likely due to rearrangements or insertion/deletion events rather than a rapid rate of nucleotide substitution in restriction sites.

Tony Oro and Virginia Walbot

Changes in somatic reversion frequency in a progeny of plants with different numbers of copies of sequences hybridizing to a mutator probe

We are interested in determining the relationship between the number of copies of Mutator or Mutator-related elements in the maize genome and the frequency of forward mutation at selected loci and the frequency of reversion from mutant to wild type at unstable alleles. For these studies we are using genes of the anthocyanin biosynthetic pathway. An interesting preliminary result concerns changes in the frequency of somatic reversion we see in the progeny of plants which differ in their Mu copy number.

An unstable bz2 mutable (bz2-mu-1) with 25% of the surface covered in purple spots of 1-20 cells was recovered in 1982 from a cross of a purple aleurone Mutator line (kindly supplied by D. Robertson) as male onto bz2 tester in a hybrid W23/K55 background (kindly supplied by E. H. Coe, Jr.). The somatic reversion seen in the unstable bz2-mu-1 mutation is assumed to involve the excision of the Mu sequence from the allele restoring essentially wild type phenotype in revertant sectors. Because mutator insertion creates a short host sequence duplication, it is assumed that the unstable nature of the bz2-mu-1 mutation is due to the insertion of a copy of the mutator into the Bz2 gene in a position in which no permanent loss of gene function occurs after the element excises. For example, the element may have inserted into a noncoding region or other position where it eliminates transcription or prevents proper processing, and upon excision the duplicated sequence does not interfere with the gene's expression. The mutant was backcrossed to the hybrid tester in the greenhouse, and progeny kernels, each with approximately 25% of the total surface area occupied by small revertant sectors encompassing 1-20 cells of the aleurone, were planted in the 1983 summer crop. At the 8-leaf stage a lower leaf was removed, DNA prepared, and slot blot hybridization carried out by M. Fromm using a cloned Mutator (Mu) element as the radioactive probe to DNA samples from each of 30 plants. The hybridization results clearly showed that there were plants with high, medium and low copies of Mu or sequences cross-hybridizing with mutator. There was approximately a 10-fold difference in copy number between the high and low classes.

Each classified plant was then testcrossed by the hybrid bz2 tester, crossed onto several hybrid tester ears, and the second ear self-pollinated. In these progeny ears we have scored the occurrence of revertant sectors. In those kernels with revertant sectors, the spots are still about 1-20 cells in size. The expectation, barring any effects of mutator copy number, is that in each outcross ear 50% of the kernels will have spots because they are bz2-mu-1/bz2 and 50% of

the kernels will be colorless (bz2). In a self-pollination, 75% of the kernels are expected to have spots: 1/4 bz2-mu-1/bz2-mu-1, 1/2 bz2-mu-1/bz2, 1/4 bz2/bz2. The table summarizes the observed distribution of kernels with somatic sectors for eight plants in the experiment:

Classification (Family Number)	Self Pollination Results	Testcross Results
Low Mutator Copy Number (MF-9, 230-1)	75% kernels with spots	1 - 5% kernels with spots
Low Mutator Copy Number (230-7, 230-8)		
Medium Mutator Copy Number (230-5, 230-6)	75% kernels with spots	50% kernels with spots
High Mutator Copy Number (230-2, 230-4)	1 - 2% kernels with spots	50% kernels with spots

With outcrosses, the frequency of spotted kernels was independent of whether the mutator plant was the ear or pollen parent. In self-pollinated ears with the expected number of spotted kernels, it is interesting to note that approximately 1/3 of the spotted kernels were 50% or more occupied by somatic sectors and 2/3 of the kernels were 10-30% occupied by somatic sectors, suggesting that the heavily spotted kernels are the homozygotes and that each copy of bz2-mu-1 can revert independently. Tests of these suppositions are underway.

The unexpected observation is that, although self-pollinated ears of low copy number parents show 75% kernels with spots, the outcross ears in two families fail to have as many revertant kernels as would be expected from the segregation of the mutable allele. Naively we can propose that in the testcross either the number of Mu copies or some other factor was lost or diluted out so that reversion becomes less frequent (or very much later in development). Conversely, in high copy number parents self-pollination, which could increase the copy number of Mu or of another factor influencing Mu behavior, drastically reduces the number of kernels with spots, although in testcrosses 50% of the kernels are spotted. This result suggests that a high copy number of Mu may also prevent somatic reversion.

It is possible that Mu encodes both a transposase which can become rate-limiting at low Mu copy number, as well as a repressor (of transposition or of transposase gene transcription) which at a high Mu copy number effectively prevents excision of the elements. In such a model only lines carrying an intermediate number of Mu copies will have an active mutator system viewed from the perspective of excision from known loci. Indeed the medium copy number parents in this experiment demonstrated expected segregation of spotted kernels in both testcrosses and in self-pollination. Mutator activity in transposition and insertion at other sites may or may not parallel the frequency of Mu excision from a known mutable allele. Tests are underway to determine whether mutation frequency, a measure of transposition and insertion, is affected to the same extent and in a manner parallel to excision frequency in somatic tissue in lines with varying numbers of Mu sequences. We are also testing whether the quiescent bz2-mu-1 allele in low copy number self ears can be activated by crossing in Mu copies from a bz2 kernel with a high number of copies of Mu, and whether the quiescent bz2-mu-1 alleles in self-pollinated high copy number ears can be activated by outcrossing the progeny plants to lines lacking mutator activity, a cross which should dilute the active copies of the Mu sequence.

Virginia Walbot

Marking pens can cause lesions in Les mutants

As part of our study of the phenotypes of disease lesion mimics (see Hoisington et al., 1982, Devel. Biol. 98:381; Walbot et al., 1983, in: Genetic Engineering of Plants, ed. T. Kosuge et al.) we measure the spread of lesions on individual leaves. The leaves are numbered using a marking pen. Inadvertently, we discovered that the MARKSALOT brand of pen induced dramatic lesion formation in Les1 and Les*-1451 leaves, which at the time had not yet made lesions. Normal sibs showed no lesions. Several other Les mutants show some lesions along pen marks. The effect occurs within 24 hr--the edge of the pen mark is covered with lesions. All colors of MARKSALOT cause this effect, suggesting that the dye is not causing the lesions. No other brand of marking pen tested causes lesions, suggesting that there is a specific chemical found in this brand of pen. From a list supplied by the manufacturer of chemicals in MARKSALOT, the "active" ingredient was shown to be n-propanol. Isopropanol, n-butanol, ethanol and methanol have no effect. The propanol works in a 5% aqueous solution; n-propanol or a contaminant may be a toxic "trigger" to plants with certain Les mutations.

Query: Has anyone ever noticed tissue damage from marking pens?

Nigel Ray and Virginia Walbot

Ears and kernel sectors differing in somatic reversion frequency

In observing the frequency of somatic reversion in bz2-mu-1 materials we have noted several classes of interesting events: (1) individual kernels with crown-only or base-only patterns of revertant sectors; (2) half kernels or smaller sectors in which one part has a low (5%) frequency of coverage by somatic reversions and the other part has a high frequency (50%); (3) an ear of medium Mu copy number of genotype bz2-mu-1/bz2, testcrossed by a bz2 hybrid tester, had a sector comprised of approximately 30 kernels which exhibited the expected 50:50 segregation for spots. Of the remaining ca. 300 kernels on the ear, however, only 5 had spots.

These observations on the distribution of spotted kernels on the ear and changes in the distribution of spots on individual kernels suggest that a change in the capacity to undergo somatic reversion can occur in cells during ear and kernel development. Changes in reversion frequency may reflect changes in the copy number of Mu in cell lineages, a phenomenon parallel to the observation that Mu copy number influences the apparent transmission of the spotted kernel phenotype, as discussed in the accompanying article.

Virginia Walbot

Quantitative genomic change in maize

In a previous newsletter note, Chris Cullis and I reported that most repeated DNA sequences in maize vary considerably in copy number between inbred lines. Testing 10 cloned sequences that differ in function and molecular arrangement and have reiteration frequencies of 100 to over 100,000 copies/genome, we found that nine showed significant quantitative diversity among inbred lines.

These differences appeared to be stable quantitative polymorphisms because the copy numbers measured are characteristic of all individual plants within a line and are the same in DNA extracted from different plant tissues. Therefore, we anticipated that when a cross was made between inbreds that differed in copy number for a particular sequence, the F1 plants would all be identical to each other and have copy numbers that were equal to the mean of the parental lines.

We found, however, that while each of the F1s had the same copy number for a particular sequence, that number was not necessarily the parental mean. In a manner that is specific to the genotypes involved in the cross, the copy number of a repeated sequence can be dramatically different from Mendelian expectation. Progeny from 25 crosses including reciprocals have been analyzed. A sample of the data is given below. Although the phenomenon is not yet fully described, the following appear to be true:

1. The change is not random. A particular genomic change is found each time a cross is made and in all progeny of that cross.
2. Reciprocal crosses result in the same genomic changes.
3. Neither the occurrence of F1 change nor the magnitude of the change is related to the copy numbers or difference in copy numbers of the parental lines.
4. The direction of copy number change is consistent for each sequence. In the data we have collected so far, rDNA and 5S sequences either show the parental mean or a decrease in number, whereas a sequence associated with chromosome knobs, pZmK10, and one that might have a role in DNA replication pZmA101, have only shown changes that are increases over expected values.

Repeated sequence copy number changes in F1s expressed as observed number/parental mean:

Sequences:	rDNA	5S	K10	A101
Cross:				*
W22 x Bl. Mex. Sweet	.70	.66	1.56	ns
Bl. Mex. Sweet x W22	.65	.55	1.44	ns
W22 x Kv21	.50	ns	1.24	ns
Kv21 x W22	.59	ns	1.26	ns
Kv21 x Bl. Mex. Sweet	ns	ns	ns	1.22
Bl. Mex. Sweet x Kv21	ns	ns	ns	1.28

* ns indicates the ratio is not significantly different from 1.0

Experiments are in progress to explore the timing, genetics and developmental ramifications of this rapid genomic change. Measurements are being made on DNA samples from early embryonic tissue of both F1 and inbred plants to determine when genomic changes take place. F2, F3 and backcross progeny are being examined to determine if there is continued change, stabilization or segregation for repeated sequence copy number. I am also looking at a new set of genotypes to ask whether change or stability of the F1 genome is correlated with hybrid vigor.

Other related work in progress is a study of the genomic stability of maize cells in culture. I have been following 3 embryo cultures started from a selfed ear of A188 over 6 months in culture. By two months, quantitative differences in repeated DNA sequences were apparent between each culture and the inbred plant genome. The changes that have accumulated are similar in magnitude and direction to those observed in the outcross progeny. I am continuing to follow these cultures to ask how differentiation and regeneration may affect or be affected by genomic fluctuations.

I welcome any questions or comments about genomic and phenotypic stability in maize. After March 1984 I can be reached at the Department of Botany and Plant Pathology, Oregon State University, Corvallis, Oregon.

Carol Rivin

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Changes in mitochondrial DNA associated with NCS2 and NCS3 mutants

"Nonchromosomal stripe" (NCS) refers to unstable, maternally inherited mutants that have drastic and variable phenotypic effects including short stature, reduced viability and vigor, abnormal growth patterns and leaf striping (Shumway and Bauman, Genetics 55:33; Coe, Maydica 28:151). The specific materials studied by Shumway and Bauman, termed NCS, have been lost; however, two others, NCS2 and NCS3, were available for our study. Each of them arose in WF9, or the closely related line H49, with T-type male sterile cytoplasm. The generation of these mutants is directly dependent on the nuclear genotype (NCS-like plants can be generated with any cytoplasmic type in the WF9 background--see Duvick, Adv. Genet. 13:1). However, once present, the inheritance of the defective phenotype appears to be strictly maternal. We decided to examine the organellar genomes of NCS2 and NCS3, to compare them with the progenitor and with normal derivatives of NCS lineages.

Differences in restriction endonuclease fragment patterns between the progenitor mitochondrial DNA (cms-T) and both NCS2 and NCS3 have been seen with two different enzymes, XhoI and PstI. NCS2 has lost an XhoI band at approximately 8 kb and gained a 20.5 kb novel fragment. A new 20 kb XhoI band and a correlated reduction of a 16 kb fragment are observed with NCS3. When chloroplast DNA from NCS3 was examined with the same enzyme no changes in restriction patterns were seen. With PstI, a novel band appears at 7.6 kb in NCS2 mitochondrial DNA, and there is a corresponding loss of a 3.7 kb band. A new PstI fragment of 23 kb has been observed with mitochondrial DNA from NCS3. While an explanation of these results is not yet clear, they do seem to rule out the possibility that NCS2 and NCS3 result from simple losses in specific XhoI restriction sites.

The severity of the NCS3 phenotype is correlated with the relative amount of DNA in the new restriction enzyme band. NCS3 gives a continuum of phenotypes which appear to be quantitatively different expressions of the same basic defect. Affected plants range from nearly normal (tall with few striations, moderately affected) to shorter and heavily striated, with asymmetries of leaf and cob, to severely affected (extremely short, highly distorted, with few stripes but large losses of leaf tissue). The most severely affected plants are so morphologically abnormal that no tassels or ear shoots are formed. Mitochondrial DNA was isolated from the ear shoots of individual NCS3 plants, which were scored for their phenotypic severity. Following XhoI digestion and gel analysis, we found that there is a positive correlation between the severity of the defect and the amount of DNA in the novel 20 kb band. As the amount of DNA in the 20 kb band increases, the amount of DNA in a 16 kb band is correspondingly decreased.

In any NCS3 family, the sib plants are highly variable in their expression of the mutant phenotype. A number of plants in a single, variably-expressing NCS3 family were pollinated by the same male inbred. Subsequently F1 kernels from ears of a normal looking plant and kernels from an ear of a strongly expressing sib plant were planted. The progeny of the normal looking NCS3 plant were all non-mutant in appearance; hence the NCS3 determinants had been lost from this lineage. The progeny of the affected plant showed a range of phenotypes, but most were visibly mutant. MtDNA was isolated from individuals of both the stable, normal plants and from the NCS3-affected plants. XhoI digests showed that the normal plants derived from an NCS3 lineage lack the 20 kb band, whereas affected plants carry this band. The plants used had exactly the same grandmother, and mtDNA in maize is strictly maternally inherited. However, only NCS3-affected

plants have the 20 kb XhoI restriction enzyme fragment. We propose that NCS3-affected plants carry a mixture of mutant and normal mitochondrial genotypes, that these genomes sort out during development of the plant and that cells carrying only NCS3 mtDNA die, resulting in stripes and tissue loss. Lineages that contain purely normal mtDNA can no longer sort out defectives.

There is also a correlation of NCS2 phenotypes with the XhoI band differences. An NCS2 derivative plant that had lost the NCS2 phenotype (striping, shortened stature etc.) had lost the 20.5 kb XhoI mtDNA band as well. We therefore propose that the cells of NCS2-affected plants, like those of NCS3, carry a mixture of normal and mutant mitochondria that sort out during development. If no mutant mitochondrial genomes are represented in the cells giving rise to an ear on an individual plant, the mutant phenotype will no longer be transmitted.

Kathleen J. Newton and Ed Coe

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A fresh look at a peculiar type of non-linear response of performance to level of heterozygosis in maize

While most investigations into the relationship between heterozygosis and performance in maize did not reveal important deviations from a linear response, there is one early reported exception still lacking a satisfactory explanation. This refers to the findings of J. C. Sentz et al. (Agron. J. 46:514-520, 1954), who compared five genetic groups representing 0%, 25%, 50%, 75%, and 100% heterozygosis. Unexpected deviations from linearity were observed with "second backcrosses" (B11 and B22) and "double backcrosses" (B12 and B21), which formed the 25% and 75% heterozygosis groups, respectively. Almost consistently over crosses, places and years, these deviations were antagonistic in direction (namely, enhancing with second, but depressing with double backcrosses) and asymmetric in size (namely, relatively larger with second than with double backcrosses). Furthermore, this peculiar pattern of curvilinear response considerably varied in expressivity over environments.

In discussing their results, Sentz et al. were led to attribute them to epistasis and, of course, to genotype-environment interactions, but not to any form of selection during development of the segregating generations. Quoting the authors: "It is difficult to visualize any natural selection for either gametes or zygotes which would simultaneously depress expression in the double backcross generations and enhance it in the second backcross generations." Nevertheless, it would seem to the present writer that those antagonistic effects could well have been brought about by an unconscious selection of more vigorous plants of the first backcrosses (B1 and B2) in producing the seed of the second and double backcrosses. Such selection would change the means of the latter generations by amounts which in sign and size depend on the covariances of B1 and B2 with these generations. Table 1 specifies averages of the pertinent covariances in terms of the model of R. E. Comstock and H. F. Robinson (Biometrics 4:254-266, 1948), assuming linkage and epistasis to be absent. It appears therefore that the average covariance of first with second backcrosses will always be positive. But the average covariance of first with double backcrosses may be positive, zero, or negative according to whether the respective average degree of dominance is below, equal to, or above one. In practice, a negative value of this covariance may result not only from the prevailing of true overdominance, but also from pseudo-overdominance due to repulsion linkages. The latter situation seems to be not

uncommon in corn, and has indeed been demonstrated for one of the two crosses used by Sentz et al. (R. H. Moll et al., Genetics 49:411-423, 1964).

Table 1. Average covariances between indicated generations.

First and second backcrosses :	$\frac{1}{2}(cov_{B_1B_{11}} + cov_{B_2B_{22}}) = \frac{1}{8}\Sigma u^2 + \frac{1}{8}\Sigma a^2 u^2$
First and double backcrosses :	$\frac{1}{2}(cov_{B_1B_{12}} + cov_{B_2B_{21}}) = \frac{1}{8}\Sigma u^2 - \frac{1}{8}\Sigma a^2 u^2$

Finally, besides genotype-environment interactions, the fact that the seed in part had been produced anew for successive testing years may account for the varying expressivity of the curvilinear response observed in the study in question.

F. W. Schnell

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The 1.94 kb mtDNA plasmid is absent in some cms-S cytoplasms

All four maize cytoplasm groups (N, cms-S, cms-T and cms-C) have been shown to contain supercoiled DNA molecules of 1,940 base pairs in their mitochondria (Kemble and Bedbrook, Nature 284:565, 1980). We have recently found, however, that not all members of the cms-S group possess these plasmids. Six cms-S cytoplasms were studied in depth; three carried the 1.94 kb plasmid (S, J, I) whereas three did not (VG, RD, ML).

We have used a full length recombinant DNA clone of the 1.94 kb plasmid (Thompson, Kemble and Flavell, unpublished) to probe Southern blots from agarose gels of mtDNAs from over one hundred combinations of cms-S cytoplasms and nuclear backgrounds. In each case, regardless of nuclear background, mtDNAs from S, J and I cytoplasms consistently hybridized strongly to the 1.94 kb probe whereas RD, ML and VG mtDNAs did not show hybridization. Inbred lines homozygous for Rf3, non-restoring backgrounds and the Laughnans' cms-S revertants to fertility all held strictly to the plus, minus pattern. Those cytoplasmic revertants arising from I cytoplasm, for instance, still retained the 1.94 kb plasmid at parental levels, while revertants to fertility from VG cytoplasm lacked the plasmid, as did their sterile parents.

Although the presence or absence of the 1.94 kb plasmid neatly subdivides the cms-S group of cytoplasms, no obvious function is yet apparent for this mtDNA entity. Of interest, however, is our observation that maize nuclear chromosomal DNA contains substantial sequence homology to this 1.94 kb plasmid and the cms-S S1 and S2 linear mtDNAs (Kemble et al., Nature 304:744, 1983). So the 1.94 kb plasmid may yet prove to be more than a "vestigial" phenomenon.

John E. Carlson and Roger J. Kemble

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Lysine-threonine inhibition in callus culture

In the previous work (Nesticky et al., Z. Pflanzenz. 86:33-40) the possibility of using lysine-threonine inhibition in embryo culture was studied. From the inbred lines used in this study, inbred line MT42 and its opaque-2 analogue were chosen to verify this inhibition on the level of callus culture in vitro. Callus cultures were initiated from the excised embryos of mature kernels on basal medium MS. During three subcultures, callus growth on selective medium with 2 mM lysine and 1 mM threonine was observed.

Callus growth of the opaque-2 analogue on the selective medium reached on average 30.1% in comparison with the control. Within every subculture, callus growth varied from 19.4% to 45.5%. Callus growth of the normal analogue on the selective medium with lysine and threonine reached 61%. This means that the growth of the opaque-2 analogue on selective medium was two-fold less than normal.

This result is in contradiction with our previous study, in which the opaque-2 analogue in embryo culture with exogenous lysine and threonine significantly outgrew the normal analogue. This disagreement in results cannot be explained by the already known hypotheses of Rapela (MNL 55:54, Plant and Cell Physiol. 23:285-291) and Phillips et al. (Crop Sci. 21:601-607) because both results were obtained on the same genotype. Knowing the results of Lukjanuk and Ignatova (pers. commun.), it is possible that the ontogenetic stage and/or level of differentiation of explant will play a role in the expression of inhibition in one genotype. Study of this problem is in progress.

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Analysis of combining ability of total grain nitrogen

Combining ability of the total grain nitrogen, yield of nitrogen and grain yield in two sets of diallel crosses was studied. In the first set high protein inbred lines Early 7, Tva200HP, Hymador 222, P270 Barilles and P263-213, were used. In the second set of diallel crosses, except for the above-mentioned inbreds, normal inbreds Tva10870 and F115 were included. The combining ability was analyzed according to Griffing (Model I, Method I). The results are as follows:

1. Analysis of genetic variability of the content of the total grain nitrogen showed significant influence of GCA on this character in both diallel sets.
2. GCA:SCA ratio (1:2) in the first diallel set indicates that grain yield in high protein inbreds is mainly determined by SCA. By including normal inbreds in the diallel set the ratio of GCA:SCA becomes more nearly equal (1:1). Comparison of this finding with our previous unpublished studies, in which combining ability of yield of normal inbreds was determined only by GCA, shows that grain yield of normal and high protein genotypes is probably controlled by different genetic systems. This conclusion is in agreement with the assumption of Pollmer et al. (Z. Pflanzenz. 80:142-147), that decreased yield potential of high protein genotypes is a consequence of long-term selection pressure, during which the genetic system controlling grain yield has changed.
3. Reciprocal differences in the content of the total grain nitrogen were found only in the F1 generation. No reciprocal effects were found in further generations. This fact confirms our previous finding from breeding of high protein inbreds, that any differences in the protein content between direct

and reciprocal crossing disappeared after three generations of self-pollination. This conclusion could be used in breeding of high protein genotypes to eliminate some negative characters of the genetic source.

4. As for the yield of nitrogen, it is more dependent on grain yield than on nitrogen content. Correlation coefficient between yield of nitrogen and grain yield was 0.885**. No correlation was found between grain yield and content of the total grain nitrogen.
5. The best hybrid combination from the diallel sets was Hymador 222 x F115, which had a nitrogen content of 15.25% and reached a grain yield of 8.60 $t.ha^{-1}$ and a nitrogen yield of 1.31 $t.ha^{-1}$. There were other hybrid combinations of the high protein x normal type, which overcame the population mean in all characters. This shows a possibility of production of high protein hybrids with good yield potential by combination of the high protein x normal genotypes.

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Brown's cytoplasmic type

In 1961 W. L. Brown reported a cytoplasmic type in corn characterized by greatly reduced plant height and general vigor, together with many short fine white streaks on the leaves. He introduced the genome of several standard lines into this cytoplasm without modifying the original phenotypic pattern. I have made additional attempts to find a restorer for this unusual syndrome. As only the more normal plants within the affected class produce seed, the line by line approach was discarded in favor of a shotgun approach. Pollen was collected, repeatedly, throughout my nursery and applied to silks of the cytoplasmic type. In 1982, one normal plant was observed in the progeny of such crosses. Selfed and backcross progeny grown in 1983 suggested that a restoration type phenomenon was involved. Due to poor stands and the unfavorable season, the data on restoration are probably inadequate but inheritance of restoration appears to be simple, possibly due to a single gene. The symbols (cgh) - cytoplasmic growth habit - and Rgh (restorer) are suggested.

G. F. Sprague

Glossy seedlings

The Coop collection contains a large number of unidentified glossy seedling types. As these are potentially useful seedling traits, I have been testing these against my standard series. As these have come from diverse backgrounds, all testing has been done with heterozygotes to minimize maturity differences and simplify intercrossing. To date, some 40 items have been tested. Similarly, a series of glossy mutants provided by M. G. Neuffer have also been tested. Thus far all have been found to be repeats of known glossies.

G. F. Sprague

Tassel silks and branched ear

This type was found in one of my stocks involved in the inheritance of yellow endosperm color. The trait exhibits pleiotropism, both tassel and ear being

affected. The tassels exhibit few to many silks but no seeds are produced. The ears exhibit varying degrees of basal branching. Both manifestations are affected by environment and background, with the tassel manifestation being the simplest to score. Tests with the recessive tassel seed indicated non-allelism. Additional F2 tests involving bm2 lg1 su y1 wx and g have given no indication of linkage. The symbol tsi is proposed.

G. F. Sprague

Dotted-6

Several mutable types have been isolated from virus exposed cultures. One of these has been designated Dt6 and is located on chromosome 4. The relation to Su su is indicated below:

Su Dt6	Su dt6	su Dt6	su dt6	
982	77	56	341	7.5% crossing-over

Three point tests involving the cross a su Gl3 dt6/a Su gl3 Dt6 were inconclusive, both gl3 and dt6 exhibiting independence with su but suggesting that Dt6 is proximal to su.

G. F. Sprague

Mutable a-mt-1 and its control by Sta (Uq)

A number of apparently different controlling element systems have been found in populations derived from my earlier AR studies. Some years ago, a number of these were supplied to P. A. Peterson and he and Friedemann (MNL 54:2, 55:6) have reported on one they designated Uq. Under my terminology this stock is designated a-mt-1.

In the original stock, this phenotype exhibited a coarse but fairly uniform mottling and gave rise to both self-colored and colorless seeds in high frequency, about 5% colored and 2% colorless. Changes were both somatic and germinal and true breeding types were easily established. A third type of change to a fine mottling or dotting was also observed but in much lower frequency. Subsequent tests indicate that the two mutant types are due to mutations at different loci. The self-colored type is due to mutation of the a-mt-1 allele to A. This new A, when homozygous, retains the brown cob color typical of standard a. The colorless mutation is at a second locus (Sta sta) which controls the mutability of a.

Results from a series of crosses involving the colored and colorless mutant types with standards are presented in the following table.

Crosses	F1	F2		
		A	Mt	a
a-mt-1 sta / a sta	a			all
a-mt-1 sta / a Sta	mottled		1593	1528
a-mt-1 sta / A sta	self-color	1480	0	487
a-mt-1 sta / A Sta	self-color	3032	780	229
A-mt-1 sta / a sta	self-color	1371	0	441
A-mt-1 sta / A Sta	self-color	1143	283	101

a-mt-1 behaves as a stable a allele. In the presence of Sta a-mt-1 is mutable, giving rise to typically mottled seeds. The behavior of the A mutant type remains

somewhat anomalous. When crossed to standard A C R stocks, the F2 results are as indicated in the last line of the table.

G. F. Sprague

Heritable differences in glucose levels among sugary corn lines and sporadic maltose accumulation in heterozygous sugary enhancer sweet corn kernels

The sugary enhancer (se) endosperm mutant was identified as a recessive modifier of sugary (su) in the sweet corn line IL677a (J. E. Ferguson et al., J. Hered. 69:377-380, 1978; and R. A. Brink, MNL 52:110-112, 1978). Homozygous sugary-sugary enhancer (su se) kernels are characterized by elevated sucrose at the eating stage (about 21 days after pollination) and by elevated maltose at the mature dry stage (J. E. Ferguson et al., Plant Physiol. 63:416-420, 1979). A breeding program was undertaken to transfer se from IL677a to a number of different su backgrounds. Selection on segregating ears was based on kernel appearance; su se kernels are thought to be slower to dry during maturation, and to be lighter colored and more finely wrinkled at the mature dry stage than su Se kernels. Immature self-pollinated ears from plants grown from the selected seed were tasted in the field to test for the expected levels of sweetness.

Sugar and sorbitol levels in mature dry seed of the lines developed in the breeding program were analyzed by gas-liquid chromatography to check the success of selection for se. Sucrose and maltose are the diagnostic sugars, but sorbitol, fructose, and glucose were included for completeness. Most lines analyzed were found to have elevated maltose levels, indicating successful selection for se, but a few lines had low maltose levels. The low maltose lines generally had higher glucose levels than their high maltose counterparts (E. E. Carey, M.S. Thesis, Univ. of Illinois, 1981). Five standard su Se inbred lines and IL677a were analyzed at the same time for comparison with the lines from the se breeding program. The glucose levels of the su Se lines varied markedly (Table 1), with IL14h having very low levels, IL442a and IL11a having intermediate levels, and IL454a and IL459a having high levels. IL442a, one of the parents of IL677a, also had low levels of maltose. That sugar levels of the inbred lines were stable over successive generations was determined by analysis of remnant seed of four successive generations (E. E. Carey, M.S. Thesis).

In an attempt to compare the behavior of the glucose trait in the lines from the se breeding program with those in the su Se lines, a number of crosses were made (some reciprocal) and sugars in the mature dry F1 seed were analyzed. Table 1 lists the sugar and sorbitol levels of the parents (selfs) and the crosses. IL11a through IL459a were the su Se lines, IL751a and ILB5765 were the high glucose-low maltose lines from the se breeding program, and IL677a and IL753a were the su se lines (IL753a derived from the se breeding program).

In crosses between the high glucose lines from the se breeding program (IL751a and ILB5765) and the high glucose su Se lines (IL454a and IL459a), glucose levels were consistently elevated. High glucose was dominant to low glucose in the cross IL454a X IL14h and its reciprocal, but not in IL14h X IL677a which accumulated almost no glucose. Heritable differences in the levels of reducing sugars in sweet corn lines might be of interest since canning quality is adversely affected by elevated levels of reducing sugars.

In crosses between su Se and su se lines, maltose levels were generally low, but in a few cases maltose accumulated when the female parent was se. It is difficult to draw conclusions about the cause of this maltose accumulation because of the limited number of crosses that were made and the inconsistency with which it occurred - for instance, IL753a X IL442a accumulated maltose but IL677a X IL442a did not.

Table 1. Sorbitol and sugar levels in the mature dry seed of [1] crosses and inbred parent lines[z]

Name of Cross or Parent line	Sorbitol	Fructose	Glucose	Sucrose	Maltose
	% of dry weight				
IL11a	0.01	0.05	0.46	2.79	0.08
IL14h	0.01	0.06	0.08	2.54	0.11
IL442a	0.06	0.18	0.24	4.06	0.56
IL454a	0.06	0.25	1.32	4.19	0.21
IL459a	0.01	0.14	1.17	2.20	0.04
IL751a	0.53	0.75	1.18	7.42	0.28
ILB5765	0.07	0.28	0.64	4.29	0.10
IL677a	0.23	0.37	0.51	4.79	1.62
IL753a	0.20	0.34	0.63	6.51	5.64
IL14h X IL11a	0.04	0.10	0.61	2.12	0.01
IL454a X IL11a	0.01	0.02	0.82	2.93	0.01
IL11a X IL454a	0.05	0.10	0.56	3.18	0.01
IL454a X IL14h	0.05	0.12	1.22	2.99	0.04
IL14h X IL454a	0.18	0.34	1.30	3.77	0.03
IL751a X IL454a	0.17	0.39	1.18	4.87	0.01
IL454a X IL751a	0.07	0.17	2.11	6.16	0.10
IL751a X IL459a	0.11	0.15	0.78	3.46	0.01
IL459a X IL751a	0.01	0.05	0.83	2.83	0.01
ILB5765 X IL454a	0.06	0.20	1.15	3.80	0.01
ILB5765 X IL459a	0.03	0.15	0.78	2.95	0.03
IL459a X ILB5765	0.01	0.08	1.00	2.69	0.05
IL11a X IL677a	0.03	0.06	0.47	2.02	0.01
IL677a X IL14h[y]	0.21	0.36	0.44	4.85	1.13
IL14h X IL677a	0.05	0.07	0.08	1.95	0.01
IL677a X IL454a	0.06	0.17	0.84	3.64	0.05
IL454a X IL677a	0.04	0.17	0.77	4.29	0.08
IL677a X IL459a	0.12	0.25	0.64	4.69	0.01
IL459a X IL677a	0.02	0.13	1.09	3.03	0.07
IL677a X IL751a	0.14	0.23	0.60	5.32	0.09
IL751a X IL677a	0.29	0.40	0.91	6.56	0.08
IL677a X ILB5765	0.05	0.33	0.56	4.87	0.05
ILB5765 X IL677a[y]	0.14	0.28	0.54	4.63	0.03
IL753a X IL14h	0.07	0.22	0.26	3.35	0.78
IL753a X IL442a	0.06	0.09	0.28	3.02	1.54
IL442a X IL753a	0.02	0.05	0.16	3.31	0.36
IL677a X IL442a[y]	0.01	0.04	0.07	2.90	0.02

[z]Unless otherwise noted, one ear was analyzed. [y]2 ears analyzed.

Accumulation of maltose in heterozygous se and non-se (i.e., IL442a) kernels indicates that conclusions about the allelic state of se should not be made on the basis of maltose alone. A more thorough understanding of the expression of se and its effects on su will require more complete genetic studies. These will probably involve analysis of individual kernels from segregating ears, and development of near-isogenic lines for more controlled physiological genetic studies.

Edward E. Carey

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Specialized traits of the oldest known maize cobs are absent in the teosinte tassel (contrary to basis of CSTT)

According to the CSTT of Iltis (Science 222:886-894, 1983), the maize ear originated by a transmutation in teosinte of the target area for expression of the secondary male traits to also include the female spike. This transmutation supposedly accounts for the archaeological record in which there seems to be a sudden despecialization of the teosinte female spike with a concomitant loss of induration, a reduction of cupules and a reactivation of the second member of paired spikelets. But the fact is that the oldest maize cobs are still more similar to the teosinte ear than to its tassel in regard to both cupule development and glume shape.

The phenotype of the tassel seed mutants of maize should be an example of what happened according to the CSTT. But the morphology of neither tassel seed maize nor of tassel seed teosinte fits the Iltis theory. In both cases female development in the tassel is associated with the fruit case derivatives for cupules, induration and glume shape typical of their normal target areas. Even with normal teosinte and maize, the female areas within the mixed (bisexual) inflorescences that usually terminate tillers are also associated with these fruit case derivatives as expressed in their ear type, contrary to the expectations of CSTT.

It is also significant that the long rachilla that is characteristic of the oldest cobs is absent in both the ear and tassel of teosinte and so it could not be derived from transmutation from the tassel. Rather the long rachilla is one of several pleiotropic effects that are controlled primarily by a series of multiple alleles at the tunicate locus. These other effects include softer, longer female glumes and some cupule reduction, which are traits that Iltis would attribute to transmutation, but more probably came from domestic selection for a tunicate allele.

The effects of a weak tunicate allele still occur in Chapalote, an ancient indigenous race of maize in Mexico. Its phenotype in Chapalote is considered by Mangelsdorf to be similar to that of its prehistoric precursor in the oldest cobs from Bat Cave, New Mexico and probably to that of certain of the oldest known cobs from caves near Tehuacan, Mexico. Since Iltis ignores any role for the tunicate locus during the origin of maize, the domestic advantages of an elongate rachilla in the ear need to be mentioned here. The long rachilla not only elevates the kernel to a level near the apex of the glumes and, thereby, exposes the kernel except for a little membranaceous tissue from the lemmas and paleas, but it also reflexes the spikelet away from the cupule and thereby makes it threshable. Protection from birds at this stage becomes dependent upon an enclosure of the entire ear by husk leaves borne on the shank.

Other serious errors of fact in the Iltis article include the statement (p. 888) "no key genes differentiating maize from teosinte have ever been found." He goes on to explain, "This is because, in fact, they do not as such exist." The fact is that genes controlling the key traits are known: (Pd pd) - paired vs. single female spikelets and (Tr tr) - many vs. two-ranked spike. All combinations of these genes occur in the F2; although the phenotypes of the teosinte alleles tend to be unstable in a highly evolved maize background, their expression can be stabilized through selection. As a single event in the origin of maize, the transmutation speculated by Iltis should segregate as a simple Mendelian factor in the F2 of hybrids and, thereby, yield only maize and teosinte. This is obviously not the case.

The morphology of the oldest maize cobs and the genetics of maize-teosinte hybrids make the contentions of Iltis untenable.

Walton C. Galinat

The introduction of alien genes, their adaptation and integration within a new genome

The expression and apparent heritability of a gene that is suddenly thrust into an unprepared genome of a complex organism such as corn is often not clear and not stable. Although the details of a trait's heritability, the type of gene action and interaction and the genetic location on the chromosomes may take years to discover, this does not preclude attempts to use said trait for crop improvement. First, however, some degree of heritability needs to be established, such as is the case with sporadic resistance or tolerance of plants to pests.

The sugary enhancer (se) gene is an example of a gene or complex of genes whose inheritance and location is not yet understood, and yet we go ahead and use it in sweet corn improvement. In the case of sh2, bt2, wx, o2 and several other endosperm mutants, their genetics was worked out long before their practical application. During the genetic experiments, the background of these mutants was usually adapted for increased viability by unconscious selection of modifying genes that affect the growth pattern of the pericarp.

Generally, the more divergent the background source of a gene, such as from an unrelated or exotic race of corn, the more difficult its integration into a new background. This is especially true with germplasm extracted from corn's wild relatives, teosinte and Tripsacum. In the case of teosinte, when its key traits are transferred to a background of modern maize they have unstable expression and variable linkage relationships (review, Galinat, 1974). Because the genes involved do not always behave in a clear-cut Mendelian manner, one taxonomist (Iltis, 1983) has concluded that they do not exist. Instead, Iltis would have "a catastrophic sexual transmutation, a unique macroevolutionary event," in which the secondary male traits of the teosinte tassel became the secondary female traits of the corn ear (see previous item).

In the case of experimental introgression from Tripsacum, penetrance of the key trait genes is even more obscure. Usually it is primarily the dominant alleles from Tripsacum masking the recessive marker genes in maize that find expression. With considerable difficulty, resistance (Ht3) to Helminthosporium turcicum was extracted from T. floridanum (Hooker). Tripsacum does not appear to have been involved in the origin of either corn or teosinte. Most of its chromosomes have a different genetic architecture from that of corn and will not pair with corn.

Walton C. Galinat

High oil, scutellum, embryo and endosperm

While the endosperm is the primary storage organ in the corn kernel and monocots in general, the scutellum may also serve this purpose. In fact, in dicots it is the cotyledons, the counterpart of the scutellum, which are the sole storage organs. There is no obvious reason why the scutellum could not be bred to become the primary storage organ in corn. This would dramatically change the composition of stored material to that of oil which resides in the scutellum and embryo. Selection for high oil by Alexander's group in Illinois has over the years produced an enlarged scutellum. Enlargement of the embryo associated with pointed kernels would further increase the oil level, as would a reduction of the endosperm with mutants such as sugary, shrunken and brittle. These mutants, by

extending the period of flow by soluble polysaccharides into the embryo, also increase the growth duration and size of embryo and, consequently, the level of oil.

The pointed shape of certain kernel types results from more rapid growth of the embryo and scutellum (the germ) relative to that of the endosperm, all under the pericarp enclosure. The pericarp and its stylar extension grow from the base. Growth of the style stops at the time of fertilization, and at the same time growth by the pericarp is reactivated. Enlargement of the pericarp is necessary in order to adequately accommodate the two products developing from double fertilization. If the reactivated pericarp grows faster or in greater duration than the germ and endosperm, it becomes loose and does not give adequate protection.

Walton C. Galinat

Direction of uppermost leaf whorling determined by chance

Like the mirror-image reversal in pattern of successive phytomers along an axis in maize, the direction of whorling by successive leaves also alternates in opposite directions along the axis. This antidromy sequence was observed previously by Weatherwax (1948) in sectioned embryos of corn. He found that the direction of overlapping by the first leaf is determined solely by chance and is not correlated with position within a pair of spikelets. It should, therefore, be expected that the whorl of the last leaf might also be noninherited.

Because corn is so super-sensitive to light intensity and because the sun's rays in the northern hemisphere appear to move clockwise from east to south to west as the earth spins from west to east, it seemed possible that corn now adapted to the northern hemisphere might have evolved a counter clockwise (left-over-right or male) whorl for its upper leaf. This would allow the uppermost leaf to benefit from having the sun rays penetrate more deeply onto its young actively growing tissue that requires extra energy. But as interesting as the hypothesis seemed, no correlation was found in 212 plants from 22 varieties of Northern Flint now adapted to northern United States and Southern Canada (Table 1). The upper

Table I: Upper Leaf Spiral in Varieties of Northern Flint.

1983		♂ orient	♀ orient
277	Pipestone	4	8
278	Bear Island-Chippewa	4	3
279	Shoshoni	3	4
280	Mandan	6	5
281	Rhee	7	5
282	Saskatchewan	5	5
283	Dakota	6	5
284	6 Nations	3	4
286	Iroquois	12	7
288	Gray's sib	3	3
294	Carpenter's	3	5
299	Longfellow	5	2
300	King Philip	2	7
302	Canada Yellow	5	3
315	Quick's	4	2
EB4	Wakefield	7	5
EB5	Wilbur's	5	6
EB6	Gray's	9	7
EB7A	Palmer	6	8
EB7B	Barber	3	9
607	Golden Midget	3	4
	Total	105	107

leaf whorling on tillers sometimes differed from that on the main stalk of the same plant. The only correlation that could be found was with a few 10-rowed ears

in which row-twisting was in the same direction as the whorling of the uppermost husk leaf. Adequate material adapted to the southern hemisphere was not available for testing.

Walton C. Galinat

Preliminary cytology of Erianthus species

Cytological examinations were conducted on the pollen mother cells of the plume grass, Erianthus sp., for chromosome number and general morphology of the chromosomes. The meiotic chromosome number of 2n = 60 is established. C. D. Darlington and A. P. Wylie in their book, Chromosome Atlas of Flowering Plants (1945), have listed only three species, namely, E. arundinaceus, E. formosanus, and E. sara (bengalense) with a chromosome count of 2n = 60. A few other species with a 2n count of 20, 30, and 40 have also been reported.

At diakenesis and metaphase, 30 bivalents are formed. Meiotic irregularities or higher associations as trivalents and/or quadrivalents are absent. Pachytene pairing was normal and complete. The chromosomes are achromatic in their entire length except in regions adjacent to the centromere, where they are heterochromatic. Terminal as well as internal knobs are absent. Due to the high number, individual chromosomes could not be identified. Further work is in progress.

This species is important to our understanding of the origin and evolution of teosinte and corn because the morphology of its paired spikelets and rachis segment represents the primitive form starting a sequential series culminating in teosinte and corn.

Chandra V. Pasupuleti and Walton C. Galinat

Tripsacum chromosome with partial homology to chromosome 6 of maize

In our cytogenetic cross-mapping of maize and Tripsacum, a chromosome from Tripsacum has been isolated which appears to carry the pygmy locus in common with corn chromosome 6.

Pollen mother cells were stained by the customary methods and observed for chromosome relationships at pachytene, diakinesis, metaphase and anaphase stages of meiosis.

The Tripsacum chromosome has been isolated and identified in the disomic (20 + 2) condition. Analysis at pachytene shows that it corresponds to chromosome 10 of the Tripsacum genome with a total length of 27.9 u and an arm ratio of 3.3. The long arm has a conspicuous large knob. The Tripsacum chromosome, though intact in a few cells, in a majority of cases showed a deletion of one of the chromatids of the entire short arm, resulting in a univalent condition of that part of the chromosome, while the long arm remained as a bivalent. This occurred in at least 75% of the cells observed. The segmented short arm, being unstable, either folds back on itself or is seen sticking on to one or the other of the maize chromosomes with no specific preference or any real association.

Though this particular Tripsacum chromosome appears to have at least one gene in common with maize chromosome 6, the nucleolus organizer chromosome of maize, it did not show any real association with this chromosome, or with the nucleolus. Nor does it resemble the nucleolus organizer chromosomes of the Tripsacum complement in any of its morphological details.

The disomic plants are pollen sterile. Though the tassels were produced, stamens were not exserted and did not shed any pollen. The monosomic plants are semi-sterile.

Chandra V. Pasupuleti and Walton C. Galinat

Stiff stalk (Evergreen stalk), an apparent component of Perennialism (Pe) on short arm of chromosome 4

At the urging of Dr. Paul C. Mangelsdorf, the following report on diploperennis - su gl3 corn backcross segregations that I grew for him is presented. My report to P.C.M. is as follows:

"Unfortunately the starchy portion of the backcross segregation was badly depleted in the three-leaf stage by a dozen or so Canadian geese that landed there. Then an early September frost did not allow good expression of evergreen stalk or of basal shoot development. I did score and stake everything for glossy-3 and I did score for stiff stalk. This was done on November 1 by a swift kick to the second internode above ground level, usually an internode about 3 inches long, and then recording whether the stalk yielded or stood stiff. I thought the trait might be related to evergreen stalk and perennialism. It already occurs in some inbreds such as B73 out of Iowa stiff-stalk synthetic.

"Starchy and sugary were planted in equal numbers but because of the geese, I had only 62 starchy compared to 139 for sugary. The data is pooled here for your 2924-4/2920 and 2927-1/2920 because it appeared identical. St stands for stiff stalk.

	St Gl	St gl	st Gl	st gl	Total
Su	29	11	14	8	62
su	29	56	13	41	139
Total	58	67	27	49	201

Changing the data to percent to cover up damage by geese -

	St Gl	St gl	st Gl	st gl	Total
Su	46.8	17.7	22.6	12.9	100.0
su	20.9	40.3	9.4	29.5	100.1

Arranging for Su vs St as follows:

Su St	Su st	su St	su st	Total
64.5	35.5	61.2	38.9	200.1

Arranging for Gl vs St as follows:

Gl St	Gl st	gl St	gl st	Total
67.7	32.0	58.0	42.4	200.1

Arranging for Su vs Gl as follows:

Su Gl	Su gl	su Gl	su gl	Total
69.4	30.6	30.3	69.8	200.1

"I cannot see that the data for St is meaningful here as I scored for it. I was just dealing with a tendency or subjective trait. You will note there were 125 St to 76 st rather than a 1:1 ratio."

I will add a comment about the possible usefulness of the evergreen stalk trait as follows: In addition to drought resistance from a more robust root system, the

evergreen stalk associated with the Pe gene should be useful both in silage corn, by allowing a late season cutting when more time is available, and in breeding high sugar-stalk corn as an alternative for sugar cane.

Walton C. Galinat

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Simple test for recognition of se (sugary enhancer) gene

The presence or absence, homozygosity or heterozygosity, as well as the linkage relationships of the se gene continue to baffle many sweet corn breeders who try with varying success to use this basic gene for quality improvement. Chemical tests for se are virtually impossible to use in a breeding program because the breeder is seldom an experienced chemist who has the necessary laboratory equipment and chemicals. There is, however, a simple genetic test for se that the breeder can make if he will grow some Il1677a for this purpose.

The test cross for se is based on the super sensitivity of the 677a background to the fine wrinkling phenotype of se expression. Any pollen from a heterozygous se plant will result in obvious differences in xenia effects, yielding two different kernel types. The finely wrinkled se kernels will stand out in bold contrast to the kinky kernels of Se (non-se). To the initiated, the differences between se and Se expression on 677a are just as obvious as those of sugary vs. starchy.

Probably the great sensitivity of 677a to se wrinkling was a major factor in the recognition and selection of the se gene by Dusty Rhodes.

David W. Galinat and Walton C. Galinat

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Improved sample buffer for LDS PAGE of thylakoid membrane proteins

The solubilization buffer most commonly used to prepare samples for SDS polyacrylamide gel electrophoresis is that described by Laemmli (Nature 227:680-685). While this solubilization buffer works well for the electrophoretic analysis of most proteins, we find that solubilization of thylakoid membrane polypeptides in Laemmli's buffer leads to a time and temperature-dependent loss of a spectrum of lamellar polypeptides. These losses are clearly seen in Figure 1A where aliquots from a single thylakoid preparation were solubilized in Laemmli's buffer at 4 C, 20 C, or 100 C for 10 minutes and subjected to PAGE at 4 C. The loss of polypeptides A-E at 100 C is accompanied by the appearance of high molecular weight material at the top of the analyzing gel, suggesting that these polypeptides aggregate when heated in the sample buffer. These losses occur at progressively lower temperatures as thylakoids are incubated in the solubilization buffer for longer periods of time (compare polypeptide A in Figs. 1A, 1B). Raising the run temperature from 2 C to 8 C or increasing the current to induce gel heating also promotes the loss of polypeptides A-E. The polypeptides lost after heating represent core components of both photosystems I and II. Note that

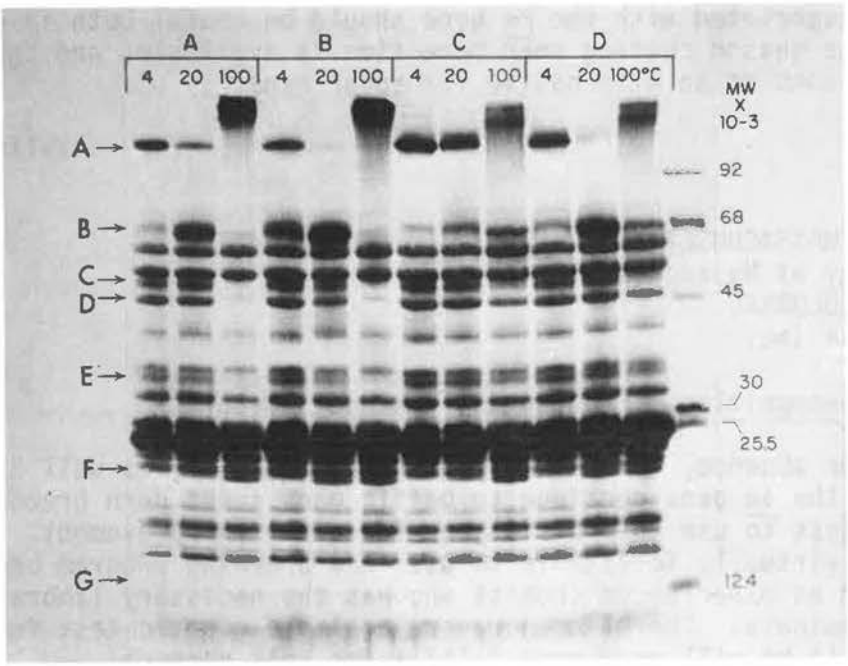

Figure 1

(A) Thylakoids were solubilized in Laemmli's buffer (with LDS substituted for SDS) either on ice (4C) or at 20C for 10 minutes, or at 100C (2 min.) followed by 8 min. cooling to 20C.

(B) Thylakoids solubilized as in (A), but incubated in solubilizing buffer for 45 min. prior to loading.

(C) Thylakoids were solubilized in 50mM Na-Tricine pH 7.8, 50mM dithiothreitol, 7%(v/v) glycerol, 2% (w/v) LDS, and 0.001% bromophenol blue as indicated in (A).

(D) Thylakoids were solubilized in the buffer indicated in (C), but were incubated in buffer for 45 min. as in (A).

polypeptides F and G increase in intensity after heating; this is consistent with the known action of LDS on multisubunit and chlorophyll-protein complexes.

We find that the loss of polypeptides A-E is minimized by replacing the buffer species (tris, pH 6.8) and sulfhydryl reagent (2-mercaptoethanol) in Laemmli's formulation with tricine, pH 7.8, and dithiothreitol, respectively, as suggested by Jim Metz (Biochim. Biophys. Acta 681:95-102). With these changes, the improved retention of polypeptides A-E is evident (Figs. 1C, 1D). We have varied the pH (6.8 vs. 7.8), reductant (2-mercaptoethanol vs. dithiothreitol), buffer (tris vs. tricine) and detergent (SDS vs. LDS) separately and in combination and find that the pH of solubilization has the greatest effect on stability. We now use the sample buffer described in Figure 1C for routine analysis of thylakoid membrane polypeptides. Lithium dodecyl sulfate is preferred over the sodium salt in this system because of its greater solubility at low temperature. Sample solubilization is carried out at 4 C for 10 minutes or by heating to 100 C for 2 minutes, followed by electrophoresis at 2-4 C in a jacketed apparatus (Bio-Rad).

All samples were solubilized at detergent: chl ratio of 20:1 (w/w). Arrows indicate polypeptides appearing or disappearing as a function of solubilization conditions.

Kenneth J. Leto and Roslyn Young

Identification of PS-II reaction center proteins using hcf*-3

Solubilization of thylakoid membranes under mild conditions prevents the complete dissociation of chlorophyll from protein and results in the appearance of a series of green bands (chl-proteins) during SDS or LDS PAGE. In 1977 Hayden and Hopkins (Can. J. Bot. 55:2525-2529) described a minor chla-protein from maize thylakoids which they assigned to the photosystem II reaction center. As techniques for preserving the association between chlorophylls and their associated apoproteins progressed, at least two minor chla-proteins were shown to occupy the gel region described by Hayden and Hopkins. Differential functions have not been assigned to these two chl-proteins, and their precise relationship to the primary photochemistry of photosystem II is unclear.

We investigated the association of these complexes with photosystem II by comparing polypeptide and chl-protein profiles from wild type and hcf*-3 chloroplasts under conditions of mild solubilization (4 C, 10 minutes, at pH 7.8) and electrophoresis (2 C, 10-17% gradient gels, in the dark). The nuclear hcf*-3 maize mutant is known to specifically lack photosystem II reaction centers in both mesophyll and bundle sheath chloroplasts (Leto et al., Plant Physiol. 69:1450-1458; MNL 54:117-118), and thus provides a reference for determining structural association of polypeptides with photosystem II. We also examined the chl-protein profile of purified photosystem II particles (Mullet and Arntzen, Biochim. Biophys. Acta 589:100-117) to determine which chl-proteins in the "CPa" region were essential for photosystem II activity. Minor chl-proteins in the "CPa" region were detected by activating chl fluorescence with a longwave ultraviolet source (Machold et al., Carlsberg Res. Commun. 44:235-254).

As shown in Figure 1A, several chl-proteins were resolved during LDS-PAGE at 4 C. The band labeled CPa-2 corresponds to the minor chla-protein originally described by Hayden and Hopkins. Illumination of the gel with ultraviolet light revealed that the "CPa" region contained three distinct bands, two of which, CPa-1 and CPa-1*, were clearly missing from hcf*-3 thylakoids (Fig. 1B). In contrast, the chl-protein profile from purified photosystem II particles contained both CPa-1 and CPa-1*, but lacked CPa-2 (Fig. 1C). These observations suggest that CPa-1 and CPa-1* are closely associated with photosystem II, while CPa-2 is not necessary for photosystem II activity. Further work with digitonin/triton fractionated lamellae indicates that CPa-2 is enriched in stroma lamellae and thus may be preferentially associated with photosystem I.

CPa-1 and CPa-2 were purified by preparative electrophoresis, the pigment extracted, and the apparent molecular weight of the constituent apoproteins determined by LDS-PAGE. The apoproteins obtained in this way were compared with the corresponding authentic chl-proteins by proteolytic mapping to establish identity (data not shown). This analysis assigns apparent molecular masses of 48kD and 42kD to the apoproteins of CPa-1 and CPa-2 respectively. By similar analysis CPa-1* was shown to be an electrophoretic variant of CPa-1. The general

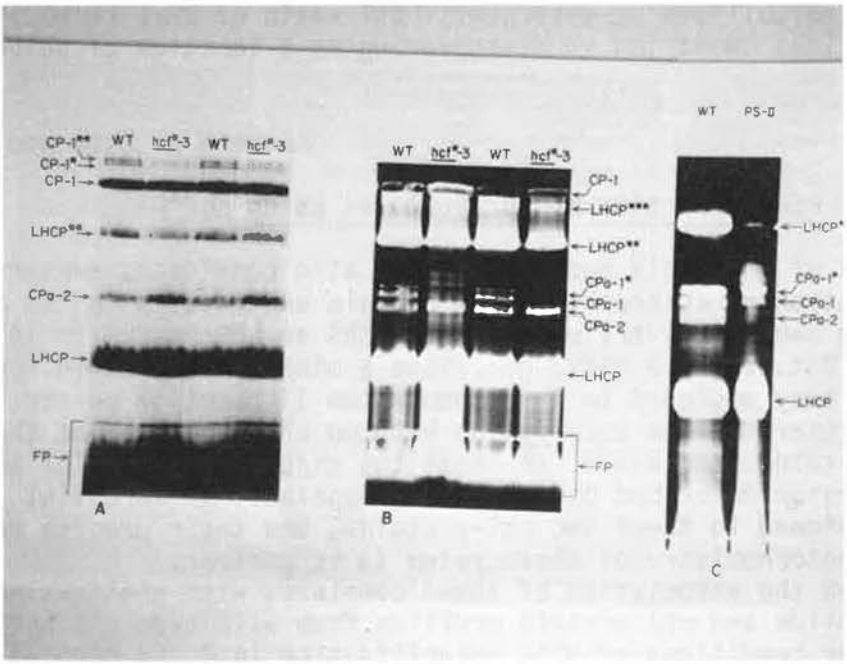

Fig. 1 - Chl-proteins resolved on LDS-polyacrylamide gels.

(A) Green bands seen during electrophoresis of unfractionated thylakoid membrane proteins.

(B) The same chl-proteins detected as red fluorescent bands.

(C) Fluorescent chl-proteins seen with unfractionated wild type lamellae and purified photosystem II particles. Note the absence of CPa-2 from the PS-II particles.

characteristics of CPa-1 make it a leading candidate for the photochemically active reaction center protein of the photosystem II complex.

Kenneth J. Leto and Roslyn Young

Accelerated turnover of photosystem II polypeptides in hcf*-3

Mutants blocked in the light reactions of photosynthesis characteristically lack functionally related subsets of thylakoid membrane polypeptides. However, the primary cause and/or mechanism underlying these "losses" is understood only in those cases where chloroplast DNA is known to be missing or altered, or where the synthesis/assembly of chloroplast ribosomes is affected. These losses are especially intriguing in cases where well characterized nuclear mutations lead to the loss of chloroplast-encoded thylakoid polypeptides.

To gain insight into the mechanisms underlying these losses we have compared the synthesis and insertion of chloroplast-encoded polypeptides into wild type thylakoids with thylakoids from the photosystem II deficient hcf*-3 mutant. Hcf*-3 is located on chromosome 1S (MNL 53:37-38); mutation at this locus results in the loss of a prominent 48kD photosystem II reaction center polypeptide (previous article) and a rapidly synthesized 34-32kD, photosystem II associated

atrazine binding protein (Leto et al., Plant Physiol. 69:1450-1458; MNL 54:115-117). Both of these polypeptides are chloroplast-encoded and are translated on chloroplast ribosomes.

We conclude from our studies that the apparent "loss" of the 34-32kD and the 48kD polypeptides from hcf*-3 thylakoids is due to accelerated turnover of these polypeptides in the thylakoid membrane. The supporting evidence is as follows:

1) Dot-blot Northern hybridization of total chloroplast RNA against a clone (pZmc427) containing the structural sequence for the 34-32kD polypeptide indicates that hcf*-3 chloroplasts contain wild type amounts of message for this polypeptide.

2) Total chloroplast RNA from both wild type and hcf*-3 seedlings synthesizes a prominent 34kD polypeptide in the rabbit reticulocyte in vitro translation system. It is known that message for the authentic 34-32kD polypeptide is the most abundant message in maize mesophyll chloroplasts.

3) Both the 34-32kD and the 48kD polypeptides are synthesized and inserted into thylakoids when intact hcf*-3 chloroplasts are incubated with ^{35}S-methionine in vitro. Thus, in the absence of the nucleus and over a short (30 min) time course, synthesis and insertion of these polypeptides is essentially normal in hcf*-3.

4) The 34-32kD and 48kD polypeptides are also synthesized and inserted normally into hcf*-3 thylakoids in vivo over the first 1-4 hours that seedlings are incubated with ^{35}S-methionine. However, subsequent "chase" (i.e., dilution) of label in the light reveals a progressive and specific loss of newly synthesized 34-32kD and 48kD polypeptides during the next 20 hours. These losses are also seen when hcf*-3 seedlings are labeled for 1 hour in the light and subsequently transferred to the dark for 20 hours, indicating that photoinhibition is not the primary cause of this accelerated turnover.

5) Turnover of both polypeptides probably occurs in the unpaired (stroma) lamellae, as fractionation studies indicate that neither the 34-32kD or the 48kD polypeptide accumulate in the grana of hcf*-3 thylakoids.

6) The authentic 34-32kD polypeptide lacks lysine residues (McIntosh, J. Cell Biochem. 7B, Abst. 1287). Comparison of in vivo synthesis with ^{35}S-methionine and ^{14}C-lysine indicates that the 34-32kD polypeptide we are following in this study is the authentic chloroplast-encoded species.

While we have apparently discovered the mechanism underlying the "loss" of these polypeptides from hcf*-3 thylakoids, we have yet to discover the primary cause for this turnover. Candidates include increased activity of membrane bound proteases known to specifically metabolize the 34-32kD polypeptide, the involvement of (uncharacterized) nuclear proteins needed for stabilization, or defects in the process of protein import across the chloroplast envelope. We have not yet determined whether the loss of lamellar polypeptides from other hcf mutants is also due to a specific increase in protein turnover.

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Effects of visible recessive alleles on grain yield in an inbred line

Recessive alleles produced and selected in an inbred line usually produce an array of pleiotropic effects involving vigor characteristics both as heterozygotes and homozygotes in the homozygous background of the inbred line, and as heterozygotes in the heterozygous background of a hybrid combination. The vigor

effects may be positive or negative for a particular characteristic. In general, the effects of an allele on vigor characteristics in the homozygous background of the inbred line are of greater magnitude than in the heterozygous background of a hybrid combination. Indeed, many visible recessive alleles may be selected in the first generation as heterozygotes since plants heterozygous for a mutant recessive allele may flower earlier, or later, or be somewhat larger or taller, etc. (alleles are produced by treating inbred material with a mutagen before meiosis and using the pollen as a screen to screen out undesirable types of mutations, i.e., pollen from treated inbred plants is used on silks of untreated inbred plants). Of the twelve alleles under investigation (in inbred line Oh43), ten alleles as homozygotes -/- yield less than the normal +/+ homozygotes. Four of the alleles as heterozygotes yield more than the normal +/+ homozygotes. In no case do the heterozygotes yield less than the normal +/+ homozygotes. In the four cases where the heterozygotes yield more than the normal +/+ homozygotes the recessive homozygotes yield less. The ora2-13 (orange endosperm-case 13) allele is a case in point. The ora2-13/ora2-13 homozygote yields 15.5% less and the +/ora2-13 heterozygote 8.6% more than the +/+ homozygote (grams of grain per plant for +/+ = 79.4, +/- = 86.2**, -/- = 67.1**). In this case the heterozygote flowers earlier and the homozygote later than the normal +/+ homozygote (days to silk for +/+ = 85.5, +/- = 83.2**, -/- = 88.3**; days to pollen shed for +/+ = 84.0, +/- = 82.2**, -/- = 87.6**). From the information available it seems that single locus heterosis is controlled by the pleiotropic effects of recessive alleles.

E. J. Dollinger

Alleles at the lty1 locus

The lty1-16 allele (light yellow endosperm-case 16) is the only allele studied thus far (only five have been studied) which increases grain yield in a hybrid combination. In the Oh43 lty1-16xOh551 hybrid (control = Oh43xOh551) the pleiotropic vigor effects are as follows: 1.2% decrease in days to silk, 1.7% increase in plant height, 5.0% increase in 100 kernel weight, 3.1% increase in per plant grain yield, 0.5% increase in ear length, 0.8% decrease in ear diameter, 3.7% decrease in kernel row number, no significant difference in number of kernels per plant or ears per plant. Two other alleles have been produced and isolated at the lty1 locus, namely lty1-17 and lty1-19. Studies in progress indicate that the three alleles will differ with respect to pleiotropic vigor effects.

E. J. Dollinger

ADDENDUM

COLUMBIA, MISSOURI
University of Missouri

A new chloroplast thylakoid gene, pdf1, is linked to Lct1

If the proteins of the maize chloroplast thylakoid are run out on a density-gradient Laemmli SDS urea polyacrylamide gel, there is between the 21.5 Kd and 31 Kd markers a cluster of at least six major proteins (and several more minor bands). Under certain gel conditions (full details upon request from the author) there is a well separated pair of pink-hued (Coomassie Blue-stained) bands which are the fastest migrating proteins of that cluster. An allele derived from Zea luxurians causes the faster of this pink pair to migrate still faster (farther on the gel). Z. diploperennis and Z. perennis mimic the phenotype of Z. luxurians. This phenotype of luxurians is constant in accessions from Guatemala, Honduras and Florida. Plants of the generation ((Z. d. x Z. m.) x Z. m.) x Z. m. are segregating this phenotype. Chalco, Nobogame, Huehuetenango, Balsas, and all maizes (Corn Belt and exotic) have the slower migrating phenotype.

No F2 data are in hand to test the mode of inheritance. However, a dominant-recessive relationship is suggested by absence of apparent band broadening on the gel. The following data for crosses involving Z. luxurians are presented:

Cross	Gel phenotype		Chi-sq		
	"Fast"	"Slow"	1:1	9:7	3:1
B73 x (B84 x Z. l.)[1]	39	31	0.7	0.0	12.9
Mo17 x (B84 x Z. l.)[2]	21	24	0.1	1.3	17.8
Combined data	60	55	0.1	0.6	30.8
(B84 x Z. l.)[3] x Z. l.	14	0			

[1] individual B-30
[2] individual B-20
[3] combined females B-20 and B-30

I designate this nuclear factor pdf1 (pink doublet, faster band). There are two alleles: pdf1-Zm, the recessive factor common in domesticated maize; and Pdf1-Zl, the dominant allele derived from Z. luxurians. I do not now designate the genotype of diploperennis, though the test for allelism is partially set up. I am advancing this factor into several standard maize lines.

The data gathered suggest a linkage relationship with Lct1 (see details of Lct1 alleles in the main Columbia, MO section). The data are:

Cross	Gel phenotypes[3]				Linkage intensity
	ss/s	ss/f	tb/s	tb/f	
B73 x (B84 x Z. l.)[1]	26	9	5	30	0.20
Mo17 x (B84 x Z. l.)[2]	21	3	3	18	0.13
Combined data	47	12	8	48	0.17

[1], [2] as above
[3] ss = single slow Lct1-Zm band, tb = two bands from Lct1-Zm and Lct1-Zl, s = "slow" phenotype from pdf1-Zm, f = "fast" from Pdf1-Zl

It is clear from the data that these two loci can not be locked in a "supergene" block by a small local inversion.

Others are welcome to work with these genes. A small quantity of seed for increase is available upon request.

Stephen A. Modena

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Further studies on zein gene structure

A series of new zein cDNA clones obtained by rescreening the previously constructed cDNA libraries were identified on the basis of cross- and differential-hybridization to previously characterized clones (Viotti et al., EMBO J. 1, 1982). The nucleotide and deduced amino acid sequences of the new clones were compared both within them and with respect to the already published sequences of zein genomic and cDNA clones.

On the basis of the four domains present in zein polypeptides, signal peptide, head piece (amino terminus), repeat block structure and tail piece (carboxyl terminus), at least four prototype sequences can be identified that do not necessarily correspond to the length of the various polypeptides detected by SDS-PAGE. As the comparison was carried out within sequences all belonging to the maize inbred W64A, the mutations found in the various clones of each prototype represent really different genes. This allows a better classification and a definition of the effects of the mutations on the various zein domains of the polypeptides belonging to each prototype. The comparison at the amino acid level among members belonging to each prototype indicates that three domains, signal, head and tail, were strongly conserved in sequence while the number, length and a.a. sequences of the blocks may vary extremely. In turn a gene can be assigned to a given prototype simply on the basis of the a.a. sequence of the three conserved regions even though its coding capacity may vary in length. This was found particularly true for cDNA clones coding for the so-called high molecular weight zein polypeptides, defined in our case by the M1 prototype (Viotti et al., EMBO J. 1, 1982). Among these, two independently isolated clones show a terminator codon twenty triplets upstream from the physiological one, resulting in truncated polypeptides and thus phenotypically belonging to the light chain zein class. A similar prematurely terminated gene has been found by sequencing a zein genomic clone (Spena et al., EMBO J. 1, 1982).

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Chromosomal distribution of cloned zein cDNA sequences in maize and teosinte

The occurrence of zein-like sequences in teosinte has been investigated using two zein cDNA clones, M1 and M6 (Viotti et al., EMBO J. 1, 1982), and the BamHI-XbaI fragment (616bp, E21) of the genomic clone E19 (Spena et al., J. Mol. Biol. 169, 1983, more than 96% homologous to pMGI cDNA clone) that define three of the four or more zein prototype sequences found in the inbred W64A. The three non-cross hybridizing zein sequences have been labelled with ^{3}H-TTP by nick translation and then hybridized in situ to meiotic spreads of W64A, of two

teosinte lines (Zea mays ssp. mexicana, race Chalco, and Zea mays ssp. parviglumis, race Balsas-Guerrero) and of F1 hybrids W64A x Z. mays mexicana and W64A x Z. mays parviglumis.

In all types of spread more than 140 meiocytes were analyzed, all having labelled chromosomes. The number of chromosomes showing hybridization and the hybridization sites per chromosome vary depending on the probe and on the line or hybrid investigated. In any case the silver grains per hybridization site were from three to five. Statistical analysis of the various parameters mentioned above indicated that M6 and E21 (pMGI) zein-like sequences were found in teosinte subspecies on the same chromosomes and sites as in maize: chr. 4 proximal 1/3 of long arm (M6+E21); chr. 7 distal 1/3 of short arm (M6+E21); chr. 10 distal 1/3 of long arm (M6). On the contrary the M1 probe showed in both teosintes in respect to maize an additional chromosomal hybridization site. In all the combinations tested (inbreds and hybrids) where teosinte chromosomes were present chromosomes 4 and 10 showed hybridization on the distal 1/3 of both long arms, while in maize line W64A only chr. 4 hybridized.

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Preliminary characterization by Southern blot analysis and in situ hybridization of five repeated DNA sequences

Maize DNA extracted from W64A was digested with HaeIII, Sau3AI or TaqI restriction enzymes and fractionated by acrylamide gel electrophoresis. Five fragments that light up in ethidium bromide staining were separated (H1 and H2 from HaeIII digest, S1 from Sau3AI digest, T2 and T3 from TaqI digest) and cloned in E. coli by ligation to pUC8 plasmid. The fragments do not cross-hybridize to each other and have a length between 150 and 220bp.

Southern blot hybridizations of nick translated fragments to total digested DNA from maize lines differing in heterochromatin content were carried out (W64A, A69Y, Black Mexican without or with seven B chromosomes). The cloned fragments appear to have a lower reiteration frequency in Black Mexican than in the other lines, suggesting a relation between these sequences and the knob heterochromatin, the knobs being absent in Black Mexican lines.

The chromosomal localization of these repeated sequences was detected by in situ hybridization on meiotic spreads of maize lines with (W64A, KYS) and without (Black Mexican with two or three B's) knob heterochromatin. The S1 fragment hybridization sites were found in Black Mexican only on the B chromosomes, but spread on seven or more A chromosomes in W64A and KYS lines, with preferential hybridization to knob heterochromatin. In all lines investigated some of the A chromosomes have hybridization sites essentially in the centromeric region for H1 and T2 probes while B chromosomes have none. The T3 fragment hybridization induces groups of spots on the B chromosomes and on few A chromosomes with different number of sites among the lines investigated. Finally the H2 fragment strongly and preferentially hybridizes to the NOR region of chr. 6 and only to the centromeric region of B chromosomes. In summary the five repeated sequences have different clustering and unique or multiple hybridization sites on both A and B chromosomes.

A. Viotti, N. Pogna, E. Privitera and E. Sala

III. MAPPING

The current mapping coordinators for each arm are as follows (* indicates the lead coordinator):

1S	*Fletcher, Patterson	7S	Steffensen
1L	*Sisco, Goodman	7L	Whalen
2S	Tracy	8S	Neuffer
2L	McCormick	8L	Neuffer
3S	Poethig	9S	O. Nelson
3L	*Sheridan, Beckett	9L	Coe
4S	Galinat	10S	(volunteer needed)
4L	*McCormick, Beckett	10L	*Kermicle, Patterson
5S	*Polacco, P. Crane	B	W. Carlson
5L	*D. Weber, Shadley	Gene list updating - Hoisington	
6S	*Phillips, Mascia		
6L	*Phillips, Mascia		

Each coordinator would appreciate receiving information about factors or data that apply to that segment.

CHROMOSOME 1, SHORT ARM

We are underway on this study but do not yet have any definitive data. We made many F1 crosses this past summer. Successful crosses were: w*-8345, w*4791, v*-8943, and v*-032-3 x sr1 zb4 P-WW, rs2, zb4 ms17 P-WW and P-RR ts2, plus w*-018-3 x sr1 zb4 P-WW, zb4 ms17 P-WW and P-RR ts2.

In addition to this we have received information from William Tracy of Cargill that he believes that gt is located on the short arm of one (see item on page 99 of this News Letter).

The o13 in the series of opaque stocks that we have been working on appears from the TB analysis to be located on the short arm of one. We will make additional tests next summer.

G. Fletcher

CHROMOSOME 1, LONG ARM

Crosses and backcrosses have been initiated toward mapping of Kn, tb and lw, all of which are closely associated, and several other visible markers in the arm, including id, D8, Mpl (miniplant). In part of the material, characterizations by Major Goodman have defined segregating variants for Adh1, Phi1 and Gdh1 that may help establish their positions with relation to visible markers.

(P. Sisco has volunteered to be the new lead coordinator for 1L).

E. H. Coe, Jr.

CHROMOSOME 2, SHORT ARM

Despite the unfavorable growing season experienced in 1983, I was able to initiate linkage tests for mutants supplied by M. G. Neuffer.

Work is underway in gathering published data and checking for ambiguities in the linkage map. Any citations, information or genetic stocks relating to the short arm of chromosome 2 would be greatly appreciated.

W.F. Tracy

CHROMOSOME 2, LONG ARM

The following 2L mutants (obtained from M. G. Neuffer) were tested for allelism within phenotypically similar sets (see item in this MNL) and were crossed with known 2L marker stocks; F2 data will be available next summer: o*-1195A, et*-1078A, ygv*-72, o*-999A, et*-789, pg-spt*-278B, w*-332, et*-496A, pg-spt*-464, w*-77, pg*-330A, pg-spt*-579B, fl*-1316A, pg*-760A, wlv-pg*-424, fl*-1414A, ogm*-1488A, cp-fl*-1024A (allelic to dek4), de*-1175, dcr*-1428, de*-660E (allelic to cp*-1225), mn*-1120A, rgh*-786.

Sheila McCormick

CHROMOSOME 3, SHORT ARM

An effort is being made to map the following mutations: g2 (allele is pg14), ra2, h, E8, rt, Rp3, and Cg. Whalen's preliminary data indicate that g2 is 32 m.u. from d1, and is unlinked or weakly linked to Rg. My test cross data indicate 50% (57/114) recombination between Lg3 and g2. Thus g2 can be tentatively placed at the distal end of 3S; more complete data on the location of g2 will be presented next year. Whalen also reports that ra2 is very closely linked to d1, but he has no further information about its location. There is some uncertainty about the location of Cg, since it shows no linkage with c1, cr or g2 (Poethig, MNL 58). An attempt is now being made to locate Cg using the T wx series.

The location of a number of 3S mutations from Neuffer's collection was confirmed using TB-3Sa, and some of these were tested for allelism with g2 or d1. All of the chlorophyll mutations in this group have a yellow endosperm, and are therefore not likely to be alleles of c1. Specifically, pgspt*-E537A (pale green diffuse spots on all leaves; plants viable but weak), yg*-E1396 (yellow green seedlings, pale green plants), wl*-E2 (albino seedling, lethal) and pg*-E570C (pale green leaves, golden leaf sheaths) are not allelic to g2; and d*-E339 and d*-E446 (extreme dwarfs) are not allelic to d1.

Scott Poethig

CHROMOSOME 3, LONG ARM

Many mutants known or thought to be on chromosome 3 have been acquired and are being crossed by all B-A translocations on chromosome 3. In addition to TB-3Sb, -3La, -3Lc, and -3Ld, 8 new translocations on 3L have been identified and are being crossed onto the chromosome 3 mutants; these are designated TB-3Lf, -3Lg, -3Lh, -3Li, -3Lj, -3Lk, -3Ll, and -3Lm. Four of J. A. Birchler's compound B-A's, TB-1La-3L4759-3, -1La-3Le, -1La-3L5242, and -1La-3L5267, all involving 3L, will also be tested. The results of these tests should aid greatly in placing both mutants and B-A translocations in proper sequence along the chromosome arm.

Preliminary results of some of the above crosses are summarized in a separate note in this News Letter. Fragmentary results indicate that TB-3Lf and -3Lg are proximal to gl6, that TB-3Lh, -3Li, and -3Lj are distal to gl6 and proximal to lg2, and that TB-3Lk, -3Ll, and -3Lm are distal to lg2. The following thirteen mutants are confirmed to be on 3L: cp*-330D, gl7 (allelic to gl12), y10, cp*-1379A, pm, yd2, d*-282, rgh*-1060B, yel*-5787, de*-932, su*-748A, dek6, vp1.

(W. F. Sheridan has volunteered to be the new lead coordinator for 3L).

J. B. Beckett

CHROMOSOME 4, SHORT ARM

(No report).

CHROMOSOME 4, LONG ARM

The following 4L mutants (obtained from M. G. Neuffer) were crossed with known 4L marker stocks; F2 data will be available next summer: et*-516E, dek8, ptd*-1130, sml*-976A, dcr*-1005A, pg*-507A.

The following mutants are confirmed for 4L (Beckett, this NL): o1, v8, ptd*-1130, wlv*-378A, py*-PI177593.

Four new B-A translocations are now available, TB-4Lc, TB-4Ld, TB-4Le, and TB-4Lf. All have breakpoints proximal to gl3. The breakpoint of TB-7Lb-4L4698 is distal to gl3 and o1.

Sheila McCormick and J. B. Beckett

CHROMOSOME 5, SHORT ARM

(Please see the item by M. Polacco on pages 64-65 in this News Letter).

CHROMOSOME 5, LONG ARM

(Please note that the location for TB-5La and TB-5Lb, given in the map on page 191A of MNL 57, is incorrect; data given by J. B. Beckett, MNL 49:132, place both of these TB's between v3 and bv1--i.e., bv1 is uncovered, but not v3, by both translocations--please correct your map accordingly).

(Please see the item by J. D. Shadley and D. F. Weber on pages 156-158).

CHROMOSOME 6, SHORT AND LONG ARMS

The following cross combinations have been carried to F2 and are to be examined for recombination data: wl x yel*-4-6(4447), wl4 x yel*-4-6(4447), wl5 x yel*-4-6(4447), wl4 x yel*-7285, wl5 x wh*-8613, wl4 x wh*-8954, wl5 x wh*-8954, wl4 x yel*-8631, y pb4 pl x yel*-4-6-(4447).

P. Mascia and R. L. Phillips

CHROMOSOME 7, SHORT ARM

A recent paper (M. Motto, M. Perenzin, F. Salamini and C. Soave, 1983, Maydica 28:25-39) reports new mapping data for the Hs-gl region of chromosome 7. Four new seedling mutants, luteus-13 (l13), white-16 (wl6), white-17 (wl7), and white yellow-green (wyg) were mapped. These new data were analyzed together with previously reported data and a new linear order of genes has been proposed. Whereas previously the Hs locus was the most distal locus on 7S and therefore occupied position 0 on the map, it now occupies position 8. Furthermore, whereas the position of the centromere was previously located between vp9 at position 25 and ra at position 32, the current map places the centromere between zp16 at position 41 and ra at position 48. The new order of genes with their map position is: (l13), zp6 (0), Hs (8), wl7 (13), zp21 (17), zp29 (20), o2 (24), y8 (26), in (28), v5 (32), De*-B30 (32), vp9 (32), wl6 (36), (cp) (37), zp1-3 (37), zp16 (41), ra (48), gl (52), Tp (62), sl (66), ij (68), Bn (87), bd (125), Pn (128). Note that the positions of l13 and cp remain uncertain.

(D. Steffensen has volunteered to be the new coordinator for 7S).

William F. Sheridan

CHROMOSOME 7, LONG ARM

Motto, Perenzin, Salamini and Soave (Maydica 28:25-39, 1983) report a new seedling lethal, white yellow-green (wyg), on 7L very near ra; the relative order is unknown. They also place collapsed endosperm (cp) on 7S rather than 7L as reported previously.

Mapping work has been initiated with ms7 and va (variable sterile); allelism tests show they are not allelic.

R. H. Whalen

CHROMOSOME 8, SHORT AND LONG ARMS

(Please see the item by M. G. Neuffer and D. J. England on pages 77-78, and the item by G. Gavazzi et al., on page 148 in this News Letter).

CHROMOSOME 9, SHORT ARM

Efforts have commenced to map a number of mutants isolated by M. G. Neuffer and located by him to the short arm of chromosome 9. Using Neuffer's designations, these mutants are (1) grainy, white, luteus virescent, (2) defective seed-1089, (3) collapsed endosperm-1054, (4) dwarf-660B, (5) adherent, glossy-512B, (6) white, luteus, virescent-pale green-585, (7) rough seed-802, and (8) collapsed endosperm-873.

Oliver E. Nelson, Jr.

CHROMOSOME 9, LONG ARM

(E. H. Coe, Jr., has volunteered to be the new coordinator for 9L).

CHROMOSOME 10, SHORT ARM

(A volunteer is needed to coordinate mapping information for this arm).

CHROMOSOME 10, LONG ARM

(J. L. Kermicle has volunteered to be the new lead coordinator for 10L).

B CHROMOSOMES

Individual genes have not been identified on the B chromosome. However, certain regions of the B have been shown to be required for nondisjunction. These regions were referred to as "factors" (Genetics 97:379-389). However, the term "regions" seems preferable and will be used here. Regions 1, 2 and 3, corresponding to the distal euchromatin, the proximal euchromatin and the centromeric heterochromatin, respectively, are each required for nondisjunction. In addition, absence of region 4 (the B short arm) seems to decrease the rate of nondisjunction, although the short arm is not required for nondisjunction.

Recently, Beckett described a new property of B chromosomes. He found that B chromosomes (in B-A translocations) confer a competitive advantage on pollen containing them over B-containing pollen (J. Heredity 73:29-34, 1982). The region or regions controlling this property have not yet been localized. Another recent finding from our laboratory is unpublished and still quite tentative. However, it seems that B^9 chromosomes can be identified which lack preferential fertilization due to deletion of most or all of the centromeric heterochromatin. These B^9-derived chromosomes show low rates of nondisjunction due to the absence or near absence of region 3, so that calculations of preferential fertilization are difficult.

Wayne Carlson

GENE LIST

The following factors were dropped from the 1983 linkage map and gene list (MNL 57:175-191B). Earlier map locations were given as indicated; none of them are available in the Stock Center collection:

ag1	1-14	lost	b12	lost
ga6	1-15	lost	b13	lost
pa1	1-58	lost	bt4	allele bt2
Ts3	1-119	lost	co1	lost
v19	1-	lost	de17	lost
z11	1-28	lost	g4	lapsed (see Brawn, MNL 36:49)
b11	2-	lost	ga4	lost
rp7	2-11+	lost	g116	allele g14
pg2	3-	lost	gm1	lost
w13	3-near lg2	lapsed	l3	lost
de1	4-0	lost (see Patterson et al., MNL 42:50)	lu2	lost
de16	4-74	lost (see Mascia, MNL 52:56)	ms4	allele po1 (Golubovskaya, MNL 55:80)
ga9	4-	lapsed (see Ashman, MNL 55:50)	ms6	allele po1 (see stocklist, MNL 50:122)
lo1	4-73	lost	pg13	lost
S1	4S	lost	Pu1, Pu2	lost
sp1	4-66	lost	rp2	lost
tn1	5-	lost	S2, S3, S4, S5	lost
yd1	6-	lost	so1, so2	lost
bp1	9-44	lost	ts8	lost
12	10-99	lost	w11	lost
18	10-near du1	lost	zg2	lost
sp2	10-near duI	lost		

D. A. Hoisington and E. H. Coe, Jr.

WORKING MAPS

In an effort to incorporate all known linkage information into the chromosome maps, we are trying the idea of a "working map" for each chromosome. These are presented at the end of this News Letter, for convenient access. The published linkage map is used as the core, with all other genes on that chromosome listed in columns to the right. Temporarily designated laboratory mutants are excluded pending assignment of permanent symbols that conform to the standards of nomenclature (MNL 49:3-4, 1975). In the first column to the right are those genes that have enough linkage information to place them "near" a gene on the linkage map. The second column lists genes located to arm, and the third column, those genes located only to chromosome. All brackets are only arbitrary and bear no reference to actual ranges of map units. It is hoped that these maps will result in the presentation of data to move the gene to the left (ultimately onto the linkage map). Placing a gene onto the linkage map is the most difficult, generally requiring accurate 3-point-linkage data; however, acquiring data to place genes near genes on the linkage map is easier. This type of information is of great importance to anyone mapping a particular region or to anyone interested in marking genes of interest. Any feedback on these maps will be most welcome. They are intended to provide a framework on which the coordinators for each chromosome can provide new working information.

D. A. Hoisington

CHROMOSOME 1

1S, 1L suggested to have homeology with 5S, 5L, based on secondary somatic associations of similar-length arms --M. D. Bennett, 1983

hcf*-41 (gr) affects electron transport; hcf*-2 (yg), -3 (gr) affect cytochromes; hcf*-4 (gr), -13 (gr) affect CO_2 fixation; hcf*-44 (pg), -50 (gr) affect chlorophyll-protein complexes --C. D. Miles, 1982

Adh1-2F11 null allele that arose in presence of bz2-m (Ds) and Ac and responds to Ac; contains 1.3 kb insertion that is transcribed in mRNA (in which hybridization to a Ds probe is found); restriction map for KpnI, SstI, BamHI, XbaI, HindIII, BglI, PvuII, BglII, SphI, HindII --H.-P. Doring &, 1984

vp5: point of action in carotenoid biosynthetic pathway --F. Fong &, 1983

Adh1 alleles -S, -F, -FkF (from Funk G4343 hybrid), -Ct (from a teosinte source); progenitors respectively of -S1951a (underexpressed in scutellum, over- in root and pollen, from -S following accelerated neon irradiation and allyl alcohol selection), -FkF3037 (overexpressed in scutellum, from -FkF spontaneously), -S3034 (underexpressed and unstable, from -S, Mu-generated) --M. Freeling &, 1983

Mdh4 alleles -10.5, -11.5; Pgm1-6, -7, -17; Phi1-n, -6, from Bolivian races --M. M. Goodman &, 1983

hcf*-3 uncovered by TB-1Sb-2L4464 as well as by TB-1Sb --K. J. Leto &, 1982

Adh1-0 (null) selectively sensitive to anaerobic conditions, resistant to allyl alcohol --K. Shimamoto &, 1983

Pgm1 alleles -1, 3, 5, 6, 7, 8, 9, 10, 11, 13, 15, 16, 16.5, 17, 18, 18.5, 19, 21, N from lines, races and teosintes --C. W. Stuber &, 1983

Adh1-Fm335 (Ds insertion); base sequences at site of Ds excision in revertants -RV1 through -RV4 and in other Ds excisions have common property of 11bp inverted repeat --M. M. Sachs &, 1983

Adh1 alleles -U725, -W586, -W190, -W182, -U327, -U1048, EMS-induced --R. J. Ferl &, 1983

Adh1 alleles -Cm, -Ct, -1S, -54S, -1F, -Fkf, -33F, restriction maps; conserved region includes 5'-3', variable outside among all alleles except -1F and -Fkf --M. A. Johns &, 1983

Adh1-S3034v allele unstable, arose in presence of Mu --M. Alleman, 58:24

Adh1-2F11 null, Ac-controlled --H. P. Doring &, 58:59

dek* mutants uncovered on 1S, 1L --M. T. Chang &, 58:61

hcf*-9 (allelic to -3) loosely linked to zb4; hcf*-2, -4, -12, -13, -41, -44, -50 on 1L --M. Polacco, 58:64

Les2 (was Les*-845) and Les*-1449 associated with wx T1-9c (1S.48) and T1-9(4995) (1L.19); Les2 - 2.2 - sr1; Les2 - 33.3 - lls1; lls1 - 18.9 - sr1; les*-501B allelic to lls1; nec*-495C - 0 - sr1; nec*-495C not allelic to zb4 or nec2; Les*-1461 associated with wx T1-9(8389) (1L.74), but unlinked with br1, an1, bz2, gs1 or bm2 --D. A. Hoisington, 58:83

Oleic-linoleic acids level in X-187 possibly associated with wx T on 1S --M. D. Jellum &, 58:88

cp*-E1113A developmentally blocked prior to leaf primordia; on 1L --J. K. Clark &, 58:91

gt1 shows low recombination with wx T1-9c --W. F. Tracy, 58:99

CHROMOSOME 2

2S, 2L suggested to have homeology with 4S, 4L --M. D. Bennett, 1983

hcf*-1 (gr) affects electron transport; hcf*-15 (yg) affects photophosphorylation --C. D. Miles, 1982

w3, al, y3: points of action in carotenoid biosynthetic pathway --F. Fong &, 1983

ws3 - 9.1 - lg1 - 13.7 - Mut - 6.9 - gl2; Mut shows no dosage effect --M. M. Rhoades &, 58:30

B1 - 19 - Ltel - fl1 --L. T. de Miranda &, 58:48

dek* mutants uncovered on 2S, 2L --M. T. Chang &, 58:61

Les*-A607 associated with wx T2-9b (2S.18) and wx T2-9d (2L.83) --D. A. Hoisington, 58:84

dek16 (was E1414) on 2L --W. F. Sheridan &, 58:98

o*-1195A, ogm*-1488b and o*-999A not allelic; fl*-1316A and cpf1*-1024A not allelic; ygv*-72 and wlv*-424 not allelic; pgspt*-278B and pgspt*-464 not allelic; de*-660C and cp*-1225 allelic; de*-1175, cp*-1225 and dcr*-1428 not allelic; w*-332 probably allelic to w3; w*-332 and w*-77 not allelic --S. M. McCormick, 58:178

CHROMOSOME 3

3S, 3L suggested to have homeology with 9S, 9L --M. D. Bennett, 1983

hcf*-19 (gr&yg) affects cytochromes --C. D. Miles, 1982

vp1: point of action in carotenoid biosynthetic pathway --F. Fong &, 1983

Trisomes found among small kernel selections --A. Ghidoni &, 1982

E8 allele -3; Got1-n, -2; Mdh3-17.8; Me1-S; Pgd2-n, -2, -4, from Bolivian races --M. M. Goodman &, 1983

a1-ruq, responds to Uq regulatory element; a1-0, not responsive to any known regulatory element --Peterson &, 1983

a1-m13 and a1-m16 alleles Uq-controlled; a1-m61138-3 En-controlled autonomous that shows chromosome breakage; vp1-m451 mutable allele --Peterson, 58:5,6

a1-SR03 allele: pale, stable, lethal or semilethal; arose in presence of Mu --D. S. Robertson, 58:9

Designations E4 and Est4 refer to the same locus --A. L. Kahler, 58:33

dek* mutants uncovered on 3S, 3L --M. T. Chang &, 58:61

TB-3La, f, and g uncover gl6 and distal markers; TB-3Ld, h, i and j uncover lg2 and distal markers, but not gl6; TB-3Lc uncovers ba and distal markers but not lg2; TB-3Lk and l uncover a3 and distal markers but not lg2; TB-3Lm uncovers a1 and distal markers but not lg2; cp*-330D uncovered by TB-3La and g; cp*-1379A by TB-3La, g, i and j; d*-292 by TB-3La, f, and g; de*-932 by TB-3Lf; dek6 by TB-3La, d and m; pm1 by TB-3La but not by d or c; rgh*-1060B by TB-3La; su*-748A by TB-3La, g, d, h, i, j, c and l; vp1 by TB-3La, g, d and h; y10 by TB-3La, f, d, h, i and c; yd2 by TB-3La, g, h, i, j and c; ye1*-5787 by TB-3La and f; ys3 by TB-3La --J. B. Beckett, 58:73

Spc1 associated with wx T3-9c (3L.09); Spc1 - 12.7 - lg2 - 43.2 - a1 --D. A. Hoisington, 58:84

dek17 (was E330D) on 3L --W. F. Sheridan &, 58:98

Cg shows no linkage with c11, g2 or cr --R. S. Poethig, 58:170

a1-mt-1 mutable, Sta-controlled --G. F. Sprague, 58:197

CHROMOSOME 4

4S, 4L suggested to have homeology with 2S, 2L --M. D. Bennett, 1983
hcf*-23 (gr) affects photophosphorylation --C. D. Miles, 1982
c2-E1, -E2. ....-E14 alleles, EMS-induced; -E11 leaky, near-colorless --H. K. Dooner, 1983
Trisomes found among small kernel selections --A. Ghidoni &, 1982
Zp6-h - (7.4) - Zp22 - 1.8 - Zp28 - 1.2 - Zp30 -2.9 - Zp27 - 4.7 - f12 - (3.5) - Zp14 - 7.1 - su1 - (3.1) - Zp12 - (2.2) - G14 - (4.9) - Zp15 - 5.6 - Zp10; Zp27, Zp28, Zp30 code for 20kd zein subunits; others for 22kd --C. Soave &, 1982
c2 alleles -m836018, -m836019, -m836024, -m836039 mutable; c2-m3 autonomous mutable --P. A. Peterson, 58:3
dek* mutants uncovered on 4S, 4L --M. T. Chang &, 58:61
TB-4Sa uncovers bt2 and distal markers; TB-1La-4L4692, TB-9Sb-4L6222, TB-9Sb-4L6504, TB-4Lb, c and f uncover gl4 and distal markers; TB-7Lb-4L4968 uncovers c2 and distal markers but not gl3; TB-4Sa uncovers d*-156A, hcf*-23, pgspt*-1269, nec*-562, py*-60-1106, shsu*-2IIC, su*-lethal, vit*-X832mut, wcb*-719A and wtcb*-10A; o1 uncovered by TB-9Sb-4L6222, TB-9Sb-4L6504, TB-4Lb, c, d, e and f but not by TB-7Lb-4L4698; ptd*-1130 uncovered by TB-9Sb-4L6222; py*-PI177593 uncovered by TB-1La-4L4692 and TB-4Lc; v8 uncovered by TB-4Lc and e; wlv*-378A uncovered by TB-1La-4L4692 --J. B. Beckett, 58:74
nec*-642A uncovered by TB-3La, but unlinked to su1, gl4, c2 --D. A. Hoisington, 58:83
Oleic-linoleic acids level in X-187 associated with wx T on 4L --M. D. Jellum &, 58:88
Dt6 - 7.5 - su1 --G. F. Sprague, 58:197

CHROMOSOME 5

5S, 5L suggested to have homeology with 1S, 1L --M. D. Bennett, 1983
TB-5La tertiary B-5La trisegmental endosperms were lighter than their disomic sibs --J. B. Beckett, 1983
vp2, ps1 (=vp7): points of action in carotenoid biosynthetic pathway --F. Fong &, 1983
Trisomes found among small kernel selections --A. Ghidoni &, 1982
Got2 allele -6; Got3-7; Mdh5-8.5, -9, -14.7, -16.4; Pgm2-6, -16, from Bolivian races --M. M. Goodman &, 1983
hcf*-18 (yg) affects photophosphorylation; hcf*-21 (gr) affects CO_2 fixation; hcf*-43 (yg) affects chlorophyll-protein complexes --C. D. Miles, 1982
Pgm2 alleles -.4, .45, .5, 1, 2, 2.5, 3, 4, 4.2, 5, 5.5, 6, 7, 7.2, 7.5, 7.8, 8, 9, 12, 14, 16 from lines, races and teosintes --C. W. Stuber &, 1983
Inv5a, ratio of long arm to short arm 2.9 (vs. normal 1.1); heterozygotes show little or no pairing failure at meiosis in other chromosomes, despite changes predicted due to non-homologous arm associations per Ashley (J. Cell Sci. 1979) --M. P. Maguire, 1983
gl8-3134 and bt2-2626 arose in presence of Mu --D. S. Robertson, 58:17
dek* mutants uncovered on 5S, 5L --M. T. Chang &, 58:61
hcf*-18 allelic to -43, linked to pr; hcf*-21 on 5L --M. Polacco, 58:65
nec*-493 not allelic to nec3; nec*-493 - 0 - a2; nec3 - 0 - bm1 --D. A. Hoisington, 58:84
dek18 (was E931A) on 5S --W. F. Sheridan &, 58:98
Oleic-linoleic acids level in GE82 associated with wx T on 5L (see also J. D. Shadley and D. F. Weber, Can. J. G&C 22:11, 1980) --M. D. Jellum &, 58:88
w*-21 (allelic to -22) and gl*-166 (allelic to gl8) uncovered by TB-5La and TB-5La-3L5521; w*-206, wl*-1308 (allelic to wl*-199), wlv*-308, wlv*-473 (allelic to pg*-735), and pg*-296 (allelic to -408, -71, -735) uncovered by TB-5La but not by TB-5La-3L7043; tn*-409 (allelic to -493) and gl*-681 on 5S --J. D. Shadley &, 58:160

CHROMOSOME 6

6S, 6L suggested to have homeology with 7S, 7L --M. D. Bennett, 1983
hcf*-26 (yg) affects electron transport; hcf*-34 (yg) affects photophosphorylation --C. D. Miles, 1982
Idh2 alleles -7, -7.5; Mdh2 alleles -.03, -5m; Pgd1 alleles -1, -2.8, from Bolivian races --M. M. Goodman &, 1983
Designations Enp1 and Ep1 refer to the same locus --A. L. Kahler, 58:33
dek* mutants uncovered on 6S, 6L --M. T. Chang &, 58:61
Modified chromosome M06, maize-Tripsacum translocation --B. Kindiger &, 58:67
dek19 (was E1296A) on 6S --W. F. Sheridan &, 58:98
Chromosome Trip10 carries Py1 --C. V. Pasupuleti &, 58:203

CHROMOSOME 7

7S, 7L suggested to have homeology with 6S, 6L --M. D. Bennett, 1983
Px3 located by monosomics (Weber, 1982) --M. M. Goodman &, 1983
Inv7-3717, ratio of long to short arm 1.5 (vs. normal 2.6); heterozygotes show little or no pairing failure at meiosis in other chromosomes, despite changes predicted due to non-homologous associations per Ashley (J. Cell Sci. 1979) --M. P. Maguire, 1983
o2 - 12.9 - wl6, wl6 - 5.0 - zp1-3, wl6 - 19.0 - gl1, o2 - 27.9 - gl1, o2 - 13.2 - zp1-3, o2 - 25.7 - wyg, l13 - 35.0 - o2, l13 - 47.1 - gl1, wl7 - 35.8 - gl1, wl7 - 10.6 - o2, o2 - 4.9 - y8, wl7 - 26.6 - y8, wl7 - 13.3 - De*-B30, wl7 - 9.3 - vp9, o2 - 7.3 - vp9, wyg - 12.5 - gl1, y8 - 23.1 - wyg, y8 - 24.9 - gl1, l13 - 43.0 - y8, l13 - 47.2 - De*-B30, De*-B30 - 11.2 - gl1; combined map --M. Motto &, 1983
gl1-5048 arose in presence of Mu --D. S. Robertson, 58:17
Designations E1 and Est1 refer to the same locus --A. L. Kahler, 58:33
dek* mutants uncovered on 7L --M. T. Chang &, 58:61
Les*-F331035142 associated with wx T7-9(4363) (7ctr.) --D. A. Hoisington, 58:83

CHROMOSOME 8

8S, 8L suggested to have homeology with 10S, 10L --M. D. Bennett, 1983
Idh1 allele -n; Mdh1 alleles -3.5, -5, -9.2, from Bolivian races --M. M. Goodman &, 1983
dek* mutants located on 8; associations with breakpoint in hypoploid TB-8Lc --M. T. Chang &, 58:61,62
Clt*-985 new designation for D*-985; Bif1 (barren inflorescence) symbol for Tht*-1440 (thin tassel); tentative map Bif1 - (ca. 20) - (pro1, Clt*-985) - 33 - v16 --M. G. Neuffer, 58:76,78

Rf4 closely associated with wx T8-9(6673) (8L.35) but not with T8-9d (8L.09) --A. Johnson, 58:101
Bif1 - 20.7 - pro1 --G. Gavazzi &, 58:148
dek20 (was E1392A) on 8L --W. F. Sheridan &, 58:98

CHROMOSOME 9
9S, 9L suggested to have homeology with 3S, 3L --M. D. Bennett, 1983
Sh1 Ds-4864A, located just distal to Sh1, transposed to give Ds sh1-6233.Ds, which gave Ds Sh1-6233Rev.; Ds-5245, located just distal to Sh1, gave Ds sh1-6258.Ds, which gave Ds Sh1-*.Ds, which gave Ds sh1-6795.Ds, which gave Ds Sh1-6795Rev.; Ds-5245 also gave Ds sh1-5933.Ds, which gave Ds Sh1-5933.Rev;
Insertion map: 3' - Ds-6258 - 5' - Ds-5933 - Ds-6233
cDNA clone 1-333[] Pst7[ ] --B. Burr &, 1983
TB-9La/TB-9Lb gave composite product 9-B(La +Sb) by crossing over; transmission through male reduced, through female normal --W. R. Carlson, 1983
sh1-5933 gave revertants Sh1-r3, -r4, ...-r11 that continue to show Ds-mediated breakage at Sh1 and have an intact Sh1 locus from which a 30kb insertion has excised, yet retain a duplicated part of the insertion and an extra 5' end of the Sh1 locus --W. Courage-Tebbe &, 1983
sh1 alleles -m6233, -m5933, -m6258 (independent origins, Ds-generated), and -M6795 (spontaneous recessive derived from Sh1 revertant of -m6258); rearrangements in each relative to progenitor Sh1; restriction sites for XbaI, HindIII, BglII, SstI, PstI, BstEII; mRNA transcripts --N. Fedoroff &, 1983
vp9: point of action in carotenoid biosynthetic pathway --F. Fong &, 1983
sh1 allele -m5933 (Ds-generated) vs. Sh1; restriction map for BamHI, PvuI, SphI, ClaI, EcoRI --M. Geiser &, 1982
bz1-m4 (sh1-bz1-m4) revertant Bz1-'1 (stable) shows UFGT activity profile in subaleurone similar to that of bz1-m4 (early peak), suggestion of an original deletion of Sh1 and fusion of its regulating sequences to structural Bz1 at the time of Ds transposition; Bz1-'7 (unstable) activity uniformly low --A. G. M. Gerats &, 1983
Trisomes found among small kernel selections --A. Ghidoni &, 1982
Acp1 designation recommended for Ap1 (as also Acph, Phos); alleles -2, -3, -4; Acp1 alleles -3*, -3.5, -3.8, from Bolivian races --M. M. Goodman &, 1983
hcf*-42 (gr) affects CO_2 fixation --C. D. Miles, 1982
c1 alleles -m55292, -m55301, -m55351, -m55453, -m68613, -m68655, En-mediated --A. R. Reddy &, 1983
Sh1 genomic clone: EcoRI, PstI, SstI, HindIII, BglI, BglII, HinfI, HpaII, TrqI restriction map; segmental base sequence contains two introns, 156bp (in which are 2 of 4 Ds insertion rearrangements) and 125bp --E. Sheldon &, 1983
9B(La+Sb) derived as a trisome from TB-9La/TB-9Sb --W. R. Carlson, 1983
bz1-m13 from Bz1-Mc progenitor, Spm-controlled; Rs receptor element --A. S. Klein &, 1983
Wx: map of restriction sites; allele wx-m6 contains Ds with insertion of 2.4kb, revertants Wx-m6r1 and Wx-m6r2 lack the insertion and are equivalent to the Wx progenitor of wx-m6 --M. Shure &, 1983
Wx: maps of restriction sites in wx-m6 (containing Ds6), wx-m9 (containing Ds9), wx-m9.Ac (containing Ac9), and Wx9-r1; Ac9 is 4.3kb while its derivative Ds9 is 4.1kb; Ds6 is 2kb --N. Fedoroff &, 1983
C1-I-m836526, -m836553, -836683, -836684, -836685, -836934, -836955, -836956, -836958, -836959, -836960, -836969, -836970, -unst836511, -unst836513, -unst836518, -unst836522, -unst836524, -unst836811, as well as C1-sh1-836882 and sh1-836660 and -836673, and wx1-836605, -836608, -836610, -836616, -836617 all arose from C1-I Sh Wx1 in the presence of En --P. A. Peterson, 58:2,3
bz1-m826301 En-controlled; c1-m816665 and c1-m816667 Uq-controlled; c1-m816666, c1-st817086, c1-m804655 tests --P. A. Peterson, 58:3,4,5
bz1-m805137 Cy-controlled, bz1-rcy isolate requires Cy for mutability; bz1-m794266 mutable --P. A. Peterson, 58:9
bz1-N1032 unstable; arose in presence of Mu --M. Alleman, 58:24
bz1-mut, Mut-controlled --M. M. Rhoades &, 58:30
dek* mutants uncovered on 9S, 9L --M. T. Chang &, 58:61
sh1: restriction map, genomic vs. cDNA shows 15 introns, exons, boxes --W. Werr &, 58:57
sh1-m5933 and sh1-m6233 contain insertions, reverse repeats, duplications; homologies defined to other Ds and Ac sequences --U. Courage-T. &, 58:57; H. P. Doring &, 58:58; E. Weck &, 58:59
wx1-m7 contains Ac, 4.3kb insertion --H. P. Doring &, 58:59
hcf*-42 on 9L --M. Polacco, 58:65

CHROMOSOME 10
10S, 10L suggested to have homeology with 8S, 8L --M. D. Bennett, 1983
y9: point of action in carotenoid biosynthetic pathway --F. Fong &, 1983
Glu1 alleles -2, -6 , -7 are those identified as beta-Glu1-d, -j, -k by Stuber et al., 1977; Glu1 alleles -3.2, -4, -5, -10.5, -13.5, -14, -16, from Bolivian races --M. M. Goodman &, 1983
R1 - 19.3 - w2 - 24.4 - sr2, confirmed order --M. M. Rhoades &, 58:28
Recombination for R1 Sr2 in +/w2 32.8%, in +/+ 28.7% --M. M. Rhoades &, 58:32
g1 - (ca. 6) - lte2 - (ca. 5) - R1 --L. T. de Miranda &, 58:48,49
dek* mutants uncovered on 10S, 10L --M. T. Chang &, 58:61
dek21 (was E1330) on 10L --W. F. Sheridan &, 58:98

UNPLACED
hcf*-5 (gr), -17 (gr), -20 (gr), -33 (gr), -35 (yg), -36 (gr), -37 (gr), -40 (gr), -325 (yg) affect electron transport; hcf*-6 (gr), -16 (gr), -38 (gr), -47 (yg) affect cytochromes; hcf*-7 (yg), -8 (yg), -30 (gr), -48 (yg), -317 (yg), -323 (gr), -324(gr) affect photophosphorylation; hcf*-11 (yg), -28 (gr), -45 (gr) affect CO_2 fixation; hcf*-14 (pg), -29 (yg), -31 (yg), -49 (yg), -311 (yg), -316 (yg), -318 (yg), -408 (yg), -418 (pg) affect chlorophyll-protein complexes --C. D. Miles, 1982
pi1, pi2: pistillate florets; development of secondary florets in ear; 15:1 interaction --V. E. Micu &, 1983
vp*-x1: point of action in carotenoid biosynthetic pathway --F. Fong &, 1983
Acp2 designation recommended for Ap2; E12, E16 relationship to E1 ... E10 unresolved --M. M. Goodman &, 1983

Sod1: superoxide dismutase; hybrid bands occur; plastidal; alleles -A, -B;
Sod3: superoxide dismutase; hybrid bands occur; mitochondrial; alleles -A, -B;
Sod4: superoxide dismutase; hybrid bands occur; cytosolic; alleles -A, -B --Baum & Scandalios, J. Hered. 1982
Cat3 alleles -8, -8.5, -10.3, -11, from Bolivian races --M. M. Goodman &, 1983
Acp4, acid phosphatase; electrophoretic mobility; alleles -1, -2, -3, -4, -5, -6 --A. L. Kahler, 1983
Rst: Restrainer of En patterns --A. R. Reddy &, 1983
Mc (Mucronate): opaque endosperm; Mc o2 combination slightly collapsed, zein reduced --F. Salamini &, 1983
Les*-1450, les*-1395, les*-F26514, Les*-A467: descriptions --V. Walbot &, 1983
RSS (Reduced Seed Set) --K. Sukhapinda &, 1983
Uq (Ubiquitous) regulatory element, affects a1-ruq --P. A. Peterson &, 1983
brn1: brown aleurone, lethal to seedling; arose in presence of Mu --D. S. Robertson, 58:18
Designations Apl, Acp1 and Acph1 refer to the same locus; Px1 and Prx1 to same locus --A. L. Kahler, 58:33
Pe*-D: perennialism from Z. diploperennis (evergreen stalks, stiff stalks, robust root system); association with sul and gl3 --P. C. Mangelsdorf &, 58:53,54
hcf*-Mu-5, Mu-induced --M. Hunt &, 58:68
Dominants from EMS treatments: Tillered-1590, Golden-1585, Semidwarf-1592, Hsf*-1595 --R. M. Bird &, 58:71
Lcs1: thylakoid membrane polypeptide, fast form in SDS gels -Zm, slow -Z1 (Z. luxurians);
Lct1, slow -Zm, fast -Z1; Lct2, presence, dominant to lct2-Ky27 (absence) --S. A. Modena, 58:82
rgh*-E1210, fl*-E1253B, dcr*-E1428 developmentally blocked prior to leaf primordia; cp*-E1399A fails to germinate --Clark &, 58:91

Rgh, restorer of cytoplasmic growth habit; tsi, tassel silks and branched ear; Sta regulatory element --G. F. Sprague, 58:196

ora2-13, orange endosperm; lty1-16, -17, -19, light yellow endosperm --E. J. Dollinger, 58:210
pdf1, thylakoid membrane polypeptide, recessive allele -Zm (slow) vs. -Z1 (fast, from Z. luxurians); association with Lct1 --S. A. Modena, 58:211

CHLOROPLAST

cms-C and cms-T differ from normals in EcoRI and BamHI restriction fragment patterns --J. Li &, 1983
Homologies and inversions relative to spinach, peas and other species --J. D.Palmer &, 1982
tRNAser3, tRNAphe, tRNAthr2, tRNAleu2, tRNAmetm base sequences and upstream promoters --A. A. Steinmetz &, 1983
Putative ribosomal protein S4 gene (-rProt"S4") base sequence --A. R. Subramanian &, 1983
Interspecific tRNA hybridizations; map locations of tRNAhis, tRNAleu2, tRNAile1, tRNAile2 --J. H. Weil &, 1981
Map locations of tRNA sequences in genome and in repeats I & II --J. H. Weil &, 1982; R. F. Selden &, 1983
Locations of promoters for tRNAval, for rRNA16S-23S-4.5S, and for rRNA5S --G. Zenke &, 1982
LS (-rcL), upstream and 3' terminus sequences; beta and epsilon of coupling factor (-cf1BE) have fused base sequences; other open reading frames; section of homology to E. coli ribosomal gene S4 (-rProt"S-4"); map locations of 32kd photogene (-PG32) and four other photogenes --L. Bogorad &, 1983
Map of SalI, PstI, EcoRI, BamHI sites, fragment lengths, and coordinates of sites; starts and termini of genes -r5 (5SrRNA, repeat I & II), -r4.5 (4.5SrRNA, repeat I & II), -r23 (23SrRNA, repeat I & II), -tA-UGC (tRNAala, repeat I & II), -tI-GAU(tRNAIle, repeat I & II), -r16 (16SrRNA, repeat I & II), -tV-GAC (tRNAval, repeat I & II), -tL-CAA (tRNAleu, repeat I & II), -rcL (large subunit), -cf1B (beta subunit of coupling factor), -cf1E (epsilon subunit of coupling factor), -tM (tRNAmet), -tF (tRNAphe), -tL-UAA (tRNAleu), -tT-UGU (tRNAthr), -rpS4 (homol. to E. coli ribosomal protein S4), -tS-GGA (tRNAser) --I. M. Larrinua &, 1983
LS segment is represented in mtDNA --D. M. Lonsdale &, 1983
Possible Mu insertion --D. S. Robertson, 58:20

MITOCHONDRION

18SrRNA (3' end) through 5SrRNA, 664-base sequence --S. Chao &, 1983
S1 and S2 bands lost in friable callus; probes for S1 and S2 hybridize intensely to mtDNA --P. S. Chourey &, 1982
cmsS subgroups I (S, R, ML); II (L, CA); III (EK) defined by principal component analysis of restoration reactions --L. Kalman &, 1982
Five fertile, resistant derivatives arising from cms-T after regeneration from tissue culture show no changes in low MW mtDNA, but various changes in restriction patterns of the high MW mtDNA --R. J. Kemble, 1982
Plasmids S1 (6.2kb), S2 (5.2), linear n (2.35), and smaller linear t, heterogeneities and homologies in 25 Mexican races; lines with t also have an S1-related 6.55 kb BamHI fragment, while lines with n have an S1-related 6.85 kb fragment, except for Reventador (n and S1-related 80kb), Zapolote Grande (t, no S1-related bands), and Bolita individuals (t, 3.5kb); n probe hybridizes to t and to S1 and S2 --R. J. Kemble, G &, 1983
Plasmids S3 (13.6kb), S4 (12.4), S5 (11.4) (possible concatemers), as well as S1 and S2, disappear on reversion of cms-S to fertile; S3 and S4 hybridize with S1 probe pZmS42; S4 and S5 with S2 probe pZmS4; S3, S4, S5 with S1S2 probes pZmS21 and pZmS40; differing S1- and S2-hybridizing sequences are found in XhoI, BamHI and SalI digests of high MW mtDNA of revertants 251, 296, and 369 --R. J. Kemble & M, 1983
S2 probe pZmS4 does not hybridize with BamHI digests of nuclear DNA of cms-Vg, of cytoplasmic revertant 296, or of nuclear revertant VgRfh; S1-related probes hybridize similarly with digests of nuclear DNA of normal (Tr, W64A, B37) and of nuclear revertants (Rf*-h, Rf*-i, Rf*-m) --R. J. Kemble, M &, 1983
Regions of genome homologous to S1 and S2 ("S regions"): variations in S1-homologous regions (3 groups--Wf9; F6, A188, W182BN; Black Mexican); linear 2.1kb in the second group in place of 2.3kb --J. W. McNay &, 1983
S1, S2, R1, R2 plasmids: BamHI and EcoRI characterization of Latin American races --A. K. Weissinger &, 1983
cms-S, VG, I, ML, RL reversion rates to male fertility in 8 inbred-line backgrounds; cytoplasmic and nuclear changes --S. Gabay-Laughnan &, 1983
Male-fertile revertants from cms-T following tissue culture, -T1 through -T6, maternally inherited; XhoI restriction fragment of 6.6kb absent in all but -T4 and progenitor -T --P. F. Umbeck &, 1983
Plasmids D1 (7.5kb) and D2 (5.5kb), double-stranded linear in cytopl-ZD (Z. diploperennis) --D. H. Timothy &, 1983
Segment homologous to ctDNA-LS, transcribed and translated --D. M. Lonsdale &, 1983
S2 base sequence (5452bp) contains two large open reading frames and is terminated with exact 208bp inverted repeats --C. S. Levings III &, 1983

Male-sterile variants from a Peruvian population, -P2, -P4, -P5, variant mtDNA patterns --A.Mezzarobba &, 58:85
Probes for 1.94kb circular and 1.4kb circular not homologous to each other or to S1, S2, 2.3kb linear or 2.1kb linear, or to restricted mtDNA of any source tested; homology for 1.4 is found in many lines and cytoplasms, accompanied by 1.94; 1.94 present without 1.4 in WF9 N, T, S, and in RB cytoplasm --A. G. Smith &, 58:90
mox1 exons, intron --R. E. Dewey &, 58:171
NCS2 and NCS3 sources each display a single change in restriction fragments as compared to T, from which they arose --K. J. Newton &, 58:192
1.94kb is found in S, J and I but not in VG, RD or ML members of S group --J. E. Carlson &, 58:194

CYTOPLASM
NCS2: nonchromosomal stripe; abnormal, pale green, clonal stripes; similar to wsp of Brown & (MNL 32:120,1958); NCS3: nonchromosomal stripe; abnormal, distorted sectors; similar to P2 of Brown & --E. H. Coe, Jr., 1983
cgl (cytoplasmic glossy) --G. F. Sprague, 1983
MET (multiple ears and tillers) trait maternally transmitted --B-h. Choe &, 58:85
Symbol cgh (cytoplasmic growth habit) suggested for P2 cytoplasm of W. L. Brown --G. F. Sprague, 58:196

cDNA/GENOMIC CLONES/PROBES
zein cDNA clones A20, A30 (of 19kd chains) and B49 (of 22kd): base sequences, maps of sites for AluI, HaeIII, RsaI, TaqI; homologies inter se --D. E. Geraghty &, 1982
12-C-6, cDNA homologous to mRNA for pyruvate, Pi dikinase --D. R. Hague &, 1983
zein genomic clone Z4, cDNA clones A30, ZG31A; base sequences and homologies; restriction map for Sau3A, RsaI, AluI, HpaII, XbaI --N-T. Hu &, 1982
zein cDNA clones ZG7, ZG14, ZG15, ZG19, ZG31, ZG124, A30; genomic clone Z4, base sequences and homologies --G. Heidecker &, 1983
zein genomic clones pML1 (of 19kd) and pMS1 (of 21kd); HincII, PvuII, HinfI, TaqI, Sau3A restriction sites and two promoter sequences, P1 and P2, with base sequences --P. Langridge &, 1983; 58:88
zein genomic clone ZA1 (heavy chain): EcoRI, Taq, Dde, HpaII restriction map and base sequence; homologies with clone E19 (light chain) --A. Spena &, 1982
zein nomenclature proposed, subfamilies SF1 through SF5, source and cDNA vs. genomic, e.g., SF1(IHP-c)A20, SF4(W22-g)Z7, etc. --I. Rubenstein &, 58:178
zein clones hybridize in situ on chromosome 4 (clone M6, E21), 7S (M6, E21), 10L (M6) --A. Viotti &, 58:213
Restriction fragment probes from repeated DNA (H1, H2, S1, T2, T3) show differential hybridization in situ on B chromosomes and A (including NOR region) --A. Viotti &, 58:213

RESISTANCE/TOLERANCE/HERITABILITY
European corn borer (Ostrinia nubilalis Hubner) tunnel length reduced significantly by 3 cycles of selection in 3 populations, 5.3 to 12.7% per cycle --D. Barry &, 1983
Nitrogen fixation in association with Azotobacter vinelandii showed dominance and heritability responsive to selection across 5 generations --S. W. Ela &, 1982
High stearic acid level in germ oil of PI175334 determined by a major recessive gene and modifiers --M. D. Jellum &, 1983; 58:87
Maize Dwarf Mosaic Virus R/S in Oh7B vs. Oh43 and Pa91: one dominant gene for Resistance --C. W. Roane &, 1983
European Corn Borer (Ostrinia nubilalis Hubner) R/S in B52 vs. CI131A showed predominantly additive genetic variance --M. Sadhedel- &, 1983
Isocitrate lyase: high activity from Illinois High Oil dominant in crosses with Illinois Low Oil, in contrast to additivity for oil content; no correlation in F2 between activity and oil content --A. S. Tsaftaris &, 1983
Stewart's wilt (Erwinia stewartii) R/S additive (general combining ability high among 6 inbreds/15 single crosses/60 3-way crosses --W. M. Forgey &, 1982
Sulfate uptake efficiency (V_{max} and K_m) in 5-inbred diallel showed additive and non-additive effects (significant general and specific combining ability), and F1 reciprocal variation --M. Motto &, 1982; M. Saccomani &, 1982
Downy mildew (Peronosclerospora philippinensis) R/S among 36 entries showed both additive and partially dominant resistance (highly significant general and specific combining ability); broad-sense heritability 45.67%, narrow 39.23% --O. B. Capuno &, 1982
Ear rot (Fusarium graminearum) R/S in 7-inbred diallel F1, F2, P1F1, P2F1; both additive and partially dominant resistance (highly significant general combining ability --M. Odiemah &, 1982
European corn borer (Ostrinia nubilalis Hbn.) R/S in 14-inbred diallel additive; resistance associated with late silking and grain maturity, stalk-rot resistance; broad & narrow-sense heritability 38-43% --F. Kaan &, 1983
Eyespot (Kabatiella zeae) R/S in crosses of two R and 3 S lines, F1, F2 and BCs indicates resistance partially dominant, one to two genes --F. J. B. Reifschneider &, 1983
Twin-ear --A. R. Hallauer, 58:21
Associations of flint character, kernel row number, earworm resistance from Zapalote Chico, earworm resistance from IAC Maya, glycoside earworm resistance, and purple-restricted-to-cob with wx translocations --L. T. de Miranda &, 58:38
Twin-stalks, 4.5% in inbred F1254, show low, incomplete penetrance --M. Pollacsek, 58:56
Morphology of TB hyperploids --M-T. Chang, 58:63
Oleic-linoleic acids level in GE82 and X-187, association with wx T's --M. D. Jellum &, 58:88
Apparent photosynthetic activity negatively correlated with upright leaves and leaf area index in Argentinian flint lines --J. R. Jatimliansky &, 58:117
Pollen grain size, Gaspe vs. Z. perennis, heritable, additive --J. L. Magoja &, 58:118
Tassel branching traits, Gaspe vs. Z. perennis, heritable; branch number closely associated with length of branching axis but not with internode length between spikelets --I. G. Palacios &, 58:122

V. REPORT OF MAIZE GENETICS COOPERATION STOCK CENTER

During 1983 there were 167 seed requests and 1,927 seed packets were sent. This was the highest figure ever. Domestic requests amounted to 146 for 1392 packets while there were 21 foreign requests for 167 packets.

The estimated uses of the seed were distributed among the following categories:

Geneticists	52%
Physiologists	15%
Breeders	13%
Educators	4%
Genetic Engineers	16%

This is the second time that the category of Genetic Engineers has been used. These requests this year amounted to over one and a half times that of the previous year.

Progress continues in our efforts to get the entire inventory on the computer terminal. Other aspects of our operation are being computerized at a good clip.

The following listing shows the stocks that are available. Translocation stocks are listed in the 1981 News Letter.

Requests for seed and information should be sent to:

Dr. Gilbert B. Fletcher
Department of Agronomy
University of Illinois
S-123 Turner Hall
1102 South Goodwin Avenue
Urbana, IL 61801

Ah, el novio no quiere dinero
Oh, the bridegroom doesn't want money
he wants a bride with a good field of maize.
I've come to wish them joy and prosperity
and all manner of good things

Oh, the bridegroom doesn't want ducats;
he wants a bride whose maize grows tall.
I've come to wish them....

Oh, the groom wants no fine bracelets;
he wants a bride who'll look happy withal.
I've come to wish them....

--A song from the Sephardic
Jewish tradition

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 msl7 P-WW
zb4 msl7 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
msl7
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
tb-8963
Kn
Kn Ts6
Kn bm2
lw
Adh1-S
vp8

Chromosome 1 (continued)

gs
gs bm2
Ts6
bm2
id
nec2
ms9
msl2
msl4
mi
D8
Lls
Les2
TB-1La (1L.20)
TB-1Sb (1S.05)

Chromosome 2

ws3 lg gl2 B
ws3 lg gl2 B sk
ws3 lg gl2 B sk v4
ws3 lg gl2 B sk f1 v4
ws3 lg gl2 B gs2 v4
ws3 lg gl2 B ts
ws3 lg gl2 b
ws3 lg gl2 b sk
ws3 lg gl2 b sk v4
ws3 lg gl2 b gs2 v4
ws3 lg gl2 b f1 v4
ws3 lg gl2 b sk f1 v4

ws3 lg gl2 b v4
al
al lg
al lg gl2 B sk v4
al lg gl2 b
al lg gl2 b sk v4
al lg gl2 b sk f1 v4
lg
lg gl2
lg gl2 B
lg gl2 B gl11
lg gl2 B gs
lg gl2 B gs2 v4
lg gl2 B gs2 Ch
lg gl2 B gs2 sk Ch
lg gl2 B gs2 sk v4
lg gl2 B sk
lg gl2 B sk v4
lg gl2 B v4
lg gl2 b
lg gl2 b gs2
lg gl2 b gs2 Ch
lg gl2 b gs2 sk Ch
lg gl2 b gs2 v4
lg gl2 b gs2 v4 Ch
lg gl2 b gs2 sk v4 Ch
lg gl2 b sk
lg gl2 b sk f1
lg gl2 b sk f1 v4
lg gl2 b sk v4
lg gl2 b wt v4
lg gl2 b f1
lg gl2 b f1 v4
lg gl2 b f1 v4 Ch
lg gl2 b v4
lg gl2 b v4 Ch
lg gl2 mn v4
lg gl2 wt
lg gl2 b gs2 wt
lg gl2 w3
lg gl2 w3 Ch
lg gl2 Ch
lg b gs2 v4
lg Ch
gl2
d5 = d*-037-9
B gl11
B ts
gl14
gl11
wt
mn
f1
f1 v4 Ch

Chromosome 2 (continued)

f1 Ht v4
f1 Ht v4 Ch
f1 w3
f1 v4 w3
f1 w3 Ch
f1 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 ; r A A2 C
r2 ; r-g A A2 C
Ch
gs2
Les
2 2^{Trip} $Trip^{2}$ /ws3 lg gl2
TB-1Sb-2L4464
TB-3La-2S6270
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 gl6
d Lg3 ts4 lg2
d Rg
d Rg ts4 lg2
d pm
d yg*-(W23)
d ts4 lg2
d ts4 lg2 a-m ; A2 C R Dt
d ts4
d gl6
d lg2 a-m A2 C R Dt
d a-m A2 C R Dt
ra2
ra2 Rg
ra2 Rg ts4 lg2

ra2 ys3 Lg3 Rg
ra2 ys3 Rg
ra2 Rg lg2
ra2 pm lg2
ra2 ts4
ra2 ts4 lg2
ra2 lg2
Cg
cl
cl ; Clm-2
cl ; Clm-3
cl-p ; Clm-4
rt
ys3
ys3 Lg3

ys3 gl6 lg2 a-m et ; A2 C R Dt
ys3 ts4
ys3 ts4 lg2
Lg3
Lg3 Rg pm
gl6
gl6 lg2 A ; A2 C R
gl6 lg2 A-b et ; A2 C R Dt
gl6 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
yl0
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-xl
a-x3
a Ga7 ; A2 C R
sh2
vp
Rp3
pgl4
a3
g5
te
h
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 f12
Ts5
Ts5 f12
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
f12
f12 su
f12 bt2
f12 su bm3
f12 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-l lst
Tu-l 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
vl7
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 gl8
vp7
bm
bm yg
bt
ms5
v3
td ae
ae
sh4
gl8
na2
lw2
ys
eg
v2
yg
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 l10
y l11
y l12
y Wl5
y pb4
y pb4 pl
y pb4 Pl
y si
y wi Pl
Y Dt2 ; a-m A2 C R
y pgl1 ; Wx pgl2
y pgl1 wi ; wx pgl2
Y pgl1 ; Wx pgl2
y pgl1 ; wx pgl2
Y pgl1 ; wx pgl2
y pgl1 su2 ; wx pgl2
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 l10
Y l12
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
wl4
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 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 mn2
Tp
ij
ms7
ms7 gl Tp
Bn
bd
Pn
o5
o5 mn2 gl
va
Dt3 ; a-m A2 C R
V*-8647
yel*-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
f13
f13 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
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 pgl2 gl15 ; y pgl1
lo2
wx*
wx-a
wl1
wx d3
wx d3 wl1
wx d3 v gl15
wx d3 gl15
Wx pgl2 ; y pgl1
wx pgl2 ; y pgl1
Wx pgl2 ; Y pgl1
wx pgl2 ; Y pgl1
wx pgl2 bm4 ; y pgl1
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
yel*-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 bf2
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
Ej r-r ; A A2 C
Ej 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 Pl ; 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
Rs
v13
ws ws2
ub
zb
zb2
zn2
l*-4923
nec*-8376

Multiple Gene Stocks

A A2 C C2 R-g Pr B Pl
A A2 C C2 R-g Pr B pl
A A2 C C2 R-g b Pl
A A2 C C2 r-g Pr B Pl
A A2 C C2 r-g Pr b pl
A A2 C C2 r-g Pr B pl
A A2 c C2 R-g Pr B pl
A A2 C C2 R-r Pr B Pl
A A2 C C2 R-r Pr B pl
A A2 C C2 R-r Pr b Pl
A A2 C C2 R-r Pr B Pl wx
A A2 C C2 R-r Pr B pl wx
A A2 c C2 R-r Pr B Pl
A A2 C C2 r-r Pr B Pl
A A2 C C2 r-r Pr B Pl
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
bm2 lg a su pr y gl j 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 6 ; Hi-haploid R-r B Pl

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
Gourdseed
Maiz Chapolote
Papago Flour Corn
Parker's Flint
Tama Flint
Zapaluta Chica

Tetraploid Stocks

P-RR
P-VV
B Pl
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 Pl

Cytoplasmic traits

NCS2
NCS3

Cytoplasmic steriles and Restorers

WF9-(T)	rf rf2	[Temp. out of seed]
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; 9:.24) * 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) *
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) *
Wx 7-94363 (7 cent.; 9 cent.) *
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-L69)
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)

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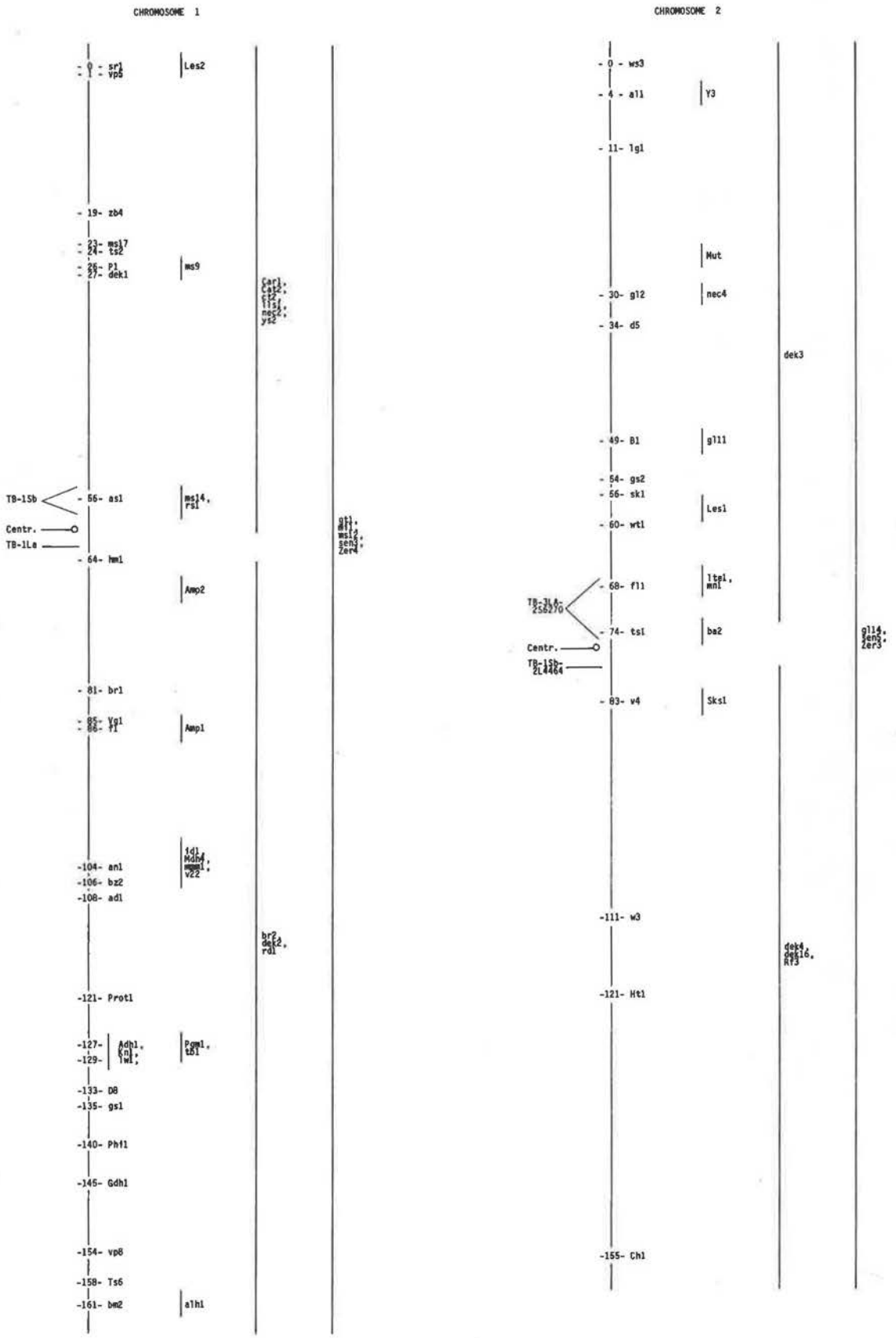
VII. SYMBOL AND CYTOGENETIC INDEX

VIII. AUTHOR AND NAME INDEX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- 26- P1
- 27- dek1
ms9
Carl, Ca12, Cr2, Ht1, nec2, ys2
TB-15b
- 56- as1
ms14, rs1
Centr.
TB-1La
- 64- hm1
Amp2
- 81- br1
- 85- Yg1
- 86- P1
Amp1
1d1, Mdh4, mm1, v22
-104- an1
-106- bz2
-108- ad1
br2, da12, rd1
-121- Prot1
-127-
-129-
Adh1, Cn1, lm1,
Pgm1, ts1
-133- D8
-135- gs1
-140- Ph1
-145- Gdh1
-154- vp8
-158- Ts6
-161- bm2
a1h1
gl1, Hk1, ms12, sen3, zer4
CHROMOSOME 2
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- 4 - a11
Y3
- 11- lg1
Mut
- 30- g12
nec4
- 34- d5
dek3
- 49- B1
gl11
- 54- gs2
- 56- sk1
Les1
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- 68- f11
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- 74- ts1
ba2
Centr.
TB-15b, 2L4464
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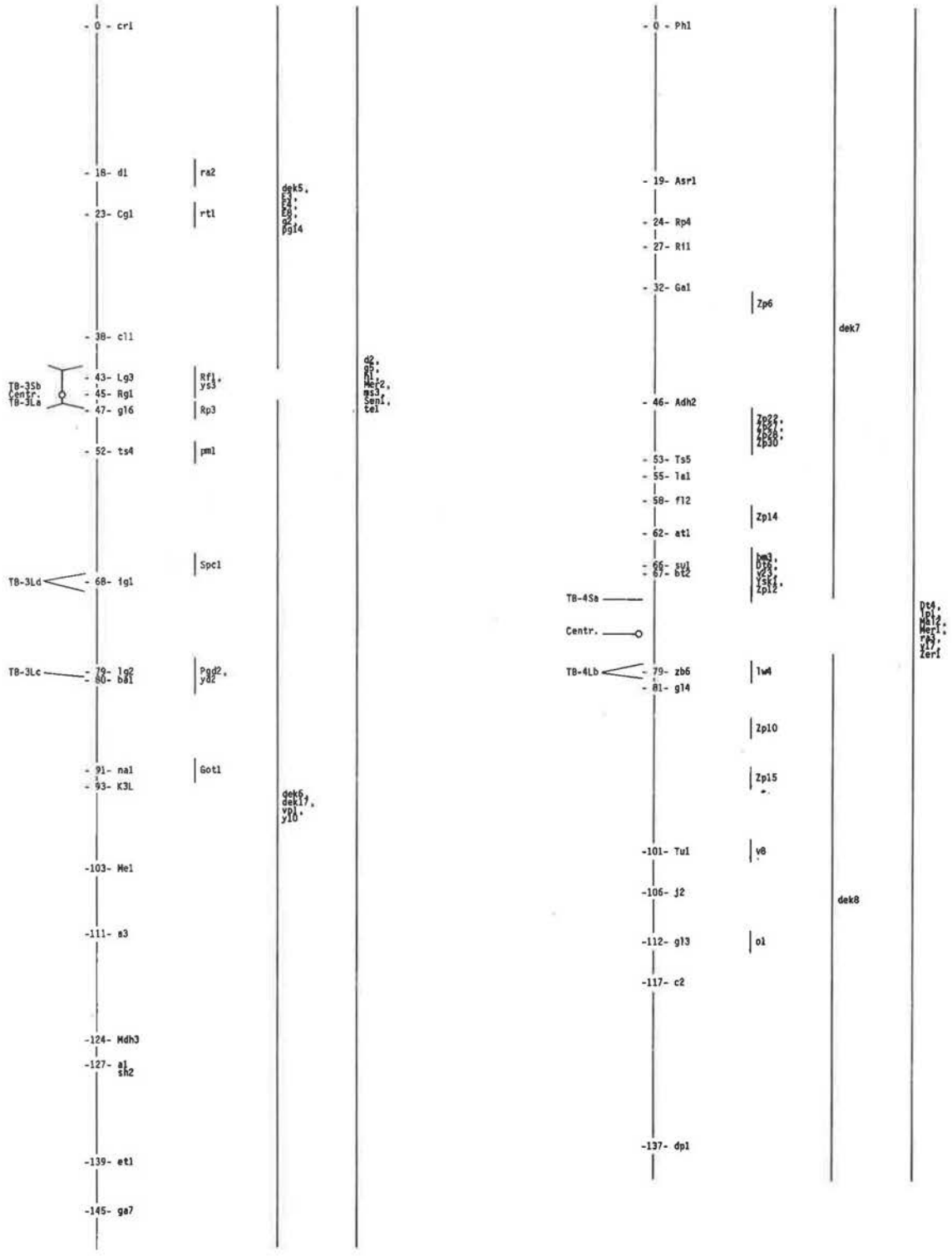
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- 47- g16
- 52- ts4
- 68- ig1
- 79- lg2
- 80- b81
- 91- na1
- 93- K3L
-103- Me1
-111- a3
-124- Mdh3
-127- al
sh2
-139- et1
-145- ga7
T8-3Sb
Centr.
T8-3La
T8-3Ld
T8-3Lc
ra2
rt1
Rf1,
ys3
Rp3
pm1
Spc1
Pgd2,
yd2
Got1
dek5,
dek6,
dek17,
vp1,
y10,
d2,
pg4,
Mer2,
as3
sen1,
tel
CHROMOSOME 4
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- 24- Rp4
- 27- R11
- 32- Ga1
- 46- Adh2
- 53- Ts5
- 55- la1
- 58- f12
- 62- at1
- 65- sv1
- 67- bt2
- 79- zb6
- 81- g14
-101- Tu1
-106- j2
-112- g13
-117- c2
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Centr.
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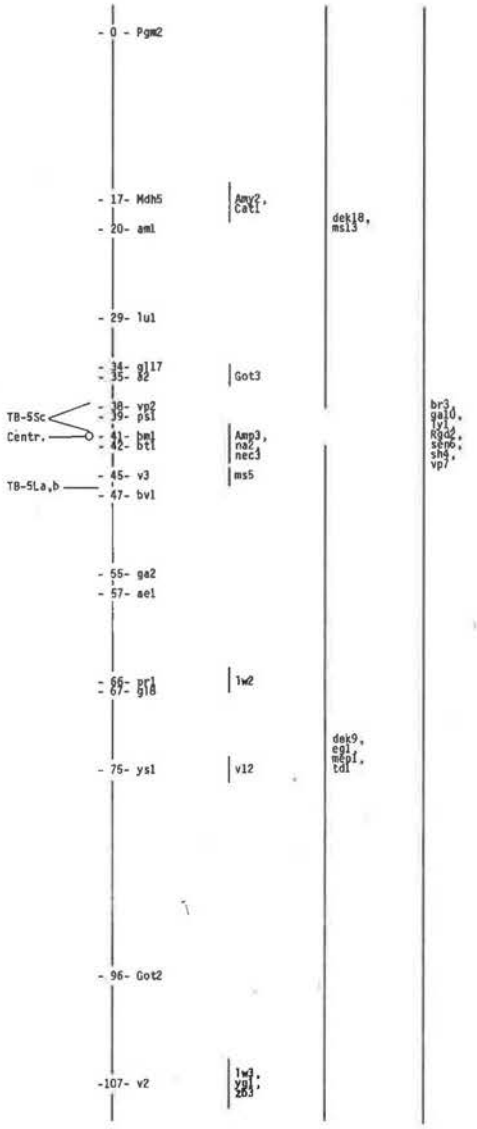

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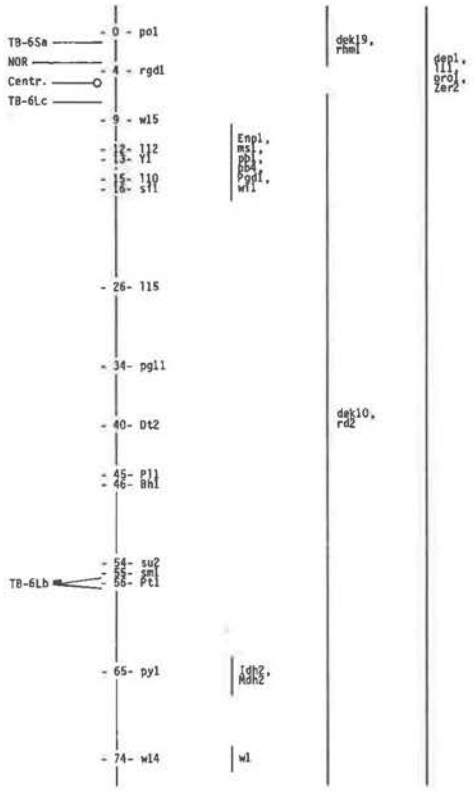

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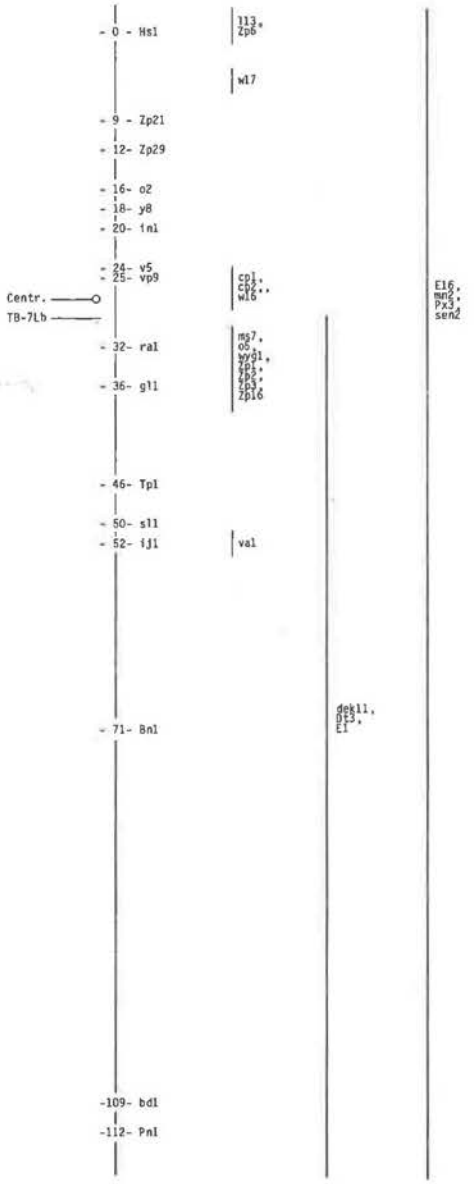

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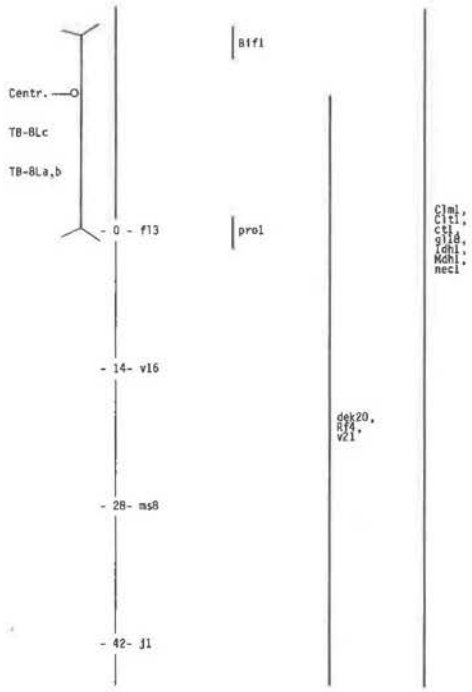

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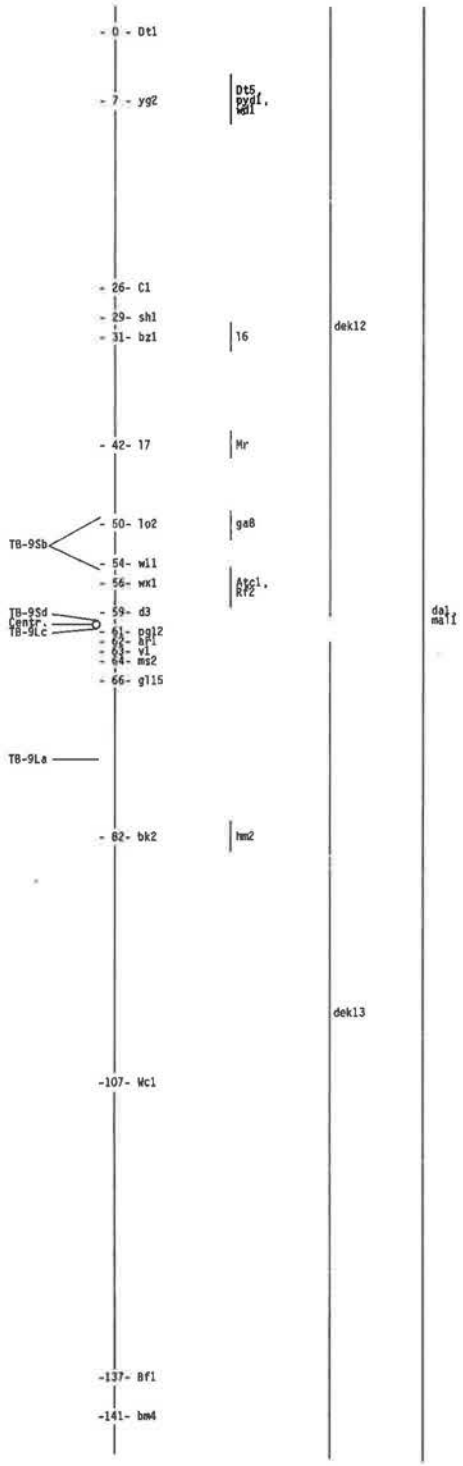

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