MAIZE GENETICS COOPERATION

NEWS LETTER

61

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and
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University of Missouri
Columbia, Missouri

TABLE OF CONTENTS

SIA MEDICINE SONG

Grant our children life
and happiness.
Send forth the good
south winds.
Send forth your breath over
the waters that our world
may be beautiful and
our people may thrive.
Far off, over there, Sun Father
awakens, and climbs up his
ladder leaving his
resting place.
May all complete life's long
road, may all grow old.
May our little ones know the
sweet smell of the sacred
breath of life.
May all our children have
maize that they may
complete their journey.
Sit down, remain here,
we give our best gift,
our best thoughts.
We inhale the sweet smell of
the sacred breath through
our prayer plumes.

I. FOREWORD

"By tradition, scientists are supposed to be objective and coldly analytical, but that view is nonsense. The best scientists convey enthusiasm and excitement and thus stimulate enhanced creative activity by their fellows." --P. H. Abelson, 1980

The data, techniques and tidbits on maize genetics in the notes in this News Letter represent for each of us the enthusiasm and openness that is the tradition among maize geneticists. The sharing of our field books, our observations and our notebooks in these pages is done with the specific understanding that the information here is not published information, and that it is not to be cited in publications without the consent of the authors.

Notes for the next issue (Number 62, 1988) should be in my hands by January 1, 1988. The writing and content should be brief and informal. Text should be in simple and efficient form, double-spaced so that it can be retyped efficiently. Tables, Figures and Charts must be compact, single-spaced, and ready for direct copying by the camera.

The U. S. Department of Agriculture and the Maize Genetics Stock Center again provided the resources needed for the assembly, compilation, production and distribution of this issue, and all of us in the Maize Genetics Cooperation appreciate the encouragement and support that is so essential for the compiling and sharing of information.

Shirley Kowalewski again skillfully edited and refined the copy and the proofs, in addition to keeping the year-round office tasks in order and aiding in screening of the literature for compilation. Mary Brazil once again produced and refined the literature with care, and Kathy Chappell and Chris Browne cheerfully and efficiently helped with assembling and other vital tasks. The University Printing Services, including Yvonne Ball and Dale Kennedy and their careful staff, efficiently and carefully made sure that the planning, typesetting, composition and printing were done promptly and well.

Dave Hoisington in particular, among my USDA and University of Missouri colleagues, continues to produce his expertly derived data compilations, the impressive working maps, and the computer trickery, as well as advice and ideas. Chang-deok Han aided with refining of Recent Maize Publications and with proofing, and I appreciate the help of Allen Wright, Evelyn Bendbow and Mary Polacco with proofing.

Mitochondrial mapping data were generously compiled again by David Lonsdale, and the impressiveness of that genome characterization challenges the nuclear maizoids to match the mitochondriacs.

The Maize Genetics Conference, held in March each year, will be in Madison, Wisconsin, in 1988. Information about the Conference can be obtained from Oliver Nelson, who is making the arrangements. His address is listed in the revised Mailing List in this issue.

Symbol Clearing House: Earl Patterson, who has now taken on the oversight of the Maize Genetics Stock Center, has proposed to me that the responsibility of the Clearing House for symbols should transfer to my hands. After some arguments and scars, I have told him I am willing to serve in that role for the present. Since Earl will not be so busy now, please see the list of stocks not in the collection, compiled by Dave Hoisington (Mapping Section), and send any of the needed stocks to the Stock Center if you have them.

Ed Coe

II. REPORTS FROM COOPERATORS

ALMORA, U.P., INDIA
I.C.A.R.

Preliminary observations on drought tolerance in Sikkim Primitive maize

The existence of primitive types of maize cultivars possessing high degrees of prolificacy (4 to 8 ears/plant) in the Northeastern Himalayas of India has generated considerable interest among the maize workers with regard to the origin and evolution of the maize plant. Recently, Sachan and Sarkar (1986), on the basis of extensive studies carried out on such primitive types of maize, have concluded that Sikkim Primitive maize is the same as the pre-Chapalote, pre-Nal-Tel and prehistoric wild corn of Mangelsdorf. In addition to a subject of interest for origin and evolution, Sikkim Primitives have considerable utility as a source of prolificacy, pest resistance, and drought tolerance due to their long history of survival against the vagaries of nature.

During *Kharif* 1986, fifteen primitive types collected from the Northeastern Himalayan region of the country were grown in a breeding nursery under rainfed, high-fertility conditions at the experimental fields of the Vivekananda Parvatiya Krishi Anusandhan Shala, Almora, situated at 1350 m a.s.l. Visual observations on drought tolerance were recorded on a 1 to 5 scale (1 no wilting, 5 all leaves wilted) on the sixteenth day of a twenty-day drought spell that occurred from 21st August to 10th September. The maize crop in the adjacent fields was severely wilted due to drought. Most of the materials in the breeding nursery also exhibited severe symptoms of moisture stress. However, of the fifteen Sikkim Primitive collections M1 and Murulia, with a score of 1.0, were highly tolerant to drought conditions as compared to S44 and S2, which had a score of 4.0 & 3.0, respectively, and showed severe signs of wilting. In the rest of the collections the tolerance was of intermediate type (1.5 - 2.0). These observations indicate that the Sikkim Primitive maize can be a potential source for drought tolerance in maize breeding programmes. However, the preliminary observations need further confirmation.

V.P. Mani, H.C. Jotshi and K.D. Koranne

AMES, IOWA
Iowa State University

c2-m826021—An autonomously controlled allele at the *c2* locus

c2-m826021 is an unstable *c2-m* allele that originated in an isolation plot (MNL 57:2) containing genotypes with *En*. The general pattern of this allele is very late in timing, though occasional early sectors appear. The frequency is largely a 6aa-type (very late-near single cell, but frequent—Reddy and Peterson, MGG 194:124), though this medium high frequency is not strictly heritable.

In tests of specific control, it has been confirmed that the control of mutability is autonomous. In 31 test crosses, the mutability cosegregated with the *c2* locus. The frequency of colorless types ranged from .4% to 2.1% among eight of the 31 crosses.

Two examples of this cross:

		Colored	spotting	cl	Total
84	1001-1/0707-6	255	273	4	532
84	1002-1/0709-7	263	236	0	499

This demonstrates the autonomous control of the mutability. Though *En* is strongly implicated, additional tests are needed for confirmation. The alternative of numerous *En* to accommodate the incidence of colorless segregants would require from 4 to 5 *En*, which is not applicable in this experiment.

Ulla Niesbach-Klosgen and Peter A. Peterson

c2-m1: the frequency of revertant colored versus colorless derivatives (cont.)

In a previous report (MNL 60), the discrepancy between the frequency of colored vs. colorless derivatives of this allele was described. With the revelation that the *Spm* of this autonomously mutating allele lies in the promoter region support is provided that excisions in the promoter region are such that colored derivatives are more likely than colorless. This would indicate that possibly excisions leading to base pair alterations in the promoter region are more likely to be tolerated and thus yield wild type products despite a change in the fidelity of the gene sequence as opposed to those changes in the structural part of the *C2* gene.

Peter A. Peterson

Deficiency *C-Sh-Bz-857003*

In an isolation plot of *C-I Sh Bz Wx En* × *En C sh bz wx* (Cross 1), a kernel with a shrunken, bronze phenotype was isolated. This would appear to be a deficiency for *C-I Sh Bz*. There is no male transmission of this chromosome segment. In crosses of this deficiency by *c-m(r)*, the resulting kernels are colored with some colored to colorless sectors. This mutation deleted shrunken and bronze action as well as that of *C-I* but the colored phenotype from the *c-m(r)* cross indicates that this *C* can complement with the *c-m(r)* allele. The colorless sectors are either a reversion to *C-I* or a loss of the *C* component.

Peter A. Peterson

wx-857027

Another mutant from Cross 1 (preceding report) was *wx-857027*. Though a number of *En*-related *wx* mutants have appeared in these crosses, *wx-857027* does not respond to *En*. This is verified by its non-mutability and that it does carry *En* based on a transactive test with an *a-m(r)* allele that is responsive to *En*. This *wx* is not a *wx-m(r)* allele.

Peter A. Peterson

C-Im857101 - an unstable *C-I* allele responsive to *En*

This is an *En*-responding *C-I-m* allele arising from a population as in Cross 1 (preceding report). This conclusion is based on results from crosses of *C2/c2-m2, C wx-m8* × *C2, C-Im857101 Wx/C-Im857101 wx*. Colorless

(or palish) and sectored (colored to colorless) were isolated. Of the 28 *wx* sectored kernels tested, all showed *wx* to *Wx*. This indicates that the *C* to *C-I* sectoring is controlled by *En*.

Peter A. Peterson

a2-m55064 - an autonomously mutating *a2* allele

In crosses such as *a2-m55064 Bt/a2 bt* × *a2 bt*, an excess of colorless-round kernels is found. This indicates either one of two sources of these kernels. Either this *a2-m* is controlled by several segregating *En* (3 or 4) or this *a2-m* is autonomously mutating and gives rise to a high frequency of colorless derivatives. Typical data are given in Table 1. A means of distinguishing between these two options is to test the colorless-round segregants (*a2 Bt/a2 bt*) that appear in greater than expected frequency. In tests of five colorless-round progeny of crosses similar to 86 4450-21 × 5134 (Table 1), none contained an active *En* as determined from a test with the *En* tester *a2-m(r)*. In another test of a sib family that arose from *A2 Bt/a2-m55064 Bt* × *a2 bt*, none (8/8) of the colored-round progeny from this cross showed the presence of *En*.

Table 1. From a testcross of fine types *a2-m Bt/a2 bt* (fine) × *a2 bt/a2 bt*) both fine and coarse types appeared in the progeny. Both these sib types, coarse (864450) and fine (864452) were testcrossed and the progeny types are given (the colorless brittle progeny are not given).

		Round			
	Coarse	fine	coarse	colored	colorless
86	4450-21 x 5134	0	125	11	34
	-23 x 5134	3	147	4	15
	Fine				
86	4452-24 x 5133	62	64	3	11
	-25 x 5133	43	39	5	12
	-27 x 5133	101	100	1	13

Table 2. Testcross of fine types - *a2-m Bt/a2 bt* (fine) × *a2 bt/a2 bt.*

		Round			
		fine	coarse	colored	colorless
86	4449Y-24 x 5134	26	25	0	4
	-25 x 5134	61	77	7	11
	-27 x 5134	71	74	0	7
	-28 x 5134	120	101	4	15

Another feature of this allele is the presence also of a "fine" factor. This "fine" factor suppresses the early mutability of the *a2-m* allele in giving a "fine" pattern (late and many). Further, this same suppressive feature of the "fine" factor manifests itself in a lower incidence of colored and colorless derivatives among testcross progenies of plants arising from "fine" kernels. This is evident in both Tables 1 and 2.

It is evident that the fine factor segregates independently and affects the mutability of the autonomously mutable *a2-m*. Note also in Table 1 (the 4450-23 progeny) that fine types arise out of progeny of the coarse testcross.

Peter A. Peterson

a2-m668140 - a basic pale allele that is modified in mutability expression by a fine factor

This mutable allele is transactivated by an independently segregating *En*. In the absence of *En*, this allele shows a light pale phenotype in the aleurone. With *En*, the kernel phenotype is a coarse, early mutating type with a colorless background. This indicates that this allele is *a2-m(r-pale)* responding to *En* to yield coarse mutability. In backcross progeny kernels with [*a2-m(r-pale) Bt/a2 bt* × *a2 bt/a2 bt*] a changed mutable phenotype arises that has a very late fine mutability pattern. In further tests of these exceptional kernels, a single factor modifying the coarse expression of the *a2-m(r-pale)* can be identified. The segregation pattern indicates that the "fine factor" (F) does not have the transactive *En* excision function but can modify the *En* that causes the coarse expression. The hypothesis for this series of interactions with *a2-m(r-pale)* is as follows:

En	F	Phenotype
+	+	= fine mutability
+	-	= coarse mutability
-	+	= pale - no mutability
-	-	= pale - no mutability

The F factor is similar in activity to other modifiers, as *En-Malt*.

Peter A. Peterson

En-controlled *I* element inserts with the *C-I* allele: *C-Im857059* and *C-Im857062*

C-Im857059 was uncovered in an isolation plot containing the cross *C-I Sh Bz Wx/C-I Sh Bz Wx En* × *C sh bz wx/C sh bz wx*. This mutant is unstable and has an *I* insert that is under the control of *En*. This was proved in several ways but one example will be given. Sectored kernels were crossed by a tester containing *c2-m2* and *wx-m8*, alleles responsive to *En* or *Spm*, *C-Im Sh Bz Wx/C sh bz wx* × *c2-m2 wx-m8*, and sectored kernels obtained from this cross were again crossed by *c2-m2 wx-m8*. Colored, sectored, pale, and colorless kernels are obtained among the progeny and are tested for the *wx* phenotype. The *wx* mutability is from *wx-m8* with *En*.

The colored are from the *C Sh Bz wx-m8* chromosome. The sectored and pales are from the other chromosome (*C-I Sh Bz Wx*) with the *wx* phenotypes arising from crossovers. All the sectored show *wx* mutability; none of the pales show *wx*-mutability (Table 1).

Table 1. Test of *En* control of *C-Im857059:* Progeny types from cross tested for *En* presence with the *wx-m8* allele.

	Colored			Sectored			Pale			ci		
Ear	Wx	wx	wx to Wx	Wx	wx	wx to Wx	Wx	wx	wx to Wx	Wx	wx	wx to Wx
#1	64	60	67	86	1	18	49	57	0	7	0	0
#2	75	27	87	97	0	26	23	21	0	5	0	2
#3	78	32	104	140	0	44	25	24	0	5	1	3

The conclusion from this analysis is that the pales represent the *C-Im(r)* allele without *En*. The sectored are the same allele with *En*. Thus, the pales are suppressed somewhat in their color-suppressive capacity and these represent an *I* insert in the *C-I* allele responding to *En* but not completely abolishing the suppressive effect of *C-I*. In summary,

Pale = *C-Im(r)/C* no *En* (none show *wx*-mutability)
Sectored = *C-Im(r)/C En* (Excisions release the modification of the color suppressing potential); all show *wx*-mutability.
Colorless = *C-Im(r)/C-I-m(r)* ± En

Peter A. Peterson

C-Im857062

This is also an *En* controlled *C-Im(r)* allele. When sectored kernels were crossed with the *a1 En*-tester,

a-m(r)/a-m-1, sectored kernels were obtained and again crossed by *a-m(r)/a-m-1*, yielding sectored and colored progeny. When these were separated and again tested, the results of these tests were:

	With *En*	No *En*	Total
Sectored	11	0	11
Colored	0	10	10

This allele as judged from tests on the *En*-tester responds to *En*. Both alleles are *En*-responsive and represent *I* inserts into *C-I*.

Ch. Jayaram and Peter A. Peterson

The *Uq* content of several breeding populations

Several maize breeding lines and populations were screened for the transposable element *Uq*. An isolation plot was used in which the lines of interest (assumed to be *c/c*) were detasseled and pollinated by a *c-ruq* tester. Resulting ears on the detasseled plants were heterozygous for *c* and *c-ruq* and the presence, absence and number of *Uq* could be determined. The following populations contained *Uq* elements:

Hays Golden (Gardner, C.O., 1976. In Proc. Int. Cong. Quant. Genet., E. Pollak et al., eds., Iowa State Univ. Press. Pp. 475-489).

Iowa Long Ear (Cortez-Mendoza, H. and A.R. Hallauer, 1979. Crop Sci. 19:175-178).

Iowa Stiff Stalk Synthetic (Hallauer, A.R., W.A. Russell, and O.S. Smith, 1983. Stadler Symp. 15:83-104).

BS11 (Hallauer, A.R., Personal communication).

Lancaster and Kolkmeier (Walejko, R.N., and W.A. Russell. 1977. Crop Sci. 17:647-651).

Illinois Oil and Protein Lines (Dudley, J.W. 1976. In Proc. Int. Cong. Quant. Genet., E. Pollak, et al., eds., Iowa State Univ. Press. Pp. 459-473).

Jean Cormack and Peter A. Peterson

Test for presence of controlling elements in populations

Tests were conducted to evaluate the presence of controlling elements in 'Iowa Stiff Stalk Synthetic' (BSSS) population, the original inbred lines for BSSS population, inbred line derivatives of BSSS population, and Lancaster populations of different sources.

Mrh element was the most prevalent among the original inbred lines. It was also exposed in two cycles of selection, however, it was not recovered in the derivative inbred lines.

Cy element expressed low activity among original inbred lines that exhibited mutability. This pattern was carried through two cycles of selection and did not appear in the inbred line derivatives of BSSS population.

Uq element was uncovered in only one original inbred line and was channelled through cycles of selection and inbred line derivatives of BSSS population. The failure to uncover the elements *Mrh* and *Cy* in two cycles of selection could be due to sampling or those elements were lost during the recombination process.

Among nine Lancaster populations tested for the presence of elements, six exhibited *Uq* mutability, one the *Cy* element, one the *En* element, and none with the *Mrh* element.

From these results the *Uq* element was the most prevalent in BSSS and Lancaster populations.

Missae Karasawa and Peter A. Peterson

Ten mutant alleles at the *C* locus: complementation potential

It is believed that two different alleles which otherwise lack the potential to produce color by themselves, might complement each other and will be able to produce color in the aleurone tissue of maize. According to this proposal for complementation, each of these two different alleles would be expected to produce a partial transcript because of the placement of the transposable element insert in their exons, which thereby would contribute to the production of a complete gene product.

In the present study 10 different mutable alleles at the *C* locus were used. Nine have either *En* or *I* of the *En* system and the tenth has the *ruq* of the *Uq* system. The alleles are *c-m611702, c-m641963, c-m655208, c-m655292, c-m655320, c-m641936, c-m655437, c-m655370, c-m641905* (all *En* mutants) and *c-m816666* (*Uq* system). All have a spotted phenotype in a null (colorless) background. (Other *c*-mutable alleles which have the same phenotype but do develop color during germination, in the light, have not been included in the diallelic crosses because the present study was aimed at observing whether there is any color development and thus complementation).

Kernels of the above ten *c*-mutable alleles were crossed to each other to create 45 different diallelic combinations (Figure 1). Approximately 20 kernels from each of these combinations have been germinated under light to observe color development in the aleurone. Very little color developed. Only 15 kernels developed a light color while the rest were without color. Further studies are in progress to confirm the above observations.

Figure 1. Color development in developing seedlings among 10 mutable alleles in a diallel series. A, B, C, etc. represent the different alleles.

	c-m	A	B	C	D	E	F	G	H	I	J
	En-I/En										
A	61 1702	-	±	-	*	-	sip	-	-	-	-
B	64 1963	-	-	-	-	-	-	-	-	*	-
C	65 5208	-	-	-	sip	-	-	-	-	-	sip
D	65 5292	sip	-	sip	-		-	±	-	-	sip
E	65 5320	-	sip	-	-	-	-	-	±	sip	sip
F	64 1936	-	sip	-	-		-	-	-	-	sip
G	65 5370	-	sip	sip	-	sip				*	
H	64 1905	sip	-	-	sip	-	-	±	-	±	-
I	65 5437	-	-	-	-	*	-	*	-	-	-
	Uq										
J	81 6666	-	sip	-	-	-	-	-	sip	-	-

Legend:

- \- = no color development
- * = light color development
- ± = kernels from one ear show color while from other of the same cross was colorless. This would be an inconclusive result.
- sip = Study in progress

Each diallelic type was crossed (reciprocally).
For ex A x B
B x A
50 45 x 2 = 90

C-Im846079: a highly mutable *C-I* allele

A highly unstable mutant allele, *C-Im846079*, has been rescued from a mottled-like kernel isolated from a population containing a *Cy* transposable element.

Results from confirmation tests and further genetic tests have indicated that the exceptional mottled-like phenotype showing heavy sectoring of colored cells in a colorless background is heritable. It is controlled by the mutant allele *C-Im846079*, very likely under an autonomous mode.

Yong-Bao Pan and Peter A. Peterson

A survey of active *Uq* elements in maize inbred lines by the use of *c-ruq* tester

The *c-ruq* tester was used to probe active *Uq* element in 11 inbred lines and 3 waxy isogenic lines. These lines are Hy(*Wx*), Hy (*wx*), M14(*Wx*), M14(*wx*), B14(*Wx*), B14(*wx*), B37, B73, B78, B80, B70, C103, C123, and 187-2. They were known to be homozygous for the recessive *c* allele. Examination of the progeny kernels of these inbred lines crossed with the *c-ruq* tester has shown the following results: 1) All these inbred lines lack an active *Uq;* 2) The *c-ruq* allele does not "slip" at all in any of the 34,631 colorless kernels derived from 158 reciprocal crosses. This is in contrast to the situation with *a-ruq* that generally gives rise to 1-spot or sector of spotting at a frequency less than 1% (Peterson and Friedemann, Maydica 28:213-249, 1983; Pan and Peterson, MNL 60:5-6, 1986). This suggests that the occasional *Uq* activity observed in these two studies might originate from the *ruq* receptor element at the *A* locus.

Yong-Bao Pan and Peter A. Peterson

Activation of *Uq* transposable element is likely a random event

In order to understand whether the phenomenon of *Uq* activation (*85 sectored*) (MNL 60:5-6) was a random event or whether there was a specific factor in the genomes of sectored kernels that stimulated *Uq* activation, the following crosses were tested.

The F1 and BF1 progenies of the 4 inbreds that had been tested previously by the *a-ruq* tester for *Uq* activity were crossed both by *85 sectored* genotypes(homozygous for *R*) and onto *a-ruq* tester. In addition, 187 plants from *85 sectored* and 67 plants from *85 colorless* sib kernels were crossed by *a-ruq* tester. If the activation of *Uq* is not a random event, we would expect a higher frequency of generating sectored kernels in crosses involving the *85 sectored* than involving *a-ruq* tester due to the enhancement effect of the factor selected. On the other hand, if the process of *Uq* activation is random, no difference would be found between the two types of crosses.

Results from this study have indicated the following: a) The *Uq* activity seen in sectors of spotting in the aleurone layer of the 187 kernels was not transmitted; b) Apparently lower frequency of sectored kernels was seen in (F1 or BF1 of B70, C103, C123) × *85 sectored* than onto *a-ruq* tester. Frequency of sectored kernels in F1 or BF1 of 187-2 by *85 sectored* was slightly higher or the same than onto *a-ruq* tester; and c) Similar frequencies of sectored kernels were observed in progenies derived from crosses by *a-ruq* tester of *85 sectored* and of *85 colorless* sibs. All these suggest that the activation of *Uq* transposable element is likely a random event.

This hypothesis is supported by the following observations. First, colorless kernels with very large area of sectoring did not produce a higher frequency of sectored kernels when crossed with *a-ruq* tester. Second, 3 sectored kernels were found in a population of 2,458 colorless kernels from crosses between *a-ruq* and *a sh* (no *Uq*) testers. Third, sectors of spotting in an otherwise colorless aleurone were very often encountered in the development of an *a-ruq* tester with *sh* and *bz* markers.

Yong-Bao Pan and Peter A. Peterson

Mutant *C-ID846627* had a deficiency distal to *C-I*

One variegated kernel was recovered from an *En* isolation plot (*C-I Sh Bz Wx/C-I Sh Bz Wx, En/En* × *C sh bz wx/C sh bz wx*) that showed sectors of color or bronze in a colorless background. Later it was found the mutant had a deficiency and the *C-I* chromosome carrying the deficiency was not transmissible through pollen. This mutant has been designated *C-ID846627*.

Table 1. Linkage test on mutant *C-ID846627*.

Plant number	Classification of progeny kernels: shrunken bronze	shrunken colorless	shrunken colored	round bronze	round colorless	round colored	Total
1. by Cshbzwx tester:							
84g200-1	49	1	0	0	52	3	105
-2	133	2	1	0	131	1	268
-3	66	0	0	0	28	1	95
Grand total	248	3	1	0	211	5	468
2. on Cshbzwx tester:							
84g200-1	308	0	4	0	5	12	329
-2	314	0	2	0	4	1	321
-3	287	0	0	0	2	6	295
Grand total	909	0	6	0	11	19	945

A preliminary linkage test was done and the results shown in Table 1 indicate that the deficiency probably starts at about 1.2 map units distal to the *C-I* locus. However, this value is only based on a few crosses on 3 plants. An additional study involving adequate numbers of plants is underway.

Yong-Bao Pan and Peter A. Peterson

R mutant alleles from mobile-element-containing populations

Thirty-seven *R* mutant alleles have been isolated from several maize populations containing different types of mobile elements. They can be divided into the following 3 groupings based on the results derived from confirmation tests and tests for genetic instability:

1. Genetically stable *R* mutants
   a) Those from *Uq*-containing populations: *r-784201, r-784214, r-817106, r-817113, r-817120, r-817123, r-817206, r-817371,* and *r-817382;*
   b) Those from *Cy*-containing populations: *r-846055, r-846058, r-846066, r-846067, r-846068, r-846074, r-846085, r-846095, r-857332, r-857336,* and *r-857343;*
   c) Those from *En*-containing populations: *r-826143, r-857299, r-857305,* and *r-857325;* and
   d) Those from *Ac*-containing populations: *r-844523, r-846142,* and *r-846155.*
2. Putative mutable *R* mutants: *r-826013, r-826014, r-826016, r-826017, r-826022,* and *r-826026.* These were isolated from one *En*-containing population.

3. Promising *R* mutable alleles
   a) One from *Uq*-containing populations: *r-846096;*
   b) Three from *Cy*-containing populations: *r-857345, r-857349,* and *r-857350.*

Further experiments are in progress on putative and promising *R* mutable alleles.

Yong-Bao Pan and Peter A. Peterson

Induction of *Uq* activity and a *mn*-type mutant by 5-aza-2′-deoxycytidine

DNA modification, particularly methylation, has been found to be inversely correlated with gene activity (for review, see Doerfler, Ann. Rev. Biochem. 52:93-124, 1983). In maize, the loss of *Mu1* activity is due to an increasing level of DNA methylation of the element (Chandler and Walbot, PNAS 83:1767-1771, 1986). Our finding on the activation of the *Uq* transposable element in somatic tissues of progeny kernels of four inbreds lacking *Uq* activity (MNL 60:5-6) has raised such a question as to whether the activated *Uq* comes from the *ruq* receptor element at the *A* locus or whether it is a methylated inactive *Uq* that becomes active.

One experimental approach to address this question is to use the deoxycytidine analog 5-aza-2′-deoxycytidine. The roots of germinating seedlings from 34 sectored BF2 progeny kernels of the 4 maize inbreds were dipped into a 30 uM 5-aza-2′-deoxycytidine solution for 72-90 hours, transferred into seed pots in the greenhouse, and grown for another 2 weeks before transplanting into the field. During this period, death of some root tips and folding of the leaves were common. Four seedlings died of the treatment, the rest were recovered and looked normal. These plants, together with 15 plants derived from the same source as a control, were crossed with *a-ruq* tester reciprocally. Examination of the progeny ears gave rise to the following results: One treated plant was totally sterile; another treated plant had one *Uq* element being activated that appeared to cosegregate with a *mn* type mutant gene, designed as *mn-866248U.* It was not transmissible via pollen:

Ear	Standard			Miniature			Total
	cl	spotted	1-spot	cl	spotted	1-spot	
866248U/*a-ruq*	129	0	3	3	143	1	279
a-ruq/866248U	322	0	6	0	0	0	328

A similar event of *Uq* activation happened in a single kernel at a frequency of about 6.1×10^{-6} in a separate experiment without any treatment. When the plant 866201X derived from this kernel of *Uq* spotting was crossed by an *a-ruq* tester, approximately 50% of the progeny kernels were miniature and *Uq* spotted and the other 50% were normally sized and not *Uq* spotted. However, tests are not available on its transmission through pollen. This *mn* mutant gene is named *mn-866201X.*

These two events are unrelated by immediate parental source and therefore represent two independent events.

Yong-Bao Pan and Peter A. Peterson

Preliminary linkage data for recessive brown aleurone (*brn1*)

As has been previously reported (MNL 60:6-7, 1986), *brown-1* (*brn1*) is a recessive brown-kerneled seedling lethal mutant located on the short arm of chromosome 3. We have now collected linkage data with respect to *d1, Lg3,* and *cl1*, which would suggest that *brn1* is located in the vicinity of *cr1* on chromosome 3. Our linkage data are reported in Tables 1 and 2. All crosses were set up to test linkage in repulsion.

Table 1. Linkage data for *brn1 d1 Lg3*, from the testcross + *d1 Lg3* / *brn1* + + × + + +.

	Parentals				Recombinants			
Family	+ d L	b + +	b + L	+ d +	b d L	+ + +	+ + L	b d +
1216	11	16	2	5	3	5	0	3
1217	16	12	3	4	8	2	0	1
1218	13	11	5	6	2	2	0	1
1219	18	13	3	3	2	5	0	0
1220	17	13	5	4	5	10	0	0
1221	15	15	6	2	1	4	1	1
1222	16	10	5	2	4	5	0	0
1223	17	10	3	6	4	2	0	0
1605	13	11	3	4	2	1	0	0
1606	3	16	5	5	2	3	0	1
Totals	139	127	40	41	33	39	1	7

% Recombination brn1-d1 = 18.7 ± 1.9
% Recombination brn1-Lg3 = 39.6 ± 2.4
% Recombination d1-Lg3 = 20.8 ± 2.0

Table 2. Linkage data for *brn1 - cl1.*

Testcross: (brn1 Cl1 / Brn1 cl1) × brn1 brn1 cl1 cl1

Family	brn1-Cl1	Brn1-cl1	Brn1-Cl1	brn1-cl1
1232*	29	3	13	1
1233*	3	36	2	11
1234	29	0	17	1
1235	0	25	0	20
1236	25	1	21	0
1237	2	28	0	13

Testcross: (brn1 Cl1 / Brn1 cl1) × brn1 brn1 Cl1 Cl1

Family	brn1-Cl1	Brn1-cl1	Brn1-Cl1	brn1-cl1
1238	14	13	4	10
1239	18	8	10	7
1609	11	17	7	7
1610	8	17	8	8
Totals	139	148	82	78

n = 447

% Recombination = 35.8 ± 2.3

* Families 1232-1237, even numbered families, yellow seeds planted. Families 1232-1237, odd numbered families, pale yellow seeds planted.

In order to test linkage with *d1* and *Lg3*, we crossed a *d1 Lg3* stock onto the first ears of heterozygous *brn1* plants whose second ears had been selfed the day before. Kernels from the first ears of plants whose selfed second ears segregated for *brn1* were planted the next season, and the *Lg3* plants were selfed and outcrossed to a standard line carrying the wildtype alleles. Kernels from the outcrosses of plants which were liguleless, and whose selfs segregated for *d1* as well as *brn1*, were planted in our selfing block, scored for *Lg3*, and self-pollinated. The resulting ears were scored for *brn1*, and kernels from each ear were planted in sand benches and the seedlings scored for *d1*. The data collected from this linkage test are presented in Table 1.

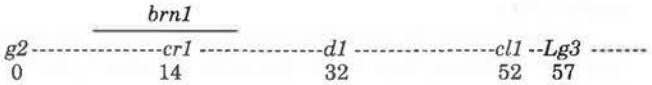
To test linkage with *cl1*, heterozygous *brn1* plants were selfed and outcrossed to homozygous *cl1 cl1 Cl3 Cl3* plants. Kernels from these crosses were planted, and the plants selfed and outcrossed to either (1) *cl1 cl1 Cl3 Cl3* plants, or (2) a standard line carrying the wildtype alleles. Kernels from the outcrosses of plants whose selfs segregated for *brn1* and *cl1* were planted in our selfing block and the plants were self-pollinated. For cross (1), the kernels planted in the selfing block were first separated on the basis of whether they were yellow (*Cl1 cl1*), or pale yellow (*cl1 cl1*), before they were planted. This is the basis of the data reported in the top half of Table 2. It should be noted that a fair amount of heterofertilization occurred (about 5%), but this was readily recognized in the selfed ears, and

did not influence the linkage results. For cross (2), the kernels were simply planted in rows, since all kernels were yellow. The data for cross (2) are reported in the bottom half of Table 2.

The data from the three point cross with *d1* and *Lg3* (Table 1) would place *brn1* at around 19 units from *d1*, and 40 units from *Lg3* on chromosome 3. The data from the two-point cross with *cl1* (Table 2) place *brn1* at around 36 units from *cl1*. Thus, on the linkage map of chromosome 3, *brn1* would be placed at position 13 (with respect to *d1*), position 17 (with respect to *Lg3*), or position 16 (with respect to *cl1*). These positions map very closely to *cr1*, which is located at position 14. Since *d1* is located closer to *brn1* on chromosome 3 than are *Lg3* and *cl1*, the placement of *brn1* at 13 units is probably the most accurate. We will be collecting more linkage data with respect to *d1*, as well as *g2* and possibly *ra2* within the next year.

We attempted to map *brn1* with respect to *cr1* and *d1* in a three point cross according to the following scheme: Homozygous *cr1 cr1 d1 d1* plants were crossed onto the first ear of a heterozygous *brn1* plant whose second ear had been selfed the day before. Kernels from the first ear of plants whose selfed second ears segregated for *brn1* were planted the next season, the second ears selfed, and the first ears pollinated by a homozygous *cr1 cr1* stock obtained from the Maize Genetics Stock Center, which was indicated as being homozygous wildtype for *d1*. The kernels from the first ears of plants whose second ears segregated for *brn1* and *d1* (*cr1* could not be scored in the seedling bench) were planted in our selfing block, the plants grown to maturity and scored for *cr1*, and the plants self-pollinated. The selfed ears were to be scored for *brn1*, and planted in the seedling bench to be scored for *d1*. As it turned out, the plants in the selfing block segregated for a dwarf, as well as crinkly, indicating that the *cr1* stock used in the second generation cross was heterozygous for *d1*. In addition, the crinkly trait was nearly impossible to score because there was a continuous range of variation in the plants from very crinkly to very smooth leaves. The *cr1* stock also was in a purple background, which made the selfs difficult to classify for *brn1*. We will try to obtain a different *cr1* stock, hopefully in a colorless aleurone background, in order to complete the linkage tests. We would be very grateful for advice on classifying for the crinkly trait.

The following diagram indicates the location of *brn1* on chromosome 3, based on our present linkage data:



Philip S. Stinard

Dappled: a putative *Mu*-induced aleurone developmental mutant

All *Mu*-induced mutable aleurone genes found to date in our studies with *Mu* have had a very characteristic spotting pattern of small spots, indicative of late events. Although there is some variation from mutant to mutant in the size of the spots, they are nonetheless always the result of late events. Large, irregular pigmented spots, which would be expected from early events, are rarely observed.

From our 1979 *a2 bt* × purple Mutator isolation plot, we recovered many seeds that had sectors of purple and yellow aleurone. These seeds did not have the typical *Mu* aleurone mutable pattern, but instead had varying sectors of purple and yellow. A couple hundred such seeds with different amounts of sectoring were planted in 1982 and self pollinated. All but a very few segregated for purple *Bt* and yellow *bt* seeds. Thus, the original sectoring was probably due to chromosome loss or somatic mutation within the endosperm. However, one plant from a sectored seed segregated for a mutable aleurone pattern we have called dappled (*Dap*). This mutable pattern is not typical of previous *Mu*-induced aleurone mutants. *Dap* seeds are yellow with sectors of purple tissue of variable sizes and shapes (Figures 1 and 2). *Dap* seeds are frequently smaller than their purple sibling seeds, and are sometimes extremely defective (Figure 2). There appears to be an

Figure 1. Close-up of seeds near the base of a dappled ear. Note that there are large and small *Dap* seeds, and that there appears to be an inverse relationship between seed size and the amount of mutant tissue present. Very small, defective *Dap* seeds are not seen in this picture because they are usually overgrown by more fully developed seeds.

Figure 2. Close-up of dappled seeds.

imperfect correlation of this defective phenotype with the extent of yellow areas on the seeds. The greater the amount of yellow tissue, the smaller the seed. Frequently, the region of the ear that matures first will have a higher concentration of dappled seeds than the tip of the ear (Table 1); these dappled seeds have more yellow tissue

Table 1. Distribution of dappled seeds on ears from heterozygous dappled plants.

Dappled parent	parent	Seeds from the base* half of the ear			Seeds from the tip* half of the ear			Total %
		P1	Dap	% Dap	P1	Dap	% Dap	Dap
633 - 1	P1 aleur	84	56	40.00	218	32	12.80	22.56
- 2	"	57	44	43.56	85	47	35.61	39.06
- 3	c1	34	8	19.05	43	22	33.85	28.04
- 5	P1 aleur	84	61	42.07	169	35	17.16	27.57
- 6	"	84	41	32.80	145	68	31.92	32.25
- 7	"	80	63	44.06	154	33	17.65	29.09
634 - 1	"	57	22	27.85	59	35	37.23	35.95
- 2	"	42	22	34.38	76	20	20.83	26.25
- 3	"	41	28	40.58	106	41	27.89	31.94
- 4	"	43	7	14.00	85	66	43.71	36.32
- 5	a1	41	29	41.43	184	46	20.00	25.00
- 6	P1 aleur	71	5	6.58	98	53	35.10	25.55
- 7	"	80	10	11.11	129	71	35.50	27.93
- 8	"	78	62	44.29	135	51	27.42	34.66
635 - 1	"	97	88	47.59	159	88	35.63	40.74
- 2	a1	37	43	53.75	102	66	39.29	43.95
- 3	P1 aleur	98	76	43.68	192	41	17.60	28.75
- 4	c2	66	41	38.32	137	47	25.54	30.24
- 6	a1	48	30	38.46	79	15	15.96	26.16
- 7	"	102	75	43.37	145	50	25.64	33.60
Totals		1324	811	37.99	2500	927	27.05	31.25

* The ears were divided in half on the basis of ear length, not on the basis of the number of seeds. Because seed set was frequently poorer in the basal half of the ear than in the tip half (see text for explanation), the tip half consistently has more seeds.

than those at the tip of the ear. In most cases where this pattern is found, there is irregular seed set in this region of the ear of the type seen on a pollinated ear in which the silks have passed the prime time for pollinating. However, this is probably not the explanation for most of the ears observed in our tests because most were pollinated within a couple of days of first silking. Because seeds with very little purple tissue are very defective, it may be that seeds without any purple sectors abort. Such seeds would be more likely in this region of the ear where the dappled expression is more intense.

It was first assumed that dappled was a mutable allele of *a2* because it was observed in an *a2 bt* × purple *Mu* cross. However, to rule out the possibility that another aleurone locus was involved, it was tested against the aleurone testers for *a1, a2, c1, c2,* and *r* loci. These tests were made reciprocally. At the same time, *Dap* stocks also were crossed reciprocally to purple aleurone lines. The plants that were used in these crosses were from *Dap* seeds. All crosses in which *Dap* plants were used as males gave nothing but purple seeds. (Two sectored seeds from male outcrosses to *a2* testers were found, but when they were planted and test crossed, they turned out not to be dappled.) However, all crosses where *Dap* plants were used as females segregated for *Dap* seeds, even the crosses with purple aleurone. Thus, *Dap* seems to behave as a dominant trait in plants crossed as females.

Although dappled is not expressed in male outcrosses, it is male transmissible, though in a reduced frequency. Last summer, we planted 10 seeds from each of 15 different male outcrosses of dappled to purple aleurone, and selfed these plants (Table 2). Had male transmission been complete, half of the plants would have segregated for dappled seeds (the male parents were heterozygous). The reduced frequency of dappled ears observed (31%) was significantly lower than 50% ($p < .01$). This summer, we will test a larger number of crosses in which *Dap* plants are used as males, to obtain a better estimate of its rate of transmission through the male.

Table 2. Distribution of dappled ears in male outcrosses of heterozygous dappled plants to purple aleurone.

Family	Female Parent	Dappled Parent	Number of Dap ears	Number of + ears	Total
1386	834-5	640-1	3	5	8
1387	832-5	634-4	4	4	8
1388	833-1	635-3	0	8	8
1389	833-2	634-7	2	8	10
1390	833-4	633-6	2	8	10
1391	833-6	633-4	4	6	10
1392	833-8	633-1	1	8	9
1393	833-9	633-3	4	6	10
1394	833-10	633-5	2	6	8
1395	834-8	634-6	1	7	8
1396	834-2	633-7	4	4	8
1397	834-3	634-2	3	6	9
1398	834-4	636-2	4	5	9
1399	834-1	636-1	3	7	10
1400	834-9	634-3	5	5	10
Totals			42	93	135

Chi-squared for 1:1 ratio = 19.2667 ($p < .01$)
Heterogeneity Chi-squared for 1:1 ratio = 13.8681 ($p < .01$)

As mentioned above, expression of dappled in female outcrosses is reduced from the expected frequency of 50% (Table 1). Furthermore, there is a higher frequency of *Dap* kernels at the base of the ear than at the tip. These observations have two possible explanations: (1) Female transmission of *Dap* is reduced, and (2) for whatever developmental reasons, *Dap* is not expressed as extremely at the tip of the ear as it is at the base; thus, more of the kernels at the tip could be heterozygous, but the trait isn't being expressed. We will test both of these possibilities this summer by growing purple kernels from both the tip and base regions of these ears, and seeing if they carry dappled.

All plants from dappled seeds so far studied have been heterozygous, even those arising from seed from selfed *Dap* plants, which suggests that the gene is lethal in the homozygous condition. If further tests bear this out, there could be a very interesting dosage pattern regulating the expression of this gene (i.e., + + + and *Dap* + + = purple aleurone, *Dap Dap* + = mutant aleurone, *Dap Dap Dap* = lethal). On the other hand, the difference in expression between male and female outcrosses could be due to male vs. female transmission effects. We will investigate these possibilities at the earliest opportunity, and welcome suggestions for further experiments.

Under the dissecting microscope, the purple areas of dappled seeds appear to be raised above the yellow sectors. If the pericarp is peeled from the seed, the cellular structure in the central portions of the yellow areas appears amorphous and irregular. The purple sectors have normal-appearing aleurone, and the portions of the yellow areas that are immediately adjacent to purple sectors are normal, too.

Cross sections through the aleurone and adjacent endosperm of imbibed *Dap* seeds (paraffin-sectioned, stained with safranin and counter-stained with chlorazol black—prepared by PSS) reveal normal-appearing, intensely staining aleurone cells in the purple sectors (Figures 3-5). In the colorless sectors, several patterns of aleurone cell morphology are observed. In some instances, the aleurone cells are lighter staining, and elongated (about twice as long in the direction perpendicular to the seed surface as they are in directions parallel to the surface—Figure 3). In other preparations, the aleurone in the colorless sectors consists of several layers of small, irregularly-packed cells (Figure 4). In still other preparations, the colorless sectors have an aleurone consisting of a few "normal" aleurone cells irregularly interspersed with what appear to be

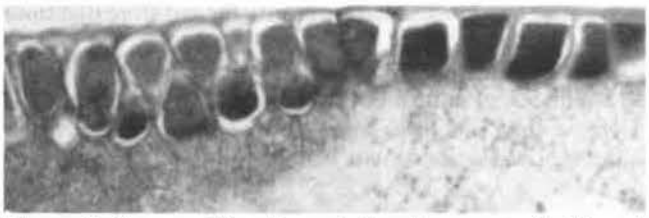


Figure 3. Cross section through the aleurone and adjacent endosperm layers of a dappled seed. The aleurone cells to the right are typical of normal aleurone cells, and are found in purple sectors, while the elongated aleurone cells to the left are from a colorless aleurone sector.

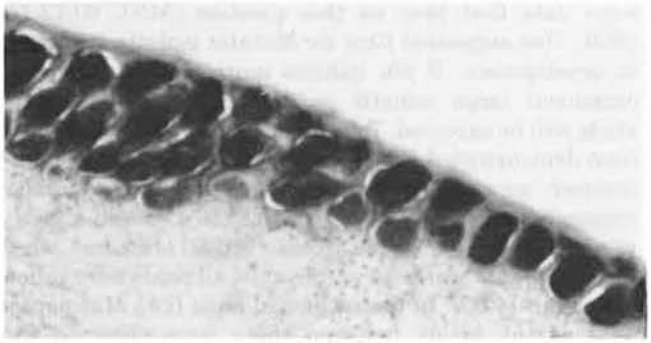


Figure 4. Cross section through a dappled seed demonstrating multiple aleurone cell layers in mutant tissues (to the left), and normal aleurone cells (to the right).

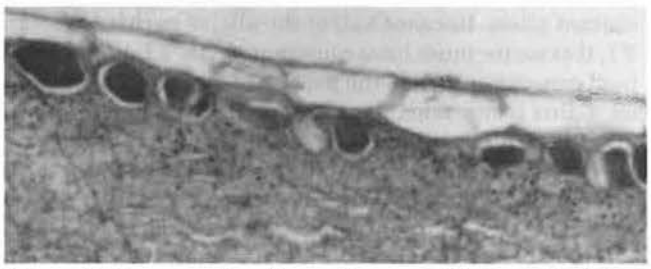


Figure 5. Cross section through a completely mutant dappled sector, showing regions missing aleurone cells.

starchy endosperm cells similar to the ones which normally underlie the aleurone (Figure 5).

Although some of these cell patterns may be the result of artifacts induced by our cytological technique, which we are still trying to perfect, it is nonetheless apparent that the *Dap* mutant is altering the normal development of the aleurone cells, and since the mutant cells are colorless, these altered cells have lost the ability to synthesize anthocyanin pigments. This alteration has more severe effects than just altering pigment synthesis as evidenced by the very defective state of seeds that are predominately yellow with very little normal aleurone tissue, and the total absence of completely yellow seeds. Whatever aleurone function is interrupted by this mutation, it appears to be essential for normal seed development. Thus, *Dap* is undoubtedly a developmental mutant.

Dappled seems to be a dominant mutable gene affecting the development of the aleurone. It has reduced transmission through the male (and possibly through the female as well), and may be dose-dependent in its expression.

Two additional mutants with aleurone patterns that superficially resemble dappled have been found in *Mu* aleurone color stocks. One arose from a cross of a purple *Mu* plant with an *a1 sh2* tester. This mutant is similar in both aleurone phenotype and transmission. It is not allelic to *a1*, and the sectored seeds are only found in female crosses and not in male crosses. It is probably allelic to *Dap*, but since we have not yet demonstrated this conclusively, we are calling this mutant *Dap*-3349*. The second mutant occurred in a *Mu*-induced bronze-1 mutable stock (*bz1-Mum8*). Plants from the sectored seeds were twisted, crinkly-leaf dwarfs with tassels having anthers that dehisce poorly. We are currently in the process of further characterizing this mutant (*Dap*-6143*), which appears to be expressed in both male and female outcrosses.

Philip S. Stinard and Donald S. Robertson

Some thoughts about the nature of *Mu*-induced *Y1* mutants

Previous studies of the genes affecting carotene synthesis in maize have indicated that many, if not all, of the same genes that are responsible for the carotene biosynthetic pathway in the leaves and other green parts of the plant are also involved in carotene synthesis in the endosperm as well.

My analyses of Guatemalan teosintes, which are perhaps the purest teosinte races, indicate that they are white seeded. Crosses with yellow-seeded corn as females give yellow F1 seeds and F2 ears that segregate 3:1 for yellow and white seeds. This white-seeded allele of teosinte is allelic to the standard *y1* gene of corn. If, as has been suggested by many, corn evolved from teosinte, early corn was probably white seeded. Thus the genes responsible for carotene synthesis were probably only "turned on" in the leaf but not in the endosperm of the primitive corn. Sometime in the development of modern yellow-seeded corn lines, corn acquired the ability to turn these genes on in the endosperm. Since yellow is dominant, it is likely that the genetic change that permitted carotene synthesis in the endosperm is involved in the regulation of this pathway. Perhaps a mutation of a site that binds an endosperm repressor of a gene regulating this pathway occurred. Thus in the white seeded progenitors of modern yellow-seeded corn, this repressor would turn off this pathway in the endosperm. However, a mutation in or near the site of repressor binding could prevent its binding and thus result in the turning on of the carotene pathway. Such a mutation would be a dominant. There are other possibilities that can be suggested that would result in an apparent dominant mutation (e.g., mutations of the locus producing the repressor substance resulting in a repressor that is no longer able to bind to the repressor site of the gene regulating the pathway).

The *y1* locus is the most likely candidate as the locus involved in this regulation because it is the locus that is responsible for the white-seeded condition of teosinte.

Over the last few years we have accumulated several hundred independent *Mu*-induced mutants at the *y1* locus. These all were originally isolated from crosses in which *Y1 Y1 Mu* stocks were used as either the male or female parents in crosses with *y1 y1 wx wx gl1 gl1* (or *gl8 gl8*) stocks.

Earlier studies (pre-Mutator studies) had revealed two classes of *y1* alleles: 1) Those in which the endosperm is white and the plant green and 2) Temperature sensitive *y1* alleles in which the endosperm is white but the plant is pale green (pastel) when grown at temperatures about

35C. These latter alleles give zebra type plants when grown in the field. To date we have tested 278 of the *Mu*-induced *y1* mutants and 71.94% have been the pastel type of allele.

A pastel *y1* allele found at the California Institute of Technology in the stocks from the post World War II atom bomb tests (*y1-wmut*) was also mutable. This allele had both mutable endosperms and mutable seedlings (plant). Because *Mu* is known to induce mutable mutants (e.g. at the *a1, a2, c2, bz1* and *bz2* loci), the *Mu*-induced *y1* mutants were screened for mutable endosperms. To date, no mutable endosperms have been observed. The percentage of mutable mutants varies from one Mutator cross to another. The last determination of the frequency of mutables was made in 1984. Of 395 seedling mutants scored, 193 or 48.86% were mutable. Thus the *Mu*-induced *y1* mutants, at first, seemed to be an exception in that none was mutable. When these *Mu*-induced *y1* mutants were seedling tested, however, 52.5% of them had mutable pastel seedlings. Thus these are not unlike the other *Mu*-induced mutants. Some indeed are mutable. Yet even those that have mutable seedlings do not exhibit endosperm mutability, even when the seeds are cut and scrutinized under a dissecting microscope. Characteristically, mutable *Mu*-induced seed mutants have very small revertant sectors. The *Mu*-induced waxy mutants have this pattern of mutability. In some of these, islands of one or a few cells scattered throughout the endosperm stain blue with the iodine stain in these mutant endosperms. If the same is true for the mutable *y1* alleles, it may be very difficult to recognize such isolated revertant cells in an otherwise white endosperm.

If the *y1* locus is indeed a gene involved in the regulation of the carotene pathway, how are the phenotypes of the *Mu*-induced mutants at this locus explained? The white-endosperm-green-plant alleles could be reverse mutations that restored the original regulation of the carotene pathway (i.e., off in the endosperm). For example, perhaps the presence of the *Mu* insertion changes the configuration of the DNA so that, whereas in the *Y1* allele the site of repressor binding was not available to the endosperm repressor, with the *Mu* insert present it now becomes available.

But how to explain the pastel alleles? The function of this allele in the endosperm seems to have been restored to the pristine condition but now its regulation in the plant is disturbed. Perhaps, for example, this locus has two regulatory receptor regions one involved in endosperm regulation and the other in plant regulation. The *Mu* insertion in the pastel allele may have restored function to the endosperm regulatory receptor but at the same time interfered with the normal function of the plant regulatory region.

The plant can tolerate a partial shutdown of the plant function of this gene. It seems, however, not to be able to tolerate its complete shutdown because no white-endosperm-albino plant allele of this locus has ever been found in the *Mu* studies or for that matter, in previous studies of this locus. Nearly all other known mutants in the carotene pathway have alleles that give albino seedlings. It may well be that the *y1* locus is involved in the regulation of another pathway (or other pathways) required for the life of the plant. If that is the case, then deletions which include this locus would not be viable. We are currently screening our *Mu*-induced mutants for putative deletions involving this locus.

Dr. David Morris is presently utilizing the mutable *Mu*-induced *y1* mutants in a program to isolate the *Y1* DNA so that this locus and its regulatory regions can be characterized molecularly.

Donald S. Robertson

A putative early *Mu*-induced mutation

The timing of germ line Mutator activity has been subject to considerable study. Last year, I summarized some data that bear on this question (MNL 60:12-14, 1986). One suggested time for Mutator activity was early in development. If *Mu* induces mutations at this time, occasional large somatic sectors containing a mutant allele will be expected. To date, no sectors of this type have been demonstrated for a known gene locus. However, last summer we made a large number of exact reciprocal crosses between *Mu*^{2} *Y1 Y1 Wx Wx Gl1 Gl1* and non-*Mu* *y1 y1 wx wx gl1 gl1* stocks. In one such set of crosses, when the *Mu*^{2} parent was used as a female, all seeds were yellow starchy (n = 65). In the reciprocal cross (i.e., *Mu*^{2} parent used as the male), however, there were observed 216 yellow starchy seeds and 106 white starchy seeds. Evidently two-thirds of the tassel shedding at the time this pollination was made consisted of a sector carrying a *y1* mutant allele. Because half of the alleles in this sector are *Y1*, this sector must have contributed 106 *Y1* alleles to the next generation. Thus, the portion of the tassel not included in this sector must have contributed the remaining *Y1* alleles (110). Since there are twice as many *Y1* alleles in this sector, it must be only half as large as the mutant containing sector. This mutant sector was not observed in the ear, thus, the mutation must have occurred after the cell lineages giving rise to the tassel and ear diverged but early enough in development to give rise to a significant portion of the tassel.

There is no way to determine by seed phenotype if this is a *Mu*-induced or a spontaneous mutant. We will test this mutant to determine if it is a mutable, temperature-sensitive, pastel allele. A positive result would suggest that this was *Mu* induced because most of the *Mu*-induced *y1* mutants are of this phenotype (see another report in this issue). Final determination that this is a *Mu*-induced mutant will depend upon its molecular characterization.

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Further evidence for Mutator activity in the male gametophyte and for differential activity of *Mu* in male and female germ lines

In last year's News Letter, we presented evidence that Mutator could induce mutants in the gametophyte (MNL 60:12-14, 1986). One line of evidence came from the frequency of discordant seeds (i.e., seeds in which the genotype of the embryo and endosperm differed) produced in reciprocal crosses. *Mu*-induced *y1* mutants produced when *Mu* plants were used as females had a much lower frequency of discordant seeds than *Mu*-induced *y1* mutants produced when *Mu* plants were used as males. To date, only white-seeded mutants have been scored for discordancy. There is a reciprocal discordant class expected (i.e., *y1 y1* embryo and heterozygous yellow endosperm).

This class is much more difficult to score, because it can not be distinguished by classifying the seeds but must be determined by scoring the ears on plants from the yellow seeds.

If mutants are induced during the development of the male gametophyte, it is possible to have pollen grains with one sperm carrying the nonmutated *Y1* allele and the other sperm with a mutant *y1* allele. If sperm from such pollen grains fertilizes an embryo sac of a *y1 y1* plant, half the time the sperm nucleus with the mutant allele will be included in the triple fusion product that produces the endosperm while the other sperm nucleus (with the nonmutant *Y1* allele) will unite with the egg nucleus, and a discordant seed with a white endosperm and a *Y1 y1* embryo will be produced. If the *Mu* plant is the female parent, however, mutations in the gametophyte would not be expected to produce many, if any, discordant white endosperm seeds. Such an event would require two independent mutations in the two cell lineages giving rise to the polar nuclei. Also, the mutation in the cell lineage that produces the egg nucleus would have to take place after the cell lineage giving rise to the polar nucleus separates from the one that produces the egg. Such a combination of events is very unlikely. In 1985, we reported a high frequency of discordant seeds in crosses involving Mu^2 plants as males and a low frequency of discordant seeds in crosses with female Mu^2 plants, as expected if *Mu* is active in the gametophyte.

Additional studies of this phenomenon (i.e., the different frequency of discordant seed in reciprocal cross) were carried out in 1986. As in the 1985 studies, *exact* reciprocal crosses were made betwen Mu^2 stocks and a *y1 y1 wx wx gl1 gl1* stock (a Mu^2 line is the progeny of the cross between two standard *Mu* lines). Mu^2 was used because of its high mutation frequency and the multiple *y1 wx gl1* stock was used so that contaminants could be recognized when this stock was used as a female parent. The results from the two years of crosses are seen in Table 1. There are over twice as many white seeds found in the crosses of Mu^2 as males than when the cross is in the reverse direction. Such a discrepancy is expected if gametophytic mutants are being produced.

Table 1. Reciprocal crosses of *Mu^2 Y1 Y1 Wx Wx* × *y1 y1 wx wx gl1 gl1*—Total of 1985-1986 experiments.

Mu^2 as ♀ parent			Mu^2 as ♂ parent		
White or pale yellow seeds	Total seeds	Freq. of white or pale yellow seeds	White or pale yellow seeds	Total seeds	Freq. of white or pale yellow seeds
50	250,736	1.99×10^{-4}	135	275,623	4.90×10^{-4}
		$X^2 = 32.9406$ $p = .01$			
Results expected if 34.29% (see text) of the y1 mutant seeds were discordant.					
50	250,736	1.99×10^{-4}	88.71	275,623	3.22×10^{-4}

To determine if putative discordant seeds, expected as a result of gametophytic *Mu*-induced mutants in the male gametophyte, could account for the difference in frequency of white seeds in these reciprocal crosses, 16 *y1* mutant seeds obtained from Mu^2 ears from 1985 crosses were planted and self-pollinated. All of the resulting ears were homozygous for the *y1*. Of thirty-five white seeds from the reciprocal cross (Mu^2 as male), twelve turned out to be discordant (i.e., these plants were *Y1 y1*) (frequency = 34.29%). Thus, we see that discordant seeds occur only in crosses in which Mu^2 was the male parent. These results are in agreement with those predicted for *Mu* activity in the male gametophyte.

The results so far obtained from the reciprocal crosses only test for one class of discordant seeds. The reciprocal class of yellow endosperm-homozygous *y1* embryo seeds cannot be scored. To do this we will plant a large sample of yellow seeds from these reciprocal crosses in an isolation plot and score for ears with 50% yellow seeds expected on a *y1 y1* plant in a field with predominantly *Y1 y1* plants. If mutations are being induced in the female gametophyte as well as the male gametophyte, they would be recognized as *y1 y1* plants produced by yellow seeds. If it is assumed that discordant seeds occur in the same frequency for the population summarized in Table 1 as it did in the 1985 reciprocal crosses, 46 of the 135 white seeds from the crosses of Mu^2 as a male would be discordant. If these 46 seeds are removed from the total of the white seeds, the frequency of *y1* mutants from the male Mu^2 crosses (i.e., 3.22×10^{-4}) now is closer to the frequency of *y1* mutants in the female Mu^2 crosses (i.e., 1.99×10^{-4}). This frequency, however, is still higher than that observed when Mu^2 plants are used as females and the difference is still significant at the 1 percent level. Thus the discordant seeds cannot account for all of the difference observed between the male and female crosses of Mu^2 plants. It appears that there are more germinal mutants induced when *Mu* plants are crossed as males than when they are crossed as females. This confirms previous observations on reciprocal crosses involving *Mu* plants (D.S. Robertson, Mol. Gen. Genetics 200:9-13, 1985).

In summary, there has been presented additional evidence that *Mu* can induce mutants in the male gametophyte. Additional tests will be necessary to determine if *Mu* is also active in the female gametophyte. Also, evidence is presented confirming earlier observation that more *Mu*-induced mutants are observed in the male progeny of a given plant than in the female progeny.

Donald S. Robertson

Mendelian ratios in crosses of mutable Mutator-induced *a1* mutants

Many mutable aleurone mutants induced by *Mu* have been obtained in our research program. These have included the loci of *a1, a2, c2, bz1*. To date, the *a1-Mum* mutants (*Mu*-induced mutable *a1* mutants) that have been studied in the greatest detail are *a1-Mum1, a1-Mum2* and *a1-Mum3*. These mutants all have the same pattern of mutability in that only small (late) revertant spots are observed. Considerable variation, however, in the intensity of spotting has been observed in both outcrosses and selfs of these mutants, varying from very intensely mutable to stable mutant phenotypes. The following scale is used in classifying seeds for intensity of mutability: Class 1 = stable, Class 2 = low mutability, Class 3 = medium mutability, Class 4 = high mutability and Class 5 = stable purple.

In the analysis of most of the early crosses of these mutable mutants, no discernible Mendelian ratios could be observed. However, as outcrossing continued with these

stocks seemingly Mendelian ratios occasionally were found. In the 1985-86 nursery, three outcross ears (two of *a1-Mum2*, and one of *a1-Mum3*), which segregated for putative 1:1 ratios in outcrosses to *a1 sh2* tester, were selected for further analysis. The percentages of stable seeds observed among the *a1-Mum* seeds on these ears were 47.51% (n = 301), 50.00% (n = 60), and 47.75% (n = 222). The first two crosses involved *a1-Mum2* and the latter was an *a1-Mum3* cross.

Stable *a1-Mum* (= *a1-Mum-stable*, i.e., a stable derivative of a mutable *a1-Mum* mutant) seeds from these outcross ears were sown and the resulting plants crossed with plants from non-*a1-Mum* sibling seeds from the same ears. If the 1:1 ratios are the result of the segregation of a single dominant controlling element (a regulator element), half of these crosses should result in ears that again give 1:1 ratios and the other half would have only stable seeds. One of these crosses produced three ears, two of which gave mainly stable seeds or an occasional seed with a few spots. One ear, however, had 49 stable plus Class 2 seeds (53.26%) and 43 mutable (33 Class 2 and 10 Class 4). The second cross resulted in six ears that had only stable or stable and few low mutable seeds and two ears that segregated for mutability and stable. One of the latter two ears had 30 stable plus low mutables (14.78%) to 173 medium and high mutable. The second segregated for 32 stable plus low mutable (48.48%) and 34 medium plus high mutable. From the third cross only two ears were recovered, one segregated primarily stable seeds and a few seeds with an occasional spot while the other segregated 1:1 for stable versus mutable (i.e., 69 stable and low mutable (43.95%) and 88 medium and high mutable).

In 1986, mutable and stable seeds from these winter nursery outcross ears were sown. The plants from the mutable seeds were outcrossed to standard *a1 sh2* stocks that had never been crossed with Mutator. The plants from the stable seeds were selfed.

Three plants from Class 4 seeds of the winter ear with 14.78% stables were crossed to *a1 sh*. One of the outcross ears had a low frequency of stables (i.e. 6.84%) like the ear from which it came. The second cross gave predominantly stable and low mutables (83.58%). However, if the low mutables are included with the medium and high mutables the stables are 59.38%; giving an approximately 1:1 ratio for mutable to stables (Note: see a later statement about how low mutables are usually counted and why). A self was obtained on the mutable parent of this first cross and it had predominantly mutable seeds of Classes 3 and 4. The third outcross ear gave 30.0% stable seeds. The self of the mutable parent in this cross had only mutable seeds of Class 4. It is possible that this line has three copies of a regulatory element although the inheritance patterns are not consistent.

One plant was obtained from a Class 4 seed from a winter outcross ear with 44.48% stable seeds. This plant on outcrossing to *a1 sh2* gave an ear that was segregating 1:1 for mutable and stable seeds (56.78% stable).

Four plants were obtained from Class 2 seeds from the winter outcross ear with 53.26% mutability. Three of the four plants when crossed with *a1 sh2* gave ears with some mutable seeds (predominantly of the low mutable class). The percent of stable seeds in these ears was 46.64%, 65.88%, and 71.69%. The fourth outcross ear had only stable seeds.

The above 1986 tests were from *a1-Mum2* stocks. The last test was with an *a1-Mum3* stock, which in the winter nursery gave an outcross ear that segregated 43.95% stable seeds. Plants from four Class 4 seeds from the winter outcross ear when outcrossed to *a1 sh2* gave ears with the following percents of stable plus low mutable seeds: ratios 66.67%, 57.89% and 54.91% and 48.94%. Results from the foregoing 1985-86 and 1986 crosses are summarized in Table 1.

Table 1. Summary 1985-86 tests of putative 1:1 (mutable: stable) *a1-Mum2* and *a1-Mum3* outcross ears and the 1986 follow-up tests with *a1 sh2*.

85-86 Plant number	85-86 a1-Mum stable cross to plants from non-a1-Mum stable sibling seed seed classification classes 1	2	3	4	% class 1 & 2	1985 cross of plants from mutable seeds from 85-86 ears to standard a1 sh2 stocks seed classification classes 1	2	3	4	% class 1 & 2
5572-3	28	2	1	172	14.78	2	6	10	99	6.84
						76	31	20	1	83.58 or 59.38*
						41	10	67	52	30.00
5572-6	28	4	2	32	48.48	65	2	4	47	56.78
5571-1	49	33		10	53.26*	118	132	3	0	46.64*
						112	58	0	0	65.88*
						157	62	0	0	71.69*
5573-2	59	10	3	85	43.95	84	44	17	47	66.67
						77	22	28	50	57.89
						70	25	12	66	54.91
						41	7	9	41	48.98

*In these crosses only stables were included in the stable class. See the text for why class 2 mutables are usually grouped with stables.

Stable seeds (*a1-Mum stables*) from each of the winter nursery ears tested in the above 1986 crosses gave plants with selfed ears that had predominantly stable seeds. On these ears, only an occasional seed was found with one or a few spots.

The results, so far presented, suggest that it is possible to derive stocks from *Mu*-induced aleurone mutants that have the classical two element pattern. Sibling seeds from the four winter nursery outcross ears from which the seeds for the 1986 outcross tests were selected were planted in the selfing block and selfed to determine if the *a1-Mum* parent plants in the 85-86 winter nursery had germinal mutator activity. Two of the plants did. One had 6.06% mutation frequency (n = 33) and the other had a 2.94% mutation frequency (n = 34). The other two winter nursery plants did not have mutator activity (i.e., no mutants were found, n = 28 and 43). These tests are very limited, thus it is impossible to generalize from them. However, the results from these tests suggest that there are different mechanisms responsible for regulating germinal Mutator activity as measured by induced mutants and somatic *Mu* activity as measured by germinal activity. This observation supports conclusions presented in last year's News Letter (MNL 60:8-9, 1986).

In the winter of 1985-1986, several new 1:1 ears were found among outcrosses of *a1-Mum/a1 sh2* stocks again to *a1 sh2*. These were tested further in 1985 by crossing plants from *a1-Mum-stable* seeds to plants from non-*a1-Mum* seeds from the same ear (i.e. *a1 sh2* segregants), as in the previous 85-86 tests. Also, in two instances, plants from mutable seeds of these 1:1 ears were crossed to standard *a1 sh2* stocks. The results of these tests are given in Table 2. As indicated in a footnote the ratios have just been estimated at this time. Also as noted in a footnote, the percent stable on these 1:1 ears was calculated by combining Classes 1 and 2 into the stable class and

Table 2. Test for segregation of a single regulator element for somatic mutability in *Mu* induced *a1* mutants. (Plants from stable *a1* derivatives from ears segregating 1:1 mutable: stable crossed to plants from non-*a1-Mum* sibling seed or mutable seeds from the same ear crossed to a standard *a1 sh2* stock—See text for more details about these crosses.)

No. of 1:1 ear	% stable of 85-86 1:1 ear	No. of tested plants in 1986	Class of seed planted	Outcross onto silks of non-a1-Mum sibling plant	Outcross using pollen of non-a1-Mum sibling plant	Outcross onto silks of a standard a1 sh2 stock
85-86 5370-5 a1-Mum3	54.80[c]	2274-1	1	stable+[a]	stable+	
		-2	1	stable+	stable+	
		-3	1	(1:1)[b]	(1:1)	
		-5	1	(1:1)	(1:1)	
		-6	1	stable+		
		-7	1	low		
		-8	1	stable+		
		-9	1	stable+		
		2295-2	4			(1:1)
		-3	4			(1:1)
		-4	4			(1:1)
		-5	4			(1:1)
		-6	4			(1:1)
		-7	4			(1:1)
85-86 5511-5 a1-Mum3	64.29	2275-1	1	(1:1)		
		-2	1	(1:1)	(1:1)	
		-3	1	stable+	stable+	
		-5	1	(1:1)		
		-6	1	(1:1)		
		-7	1	stable+		
		-8	1	stable+	stable+	
		-9	1		stable+	
		10	1	stable+	stable+	
		2300-3	3		--	
		-5	3		--	
		-6	3			(1:1)
		-7	3			(1:1)
85-86 5511-1 a1-Mum3	59.09	2280-1	1	(1:1)	(1:1)	
		-2	1	(1:1)	stable+	
		-3	1	(1:1)		
		-4	1	(1:1)		
		-5	1	(1:1)	(1:1)	
		-6	1	stable+		
		-7	1	(1:1)		
		-8	1	(1:1)		
		-9	1	(1:1)		
		10	1	(1:1)		
85-86 5510-2 a1-Mum3	61.63	2281-1	1	stable+		
		-2	1	stable	stable+	
		-3	1	(1:1)	stable	
		-4	1	stable+	stable	
		-5	1	stable+		
		-6	1	stable		
		-7	1	(1:1)	(1:1)	
		-8	1	stable+		
		-9	1		stable	
		10	1	stable+	stable	
85-86 5578-5 a1-Mum3	45.40	2282-1	1	stable+		
		-2	1	stable+		
		-3	1	stable+		
		-4	1	stable		
		-6	1	stable	stable+	
		-7	1	(1:1)	(1:1)	
		-8	1	stable+	stable+	
		-9	1	high		

a. stable+ = an ear that has predominantly stable seeds but also has an occasional spotted seed, usually with a low level of mutability (i.e., Class 2 or rarely 3).

b. (1:1) = estimated 1:1 ratio of mutable to stable. Time has not permitted the counting of seeds on these ears at this time. Thus these are only estimates.
Note: In the case of the reciprocal crosses, the ears in which the a1-Mum parent was crossed as a female were classified first and then the outcross of the a1-Mum plants as males were classified with no reference being made to the results of the reciprocal crosses.

c. To determine these ratios, Class 1 and 2 were considered to be stable and Class 3 and 4 as mutable. See text for explanation of this procedure.

Classes 3 and 4 into the mutable class. The Class 2 mutables were usually pooled with the stables because, frequently Class 2 seeds will give rise to plants that have predominantly stable seeds in the next generation. Also plants from stable seeds when selfed and/or outcrossed to *a1 sh2* will frequently give ears with predominantly stable seeds plus, sometimes, a few Class 2 seeds as well. Thus Class 2 seeds seem to be incipient stables. It is obvious from Table 2 that mutability can be restored to *a1-Mum-stable* stocks by crossing to plants from sibling non-*a1-Mum* seeds from the same 1:1 ear. When this is observed, the ratio obtained is again 1:1. Approximately half of the crosses result in this 1:1 reactivation. These results are expected if a single regulator of *a1-Mum* mutability is segregating in these stocks. The fact that the same results are observed in all but two of the reciprocal crosses reinforces this conclusion, as does the 1:1 ratio observed in the outcrosses to *a1 sh2* of the plants from mutable seeds of these 1:1 ears. Samples of seeds from these 1:1 ears were planted in the 1986 selfing block to screen for germline *Mu* activity in these stocks. As of this writing, we have the results from only three of these tests. Two had germinal activity, one did not. In both positive tests, the population size was 37 and the mutation frequency 2.70%. The population in the negative test was 36. These results provide additional support for the hypothesis that different mechanisms are responsible for germinal and somatic *Mu* activity.

In summary, the results reported here suggest that it is possible to derive an apparent two element system from *Mu*-induced mutable aleurone mutants. What role if any the putative regulator element plays in the regulation of germinal activity of the Mutator system is not known at this time. In fact, the evidence here agrees with that presented last year (MNL 60:8-9, 1986) and in other reports in this year's News Letter that would suggest somatic mutability and germline mutability are not necessarily correlated.

Donald S. Robertson

Additional evidence on the correlation of somatic mutability and germinal mutator activity in *Mu*-induced aleurone mutants

In last year's News Letter we presented the first results of a study to determine if somatic instability in *Mu*-induced aleurone mutants could be used as an indicator for *Mu* germinal activity (MNL 60:8-9, 1986). Last year's data suggested that lines having predominantly seeds with a high level of mutability sometimes did and sometimes did not have germinal activity, while lines that had predominantly medium or low levels of mutability had no germinal activity.

This year we have partial results to report (at this writing we still have more crosses to analyze) from an additional generation of testing (Table 1). Seeds from lines previously outcrossed to *a1 sh2* tester stocks, which produced ears with predominantly low (= Class 2), medium (= Class 3) or high (= Class 4) seeds, were again

Table 1. Result of tests of germinal mutator activity for mutable *Mu*-induced *a1* mutants.

Family No.	a1 allele	Mutability class of the O.C. ear	Average Mutability score of seed on O.C. ear	Mutability classes of the seeds planted	Average Mutability class of all @ ears from the family	No. of @ ears scored	% of ears segregating for new mutants
85-86-3501	a1Mum2	low	1.31	#1	1.37	35	0.0
-3502	a1Mum2	low	1.51	#1 & #2	1.39	26	0.0
-3503	a1Mum2	low	1.10	#1	1.75	14	0.0
-3504	a1Mum2	med.	2.77	#2 & #3	2.44	12	0.0
-3505	a1Mum2	med.	2.10	#3	2.41	22	0.0
-3507	a1Mum2	med.	2.70	#3	2.35	25	0.0
-3506	a1Mum2	high	3.86	#4	3.86	25	0.0
-3508	a1Mum3	low	1.68	#1 & #2	1.36	20	0.0
-3509	a1Mum3	low	1.20	#1	1.44	27	0.0
-3510	a1Mum3	med.	2.30	#3 & #4	2.68	33	0.0
-3511	a1Mum3	med.	2.30	#3	2.30	26	(3.85)**
-3512	a1Mum2	high to stable*	1.13	#1	1.66	28	3.57
-3513	a1Mum2	high to stable	1.09	#1	1.35	29	6.90
1986-1201	a1Mum1	low	1.40	#1 & #2	1.37	41	0.0
-1202	a1Mum1	low	1.33	#1 & #2	1.30	47	0.0
-1203	a1Mum1	low	1.52	#1 & #2	1.24	44	0.0
-1208	a1Mum2	low	2.08	20-#1,20-#2,10-#3	1.66	44	0.0
-1204	a1Mum2	med.	3.18	10-#1,10-#2,10-#3,20-#4	2.25	40	0.0
-1205	a1Mum2	med.	3.21	10-#1,10-#2,10-#3,20-#4	2.37	39	0.0
-1206	a1Mum2	med.	3.32	10-#1,10-#2,10-#3,20-#4	2.49	42	7.14
-1207	a1Mum2	med.	3.11	5-#1,5-#2,25-#1,15 ybt	2.44	56	0.0
-1209	a1Mum2	high	3.89	#4	3.60	46	0.0
-1210	a1Mum3	low	1.23	40-#1, 10-#2	1.18	40	0.0
-1211	a1Mum3	low	1.37	20-#2, 30-#3	1.21	42	0.0

*Stock in which the previous generation was highly mutable but on outcrossing gave predominantly stables.

**One doubtful mutant.

outcrossed to an *a1 sh2* tester and ears selected that gave again the type of ear of the parental *a1-Mum* stock. In some crosses, ears that differed from the parental stock were observed (e.g., ears that switched from high to stable, had a range of mutable classes instead of predominantly one class, etc.). Some of these latter types of ears also have been tested.

The 1985-1986 winter nursery had a very poor stand, thus the numbers in each test are not large. If one, however, sums all the plants in the high and medium crosses, the total population is 84. This also is not a large population but in the vast majority of *Mu* outcross populations of this size some mutants would be expected. As in previous tests most of the lows have no germinal *Mu* activity. The 1986 families 3512 and 3513 are of special interest. Two generations ago in the ancestry of these lines, two independent high mutable plants were crossed to *a1 sh2*. In this outcross generation, which was grown in 1985, some plants from each line had ears with predominantly stable (Class 1) seeds in both selfs and outcrosses to *a1 sh2*. The seeds from two of these *a1 sh2* outcrosses, in which only stable (*a1-Mum-stable*) seeds were observed, were planted in families 86-3512 and 86-3513. These families unexpectedly showed a high germinal mutation frequency. In all previous tests of stable derivatives, they have never shown germinal mutator activity. Thus, it had been assumed prior to this observation that the loss of somatic and germinal *Mu* activity occurred concomitantly. These observations suggest, however, that germinal activity can remain in the absence of somatic activity. In the ancestry of 3512, the parent two generations ago was *a1 sh2/a1-Mum Sh2*. Plants of this genotype can rarely be expected to give a stable *a1* (standard allele) *Sh2* seed as the result of crossing over between *a1* and *sh2*. However, since mutable seeds were planted from this heterozygous ear, a homozygous stable plant would not be expected unless heterofertilization also occurred, involving a pollen grain with the crossover product and one with the *a1-Mum* allele, an extremely unlikely event. Even this explanation is not possible for 3513 because two generations ago the genotype was purple aleurone/*a1-Mum2* and thus there is no standard *a1* allele available and the stable must be derived from *a1-Mum2*.

In 1986, additional crosses of the same material planted in the winter nursery were made. Only one of these crosses had germinal mutator activity (1206, medium seed mutability). Three lines with medium mutable seeds and one with high mutable seeds did not show any germinal activity.

The evidence is accumulating that somatic mutability is not a reliable predictor of germinal activity. It neither predicts the loss or the retention of the ability of *Mu* stocks to induce germinal mutation. Crosses described in other work from our laboratory and presented in the report by Brad Roth and me provide further confirmation of this observation. The fact that these two different aspects of *Mu* activity can be disassociated suggests that the two phenomena are dependent on different aspects of the *Mu* system.

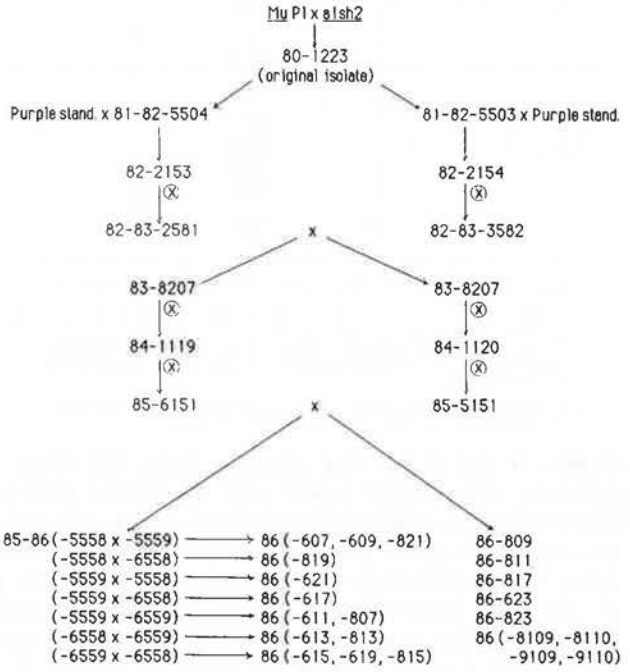
Donald S. Robertson

The effect of inbreeding on the expression of mutability in *a1-Mum2*

In 1983, Robertson reported (Mol. Gen. Genet. 191:86-90, 1983) that inbreeding *Mu* stocks would result in the inactivation of germinal mutator activity. Others have shown that this is accompanied by modification of the *Mu1* and *Mu1*-like elements present in these stocks (Bennetzen, Plant Genetics, M. Freeling, ed., pp. 343-353, 1985). Similar *Mu1* element modification has been shown to accompany the loss of somatic mutability in *Mu*-induced aleurone mutants (Chandler and Walbot, PNAS 83:1767-1771, 1986). Thus, it seemed reasonable to assume that inbreeding a *Mu*-induced aleurone mutant would eventually result in the loss of mutability accompanied by modification of *Mu* elements.

In 1985, crosses were made between two different stocks of a *Mu*-induced *a1* mutable mutant, *a1-Mum2*, which exhibited a consistent expression of an intense mutable pattern (mutability scale used in this report: Class 1 = stable, Class 2 = low mutability, Class 3 = medium mutability, Class 4 = high mutability, Class 5 = purple). With the 1985 cross there had been 5 generations of inbreeding (either selfing or intercrossing, see Figure 1). This is equivalent to Mu^{32} *per se* in our previous inbreeding experiment (Robertson, Mol. Gen. Genet.

Figure 1. Pedigree of the a1Mum2 class 4 stocks crossed in 1985.



191:86-90, 1983). In the previous work, modification and loss of germinal *Mu* activity had been observed to be complete by Mu^{16}, at least one generation earlier than that of the *a1-Mum2* stocks crossed in 1985. The inbreeding in the *a1-Mum2* stock has been more intense than in the previous work because it involved 3 generations of selfing which was never practiced in the original inbreeding experiment.

Sixteen ears were produced as a result of intercrossing in 1985. Seven of these had nothing but Class 4 seeds; six had predominantly Class 4 seeds with a few Class 2; one had predominantly Class 4 with a few stable seeds (Class 1) and two ears had a spectrum of mutable classes. On one

of the latter two ears, the mutable classes ranged from Class 4 to Class 2. On the other, the range was from Class 4 to Class 1.

In the 1985-86 winter nursery, seeds from four of the ears from the summer crosses were sown (as families 5558, 5559, 6558, 6559) and the intercrossing was continued. The results of these crosses are given in Table 1. Although

Table 1. Results of intercrossing *a1-Mum2* Class 4 stocks in the 1985-86 winter nursery.

85-86 Family & Plant #	Plant & Family # (♂parent)	Mutability Classification[a] Intercross Ear Major Class	Intercross Ear Minor Class	Exact Reciprocal[b] Major Class	Exact Reciprocal[b] Minor Class
(Classification for 6558 parent ear: 230 class 4 seeds)					
6558-1	6559-2	4	3	NT[c]	NT
6558-2	6559-6	4	-	NT	NT
6558-4	5559-4	4	-	NT	NT
6558-5	5558-5	4	-	NT	NT
6558-6	5559-5	4	2	NT	NT
6558-7	6559-5	4	-	4	-
6558-8	6559-3	4	3	NT	NT
6558-8T	5559-2	4	-	4	3,2,1
(Classification for 6559 parent ear: 310 class 4, 1 class 3, 1 class 2 seeds)					
6559-1	5558-7	2	3	3,2	-
6559-2	5559-8	2	3	-	-
6559-3	5559-1	2	3,4	-	-
6559-6	6558-5	4	3,2,1	-	-
6559-7	6558-8	4	3,2	-	-
6559-8	5559-9	2	1	-	-
6559-9	5559-9	2	1	2,1	-
(Classification for 5558 parent ear: 234 class 4, 1 class 3 seeds)					
5558-1	6558-1	4	3,2	-	-
5558-2	5559-6	2	1	3,2,1	-
5558-3	6558-2	4	3,2,1	-	-
5558-4	6558-4	4	3,2	-	-
5558-5	5559-7	2	1	-	-
5558-6	5559-3	3,2,1	-	4,3,2,1	-
(Classification for 5559 parent ear: 292 class 4 seeds)					
5559-1	6559-7	3,2	-	-	-
5559-4	5558-4	3,2,1	-	-	-
5559-7	5558-3	2	1	-	-
5559-8	5558-1	2	1	-	-

[a] Mutability scale as in text. Major Class is predominant class (multiple classes indicate mixture), minor classes contain relatively few seeds.
[b] Exact reciprocal crosses are recorded only once.
[c] No test

Table 2. Results of intercrossing *a1-Mum2* Class 4 stocks (seeds from sibling ears of material planted in 1985-86) in the 1986 summer nursery.

1986 Family & Plant#	Plant & Family# (♂parent)	Mutability Classification[a] Intercross Ear Major Class	Intercross Ear Minor Class	Exact Reciprocal[b] Major Class	Exact Reciprocal[b] Minor Class
(Classification for 8109 parent ear: 188 class 4 seeds)					
8109-1	9109-3	2	1	2	1
8109-3	9109-1	1	2	2	1
8109-5	9109-2	2	1	2	1
8109-6	9109-2	2	1	NT[c]	NT
(Classification for 8110 parent ear: 182 class 4 seeds)					
8110-1	9110-5	4	-	NT	NT
8110-2	9110-1	4	-	NT	NT
8110-5	9109-4	4	3,2	4	3,2
8110-6	9110-10	4	-	NT	NT
8110-7	9110-7	4	-	4	-
8110-8	9110-2	4	-	NT	NT
Results of all crosses with family 9109 are recorded in exact reciprocal column. (Classification for 9109 parent ear: 223 class 4, 2 class 2 seeds)					
(Classification for 9110 parent ear: 132 class 4)					
9110-3	8110-8	4	-	NT	NT
9110-4	8109-4	4 (nubbin)	-	NT	NT
9110-5	8110-6	4	-	NT	NT
9110-6	8110-1	4	-	NT	NT
9110-10	8110-3	4	-	NT	NT
9110-11	8109-6	4	-	NT	NT

[a] Mutability scale as in text.
[b] Exact reciprocal crosses are recorded only once.
[c] No test

all ears from which seeds were selected for planting had only Class 4 or predominantly Class 4 seeds, intercrosses between three of the families resulted in ears which showed a marked loss in mutability. The one exception was family 6558. Whenever this family was involved in an intercross, the ears had only Class 4 or predominantly Class 4 seeds. The results were the same regardless of the direction of the cross. In all cases where the exact reciprocal crosses were made, the results were basically the same in both directions.

In the 1986 summer nursery, intercrosses between plants from the four additional 1985 ears were made. The results, shown in Table 2, are similar to those obtained in the winter nursery, in which seeds from sibling ears were planted. Again two types of families are seen. One type produces ears with primarily Class 4 seeds and the other type gives ears with predominantly low mutability seeds. Again, in crosses between high and low mutability lines, the high condition is dominant whether the high line is crossed as a male or female.

One of the 1985 ears, 6161-8/5151-3T had 237 Class 4, 64 Class 3 and 28 Class 2 seeds. Ten Class 2 seeds were planted from this ear. Eight plants in this family were either selfed and outcrossed to *a1 sh2* or reciprocally crossed to *a1 sh2*. No mutable seeds were observed on any of the progeny ears.

During the summer of 1986, seeds from the 1985-1986 winter nursery families 5558, 5559, 6558 and 6559 as well as some seed from the 1985 intercross ears were planted. The resulting plants were intercrossed reciprocally. Intercross results are reported in Table 3. All seeds used in these crosses were either 1985 class 4 seeds (as indicated) or derived from the 1985-86 intercrosses shown in Table 1. The following define the parents of the various crosses shown in Table 3: 1) Low/low-the result of intercrosses involving combinations of 85-86-5558, -5559, or -6559 families. 2) High/low-the result of intercrosses between family 85-86-6558 as a female times 85-86-5558, -5559 or -6559. 3) Low/high-the result of reciprocal crosses in which 85-86-6558 is the male parent. The specific family origin of the 86-600, and -800 families from Table 3 is shown in the pedigree in Figure 1.

When plants from Class 2 seeds from a low/low (607, 807) were intercrossed, the resultant progeny were without exception Class 1 stables. Similar results were obtained in exact reciprocal crosses.

Intercrosses between plants from Class 2 seeds from a low/low (609 and 611) and plants from a 1985 Class 4 (809 and 811) gave variable results. Seed mutability ranged from Class 4 to Class 1 with most ears segregating for Class 4 as a major class. Notable exceptions were ears from 609-1/809-6, 609-2/809-4 and 611-9/811-8 which gave primarily low seeds. Reciprocal crosses from these same plants showed higher mutability, however, all other sets of reciprocal crosses in these two families showed only a slight bias toward higher mutability when the Class 4 parent was the female.

Intercrosses between plants from Class 4 seeds from two high/low lines (613,813) gave only seeds of low mutability when crossed in either direction. Similarly, intercrosses between plants from Class 4 seeds from a low/high (615,815) gave seeds of low mutability. When plants from Class 2 seeds from a low/high (617,617.1) were inter-

Table 3. Results of intercrossing additional *a1-Mum2* stocks in the 1986 summer nursery.

1986 Family & Plant#	Plant & Family# (♂parent)	Mutability Classification[a] Intercross Ear Major Class	Minor Class	Exact Reciprocal Major Class	Minor Class
Cross[b]: Class 2 from low/low[c] x same.					
607-1 (2)[d]	807-2 (2)[d]	1	1	1	1
607-2 (2)	807-7 (2)	1	1	1	1
607-3 (2)	807-8 (2)	1	1	1	1
607-4 (2)	807-6 (2)	1	1	1	1
Cross: Class 2 from low/low x Class 4 (85-6161-1/5151-8).					
609-1 (2)	809-6 (4)	2	4,3,1	4,3,2,1	-
609-2 (2)	809-4 (4)	1	2	1	2
609-3 (2)	809-3 (4)	4,3,2,1	-	4	3,2,1
609-6 (2)	809-7 (4)	4	3,2,1	4	3,2,1
609-7 (2)	809-8 (4)	4,3,2,1	-	4,3	2,1
609-8 (2)	809-5 (4)	4,3,2,1	-	4,3,2,1	-
Cross: Class 2 from low/low x Class 4 (85-6161-1/5151-8).					
611-2 (2)	811-5 (4)	4,3,2,1	-	4,3,2,1	-
611-9 (2)	811-8 (4)	1	3,2	4,3,2,1	-
611-10 (2)	811-1 (4)	4,3,2,1	-	4,3	2,1
Cross: Class 4 from high/low[c] x same.					
613-1 (4)	813-3 (4)	2	3,1	2	1
613-6 (4)	813-8 (4)	2,1	-	2	1
613-7 (4)	813-10 (4)	1	2	1	2
613-9 (4)	813-6 (4)	2	3,1	2	1
Cross: Class 4 from low/high[c] x Class 4 from low/high.					
615-1 (4)	815-3 (4)	1	2	1	2
615-2 (4)	815-7 (4)	2	1	2,1	-
615-3 (4)	815-4 (4)	2,1	-	2,1	-
615-4 (4)	815-1 (4)	1	2	1	2
615-6 (4)	815-8 (4)	2,1	-	2	1
Cross: Class 2 from low/high x Class 4 (85-6161-1/5151-8).					
617-1 (2)	817-1 (4)	4,3,2,1	-	4	3,2,1
617.1-1 (2)	817-4 (4)	4,3,2,1	-	4,3	2,1
617.1-2 (2)	817-5 (4)	4	3,2,1	4	3,2,1
617.1-3 (2)	817-8 (4)	4,3	2,1	4,3	2,1
Cross: Class 1 from high/low x Class 1 from low/high.					
619-1 (1)	819.1-1 (1)	1	-	1	-
Cross: Class 1 from high/low x Class 4 (85-6161-1/5151-8).					
619-1T (1)	823-5 (4)	4,3,2,1	-	4	3,2,1
Cross: Class 2 from low/low x same.					
621-1 (2)	821-3 (2)	1	-	1	-
621-3 (2)	821-2 (2)	1	-	1	-
621-6 (2)	821-5 (2)	2,1	-	1	2
Cross: Class 4 (85-6161-1/5151-8) x same.					
623-4 (4)	823-7 (4)	4	-	4	3,2,1
623-5 (4)	823-1 (4)	4	3	4	-
623-7 (4)	823-2 (4)	4	3,2	4,3,2,1	-

[a] Mutability scale as in text.
[b] See Figure 1 for details of 600 and 800 family progenitors.
[c] See text for definition of low/low, low/high and high/low parent lines.
[d] Mutability class of seed planted in ( ).

crossed with plants from 1985 Class 4 seeds (817), all classes of mutability resulted on the progeny ears. Exact reciprocal crosses showed no outstanding reciprocal differences.

Intercrosses between two plants from Class 1 seeds, one from a high/low (619) and one from a low/high (819.1), produced only stable seeds when crossed in either direction. The tiller from 619-1 was crossed as a female with a plant from a 1985 Class 4 seed (823-5). This cross produced all classes of seeds, whereas the exact reciprocal cross produced predominantly Class 4 seeds with a few of each of the other classes. Intercrosses between plants from Class 2 seeds from a low/low (621,821) without exception resulted in ears with seeds of low mutability, or Class 1 stable seeds. The latter class was the predominant class on these ears.

The inbreeding of 1985 Class 4 plants was continued (623,823) with sibling seeds of those planted in the 1985-86 family 6558. Although some reduction in the level of somatic mutability was observed, the majority of the seeds produced were of high mutability. This was also the case for plants that were selfed (data not shown).

Levels of somatic mutability and germinal mutator activity are shown in Table 4 for individual plants from the pedigree of the stocks described in this report. An outcross of 82-2154-5 was selfed and seedling-tested for the production of new mutants. Of the outcross ears, 21.7% segregated for new mutations. After one generation of selfing and an intercross generation, 3 of 4 Class 4 siblings tested in the 83-8207 family had no germinal activity. Plants 83-8207-1 and -8207-2 were used in the production of the two Class 4 lines that were crossed in 1985 (see Figure 1). An outcross of 8207-2 has been planted and will be selfed in our 1986-87 winter nursery, then seedlings tested later this spring. This information is needed before we can fully interpret the data in this report. The first time we saw a significant reduction in the level of somatic mutability was in 1985. No outcrosses of the 1985 plants were made and thus germinal activity of these plants could not be determined. However, the 1985-86 families have been tested for germinal mutator activity. Five of the six plants from family 6558 which showed high somatic mutability, had germinal activity. The other outcross had no mutator activity. On the other hand, plants from the low families, 5558, 5559 and 6559, which when outcrossed gave mainly low seed mutability, showed no correlation between loss of somatic mutability and the presence or absence of germinal mutator activity. For example, outcrosses of 5558-1 and 5558-3 both resulted in seeds of low mutability, yet 5558-1 had no germinal activity and 5558-3 had germinal activity. Both 5559 siblings and 2 siblings in family 6559 showed such activity, while one did not.

Table 4. Comparison of germinal mutator activity and level of somatic mutability through six intercross generations of *a1-Mum2*.

Plant#	#Generations Selfing or Intercrossing	Germinal Mutator Activity (%)	Level a1 Somatic Instability (avg. class)
82-2154-5	1	21.7	4
82-83-3582-6	2	a	4
83-8207-1	3	0.0	4
8207-2	3	b	4
8207-3	3	0.0	4
8207-4	3	0.0	4
84-1120-4	4	c	4
85-6151-8 5151-3T	5	NT[d]	3.64
85-86-5558-1	6	0.0	1.84[e]
-5558-3	6	27.27	1.85
85-86-5559-2	6	4.55	1.42
5559-8	6	8.70	1.42
85-86-6558-2	6	17.95	3.35
-6558-4	6	19.15	3.71
-6558-5	6	9.30	3.64
-6558-6	6	9.09	3.64
-6558-7	6	0.0	3.62
-6558-8	6	17.50	3.53
85-86-6559-5	6	2.27	2.03
-6559-6	6	0.0	2.21
-6559-7	6	18.60	2.28

[a], [b], & [c] Test in 86-87 winter nursery.
[d] No test, no outcross ears available
[e] Level is an average of estimated seed counts of approx. 50 outcross ears.

Table 5. Summary of Southern blot hybridization results to date.

Intercross ear #[a]	Level of a1 Somatic Mutability	Mu sizes	Copy # (Total)[b]	Modification (HinfI)	Germinal Mutator Activity
82-2154-5	4	Mu1,1.7	15-20	-	+
83-8207-1	4	Mu1,1.7	15-20	-	-
84-1120-4	4	Mu1,1.7	15-20	-	c
85-6151-8[d] 5151-3T	4	Mu1,1.7	15-20	+/---[e]	f
	3	Mu1,1.7	15-20	+/--	
	2	Mu1,1.7	15-20	+++/-	

[a] Individual plants grown in growth chamber sacrificed for DNA isolation. Results are consistent between several individual sibling plants.
[b] Copy number determined by 1) EcoRI hybridization profile and 2) comparison of TthIII profile to Mu1 copy number equivalents.
[c] Test in 1986 winter nursery.
[d] This plant segregated for mainly class 4, with few class 2 and 3 seeds.
[e] Relative amounts of modified (+) and unmodified (-) elements.
[f] No test, no outcross ears available.

A limited amount of molecular work on these lines has been initiated on selected plants in this pedigree. Table 5 summarizes information obtained to date as the result of Southern blot hybridization analyses. Individual plants from the pedigree in Figure 1 have been analyzed for *Mu* sizes, copy number, and modification of *Mu* element *Hinf*1 sites. All plants tested contain both *Mu1* and *Mu1.7*. The total copy number of these elements is approximately 15-20 per diploid genome. The copy number remains constant through the fifth generation of inbreeding. Extensive modification of the *Mu* elements does not occur until there is a loss of somatic mutability. This marked loss occurred in several of the 1985-6161/5151 crosses. When DNA from plants derived from Class 4, Class 3 and Class 2 seeds (85-86-6161-8/5151-3T) is analyzed for the presence of modification, there is a correlation between the amount of modified sequences and the relative level of somatic mutability. Although this has not been quantified, qualitatively, plants from Class 4, Class 3 and Class 2 seeds show increasing amounts of modified *Mu* elements relative to unmodified elements respectively. However, it is important to note that both modified and unmodified *Mu* sequences are present in all plants tested from this ear.

Germinal mutator activity and modification results for plants 82-2154-5 and 83-8207-1 indicate that in this material there is not a correlation between element modification and mutator activity. This may be further substantiated when the seedling test of 84-1120-4 is completed later this spring.

Plants 82-2154-5 and 83-8207-1 have the same number of *Mu* elements, none of which are modified, yet 82-2154-5 has germinal *Mu* activity while 83-8207-1 does not. Thus plants from seeds with high somatic mutability and possessing the same copy number of *Mu* elements do not necessarily have the same germinal activity. This lack of correlation of germinal activity with *Mu* copy number and somatic mutability would suggest that neither of these criteria are good predictors of *Mu* germinal activity. The results presented in Table 5 would suggest that the lack of modification also is not a good predictor of *Mu* germinal activity. The lack of any criteria that will predict germinal *Mu* activity suggests that the regulation of such activity is still unknown.

The following conclusions can be drawn from this work:

1) High somatic mutability can be retained after six generations of inbreeding.

2) However, in some sixth generation offspring, somatic mutability has been greatly diminished (low lines). This takes place suddenly in one generation.

3) Results from crosses between plants from high mutability seeds and those from low mutability seeds are the same, no matter what direction the cross is made. Thus there is no female effect observed.

4) When plants from Class 4 seeds from high/low and low/high crosses are crossed together, only stable or very low mutability seeds are observed in the progeny. Low/high and high/low do not behave differently in this regard. Again, no female effect is observed. In these crosses the low mutability state seems to prevail in both high/low or low/high hybrids because most progeny seeds are of low mutability.

5) Plants from seeds of different levels of mutability from the same ear show a progressive degree of modification of the *Mu* elements present that is inversely proportional to the level of somatic mutability.

6) There is little or no correlation of *Mu* germinal activity with the total *Mu* element copy number, the *Mu* element modification or somatic mutability.

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Results from a second series of inbreeding crosses of Mutator (*Mu*) stocks

In 1983, I reported on the effect of several generations of inbreeding of *Mu* stocks (Mol. Gen. Genet. 191:86-90, 1983). In this first experiment, different levels of inbreeding were produced by intercrossing between *Mu* stocks in which the putative level of *Mu* was doubled each generation for up to 4 generations of inbreeding. In these previous experiments, it was found that *Mu* activity, as measured by the standard *Mu* test (i.e., the frequency of seedling mutants transmitted by a *Mu* plant to its progeny), peaked at the second generation of inbreeding and fell off rapidly thereafter and was lost by the fourth generation of inbreeding. This inbreeding loss phenomenon has been subject to molecular analysis and has been found to be accompanied by modification of *Mu1*-like transposons.

In 1982, we undertook to repeat this inbreeding experiment to determine if the first results were repeatable. We have now obtained the Mu^{16} *per se* generation and the Mu^{8} o.c. (See Table 1 for definitions). In this experiment, we not only made the doubling crosses but also some of the intermediate crosses such as (1×3), (1×4), (2×3), (3×4), $(3/4\times1)$ etc. The results of the *per se* tests and tests of parents are shown in Figure 1 and the results of the outcross tests of the various *per se* types are given in Figure 2. We have not yet run a statistical analysis on these results and thus only generalizations can be made at the present time.

Results of the previous experiment, with respect to early generations of inbreeding, are confirmed in that there is an increase in mutants at this level of inbreeding. As inbreeding gets more intense there is a general drop-off of activity exhibited in the *per se* populations. But there is considerable variation in response depending upon the particular combination of parents. Some more-or-less consistent patterns can be discerned. For example, whenever an Mu^{3} is involved there tends to be a lower level of

activity. The more *Mu*^{3} contributes to the *per se* the greater the reduction in *Mu* activity; with *Mu*^{12} *per se* $(3\times3)^2$ showing the least activity. Note also that the straight doubling series (i.e. 1×1, 2×2, 4×4, 8×8) behaves as the previous experiment until *Mu*^{16} *per se*. In the previously reported work, there was a sharp drop of activity from *Mu*^{8} *per se* to *Mu*^{16} *per se*. In this present experiment there is instead a sharp increase when going from *Mu*^{8} *per se* to *Mu*^{16} *per se*. We will better be able to determine the significance of this when we can test the *Mu* activity of *Mu*^{16} *per se* plants by the standard *Mu* outcross test.

Generally speaking the *per ses* have more activity than their parents. This is as expected since the *per se* test is expected to measure the sum of the activity of the parents. However, the *per se* activity very seldom equals the sum of the individual parent activities or twice the parental activity, as the case may be. The only exceptions are *Mu*^{16} *per se* $(4\times4)^2$ and *Mu*^{16} $(3/4\times1)^2$. This tendency of the *per ses* to have fewer mutants than predicted from the parental frequencies suggests either a female effect exhibited by the female *Mu* parent or a depression of a possible *Mu* activity that is confined to the zygote or early developmental stages. Such *Mu* activity has been suggested by previous studies in our laboratory (MNL 60:12-114, 1986).

Symbol

Mu^1 - An Mu line that has only been propagated by outercossing.

Mu^2 per se - The F_1 generation of a $Mu^1 \times Mu^1$ cross.

Mu^3 per se - The F_1 generation of a $Mu^1 \times Mu^2$ cross.

Mu^4 per se (2 x2) - The F_1 generation of a $Mu^2 \times Mu^2$ cross.

Mu^4 per se (1 x 3) - The F_1 generation of a $Mu^1 \times Mu^3$ cross.

Mu^5 per se (1 x 4) - The F_1 generation of a $Mu^1 \times Mu^4$ cross.

Mu^5 per se (2 x 3) - The F_1 generation of a $Mu^2 \times Mu^3$ cross.

Mu^6 per se (3 x 3) - The F_1 generation of a $Mu^3 \times Mu^3$ cross.

Mu^6 per se (2 x 4) - The F_1 generation of a $Mu^2 \times Mu^4$ cross. The Mu^4 was a (2 x 2).

Mu^7 per se (3 x 4) - The F_1 generation of a $Mu^3 \times Mu^4$ cross. The Mu^4 was a (2 x 2).

Mu^7 per se (1/3 x 3) - The F_1 generation of a $Mu^1 \times Mu^6$. The Mu^6 was a (3 x 3).

Mu^7 per se (2/2 x 3) - The F_1 generation of a $Mu^2 \times Mu^5$ cross. The Mu^5 was a (2 x 3).

Mu^8 per se (4 x 4) - The F_1 generation of a $Mu^4 \times Mu^4$ each Mu^4 was a (2 x 2).

Mu^8 per se (3/4 x 1) - The F_1 generation of a $Mu^3 \times Mu^5$. The Mu^5 was a (4 x 1) in which the 4 was a (2 x 2).

Mu^{10} per se (2 x 2/2 x 4) - The F_1 generation of a $Mu^4 \times Mu^6$. The Mu^4 was a (2 x 2) and the Mu^6 a (2 x 4).

Mu^{10} per se (5 x 5) - The F_1 generation of a $Mu^5 \times Mu^5$. Each Mu^5 was a (2 x 3).

Mu^{12} per se $(3 \times 3)^2$ - The F_1 generation of a $Mu^6 \times Mu^6$. Each Mu^6 was a (3 x 3).

Mu^{12} per se $(2 \times 4)^2$ - The F_1 generation of a $Mu^6 \times Mu^6$. Each Mu^6 was a (2 x 4) in which each 4 was a (2 x 2).

Mu^{16} per se $(4 \times 4)^2$ - The F_1 generation of a $Mu^8 \times Mu^8$. Each Mu^8 was a (4 x 4) and each Mu4 was a (2 x 2).

Mu^{16} per se $(3/4 \times 1)^2$ - The F_1 generation of a $Mu^8 \times Mu^8$. Each Mu^8 was a Mu^3 x Mu^5 and each Mu^5 was a (4 x 1) and the Mu^4 was a (2 x 2).

Mu^{16} per se $(2 \times 3/3)^2$ - The F_1 generation of a $Mu^8 \times Mu^8$. Each Mu^8 was a (5 x 3) and each Mu^5 was a (2 x 3).

Mu^2 o.c., Mu^3 o.c. Mu^5 (1 x 4) o.c., Mu^8 (3/4 x 1) o.c., etc. = an outcross of the respective per se line.

Mu^2 parents, Mu^3 parents, Mu^5 (1 x 4) parents Mu^8 (3/4 x 1) parents, etc. = The results of a mutator test of the parent lines that produced the indicated Mu per se line.

Figure 2.

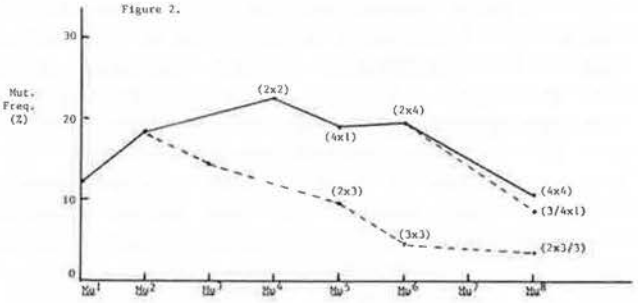
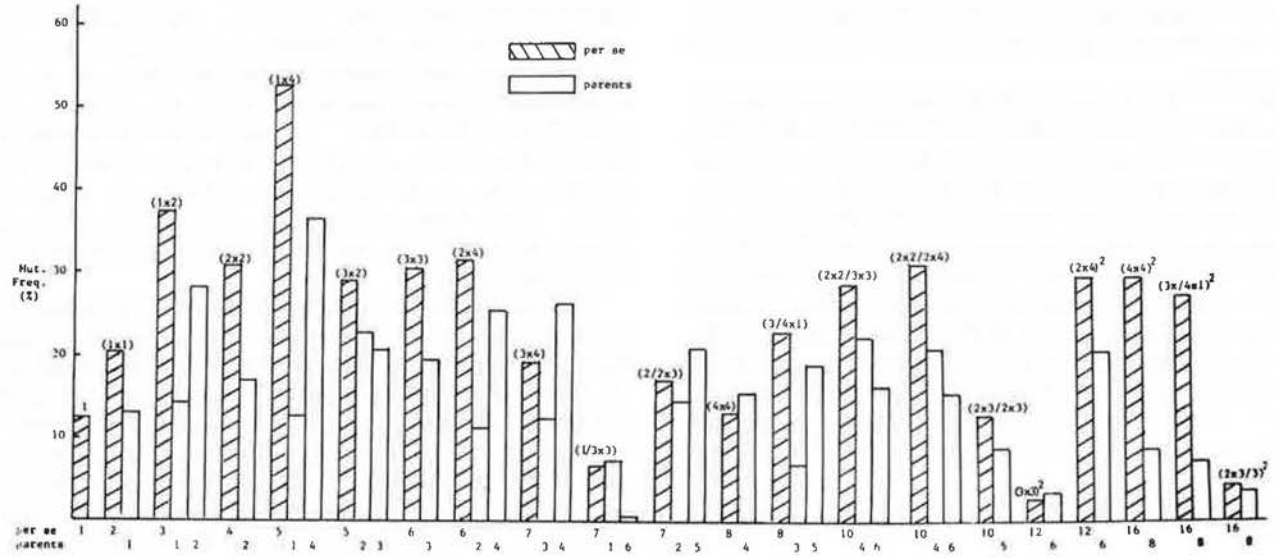


Figure 2, which gives the results of the outcrossing of the *per ses*, gives an indication of the *Mu* activity present in the *Mu per ses* themselves. Here we see that *Mu* activity peaks at *Mu*^{4} and falls thereafter. There is a bifurcated pattern, however. One in which *Mu*^{3} is not involved and those with *Mu*^{3}. The depressive effect of *Mu*^{3} that was hinted at in the *per se* data becomes obvious in the outcross tests. The exact nature of this *Mu*^{3} effect is not known.

Figure 1.



The results of this experiment taken together with those from the previous work points up a fact with regard to the Mutator system that cannot be emphasized too strongly. That is, that it is dangerous to generalize from just one experiment or one series of experiments. (For example, one could conclude from the work reported here that Mu^3s will be strong inactivators. But we do not know if this response is characteristic of Mu^3s in general or just the Mu^3 in this experiment.) There are probably several reasons for this unpredictability. For example, copy number of *Mu* elements and state of modification can vary from experiment to experiment and from generation to generation. With just those two variables alone one would not be surprised if two experiments, which superficially appeared to be identical, gave markedly different results. We need to know much more about the *Mu* system and find some way to standardize our stocks before we can start drawing generalizations from one experiment. This lack of standardization undoubtedly accounts for varying results from different laboratories.

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A new isozyme marker for the short arm of chromosome 6

Wendel et al. (MNL 60:109-110) reported isozyme segregation data that suggested *Adk1* (adenylate kinase) is located near the centromere on chromosome 6, and tentatively proposed a location on 6S. Subsequent work has confirmed that *Adk1* is on 6S, which is significant in that this is one of the most poorly mapped chromosome arms of maize. New F2 data (Table 1), in conjunction with

Table 1. Joint F2 segregation data and maximum likelihood estimates of the recombination fractions (r) for four markers near the centromere of chromosome 6.

		Genotypes[1]											
Loci	n	X_1Y_1	X_1Y_2	X_1Y_3	X_2Y_1	X_2Y_2	X_2Y_3	X_3Y_1	X_3Y_2	X_3Y_3	χ^2(df)	r/(se)
Adk1-rgd1	181	46	0		74	5		10	46		117.3(2)	.081 (.021)
Adk1-Pgd1	181	0	14	32	9	57	13	44	12	0	143.3(4)	.141 (.020)
Adk1-Enp1	181	0	16	30	9	55	15	39	17	0	116.3(4)	.170 (.022)
rgd1-Pgd1	181	8	77	45	45	6	0				119.9(2)	.076 (.020)
rgd1-Enp1	181	6	79	45	42	9	0				115.1(2)	.082 (.021)
Pgd1-Enp1	181	46	7	0	2	79	2	0	2	43	291.9(4)	.037 (.010)

[1]X and Y refer to the first and second locus listed for each locus pair. Genotypes are as follows: *Adk1*: X_1=5/5; X_2=4/5; X_3=4/4. *rgd1*: X_1=+/+ and +/*rgd* combined; X_2=*rgd*/*rgd*, except substitute Y for X in the *Adk1-rgd1* line. *Pgd1*: Y_1=3.8/3.8; Y_2=2/3.8; Y_3=2/2, except substitute X for Y in the *Pgd1-Enp1* line. *Enp1*: Y_1=10/10; Y_2=6/10; Y_3=6/6.

the previously presented data, indicate the following gene order and map distances for various markers near the centromere of chromosome 6:

Adk1 - 8.1 - *rgd1* - 7.6 - *Pgd1* - 3.7 - *Enp1*

These data are in agreement with previous distance estimates for the loci *Adk1, Pgd1,* and *Enp1*. Additional evidence that *Adk1* is on 6S was gained from a cross of TB-6Sa (as male) onto the inbred line Mt42, which carries the rate variant *Adk1-5*. Seven hypoploids (those that display only the tester allele) were recovered out of 51 seedlings analyzed. These same seven plants were heterozygous for numerous other isozyme markers, ruling out the possibility that they resulted from contaminating self-pollinations. These data thus confirm that *Adk1* is on 6S. Because neither *rgd1* nor *Pgd1* is uncovered by either TB-6Sa or TB-6Lc (*Enp1* is uncovered by TB-6Lc, however; Wendel et al., MNL 59:89-90) their placement relative to the centromere is still uncertain.

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Chiasma frequency in the distal 5% of the long arm of chromosome 1

In a recent study of chiasma frequencies in the various segments of heterozygotes for complex B-A chromosome rearrangements, in one case (TB-1La5S8041) the frequency was unexpectedly low for the segment representing 20% of the terminal region of the long arm of chromosome 1 (M.P. Maguire, Chromosoma 94:71-85). In fact where a chiasma was present in this region there was almost always a chiasma also present in each of the other arms of the complex configuration, including the opposite arm which is also estimated to be genetically short. This raised the question whether the terminal region of the long arm of chromosome 1 may be essentially devoid of "pairing centers" and depends mainly upon zipping-up of pairing initiated elsewhere for the establishment of crossover pairing. The observation was especially surprising since in another recent study estimates of crossover frequencies in a series of overlapping inversions suggested that the genetic map of the long arm of chromosome 1 is approximately uniformly distributed for the region between about 0.55 and 0.92 of the physical length from the centromere, and crossover pairing is efficiently established in regions heterozygous for these inversions (M.P. Maguire, Genet. Res. 46:273-278). Now the chiasma frequency has been studied in heterozygotes for simple reciprocal translocation T1-4g, with breakpoints located at 1L.95 and 4L.35. Given the fortunate presence of a large chromosome 4 knob, diakinesis configurations were not difficult to classify. For the segment representing the distal 5% of chromosome 1L the total observed chiasma frequency was 8.8% (in 62/704 cells). This is close to expectation from assumption of uniform genetic map distribution throughout the arm. It seems likely that either zipping-up for crossover pairing is very efficient in this system (with pairing facilitation across breakpoints) or "pairing centers" in fact exist in the distal region of the long arm of chromosome 1.

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Analysis of several derivatives of the *a-m2 Spm* insertion allele

We have done genetic and molecular studies on several derivatives of McClintock's *a-m2 Spm* insertion allele. These alleles have the unique property that the *a* gene is either co-expressed with, or expressed under the control of, the *Spm* element, while most *Spm* insertions inhibit gene expression. We have analysed 9 derivative alleles isolated by McClintock from the original *a-m2* allele (now lost). Each derivative has a different mutation affecting the inserted *Spm* element, expression of the *a* gene, or

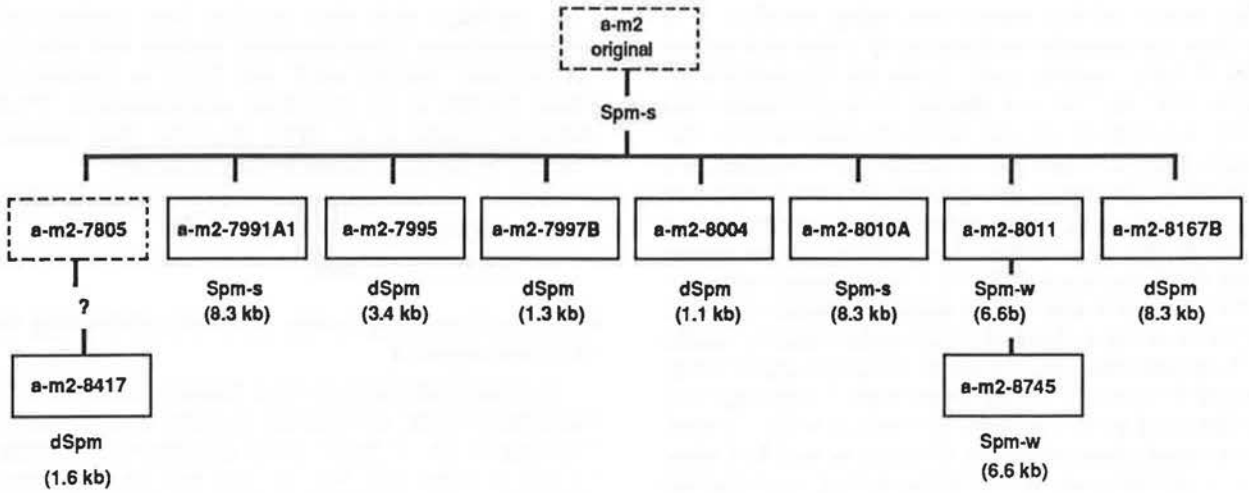

Figure 1. Derivation of the *a-m2* alleles.

both. The genetic relationship among the derivatives and their designations are shown in Figure 1. Our interest in these alleles stems from the observation that the manner in which *a* gene is expressed in these derivatives reflects the *Spm*'s own regulatory mechanisms, suggesting that the gene has come under the control of the element and that the behavior of the derivatives therefore offers insight into *Spm* regulation. Moreover, there are several mutant *Spm* elements among the derivatives, the analysis of which extends our understanding of the element's genetic organization.

We have found the insertion site to be identical in all of the *a-m2* derivatives; recent analyses of the *a* locus by Schwarz-Sommer et al. (EMBO J., in press) indicate that the insertion site is 99 bp upstream of the *a* gene's transcription start site. The derivatives differ from each other by mutations either within or very near the element. Based on genetic analyses, two of the derivatives have *standard Spm* (*Spm-s*) elements (*a-m2-7991A1* and *-8010A*), two have late-acting *weak Spm* (*Spm-w*) elements (*a-m2-8011* and *-8745*) and five have *transposition-defective Spm* (*dSpm*) elements (*a-m2-7995, -7977B, -8004, -8167B,* and *-8417*). The two derivatives with *Spm-s* elements have 8.3-kb insertions, while most of the derivatives with *Spm-w* or *dSpm* elements have shorter insertions which differ from the 8.3 kb *Spm-s* element by internal deletions. The type and length of the *Spm* element in each derivative is given in Figure 1. Most derivatives have a single insertion at the *a* locus, but the *a-m2-8745* allele has two insertions, the 6.6-kb *Spm-w* insertion of the *a-m2-8011* allele from which it was derived and a second nearby insertion (or duplication) that has no effect on the element, but inactivates the *a* gene. Most derivatives have a single large deletion within the

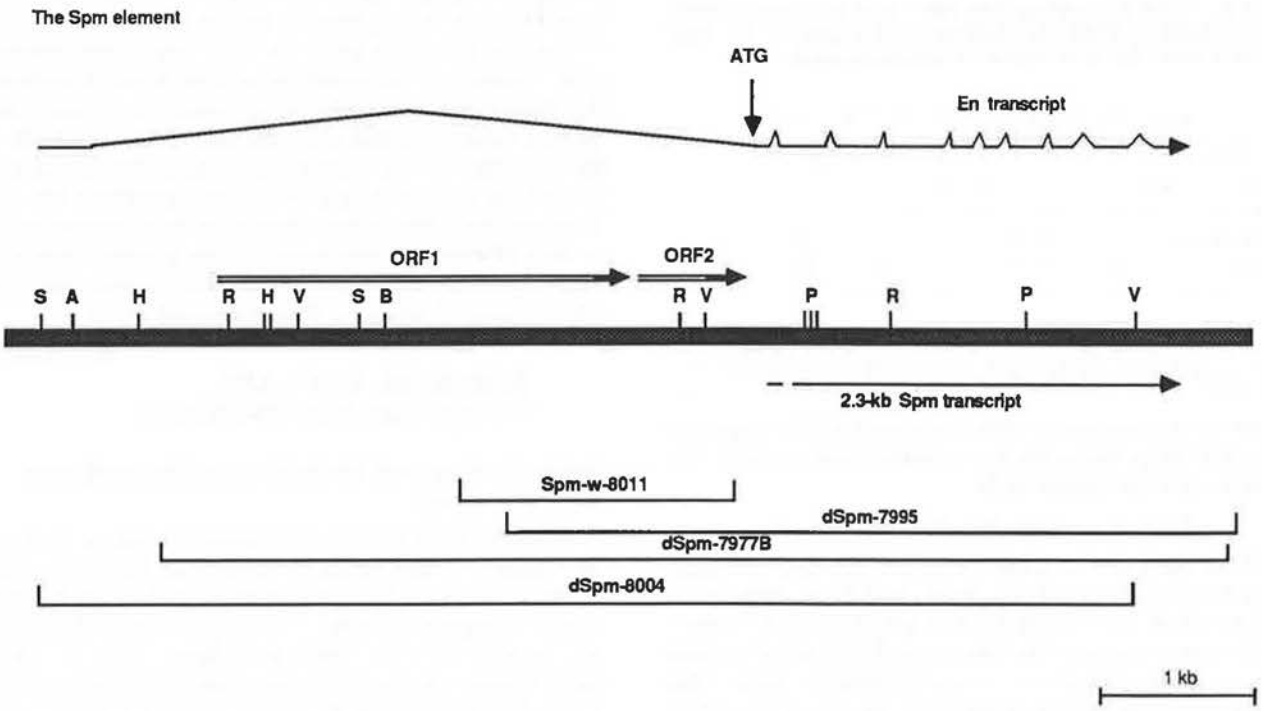

Figure 2. The *Spm* element. The parts of the *Spm* sequence deleted from each mutant element are indicated by the lines below the diagram of the complete element. The structure of the *En* transcript is reproduced from Pereira et al.

Spm element, while the *a-m2-8417* derivative has two deletions, consistent with its derivation in two steps from the original *a-m2* allele.

The 8.3-kb *Spm-s* element of the *a-m2-7991A1* allele has been cloned and sequenced; its structure is shown in Figure 2. The *Spm-s* element is virtually identical with the Enhancer (*En*) element sequenced by Pereira et al. (EMBO J. 5:835, 1986); it is shorter by 4 bp and differs at 6 additional nucleotides, only one of which affects the structure of the putative protein encoded by the element's major transcript. The two *Spm-s* alleles differ from each other and from the original *a-m2* allele primarily in the respective levels of *a* gene expression and the phenotypes of stable alleles resulting from excision of the element. We believe these differences may be attributable to small changes at the insertion site immediately adjacent to the element because we have found a 3-bp insertion adjacent to the sequenced *Spm-s* element that is not present in any of the other alleles analysed.

The 6.6-kb *Spm-w* element cloned from the *a-m2-8011* allele has an internal deletion, the location of which is shown in Figure 2. The abundance of the 2.3-kb major *Spm* transcript homologous to sequences in the element's right half (Figure 2) is lower by a factor of 5-9 in plants with an *Spm-w* element than in plants with an *Spm-s* element. The *Spm-w* element transposes less frequently and later in development than an *Spm-s* element, but retains the element's 'suppressor' function, as judged by its ability to inhibit expression of an *Spm-suppressible* insertion allele, such as the commonly used *a-m1-5719A1* allele of McClintock. Since the deletion in the *Spm-w-8011* element eliminates parts of both large open reading frames (ORFs) located in the element's left half, we conclude that the integrity of neither ORF is essential for expression of either known element-encoded function. The low transposition frequency of the *Spm-w* element is correlated with a reduction in the element's major transcript, suggesting that the transcript encodes the element's transposase. The observation that the element in the *a-m2-7995* allele is completely transposition defective and has an overlapping deletion that eliminates sequences hybridizing to the 2.3-kb *Spm* transcript supports this inference (Figure 2). The structure of the closely related *En* element's transcript is reproduced from Pereira et al. (op. cit.) in Figure 2. Since there are no differences in structure between the *En* and *Spm* elements that are likely to affect transcription, it is likely that the *Spm* transcription unit is the same as that of the *En* element. The *Spm-w* deletion is confined to the large intron that occupies most of the element's left half. We do not know whether the deletion's quantitative effect on *Spm* mRNA abundance is the result of decreased RNA stability or processing or the consequence of decreased transcription initiation.

Several lines of evidence suggest that the *Spm's* 'suppressor' and 'mutator' functions are not encoded by separate, complementing genes. All of the *dSpm* elements in the present series were tested genetically for each function and all lack both functions. The *a-m2-8167B* allele, which has a full-length element, was also tested for its ability to complement an *Spm-w* element and it could not. These observations, taken together with the observation that the *Spm-w-8011* deletion eliminates neither function, suggest that the element's two genetically defined functions either reside in the same protein or in proteins that share a subunit or domain and therefore coding sequences.

The frequency with which an internally deleted *Spm* element can be *trans*-activated to excise depends on its structure. Among the deleted elements whose structure is depicted in Figure 2, the *Spm-w-8011* element excises at a very high frequency in the presence of an *Spm-s*, the *dSpm-7995* and *-7977B* elements excise at an intermediate frequency, and the *dSpm-8004* element excises at a very low frequency. It has been shown in other studies (Schiefelbein et al., PNAS 82:4783, 1985; Schwarz-Sommer et al., EMBO J. 4:2439, 1985) that deletions that leave 1-1.5 kb of each element end do not affect excision frequency, while deletions that extend to within a few hundred nucleotides of an element end reduce excision frequency. The *Spm* element has 13-bp terminal inverted repeats (CACTACAAGAAAA) and a subterminal repetitive region at each end comprising several copies of the 12-bp sequence CCGACACTCTTA repeated in both orientations. It has been suggested by Schwarz-Sommer et al. (EMBO J. 4:2439, 1985) that the subterminal repeats form intramolecular duplexes in transposition and that deletions extending into the subterminal repeats reduce transposition frequency by disrupting secondary structure. Sequence analysis of cloned copies of the *dSpm-7995* and *-7977B* elements has revealed that the deletions extend into the subterminal repetitive region at the element's right end, eliminating 5 and 4 of the repeats, respectively. The excision frequency of these elements is lower than that of the longer *Spm-w-8011* element, but not much lower. We also find that the endpoints of intra-element deletions often occur within or at one end of the subterminal 12-bp repeats or a homologous sequence elsewhere within the element. Since it has been known for some time that intra-element deletions are element-catalysed, the implication is that an element-encoded protein capable of promoting the cleavage and religation of DNA (the transposase?) recognizes the 12-bp repeats. Thus we offer the alternative suggestion that the 12-bp sequence repeated near element termini is a recognition sequence for binding of an element-encoded protein, possibly the transposase itself.

Sequence analysis of the virtually immobile *dSpm-8004* element suggests the existence of an additional, non-repetitive determinant of excision (and, presumably, transposition) frequency. The *dSpm-8004* element retains all of the repeats at both element ends and has normal termini. The sequence present in the more frequently excising 1.3-kb *dSpm-7977B* allele that is missing from the 1.1-kb *dSpm-8004* allele comprises most of the element's first exon and a few hundred nucleotides of its first intron. Thus it appears that a non-repetitive sequence near, but not including, the element's transcription initiation site influences the element's mobility.

Perhaps the most interesting aspect of the *a-m2* alleles is that the *Spm* element controls expression of the *a* gene. The original *a-m2* allele exhibited an intermediate level of *a* gene expression, giving a palely pigmented kernel phenotype with small, deeply pigmented spots of normal *a* gene expression resulting from excision of the element. Expression of the *a* gene was affected by reversible chang-

es in expression of the element, as well as mutations within the element. McClintock (CIW Yrbk. 61:265,1962) reported that when the *Spm-s* element of the original allele became inactive, so did the *a* gene. The *a-m2-7991A1* allele resembles the original allele in *a* gene expression and we have isolated a derivative in which both element and locus are reversibly inactivated. We have observed that the inactive *Spm* element can be readily reactivated by the introduction of an *Spm-w* element, although we do not yet know whether the reactivation is heritable. This behavior suggests that 1) the *a* gene has come under the control of a mechanism that inactivates the *Spm* element and 2) that an element-encoded gene product can overcome inactivation of the element.

These inferences are strengthened by the behavior of the *dSpm* alleles. The phenotype of all of the *dSpm* derivatives studied here is colorless in the absence of an *Spm-s*. However, in the presence of an *Spm*, all except the *a-m2-8417* allele exhibit intermediate *a* gene expression. Hence intra-element deletions that inactivate the element also inactivate the gene. Yet expression of the *a* gene in *dSpm* derivatives remains under *Spm* control: the gene is expressed when an *Spm* element is present elsewhere in the genome. It follows that an *Spm*-encoded gene product can reactivate expression of the gene in *a-m2* derivatives with *dSpm* elements, just as it can reactivate an inactive *Spm* element. Thus it appears that expression of the *a* gene of the *a-m2* alleles reflects the operation of the *Spm*'s own regulatory mechanisms and suggests that the element encodes a positive regulatory function. Most of the derivatives used in the present study can be and have been tested for their ability to activate expression of the *a* gene of *dSpm* derivatives. We find that the *Spm-w* element can *trans*-activate *a* gene expression, while none of the *dSpm* elements can, suggesting that the element's positive regulatory function is not encoded by a separate, complementing gene.

Because *dSpm* elements as short as 1.3 and 1.1 kb are sufficient to mediate *a* gene activation, it appears that the target sequences for the element's regulatory protein are near its left end, which is located near the *a* gene's transcription start site. Differences among the *dSpm* derivatives in the capacity for *Spm*-dependent expression suggest that the minimal requisite element sequence is probably less than 300 bp of the element's left end, comprising the terminal inverted repeat, the subterminal repetitive region and the element's transcription initiation site. However, elements with at least an additional 1 kb of the element's left end express the *a* gene at a substantially higher level, suggesting that an internal element sequence can serve as an enhancer of *a* gene expression. The observation that *Spm* control of *a* gene expression is mediated by sequences around the element's own site of transcription initiation suggests that the element's positive regulatory mechanism functions at a transcriptional level. Thus we propose that an element-encoded protein interacts with sequences around the element's transcription start site to activate transcription of the element. We also suggest that the element's positive regulatory function can overcome the negative mechanism that reversibly inactivates the *Spm* element (as well as the *a* gene). The regulatory scheme that we propose is depicted in Figure 3.

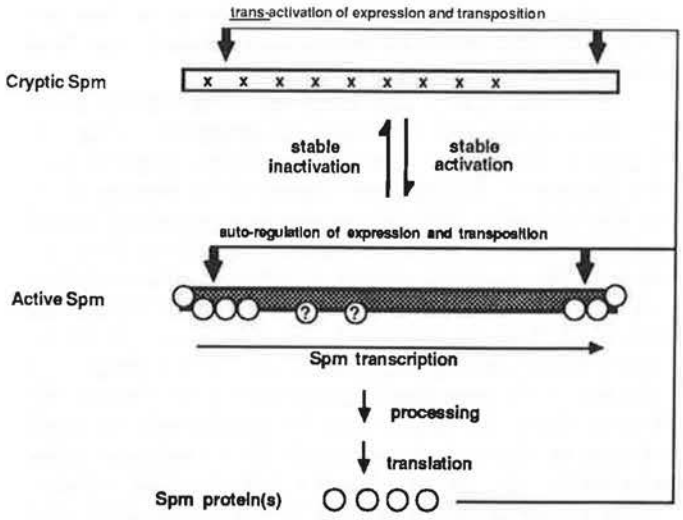

Figure 3. A model of *Spm* autoregulation. The open circles represent *Spm*-encoded proteins involved in transposition and positive autoregulation. The x's represent the mechanism by which the element is inactivated.

Although the nature of the negative mechanism that inactivates the *Spm* element has not yet been elucidated, there is growing evidence that maize transposable elements are inactivated by methylation (Chandler and Walbot, PNAS 83:1767, 1986; Cone, Burr and Burr, PNAS, in press). The results of the present study suggest that the inactivation system is probably not element encoded. This follows from the observation that the *a* gene is inactive in derivatives with extensive intra-element deletions. However, the possibility cannot be excluded that defective *Spm* elements in the genome participate in the inactivation process.

There is some evidence that *Spm*'s regulatory system not only overrides the negative mechanism, but can also interfere with it heritably. McClintock (CIW Yrbk. 63:592, 1964) reported that the *a* gene of the *a-m2-7995* and *-7977B* derivatives could be 'preset' for expression after segregation of the resident *Spm* element away from the allele at meiosis. Under such circumstances, kernels appear to commence development with an actively expressed *a* gene that returns to the inactive state during development, yielding kernels with an irregular and distinctive pigmentation pattern. Assuming that the same mechanism effects *a* gene inactivation in the *a-m2* dSpm derivatives and reversible inactivation of the *Spm* element, it follows that an *Spm*-encoded gene product can both directly overcome the inactivating mechanism and interfere with its propagation. This interpretation is supported by our previous report (MNL 60:18) that an *Spm-w* element substantially enhances the frequency of activation of cryptic elements. We suggest, therefore, that the *Spm* element's positive autoregulatory circuit functions to maintain the element in an active state both by promoting expression of the element directly and by interfering heritably with a negative, probably non-element-encoded, inactivating mechanism (Figure 3).

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Tissue-specific hypomethylation of maize rDNA

Last year we reported on a survey of the patterns of ribosomal DNA (rDNA) fragments in inbred lines of maize produced by digestion of leaf tissue DNA with the methylation-sensitive restriction enzyme *Hpa*II. Inbred lines were heterogeneous with respect to the number of bands produced by *Hpa*II digestion of nuclear DNA with either one, two, or three bands being present. R. Phillips et al. (Keystone Conference, 1985) have reported that DNA purified from leaf tissue of A188 has a single hypomethylated *Hpa*II site, but DNA from endosperm tissue harvested at 17 days after pollination (DAP) displayed four bands indicating that additional *Hpa*II sites were undermethylated.

We have initiated a survey of rDNA patterns from specific tissues of our previously assayed inbred lines to determine if there is a relationship between the heterogeneous methylation pattern in leaf tissue and the methylation pattern of other tissues. Figure 1 shows the result of

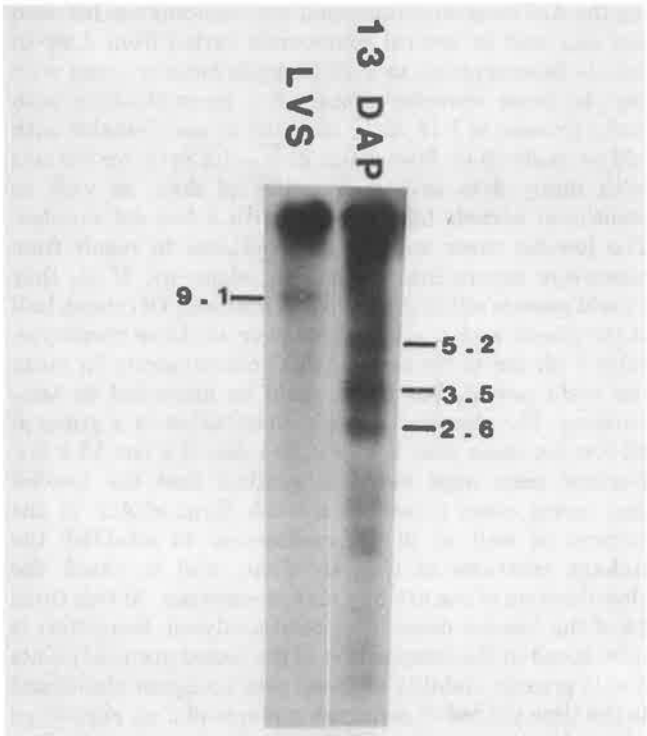

Figure 1. SX19 DNA, isolated from seedling leaves or from endosperm collected at 13 DAP, was digested with *Hpa*II, and fragments were separated by electrophoresis on 0.8% agarose gels. Fragments were transferred to nitrocellulose by Southern blotting and probed with a maize rDNA probe (pZmr1) containing the entire repeat unit (M.D. McMullen et al., Nucl. Ac. Res. 14:4953, 1986). Fragment sizes were derived from lambda DNA cut with *Hind*III. We wish to thank Pioneer Hi-Bred International, Inc. for providing SX19 seeds, and M.D. McMullen for providing the plasmid pZmr1.

*Hpa*II digests of DNA isolated from the hybrid SX19 (B73 X Mo17). *Hpa*II digestion of SX19 rDNA from leaf tissue produced a single 9.1 kbp band and left a significant uncut fraction. This result is identical to those we reported for the inbred parent lines in our communication last year. *Hpa*II digestion of 13 DAP endosperm rDNA produced three distinct new bands at 5.2, 3.5, and 2.6 kbp. The 9.1 kbp band is no longer visible, and there is considerable background smear in this lane indicating that, in general, the rDNA is less methylated. However, a significant fraction of the rDNA still is not accessible to *Hpa*II cleavage in the endosperm tissue. These results are similar to those previously reported by Phillips et al. in 17 DAP endosperm from A188. We are now examining rDNA hypomethylation in other tissues from this hybrid, in specific tissues from its progenitors, and from B37N.

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University of California

Programmed periclinal divisions of epidermal cells during glume development

We are using X-ray induced marked somatic clones to study floral development in plants carrying the Miniplant (*Mpl*) mutation. *Mpl* is a mild dominant dwarfing mutation, possibly allelic to *D8*, causing the formation of anthers in the florets of the ear. The clones are marked with the absolutely white phenotype conferred by the recessive lemon-white (*lw*) allele approximately 60 map units out on the long arm of chromosome 1. *Mpl* is also on this arm and is approximately 4 map units distal to *lw*. Plants heterozygous for *lw* (*Mpl Lw*/+*lw*) are irradiated (X-ray, 1000R) shortly after germination. Tissue derived from cells that have lost a region of the chromosome arm carrying the functional (*Lw*) allele are seen as white sectors in the mature plant. Each sector represents a patch of tissue developed clonally from a single cell. We have observed a number of sectors in ear husks. These husks were removed in order to permit greening of the florets, cupules and stem tissue beneath. In the normal ear exposure to light causes greening of the stem, cupules and glumes, just as it does in the homologous organs of the tassel. We identified several *lw* sectors which extended into the floral tissues resulting in completely white glumes, cupules and stem. These have enabled us to determine that the *Mpl* character is cell autonomous (manuscript in preparation). We also observed sectors which were almost completely white in appearance except for the presence of some narrow green streaks beginning at the glume tips and radiating outwards and downwards (Figure 1). Analysis of the white cupule tissue adjacent to these florets by fluorescence microscopy (illuminated at 395-440nm, fluorescence observed at 470nm) revealed the presence of epidermal guard cells containing chlorophyll indicating that this region consisted of a -/*lw* mesophyll covered by a *Lw*/*lw* epidermis.

These observations suggest the occurrence of programmed periclinal cell divisions of the epidermis during glume development. These divisions into the plane of the glume result in *Lw*/*lw* internal cells derived from the epidermis. The divisions are programmed because the green streaks are found in glumes of 15-20 adjacent florets in the same sector. They are programmed loosely because the streaking pattern in each floret is similar but not identical. It is interesting to note that the tip of the leaf, a homologous organ to the glume, is almost entirely derived from periclinal divisions of cells in the epidermal layer. This similarity between the glume and the leaf tip was pointed out to us by Scott Poethig. Maybe the differences in spatial

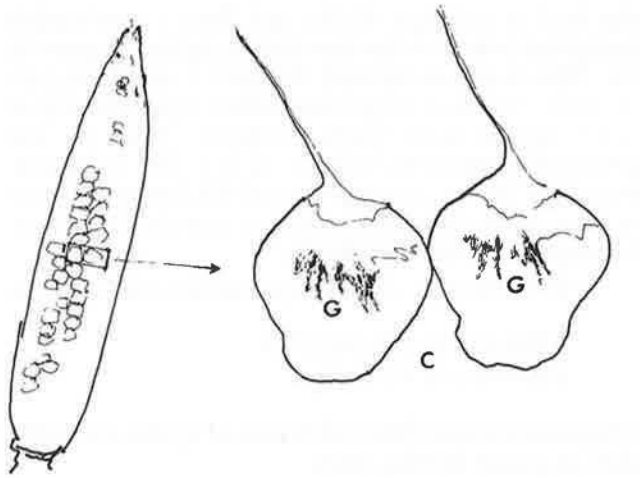

Figure 1. Two female florets from a sectored *Lw/lw* ear. In this sector the tissue was completely white (-/*lw*) except for the green streaks in the glumes (G) as indicated by the shading. The C indicates the cupule tissue that was examined by fluorescence microscopy and found to have *Lw/lw* epidermis.

organization of these epidermal divisions represent 'glume' and 'leaf' variants of homologue specific epidermal cell division programmes.

Nicholas Harberd, Sarah Hake and Michael Freeling

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Funk Seeds International

Mutation rates from sodium azide treatments

Last year (MNL 60:26, 1986) we reported on treatment procedures and physiological damage from treating maize kernels with sodium azide (NaN_3). This material has been selfed twice so that in the summer of 1986 we observed seedlings and plants (M3) for mutations (Table 1). The material is displayed similarly to last year's report in that maturity of the line and germplasm type are noted.

Table 1. Inbred designation, maturity, germplasm type and mutagen information from sodium azide kernel treatments

Inbred	Maturity[1]	Germplasm Type[2]	NaN_3 conc. Mol.	No. rows M3 plants	No. mutants	Mutation rate
42	M	LA	.001	100	24	24.0
42	M	LA	.01	11	1	9.1
43	M	LA	.001	20	8	40.0
51	M	LA	.001	11	2	18.2
32	E	SS	.001	100	22	22.0
33	M	SS	.001	9	1	11.1
54	M	SS	.001	11	0	0
B73	M	SS	.001	1	1	100.0
64	L	SS	.001	50	5	10.0
				313	64	20.4

[1]E, M, L - early, medium or late maturity respectively
[2]LA, SS - Lancaster or Stiff stalk germplasm respectively

In this study the following seedling and plant mutations were observed: various chlorophyll mutants, chlorotic leaf striping (both length and width), seedling lethality, leaf tip lethality, narrow leaf, crinkly leaf, round white spots on leaves, stature mutants and green plant base. All of these mutations were observed prior to pollination.

The 0.01M concentration under our treatment procedures is too high a dose since in Table 1 there is only one inbred, 42, that had survivors that were propagated to the M3 plant generation. However, in this instance the lower 0.001M concentration gave a higher mutation rate. The overall mutation rate is 20.4%.

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Do strong *Ac2* alleles represent duplications or triplications of a single *Ac* element?

When *Ac2/Ac2; bz2-m/bz2-m* plants are used as male parents in crosses to *bz2-m ac* testers, the endosperms of *ac ac Ac2* constitution show no dots. Occasional kernels are found with numerous dots and these have proved either to possess a more potent *Ac2* allele (which we have designated $\overline{AcAc}$) or to have two unlinked *Ac*'s. These alternative types were equally frequent and we were unable to predict from the number of *Bz* dots whether a kernel had $\overline{AcAc}$ or possessed two unlinked *Ac*'s. We have attempted to test the hypothesis that the potent *Ac2* allele is actually a duplication. These tests involve the closely linked marker genes, virescent-16 and male-sterile-8, flanking the *Ac2* locus on chromosome 8 (recombination between *v16* and *ms8* in several compounds varied from 3.3% in female heterozygotes to 7.4% in male heterozygotes with the *Ac* locus showing about 0.6% recombination with *ms8*.) Crosses of *V16* $\overline{AcAc}$ *Ms8/v16 ac ms8* females with *v16 ac ms8/v16 ac Ms8* males give a 1:1 ratio for kernels with many dots to kernels with no dots, as well as occasional kernels (about 1-2%) with a low dot number. The low-dot cases were at first believed to result from crossovers separating the two *Ac* elements. If so, they should possess either *v Ms* or *V ms* markers. Of course, half of the plants with the *V ms* crossover would be phenotypically *V Ms* due to the unavoidable heterozygosity for *ms* in the male parent, but these could be identified by testcrossing. The observed marker distribution in a group of 42 low-dot cases was: 8 *V Ms:* 19 *v Ms:* 0 *v ms:* 15 *v ms*. Further tests were made to confirm that the low-dot endosperm cases possessed a weak form of *Ac2* in the embryo as well as in the endosperm, to establish the linkage relations of the *Ac* allele, and to check the classification of the *v16* and *ms8* phenotypes. At this time, 16 of the low-dot cases have been analyzed. Some bias is introduced in the composition of the tested group of plants due to greater viability of green over virescent plants and to the time needed in genotyping plants of *v ms* phenotype where dominant marker alleles must be introduced before linkage of *Ac* on chromosome 8 can be tested. The 16 fully analyzed plants consisted of nine plants with *Ac* linked to chromosome 8, 8 *V Ms* and 1 *v Ms*, and seven plants with transposed *Ac*, 6 *v Ms* and 1 *v ms*.

There was no evidence that crossing over between *v* and *ms* was involved in the origin of the low-dot cases. The near equality of *v Ms* and *v ms* phenotypes in the group of 42 (due to the *Ms/ms* constitution of the male parent) suggests that most of the *v* plants are noncrossovers of *tr-Ac/ac* constitution arising when one component of the presumed duplication was transposed to a heterologous chromosome. Tests for linkage indicated that seven of the eight virescent plants possessed a transposed *Ac (tr-Ac)*. The absence of the *V ms* phenotype indicates that the *V Ms* plants are either *V Ac Ms/v ac Ms* or *V Ac Ms/v ac ms*

and not *V Ac ms/v ac ms*. The linkage phase of the flanking markers in the *V Ms* plants is the same as in the parent chromosome but one *Ac* component has been lost from the $\overline{AcAc}$ allele. Linkage tests with eight such plants showed the *Ac* was linked to chromosome 8 in every case.

Although attempts to demonstrate the duplicated nature of strong *Ac* alleles by association of crossing over with the origin of weak derivative alleles gave negative results, we still favor the interpretation of strong *Ac* alleles as duplications or triplications for the following reasons:

1. Similarity of the dot number in the endosperm of kernels with *Ac/ac/ac; tr-Ac/ac/ac* constitution and that found in $\overline{AcAc}$*/ac/ac* kernels. When unexpected dotted kernels occur in testcrosses of *Ac/Ac* male parents, the phenotype of those kernels shown to possess two independent activators is similar to that of individuals having the presumed duplication at the original locus. In female testcrosses, the *Ac/ac; tr-Ac/ac* plants produce 1 high-dot: 2 low-dot: 1 no-dot kernel ratios while $\overline{AcAc}$*/ac* plants give 1 high-dot: 1 no-dot ratios. Kernels in the high-dot classes in two testcrosses, although differing in genotype, cannot be distinguished phenotypically from each other.

2. Few, if any, one-step changes from a potent allele to a null form. In testcrosses of *V* $\overline{AcAc}$ *Ms/v ac ms* female parents, a change from $\overline{AcAc}$ to *ac* by excision of the $\overline{AcAc}$ as a unit would give a chromosome 8 with *V ac Ms* markers. From 1031 no-dot kernels, there were eight green normal and thirteen green male-sterile plants in addition to 894 virescent individuals. If the $\overline{AcAc}$ was completely excised, all the green plants should have normal tassels. Since more than half of the green plants were male-sterile, the 21 green plants can be accounted for as crossovers possessing the *V ac ms* chromosome with either the *v ac ms* (the thirteen green male-sterile) or the *v ac Ms* (eight green normal) chromosome contributed by the male parent. The absence of plants with a *V ac Ms* chromosome argues against the simultaneous loss of both *Ac* components and is in agreement with the map of *v16 - Ac - ms8*, a region so short that no double crossovers are expected.

Further evidence of the same sort comes from testcrosses of putative *V* $\overline{AcAc}$ *ms/v* $\overline{AcAcAc}$ *Ms* female parents where one chromosome possessed a noticeably stronger *Ac* than the homologue. Twenty-five kernels with low or no dots were selected from a testcross population of 897. Twenty-one of these produced plants possessing a *V Ac ms* chromosome (11 green normal and 10 green male-sterile) indicating a stepwise loss of a single component from the weaker of the two parental alleles. Tests of linkage with *v16* showed the remnant *Ac* is still on chromosome 8. The remaining four individuals consisted of one *v Ms*, two *v ms*, and one *V Ms* plants. The two *v ms* plants are crossover individuals derived either by loss of one component of the $\overline{AcAc}$ allele with a concomitant crossover between *Ac* and *v16* or by a crossover within the *Ac* complex following oblique synapsis of the components. The *v Ms* individual can also be accounted for by the same events but it received the *Ms* marker from the male parent. The linkage of *Ac* has not yet been determined in these three plants. The only exception to the proposed hypothesis of stepwise loss of *Ac* components is the single *V Ms* plant which seems to have lost both *Ac* components from the *V* $\overline{AcAc}$ *ms* chromosome but possesses the *Ms* allele from the male parent. In the great majority of cases where changes occurred in the *Ac* allele, a stepwise loss of a single component appears most likely. Similarly, newly transposed *Ac* alleles seem to consist of a single component. Cryptic transpositions of *Ac* from $\overline{AcAc}$*/ac* parents were identified by repeated testcrossing of all the dotted progeny and searching for ears with 3 mutable: 1 stable ratios. These represent $\overline{AcAc}$*/ac; tr-Ac/ac* individuals and the *tr-Ac's* have turned out to be weak *Ac* alleles with only one exception. A strong *Ac* allele consisting of a single element should be able to give rise to equally strong transposed alleles. For these reasons, we favor the duplication hypothesis as the explanation of strong *Ac2* alleles, but confirmation or rejection must await molecular analyses.

M.M. Rhoades and Ellen Dempsey

Do transposed *Ac2* elements arise by extra replication of existing elements or by excision from a parent allele?

Transpositions of *Ac* elements from $\overline{AcAc}$ alleles so far detected have included the low-dot cases mentioned above, where it is believed that a single component was transposed from the parent $\overline{AcAc}$ to a heterologous chromosome. In addition, transpositions have been identified in plants still possessing the parental $\overline{AcAc}$ (i.e., $\overline{AcAc}$ + *tr-Ac*). Other possible transpositions are very strong alleles ($\overline{AcAcAc}$) where the additional component may have come by transposition from the sister chromatid bearing $\overline{AcAc}$. We do not now believe these *Ac* elements arise by extra replication although this is very difficult to rule out. The evidence favors excision of one *Ac* component from a chromosome 8 chromatid having the $\overline{AcAc}$ allele and its insertion into either a homologous or heterologous chromatid. The remnant single *Ac* component on chromosome 8 has been found among our low-dot kernels from $\overline{AcAc}$*/ac* parents and similar remnant *Ac* alleles (weak alleles on chromosome 8) were present in testcross progeny of $\overline{AcAc}$*/*$\overline{AcAcAc}$ plants. Thus, changes in *Ac* constitution at the original location do occur and these weaker alleles cannot arise by extra replication. However, both the weak alleles (remnant *Ac*'s) and the transposed *Ac*'s can be explained by excision of an *Ac* component followed by its loss in the first case or its transposition and recovery in the second case. There is no need to invoke extra replication; all of the data can be accounted for by the excision hypothesis and by assuming the stronger *Ac* alleles are either duplications or triplications.

M.M. Rhoades and Ellen Dempsey

Bz reversions in the *bz2-m Ac2* system

Endosperms with three doses of the *bz2-m* responding allele and two or three doses of the *Ac2* controlling element display dots of *Bz* tissue. With rare exceptions, the dots are uniformly small indicating the *bz2-m* to *Bz* mutation occurs late in development of the endosperm. The *bz2-m* to *Bz* mutations also occur in plant tissue when high activity *Ac* elements ($\overline{AcAc}$) are present but with a reduced frequency. Again, the *Bz* sectors consist of a small number of cells and have been observed only in the anther wall. The question arose about the timing of *bz2-m* to *Bz* germinal mutations: is the opportunity for germinal mutation also restricted to a specific time and place?

A number of *bz2-m* plants of $\overline{AcAc}/Ac$ and $\overline{AcAc}/ac$ genotypes were used as male and female parents in crosses to *bz2-m ac* testers. A population of 21,892 kernels from female testcrosses showed only *bz2-m* mutable or *bz2-m* stable phenotypes, i.e., no *Bz* revertants were obtained. Reciprocal crosses produced 36 *Bz* kernels in a population of 8744, a frequency of 0.4% reversions in the male inflorescence. Not only does it appear that germinal mutation is confined to the staminate inflorescence but it could be localized even more precisely to a specific mitosis in the microspore. A number of *Bz* kernels from testcrosses of $\overline{AcAc}/ac$ or Ac/ac male parents were selected for further study. All thirteen cases analyzed proved to have the *Bz* allele in the embryo as well as in the endosperm and possessed either the $\overline{AcAc}$ or *Ac* allele from the heterozygous parent. If the *bz2-m* to *Bz* event took place before meiosis, one half of these cases should possess *ac* because of independent assortment of *Bz* on chromosome 1 and *Ac2* on chromosome 8. All *Bz* reversions possessed an *Ac* allele. If the *bz2-m* to *Bz* event occurred at the second microspore division, noncorrespondence of the two sperms would result and *Bz* endosperms should be associated with *bz2-m* embryos or vice versa. The 13 *Bz* cases all showed concordance of embryo and endosperm and, in a comparable test of *Bz* reversions from *Ac/Ac* and $\overline{AcAc}/\overline{AcAcAc}$ male parents, a total of 22 *Bz* individuals showed concordance in 21 and noncorrespondence in only one. The data clearly indicate that the first microspore division is the time when *bz2-m* to *Bz* mutations occur. Mutations after meiosis and before the second microspore division would lead to concordance of endosperm and embryo and the newly arisen *Bz* allele would be associated with *Ac*; this was observed.

It is not surprising that a somewhat higher rate of *Bz* germinal reversions was found in testcrosses of *V* $\overline{AcAc}$ *ms/v* $\overline{AcAcAc}$ Ms male parents. In a population of 1189, twelve reversions occurred, a frequency of 1.0%. All but one of the *Bz* plants carried the *v* marker of the more potent $\overline{AcAcAc}$ allele.

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Studies on a somatically unstable line

A maize ear isolated locally (by Poornima Rao) showing somatic instability for anthocyanin pigmentation in the aleurone tissue (purple dots and sectors), which is indicative of the presence of transposable elements, has been grown for two generations. Self-pollinated progenies of this ear showed unusual segregation for endosperm and plant characters. The following mutations have been isolated from this unstable line.

Chlorophyll mutations: High frequency of chlorophyll mutation has been observed in selfed progenies. Nearly 44 selfed ears showed abnormal segregation for yellowish green, virescent, japonica, zebra stripes and albino. A few of the yellowish green mutants are breeding true.

Opaque/floury: Nearly 6 out of 803 selfed ears showed sectors of varying sizes of opaque/floury phenotypes. In two cases a few kernels had fully opaque phenotype.

Shrunken: In the first generation, one ear showed few kernels (4 out of 152) with shrunken-like phenotype. These shrunken kernels bred true, but the expression of shrunken phenotype was varying from near shrunken to hollow endosperm. Ears derived from non-shrunken kernels from the same line showed shrunken sectors of varying sizes. Three more shrunken-like mutations have been isolated from 803 selfed ears in the second generation.

Instability in the aleurone tissue: Instability for anthocyanin pigmentation was also observed in the second generation.

These mutations are being studied for their breeding behaviour and allelism tests. Studies on the presence of Mutator element will be undertaken with the help of *Mu* and *Ac* clones.

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Differentiation and functional organization of the shoot apical meristem

I would like to summarize my thinking about the differentiation and functional organization of the shoot apical meristem of maize. The analysis of cell lineages during embryogenesis shows that the planes of early cell division neither specify the axis of bilateral symmetry nor demarcate the separation of major parts of the embryo or plant (R.S. Poethig et al., Dev. Biol. 117:392, 1986). This observation and the fact that more than one pattern of cell lineages during embryogenesis can be present in a plant strongly argue against the developmental significance of early cleavage patterns. In carrot, cell lineages of zygotic embryos correspond to Solanad type while those of the somatic embryos correspond to the Onagrad type (A.A. McWilliams et al., Ann. Bot. 38:243, 1974). In plants, by and large, the position of a cell rather than its lineal descent seems to determine the cell's fate.

The progenitors of the plumule-radicle axis become morphologically distinct approximately 10 to 12 days after pollination in the transition stage embryos (L.F. Randolph, J. Agr. Res. 55:881, 1936). The determination of the shoot meristem occurs just prior to or during its differentiation (Poethig et al., 1986). Auxin transport in immature embryos is known to be strictly basipetal and during embryogenesis physiologic polarity (auxin transport and auxin-cytokinin ratio) precedes morphogenetic polarity (S.C. Fry and E. Wangermann, New Phytol. 77:317, 1976; T. Przybyllok and W. Nagl, Z. Pflanzenphysiol. 84:463, 1977). It is reasonable to propose that because of polar transport, auxin accumulation at the radicle end could result in the induction and determination of the root meristem. The number of embryonic cells giving rise to root meristem is not known at this stage. Auxin and cytokinin could also be involved in the determination of the shoot meristem.

The fate of cells becomes progressively more restricted. The restriction process proceeds centripetally starting in the presumptive meristem field during the determination of the shoot meristem (Poethig et al., 1986) and continuing in the cells of the shoot apex during the formation of individual nodes (Figure 1).

The destiny of L-I cells of the apical meristem is shown schematically in Figure 1. These are two zones where cells

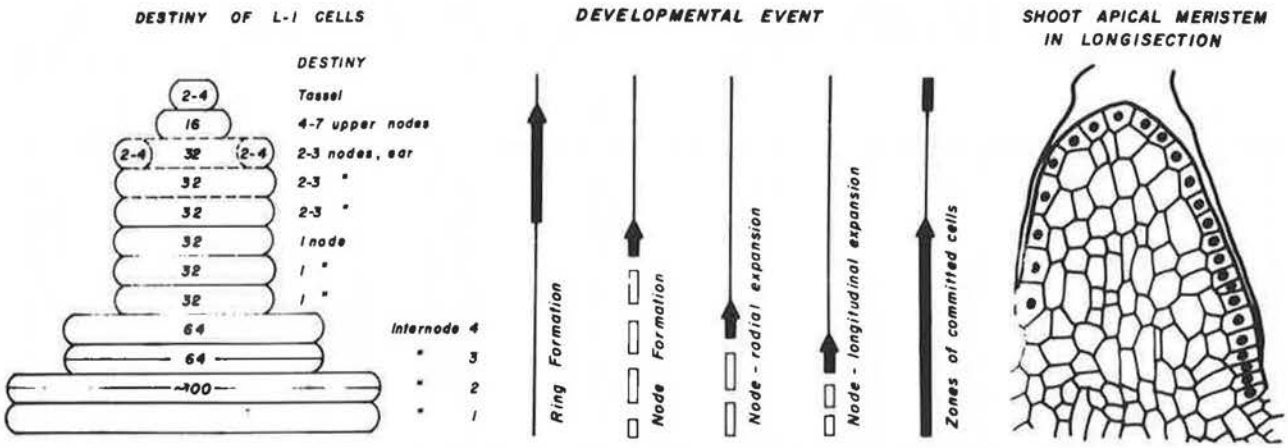

Figure 1. The destiny of L-I cells in the shoot apical meristem of the corn embryo (dry kernel stage) is depicted at the left. The solid arrows indicate the direction and location respectively of rings, differentiating nodes, radially expanding and lengthening internodes as deduced on the basis of lineage analysis. Shoot apex in longisection is shown at right.

have become committed to produce specific structures. The cells at the distal end and at the base are committed to produce, respectively, the tassel and the lower 6-7 nodes. The cells in between the two zones are relatively uncommitted and show a gradation of proliferation capacity. What does this gradation signify? Clearly these cells are in transition from an uncommitted state (i.e., cells of the ring) to become committed (the committed state being represented by cells destined to produce a specific node). The gradation therefore reflects the successive steps on the way to node differentiation from a 'ring'. While the embryo is developing, the potential of the cells committed to produce a node starts getting expressed and leads to the formation of leaves and internodes. The formation of nodes can be thought of as a wave of commitment process progressing towards the distal end. The development of the corn plant thus involves three successive and interrelated events, (a) formation of 'rings', (b) a commitment process to produce single nodes, and (c) expression of this commitment leading to the development of internodes. These events are separated from each other temporally and spatially only slightly and the shoot apical meristem represents a structure where the above events have been frozen at a specific instant of time in development.

To present an overview, it is visualized that first a rough outline of the shoot is laid down. This outline consists of tassel initials and a stack of 'rings' each of which is capable of producing a group of nodes. Then in the later part of embryonic development and continuing during the post-germination phase, a refinement process seems to occur. This process results in the production of specific individual nodes.

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A summing up of the latente systems, latente-1 stomatal control and linkages in chromosome 2S

In MNL 58:46-48 and 48-50 we demonstrated linkages of the Latente loci in a Cateto line: *Lte2* in chromosome 10 near position 56, and *Lte1* 19 units from *B* in chromosome arm 2S. This was shown by heat tolerance for the first locus and by aluminum tolerance for both loci. For heat and aluminum tolerance the Cateto system depends on the action of two pairs of complementary dominant genes. So far the latente-1 from Michoacán has shown effects only on chromosome 2 (see also MNL 56:28-30). Using the heat test *lte1* is linked with *wx* T2-9b and Inv2a, and not with *wx* T9-10b. So far our results show that the Cateto *Lte1 Lte2* is dominant or epistatic over the Michoacán *lte1* system.

In crosses with non-Cateto flint, and dents, the *lte1* is dominant. The latter statement is confirmed by the results of R.M. Castleberry and R.J. Lerette (Proc. 34th Ann. Corn and Sorghum Res. Conf., 1979). These germplasms have a bottom-most allele, for which we propose the symbol *lte-0*, or *lte-b*, for bottom.

L.T. de Miranda, L.E.C. de Miranda and E. Sawazaki in Ecological Genetics and Maize Breeding in Pt, Cargill Foundation ed. 30p. 1984, Campinas, SP, Brazil, and O. Brunini, L.T. de Miranda and E. Sawazaki in the Conference Internationale, Paris 11-14 September 1984, Les besoins en eau des cultures, in two papers in English, p. 205-212 and 361-368, present extensive comparative measurements of physiological effects in latente-1 and other maize genotypes. We suggested *lte1* has near it a factor for stomatal control, which is the object of this report.

A floury-1 originally received from Pennsylvania was crossed and backcrossed four more times before selfing to the dent lines IAC SLP103 (which is an old line selfed from the Yellow Tuxpeño collection San Luis Potosi 103, from former Oficina de Estudios Especiales, now CIMMYT) and IAC 723 (an old line selfed from Tuxpan, a Tuxpeño variety from Texas, reportedly obtained by introgressing yellow colour from Creolle Yellow Flint to a typical White Tuxpeño). So we had SLP103*fl1*(5) and a 723*fl1*(5). These *fl1* lines were crossed and backcrossed to two different *Fl1 lte1* IAC Maya latente lines. The *Fl1* and *fl1* seeds we planted in split-plot in the field nursery in Campinas, SP, Brazil. The readings were done between 1400 and 1600 since at this period the papers cited show the greatest differences among genotypes, on March 17, for the first and March 18, 1986, for the second family. The measurements were made 10-20 days after pollination, preferably on the ear leaf or the next below or above, depending on which was healthier. The equipment used was a Licor 1600 with which we measured percent relative humidity at equilibrium of the chamber with the leaf blade, leaf stomatal

IAC Maya latente line x IAC SLP103fl1 (2)							IAC Maya latente line x IAC 723fl1 (2)						
		Fl / fl	R.H.	Res	Tra	Score		Fl / fl	R.H.	Res	Tra	Score	Krn2 / krn2
1	2	3	4	5	6	7	8	9	10	11	12	13	14
1	5	+	53.6	3.54	4.33	+5.7	8	+	47.6	3.50	5.56	+5.0	+14
2	21	+	54.0	3.93	4.84	+5.7	16	+	50.0	2.97	5.42	+3.6	+16
3	30	+	48.8	3.61	5.77	+5.1	18	+	51.6	2.93	5.35	+3.3	+14
4	31	+	50.4	3.45	5.97	+4.4	17	+	51.6	2.89	5.42	+3.1	+14
5	29	+	52.4	3.25	6.15	+3.6	6	+	51.6	3.47	(6.48)	+3.1	+14
6	26	+	52.4	2.91	6.15	+3.1	20	+	54.4	2.66	5.48	+2.0	-12
7	32	+	54.0	2.63	6.35	+2.2	15	+	53.2	2.73	5.90	+1.9	-10
8	13	+	58.4	2.66	5.72	+2.1	2	+	54.4	2.94	(6.39)	+1.8	-10
9	18	+	55.6	2.57	6.61	+1.5	4	+	56.8	(2.35)	(6.92)	+0.7	-12
10	19	+	55.2	2.49	6.64	+1.4	19	+	56.8	(2.32)	5.71	+0.6	—
11	20	+	56.8	2.54	6.61	+1.3	7	+	55.6	2.28	6.93	-0.6	+16
12	10	+	58.8	2.58	6.57	+1.0	13	+	(57.2)	2.19	6.50	-0.6	-12
13	4	+	56.8	(2.16)	6.35	+1.0	5	+	60.0	2.34	7.40	-1.9	+14
14	12	+	58.4	2.50	6.66	+0.9	11	+	64.8	1.69	7.64	-4.3	-12
15	1	+	58.8	(2.07)	6.29	+0.5	1	+	64.8	1.61	7.58	-4.5	+14
16	6	+	59.6	2.30	6.66	+0.4	19	-	56.8	2.89	4.60	+3.0	+16
17	25	+	58.4	2.32	(6.95)	+0.3	9	-	52.4	2.98	6.14	+2.3	+14
18	15	+	(59.6)	2.19	6.95	-0.1	16	-	54.8	2.70	5.25	+2.3	-12
19	16	+	60.8	2.06	7.09	-0.7	8	-	52.8	3.01	6.19	+2.2	+14
20	20	+	62.0	1.72	6.87	-1.2	14	-	52,8	2.59	5.55	+2.1	+14
21	28	+	62.4	1.83	7.36	-1.6	18	-	54.8	2.59	5.20	+2.1	+16
22	11	+	63.2	1.78	7.22	-1.7	21	-	57.2	2.50	5.24	+1.4	+16
23	7	+	63.2	1.64	7.16	-2.2	1	-	55.2	2.53	6.20	+0.8	-12
24	23	+	64.4	1.71	7.61	-2.4	15	-	56.0	2.31	5.77	+0.7	-12
25	14	+	67.2	1.69	7.69	-3.0	11	-	54.8	2.36	6.15	+0.6	+14
26	8	+	67.6	1.37	7.58	-3.4	13	-	55.6	(2.31)	5.98	+0.5	+14
27	20	-	54.4	2.95	6.32	+2.6	7	-	(55.6)	2.36	6.61	-0.1	-12
28	5	-	56.8	2.40	6.35	+1.3	23	-	60.4	2.14	(5.61)	-0.4	-12
29	9	-	58.4	2.12	6.44	+0.5	12	-	(57.2)	2.16	(6.23)	-0.4	-12
30	21	-	58.8	2.37	(7.01)	+0.2	3	-	59.2	2.27	7.06	-1.5	-12
31	18	-	60.8	(2.27)	6.94	-0.2	22	-	62.8	1.81	(6.13)	-2.1	-12
32	7	-	(59.6)	1.81	(6.51)	-0.2	20	-	62.8	1.81	(6.14)	-2.1	+14
33	1	-	61.2	1.87	(6.54)	-0.4	24	-	63.6	1.68	(6.22)	-2.6	-12
34	2	-	69.6	1.51	7.65	-0.9	6	-	60.8	2.18	7.69	-2.7	-12
35	14	-	62.8	1.96	7.11	-1.2	25	-	65.6	1.54	6.34	-3.4	-12
36	16	-	63.2	1.93	7.16	-1.3	2	-	64.0	1.90	7.56	-3.8	-12
37	12	-	63.2	1.60	7.08	-1.8	4	-	65.2	1.81	8.07	-4.8	+14
38	23	-	66.8	1.53	7.99	-3.5	5	-	67.2	1.52	8.33	-6.0	-12
39	17	-	68.4	1.50	7.74	-3.5							
40	10	-	69.2	1.50	7.95	-3.9							
41	19	-	69.2	1.36	8.10	-4.3							
42	13	-	74.0	1.09	8.42	-5.9							
Mean			60.2	2.22	6.80				57.3	2.39	6.29		
s.e.			±0.9	±0.11	±0.13				±0.8	±0.08	±0.14		
c.v.%			9.4	29.2	12.0				8.6	21.3	14.1		
Lsc mean			55.7	2.73	6.23				53.9	2.73	5.76		
s.e. mean			±0.7	±0.12	±0.14				±0.5	±0.08	±0.12		
c.v.%			5.4	19.51	10.3				4.6	13.0	9.3		
lsc mean			64.7	1.71	7.37				61.6	1.96	6.94		
s.e.			±0.8	±0.06	±0.11				±0.9	±0.07	±0.19		
c.v.%			6.0	16.7	6.9				5.9	15.1	11.4		
7 & 13 score			$\frac{(60.2-R.H.)}{5.69}+\frac{(Res-2.22)}{0,67}+\frac{(6.80-Tra)}{0.94}$						$\frac{(57.3-R.H.)}{4.89}+\frac{(Res-2.39)}{0.57}+\frac{(6.27-Tra)}{0.89}$				

Table 1. Data from two families of backcross of floury-1 lines on lines with latente factor originated from Michoacán 21 Comp 1-104. Columns are: 1, classification by the score described at bottom of the table; 2, order of the plant in the field nursery; 3, flinty (+) vs. floury (-) endosperm; 4, relative humidity; 5, stomatal resistance; 6, transpiration; 7, score explained at bottom; 8 = 2; 9 = 3; 10 = 4; 11 = 5; 12 = 6; 13 = 7; 14, kernel row number; *Lsc* is *L*atente *s*tomatal *c*ontrol; Brackets, ( ), individual measurement not conforming to the score.

resistance (s.cm^{-1}), and transpiration in μg dm^{-2}s^{-1}. The measurements were made in zig-zag, alternating whenever possible in *Fl1* and *fl1* rows. The results and part of the analysis are shown in Table 1. The three physiological measurements made it possible to build a score for latente contrasting with the mean to give positive values for latente characteristics and dividing each by the standard error. The highest positive values give a greater degree of ascertainment for latente and the highest negative values more ascertainment for non-latente. In the harvested ears the kernel row numbers were counted. The χ^2 results for 103, 723, the total and the interaction results respectively were, for *Lsc1* and *Fl1*; 4.92*, 1.30, 5.80* and 0.42 with p = 29.6 ± 5.0, p = 40.4 ± 8.0 and mean of p = 35.0 ± 5.3. For *Lsc1* and *Krn2* only the second line gave indication with χ^2 = 2.78 and p = 29.7 ± 7.4. For *Fl1 Krn* the same line gave χ^2 = 0.30 and p = 45.3 ± 8.1. The values presented were calculated by the product moment method

since by additivity they give looser results. Transforming the values to map units we have the distances *Lsc Krn* = 34.3, *Lsc Fl* = 53.1 and *Krn Fl* = 67.2, which would lead to a *Krn Lsc Fl1* sequence. We think a more correct value will be arrived at by applying the "theorem of the differences" as illustrated empirically in MNL 58:38-46 with the standard *wx* translocations. In the present case the distance *Lsc Krn* is obtained by *Krn Fl1* - *Lsc Fl* = 67.2 - 53.1 = 14.1. *Lsc Fl* by *Krn Fl* - *Krn Lsc* = 67.2 - 34.3 = 32.9. We get *Krn* 14.1 *Lsc* 32.9 *Fl*. Subtracting from 32.9 the distance *B Fl* (19.0) we get 13.9 as the distance *Lsc B*, which agrees nicely with the previously reported *lte1 B* equal to 19.0. So latente-1 must be near position 30, and *Lsc* in 35. *Lsc* and *lte1* must be very near because of epistatic effects. There is no profit in closing the stomata more at highest heat, if you do not also have higher heat tolerance. The estimation of the relative position of *Krn* will agree with our previous work only if the directions of the genes were inverted in the first report, unless there is another gene for kernel row number, which is less probable. For *B Krn* distance, reported as 39, we now have 28. Note that both latente loci are very near mutagenic factors, *lte1 Lsc1* near *Mut*, mutator, and *Lte2* near *cm1*, chloroplast mutator. This must have an evolutionary meaning.

More precise location and study of *lte1 Lsc* probably will be obtained using *lg1 gl2* and also leaf water potential measurements, since latente-1 has 3-4 Bars less negative leaf water potential at all times of day. If or when available, isozymes of the abscisic acid biochemical pattern should be included, as should the substitution of cysteines responsible for the bridges of sulfhydryl-disulfide bonds (SH-SS), to understand its fine structure.

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Linkages in chromosomes 4, 5, 7 and 8 among kernel row number, fasciation, and T *wx* in crosses of adapted standard translocations with Português Fasciado

The authors (MNL 58:38-46), using 13 of the adapted standard translocations, T *wx* and Cateto Palha Roxa, reported positions and distances in centimorgans involving T, *wx*, *Krn* (kernel row number), *Flt* (flint against dent endosperm), and *Ger* (glucoside earworm resistance, which includes *Zer* and *Mer*). Now results are reported from crosses of 7 of the adapted standard translocations with a white dent variety with high kernel row number, Português Fasciado (PF, Fasciated Portuguese), probably HB 19.

S.E. Pego in a Ph.D. thesis (Genetic potential of Portuguese maize germplasm with abnormal ear shape, Iowa State University, 1982), studied PF. He concluded fasciation is not associated with *ra1*, *ra2* and *ra3* (ramosa), and a complex system of inheritance was proposed. In our experiment in direct comparisons PF gave 16.0 mean kernel row number, while our local IAC Maya gave a 12.3 mean. The cross with translocations was backcrossed to IAC Maya *wx*, and in one case we also obtained results from a self. The field plots consisted of one row 10m long planted in hills 0.4m within the row, with rows 1m apart. 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 waxy as pollinator and the experiment was detasseled. The dry ears were harvested indiscriminately, unhusked. They were classified for presence and absence of exit holes of corn earworm, for fasciated vs. non fasciated, for *Wx* vs. *wx*, for normal vs. semi-sterile ears, and kernel row number. In the crosses with the translocation at 5S.07 there was a segregation for

Table 1. Data from (Português Fasciado × translocations) × Maya *wx*. Abbreviations: *U* Unholed husks, *H* Holed husks by corn earworm; *Fas* Fasciated ears, *fas* Non fasciated ears; *Wx* Normal endosperm, *wx* Waxy endosperm; *N* Non-sterile ears, *S* Semi-sterile ears; *Krn* Kernel row number from 18 to 8 kernel row numbers; BC Back crosses; X/Y, X Colored aleurone, Y Non colored aleurone. *A2* Colored aleurone, *a2* uncolored aleurone?

				4L.33 BC			4L.90 BC			5S.07 BC(A2/a2)			5S.14 BC			7L.63			8L.35 BC			8L.35 Self	
U	Fas	Wx	N	Krn			Krn			Krn			Krn			Krn			Krn				
H	fas	wx	S	16	14	12	16	12	10	16	14	12	18	14	10	18	14	12	18	14	12	18	14
							14		8				16	12	8	16		10	16		10	16	12
+	+	+	+	1	0	0	0	0	0	0	0	0	4	2	0	0	0	0	3	3	0	0	0
+	+	+	-	2	0	0	0	0	0	0	0	0	2	2	0	1	1	1	1	1	1	1	0
+	+	-	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
+	+	-	-	0	0	0	1	5	1	0	0	0	1	6	1	0	0	0	1	0	0	1	1
+	-	+	+	2	6	4	0	5	4	0	0/1	0/3	2	4	0	2	3	6	0	3	3	0	0
+	-	+	-	0	1	8	3	0	2	0	1/1	0	0	7	0	0	4	7	1	4	3	0	2
+	-	-	+	2	0	4	1	2	0	0	0/3	2/0	2	2	0	2	4	4	3	0	2	1	0
+	-	-	-	2	4	4	2	0	0	0	0	0/5	6	13	0	0	4	6	0	3	8	1	0
-	+	+	+	0	2	0	0	0	0	0	0	0	9	3	0	0	1	0	4	1	0	1	0
-	+	+	-	0	0	0	0	0	0	0	0	1/0	2	0	0	0	0	0	2	2	0	3	0
-	+	-	+	1	0	0	0	0	0	0/1	0	0	0	0	0	0	0	0	0	0	0	1	0
-	+	-	-	2	0	0	0	0	0	0	0	0/1	2	1	0	0	1	2	1	1	0	7	1
-	-	+	+	4	5	3	1	3	2	1/0	4/2	5/0	9	6	0	5	5	5	1	5	0	0	1
-	-	+	-	0	3	4	1	1	3	0	1/0	3/4	2	12	1	0	2	8	2	5	3	0	1
-	-	-	+	2	0	1	0	1	0	0	2/0	5/4	0	2	1	0	3	8	0	1	2	4	2
-	-	-	-	2	4	7	0	4	4	0	0/2	5/1	7	21	0	1	14	18	0	3	11	3	3
				20	25	35	9	21	16	1/1	8/9	21/18	48	81	3	11	42	65	19	32	33	23	12
						80			46			30/28			132			118			84		35

Table 2. χ^2 analysis from data of Table 1.

Cross with	Wx N	Wx Krn	Wx Fas	Wx U	N Krn	Nx Fa	N U	Krn Fa	Krn U	Fa U
4L.33 BC	7.82**	+0.97	+0.14	+0.46	+0.40	+0.06	+0.05	11.85**	-1.27	-0.28
4L.90 BC	7.90**	0.01	-8.73**	-0.01	-1.51	-4.19	0.87	0.45	1.63	4.74*
5S.07 BC	0.11	1.46	-0.22	-0.70	3.24	-0.72	-0.00	0.00	0.34	-0.04
5L.14 BC	33.09**	4.16*	6.05*	-2.44	12.40**	5.77	-1.10	8.89**	-0.94	2.25
7L.63 BC	5.60*	4.30*	0.66	4.51*	8.51**	-2.14	1.08	0.41	0.42	0.07
8L.35 BC	5.62*	15.99**	9.33**	0.19	7.17**	2.47	1.03	23.25**	0.33	0.00
8L.35 Self	0.48	-0.40	0.24	0.97	-0.03	-2.91	-0.20	4.06*	-1.14	0.01

5S.07 A2 Wx = 1.15 A2 T = 1.05 A2 Krn = -0.21 A2 Fas = -0.42 A2 U = -9.62**

Table 3. p value calculated by the allometric method, with their accompanying allometric effects α and β with the data from Table 1. Extra data from 5S.07 at table bottom. Values within brackets are by the product moment method since by the allometric method the programed calculator did not give a solution.

	Wx T	Wx Krn	Wx Fas	Wx U	N Krn	T Fas	T U	Fas Krn	T Krn	U Fas
4L.33										
p=	34.0	48.6	42.2	46.2	39.1	49.5	48.8	38.7	43.7	43.0
α=	-0.100	-0.089	-0.042	0.002	0.061	0.024	0.054	0.776	-0.003	0.001
β=	0.066	-0.089	0.790	-0.091	-0.096	-0.791	0.000	0.183	0.091	-0.798
4L.90										
p=	28.4	47.7	-25.0	-49.2	34.1	-26.1	43.8	27.8	41.2	26.0
α=	-0.087	-0.053	-0.007	-0.063	0.126	0.117	0.130	0.627	-0.081	0.624
β=	0.148	0.509	-0.642	0.095	-0.525	-0.619	-0.098	-0.098	-0.224	-0.097
5S.07										
p=	47.6	42.2	-38.8	-43.0	36.0	-44.3	48.9	(49.6)	-49.3	-29.6
α=	0.051	0.037	-0.028	0.352	-0.116	-0.022	0.346	(1.049)	0.329	0.079
β=	0.101	0.260	-1.055	-0.056	0.273	-1.062	-0.092	(0.261)	-0.233	-0.880
5L.14										
p=	23.3	40.8	37.8	-43.2	35.9	42.2	-43.9	39.0	-44.4	45.1
α=	-0.058	-0.021	-0.031	-0.006	0.212	0.190	0.228	0.356	0.134	0.110
β=	0.265	0.206	0.378	-0.133	0.184	0.351	-0.135	0.165	-203	0.362
7L.63										
p=	39.5	38.2	49.5	40.8	32.6	(-32.2)	39.1	48.4	-46.8	-41.2
α=	0.091	0.041	0.017	0.088	0.055	(0.094)	0.133	0.998	0.161	0.976
β=	0.132	0.845	1.002	0.171	0.874	(-1.045)	0.213	0.014	-0.037	-0.016
8L.35 BC										
p=	35.2	(27.8)	27.3	47.6	32.6	44.2	44.6	15.1	47.1	-49.4
α=	-0.123	(-0.090)	-0.139	-0.104	0.216	0.168	0.193	0.474	0.031	0.396
β=	0.208	(0.156	0.439	0.001	0.179	0.366	0.028	0.098	0.157	-0.031
8L.35 S										
p=	-41.2	-41.7	44.7	39.5	-48.0	-32.5	44.4	30.0	-38.0	49.0
α=	0.029	0.570	0.919	1.141	1.583	0.964	-0.023	0.455	1.011	1.041
β=	0.109	0.926	0.669	1.091	1.182	0.778	0.103	0.188	0.506	0.691

5S.07	A2 Wx	A2 T	A2 Krn	A2 Fas	A2 U
p=	42.9	43.4	-47.1	-41.5	-27.4
α=	0.051	-0.100	-0.020	-0.026	0.383
β=	-0.027	-0.021	-0.259	-1.062	-0.022

aleurone colour which turned out to be due probably to *A2* and *a2* segregation. If anybody noted this in the original translocation material please report in MNL or write to us. Although the initial stand should be up to 450, leading after thinning to 300 plants at harvest, we got analyzable data from families of only 132 to 35 plants. This was due to the very low adaptation of PF. The plants simply died out. This is important to note because we used all treatments up to this number. We did not choose the results presented. At flowering time the overall conditions were unfavorable leading to very bad pollination. This explains the difficulty in separating normal from semi-sterile ears, leading to loose direct values of p for the T *wx* linkage. The results are presented in Table 1. In Table 2 the χ^2 analysis is done. The p values are presented in Table 3. The p values were calculated by the allometric method presented in a prior work, the formula being:

$$\frac{b(1+\alpha-\beta)+c(1-\alpha+\beta)}{a(1+\alpha+\beta)+b(1+\alpha-\beta)+c(1-\alpha+\beta)+d(1-\alpha-\beta)}-p=0$$

The p values were transformed into cM and using our "theorem of the differences" we got the cM values as the mean of the k-2 estimates available, k being the number of factors including T.

We made a modification in relation to the MNL 58:39-46 report in taking the algebraic mean of the differences, and not the mean of their absolute values as in the prior work. The distances, with their standard error, are presented in Table 4. Here we worked out the variances of the estimates.

Table 4. Linkage sequences in cM of T *wx Krn Fas* in crosses of Português Fasciado with the indicated translocations. p values of Table 3 were calculated by the allometric method. Then it was transformed to cM and the distances arrived at by the theorem of the differences (see MNL 58:38-46), taking the algebraic mean of the differences. Variances were gotten by first transforming V_p to V_{cM} which is $V_{cM} = (cM / p)^2 \times V_p$. A first error estimate is the sum of the four V_{cM} divided by 4 (two contrasts squared). In 5S.07, six V_{cM} divided by nine (three squared). The relative size and sign of the covariances was calculated with the cM values used and subtracted two times if positive or added if negative, to the first error estimate.

											n
4L.33			Wx	0.7 ±7.4	Krn4	0.8 ±12.4	T	7.7 ±13.0	Fas4		80
4L.90	Krn4	6.1 ±3.6	Wx	18.7 ±6.8	T	7.0 ±4.0	Fas4				46
5S.07	T	0.8 ±13.2	Wx	2.0 ±7.8	a2	6.9 ±9.6	Fas5	2.9 ±12.4	Krn5		58
5L.14	Fas5	12.4 ±6.6	Wx	12.6 ±5.4	Krn5	22.8 ±8.0	T				132
7L.63	T	22.8 ±6.6	Krn7	12.6 ±7.3	Wx	12.4 ±6.6	Fas7				118
8L.35(BC+S)	T	8.8 ±4.4	Wx	3.7 ±7.9	Fas8	5.4 ±3.4	Krn8				52 35

V_p was calculated by the classical method since we saw by the exact allometric method variance formula the 2 added in the numerator is balanced by the value within brackets in the denominator which is very near 2. The covariances are a much more important component of the error estimate. To transform V_p to V_{cM} we make $V_{cM} = (cM/p)^2 \times V_p$. A first variance of the measure obtained by differences is the sum of the four V_{cM} used in the two contrasts, divided by the square of the number of contrasts (two squared). As this doesn't take into account the covariances with the four cM values, we calculated a second new variance and a covariance. Remember $V_{a+b} = V_a + V_b - 2\,Cov_{ab}$. With this we know the proportions and signal of covariance, and the variance. With this proportion we correct the first variance calculated. If the covariance is positive it will be diminished, if negative increased.

In estimates of T 8L.35 backcross and selfs the p values were combined by their quantity of information. For 5S.07 the estimate *Fas Krn* was not utilized in the calculus. It is apparent that, as for kernel row number, factors for fasciation are as, or nearly as, ubiquitous. Really it seems they go around in pairs as *tr*, two ranked, and *pd* paired to which they are probably allelic, domesticated and wild ones.

At loose linkages allometric and classic methods gave very similar results. There is an increasing difference as p tends to zero. This is the same as reported for the F2 case.

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Allometric genetics: development of methods which permit mapping of non-Mendelian unit segregations (low penetrance)

In MNL 55:18-19 we presented a graphical interpretation of what we called allometric genetics. Here we present a mathematical demonstration. It is based on a derivation by I. L. Gridi-Papp (Dr. Thesis Agronomy School, Piracicaba, SP, Brazil, 1970) which, although correct, was not developed to our present full interpretations.

The velocity of growth of one certain character in a certain moment is a function of the dimension already attained (X), of time (t), and of a factor of proportionality that depends on the character and the genotype:

$$dX/dt = K_X\, f(X,t) \qquad (1)$$

Time (t) is considered as a measure not only from the organ but also from the variations of environment.

Formula (1) expresses a hypothetical relationship because the function f(X,t) remains unknown. Nevertheless, because at time $t=0$, $X=0$, its primitive has the form:

$$X = K_X\, F(X,t) \qquad (2)$$

F (X,t) is also unknown. To get some information about this function it is necessary to consider within the same organ a second dimension, that which presents itself as the character most influenced by the size of the organ. If (Y) is this character it can be formulated:

$$Y = K_y\, F(Y,t) \qquad (3)$$

Thus equal values of (t) correspond to a (Y) and an (X).

The relative behaviour of two characters of the same organ such as (Y) and (X) has been the object of investigations of several authors in the past and was described by the so-called allometry law proposed by J.S. Huxley in 1932. This law is based on constant growth velocities, which can be written as $d(\log X)/dt = \alpha\, d(\log Y)/dt$, where α is a constant. Integrating we get $\log X = \alpha \log Y + \log A$. We substitute A for C, the usual constant of integration, because A is really C and is the usual symbol used in allometry.

Log A is constant for a given pair of characters. It follows that:

$$X = AY^{\alpha} \text{ or } X/Y^{\alpha} = A \qquad (4)$$

By (2) and (3) comes

$$\frac{X}{Y} = \frac{K_X F(Y,t)}{K_y F(X,t)} \qquad (5)$$

A comparison between (4) and (5) suggests choosing the constant A such that $A = a(K_X/Ky)$, where a is a factor of proportionality, which means that $X = a(K_X/K_y)Y^{\alpha} = AY^{\alpha}$ and that $F(X,t)/F(Y,t) = AY^{\alpha-1} = X/Y$, with $X' = X/Y$, then $X' = AY^{\alpha-1}$ and we have shown in our prior work that $X' = (100/Y)^{1-\alpha}$. $1-\alpha$ is the more nearly biologically correct value of half the difference between the different homozygotes for the same locus. $2(1-\alpha)$ is the logarithm of A, the constant of integration of the "law of allometry".

Using α in the expectations of the appropriate classes in the observed phenotypes a mathematically exact solution can be derived. With only one type of family we must have also an allometric measure of the homozygotes, say *BB* against *bb*. With two different types of families a simultaneous solution can be derived for α and p, even if only by iterative methods, though not necessarily.

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Linkage between *Ga-S* and *o1*

In MNL 59:24 the authors reported data which would put *o1* at about position 80 in chromosome four. As its prior position in the working maps was not changed we present more data to improve our earlier estimates. *Ga-S 01* was crossed to *ga o1* and selfed. The data from three ears together with that from the prior report are in Table 1, including the analysis.

Table 1. Results from the selfed cross, *Ga-S 01* × *ga o1*. First line data from MNL 59:24. Distance *Ga-S* to *o1* turns out to be 52.9 units (cM).

Fenotypes				χ^2 deviations from 3:1	Recombination values with standard errors
	01	o2	Total		
fam 1	216	51	267	4.95*	38.2±4.8
fam 2	226	58	284	3.17	40.8±4.8
fam 3	321	77	398	6.78**	38.7±4.0
fam 4	340	92	432	3.16	42.6±3.9
Sum of individual chi-squares				18.07	
Total	1103	278	1381	17.46**	40.3±2.2
Interaction among families				0.61	0.90>P>0.80

The value of p with the total of the four families is 40.3 ± 2.2, which in cM is 52.9. Among the four families there is insignificant interaction. Based on *Ga-S* position in the working map this puts *o1* at about position 85 in chromosome four. The strong linkage depicted around glossy-3 should be depicted around glossy-4 which possibly the original report established, if the linkage were so strong.

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Allometric genetics III: A universal mathematical solution for the calculation of recombination values

Since the report "Allometric genetics" in MNL 55:18-19 we have been trying to unify allometric and genetic parameters in one body of theory and practical use. In that first report a graphic interpretation of gene effects was made in terms of α, the coefficient of allometry. Also it was shown how it could be used in quantitative genetics. In a preceding report in this volume a mathematical demonstration was done, completing the graphic one. It remains now to show how to use it in Mendelian genetics, and this is the objective of the present report.

For generations biometrists have calculated from a 2x2 table four parameters—a mean, a linear effect for the lines, another for the rows, and the interaction between them. Mendelian geneticists compute only one parameter, p, the recombination value. This seriously limits the range of work to near perfect segregations, 1:1:1:1, 3:1, etc. Segregations giving significant decimal digits are ignored. As a result, this field of work comprises only a small sample of biological variability. The usual method used is the Maximum Likelihood (K. Mather, "The measurement of linkage in heredity"). For more disturbed segregations, the Minimum Moment of the Products Method, also known as the Product Ratio, is used. We have never found a derivation, and for the particular case of the backcross we will show also how it can be done, as one more proof of the mathematical precision of the allometric solution proposed.

Looking at the most upward diagonal in MNL 55:18-19, we can imagine that the effect of a genetic factor *A* is $+\alpha$, and of *a*, $-\alpha$. Taking a second pair of allelles, *B* is $+\beta$, and *b* is $-\beta$. The combinations of these allometric coefficients are put in the theoretical frequency expectations, and equated to the observed ones. *AA* is a, *Ab* is b, *aB* is c and *bb* is d, and n is the family size as usual.

	B	b
A	$\left(\frac{1-p}{2}\right)^{1+\alpha+\beta} = \frac{a}{n}$	$\left(\frac{p}{2}\right)^{1+\alpha-\beta} = \frac{b}{n}$
a	$\left(\frac{p}{2}\right)^{1-\alpha+\beta} = \frac{c}{n}$	$\left(\frac{1-p}{2}\right)^{1-\alpha-\beta} = \frac{d}{n}$

If we make

$$-\frac{a+n}{\left(\frac{1-p}{2}\right)^{1+\alpha+\beta}} + \frac{b+n}{\left(\frac{p}{2}\right)^{1+\alpha-\beta}} + \frac{c+n}{\left(\frac{p}{2}\right)^{1-\alpha+\beta}} - \frac{d+n}{\left(\frac{1-p}{2}\right)^{1-\alpha-\beta}} = 0$$

we have the exact numerical solution for the product moment method which is the solution of $(1-p)^2/p^2 = (a \times d) / (b \times c)$, although nobody ever used it but us.

By the maximum likelihood method we take from the 2 x 2 table

$$L = \frac{a}{n}(1+\alpha+\beta)\ln\left(\frac{1-p}{2}\right) + \frac{b}{n}(1+\alpha-\beta)\ln\left(\frac{p}{2}\right) + \frac{c}{n}(1-\alpha+\beta)\ln\left(\frac{p}{2}\right) + \frac{d}{n}(1-\alpha-\beta)\ln\left(\frac{1-p}{2}\right)$$

Deriving in relation to p, after algebraic manipulation we arrive at

$$\frac{\delta L}{\delta p} = -\frac{a(1+\alpha+\beta)}{1-p} + \frac{b(1+\alpha-\beta)}{p} + \frac{c(1-\alpha+\beta)}{p} - \frac{a(1-\alpha-\beta)}{1-p}$$

And the value of p is expressed by the equation

$$p = \frac{b(1+\alpha-\beta)+c(1-\alpha+\beta)}{a(1+\alpha+\beta)+b(1+\alpha-\beta)+c(1-\alpha+\beta)+d(1-\alpha-\beta)}$$

The second derivative is

$$\frac{\delta^2 L}{\delta p^2} = -\frac{n}{2}\left[\frac{p(1-p)^{\alpha+\beta}(1+\alpha+\beta)+(1-p)p^{\alpha-\beta}(1+\alpha+\beta)+(1-p)p^{-\alpha+\beta}(1-\alpha+\beta)+p(1-p)^{-\alpha-\beta}(1-\alpha-\beta)}{p(1-p)}\right]$$

And the variance of p is

$$V_p = \frac{2p(1-p)}{n\left[p(1-p)^{\alpha+\beta}(1+\alpha+\beta)+(1-p)p^{\alpha-\beta}(1+\alpha-\beta)+(1-p)p^{-\alpha+\beta}(1-\alpha+\beta)+p(1-p)^{-\alpha-\beta}(1-\alpha-\beta)\right]}$$

The computation of the p value is cumbersome, but it can be arrived at by iterative methods and the logical values used compared with the results obtained by the classical methods.

For any other type of family to get the solution for p it is necessary just to add the appropriate α and β to the solution arrived at by the maximum likelihood expression.

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Allometric genetics IV: calculation of recombination values in F2 of *tr* and *pd* with genetic markers from chromosomes 1 to 6

In a preceding report we have shown the exact procedures, expurgated from allometric effects, to calculate p from backcross progenies. Here we will derive the formulas for F2 and illustrate them with data from D.H. Langham (Genetics 25:88-107).

The observed and expected values in a F2 segregation can be described as follows in a 2x2 table:

	B	b
A	$\left(\frac{2+p}{4}\right)^{1+\alpha+\beta} = \frac{a}{n}$	$\left(\frac{1-p}{4}\right)^{1+\alpha-\beta} = \frac{b}{n}$
a	$\left(\frac{1-p}{4}\right)^{1-\alpha+\beta} = \frac{c}{n}$	$\left(\frac{p}{4}\right)^{1-\alpha-\beta} = \frac{d}{n}$

The symbols are standard, and use β for the allometric effect of *B* gene and α for *A* gene to account for differential penetrance or viability.

The logarithmic likelihood expression turns to

$$L=\frac{a}{n}(1+\alpha+\beta)\ln(\frac{2+P}{4})+\frac{b}{n}(1+\alpha-\beta)\ln(\frac{1-P}{4})+\frac{c}{n}(1-\alpha+\beta)\ln(\frac{1-P}{4})+\frac{d}{n}(1-\alpha-\beta)\ln(\frac{P}{4})$$

Deriving and making some algebraic manipulations we arrive at:

$$\frac{\delta L}{\delta P}=\frac{a(1+\alpha+\beta)}{2+P}-\frac{b(1+\alpha-\beta)}{1-P}-\frac{c(1-\alpha+\beta)}{1-P}+\frac{d(1-\alpha-\beta)}{P}$$

The value of P is expressed by the equation

$$P^2\{a(1+\alpha+\beta)+b(1+\alpha-\beta)+c(1-\alpha+\beta)+d(1-\alpha-\beta)\}+$$

$$-P\{a(1+\alpha+\beta)-2b(1+\alpha-\beta)-2c(1-\alpha+\beta)-d(1-\alpha-\beta)\}+$$

$$-2d(1-\alpha-\beta)=0 \qquad (1)$$

The second derivative is

$$\frac{\delta^2 L}{\delta P^2}=-\frac{n}{4}\left\{\frac{(2+P)^{\alpha+\beta}(1+\alpha+\beta)}{2+P}+\frac{(1-P)^{\alpha-\beta}(1+\alpha-\beta)}{1-P}+\frac{(1-P)^{-\alpha+\beta}(1-\alpha+\beta)}{1-P}+\frac{P^{-\alpha-\beta}(1-\alpha-\beta)}{P}\right\}$$

And the variance of P is

$$V_P=\frac{4}{n}\left\{\frac{1}{(2+P)^{-1+\alpha+\beta}(1+\alpha+\beta)}+\frac{1}{(1-P)^{-1+\alpha-\beta}(1+\alpha-\beta)}+\frac{1}{(1-P)^{-1-\alpha+\beta}(1-\alpha+\beta)}+\right.$$

$$\left.\frac{1}{P^{-1-\alpha-\beta}(1-\alpha-\beta)}\right\}$$

D.H. Langham studied linkages of *pd* and *tr* of Durango teosinte with maize markers. His chief conclusion is that the gene *tr* is linked to the gene *pd*, with 20 percent recombination between only one pair of loci. We will try to show that his conclusions are very conservative. In Table 1 we present the results by the allometric method here proposed; with only one more digit than Langham used. He calculated p by the product moment method. In 28 cases we have 23 cases with a value of p below 0.5. The chi-square gives 11.57**, significant beyond any reasonable doubt. This means that for the whole, the markers in general are linked to *tr* and *pd*, and so there are *tr* or *pd* genes or both in all or nearly all chromosomes tested. Langham's data do not permit the calculation within each chromosome of *pd* with *tr* nor the linkages among the different markers used. This removes much of the efficiency of our method of the differences, but with the available data we applied it making algebraic means of the differences.

Comparing Langham's p values with ours we verify a very close agreement for values of p between 0.4-0.5 (within 1%). Below 0.4 downwards to 0.3 the difference increases to nearly 2%. That is, it seems that the product ratio method is almost fully efficient in eliminating allometric effects at loose linkages in F2. Its efficiency falls as p tends to zero and then our exact method should be used profitably. Regarding fasciation, Langham reported "A mutation to single spikelets in an inbred line of maize was discovered by the writer. When he crossed maize with teosinte the F1 ears had single spikelets instead of the usual paired spikelets. The F2 ears likewise, were homozygous for single spikelets. The inbred line of maize used in this cross has a fasciated type of ear, and its single spikelets would not have been noticed except for the cross with teosinte" [sic]. We regard *tr* and *pd* as allelic to our *Krn* and *Fas* reported elsewhere.

The error variance was first calculated by the standard F2 formula (the p values by both methods are very near). The variance of cM was first calculated as $V_{cM} = (cM/p)^2 \times V_p$. A covariance was calculated with the cM values used in the calculus. If positive we let it be. If negative it was added two times to the original variance of cM. Thus, it is a maximum error variance.

For #1 we have arrived at direct solitary measures in cM of *F1 Pd1* 52.0 ± 7.2 and *Bm2 Tr1* 48.4 ± 7.1. With four measures by differences we have *Pd1 Tr1* 23.8 ± 12.4, between *F1 Bm2*. Intrapolating the sum of the three measurements for the *F1 Bm* distance we arrive at (*F1* 86) (*Pd1* 117) (*Tr1* 132) (*Bm2* 161).

For #2 by differences, with four measurements we have *Pd Tr* 13.2 ± 6.1 *lg1 gl2* by one pair difference gives 2.0 ± 10.1. *Lg1 Tr* gave 59.9 ± 7.0. *B* results are unreliable and *V4* is too far away although congruent and we

Table 1. Reanalysis of Table 7 and 9 from D.G. Langham in Genetics 25:88-107, 1940. His values were calculated by the product moment method. Ours were by the allometric method described in the text. There are more differences between both methods only in the p values below 0.4. With these p values we calculated the linkage sequences. See text for details. α and β are the allometric effects of A and B genes, lines and rows.

			Pd (Fas)			Tr (Krn)			
Chrom	gene	Linkage phase	p	α	β	p	α	β	n
1	Fl	R	39.9	-0.123	-0.106	48.3	-0.128	-0.031	225
	Bm^2	R	49.9	-0.046	-0.102	38.3	-0.036	-0.029	225
2	Lgl	R	45.9	-0.043	-0.127	43.1	-0.029	-0.005	257
	Gll	R	48.0	0.024	-0.137	45.1	0.031	0.002	257
	B	C	64.5	0.289	0.207	46.2	0.251	-0.107	115
	V_4	R	48.4	-0.091	-0.129	48.1	-0.117	-0.008	257
3	Al	R	43.1	0.062	-0.187	42.5	0.066	-0.070	160
	Lg2	R	31.2	-0.031	-0.197	42.9	-0.027	-0.058	160
4	Ts5	C	41.7	0.089	0.038	36.7	0.502	-0.110	93
	Su	R	51.8	-0.214	0.173	58.2	-0.237	-0.149	83
5	Pr	R	49.5	0.032	0.032	42.5	0.066	0.210	101
6	Y	C	50.4	0.012	-0.123	46.5	0.018	-0.000	257
	Pl	C	48.6	0.125	0.034	53.8	0.151	0.205	145
	Py	R	41.2	-0.164	0.107	29.7	-0.256	0.033	300

arrive at (*Pd2*-45) (*Tr2*-32) (*lg1* 28) (*gl2* 30). In the mapping of *Lsc* (latente stomatal control) a *Krn2* was found in position 10. Here *Tr2 lg1* distance is grossly overestimated by direct measurement.

For #3 the single measure *lg2 Pd3* is 36.5 ± 8.3. *Tr3 a1* gives 42.6 ± 8.8. By difference with two pairs we have *Pd3 Tr3* 10.7 ± 9.0. Intrapolating the total of the three measures in the known distance *lg2 a1* we have (*lg2* 93) (*Pd3* 113) (*Tr3* 118) (*a1* 141).

For #4 distance *Pd4 Tr4* by one single pair difference 10.2 ± 12.4 *Tr Ts* directly is 45.6 ± 14.4. Most probable sequence is (*Pd4* 3) (*Tr4* 7) (*Ts5* 53).

For #5 by difference of the only pair available *Pd5 Tr5* is 41.0 ± 20.4, and the order is (*Pr* 67) (*Tr5* 125) (*Pd5* 166) with no indication of orientation.

For #6 we have a single estimate for *Py Tr6* 34.1 ± 6.0 and a single pair difference for *Tr6 Pd6* 20.8 ± 8.7 which gives the sequence (*Py1* 65) (*Tr6* 99) (*Pd6* 119) in the map positions.

There seems to be a certain correspondence between *Pd Tr* and knob position within each chromosome in all cases.

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Following the paths from wild to cultivated maize: a cytogenetic mine map

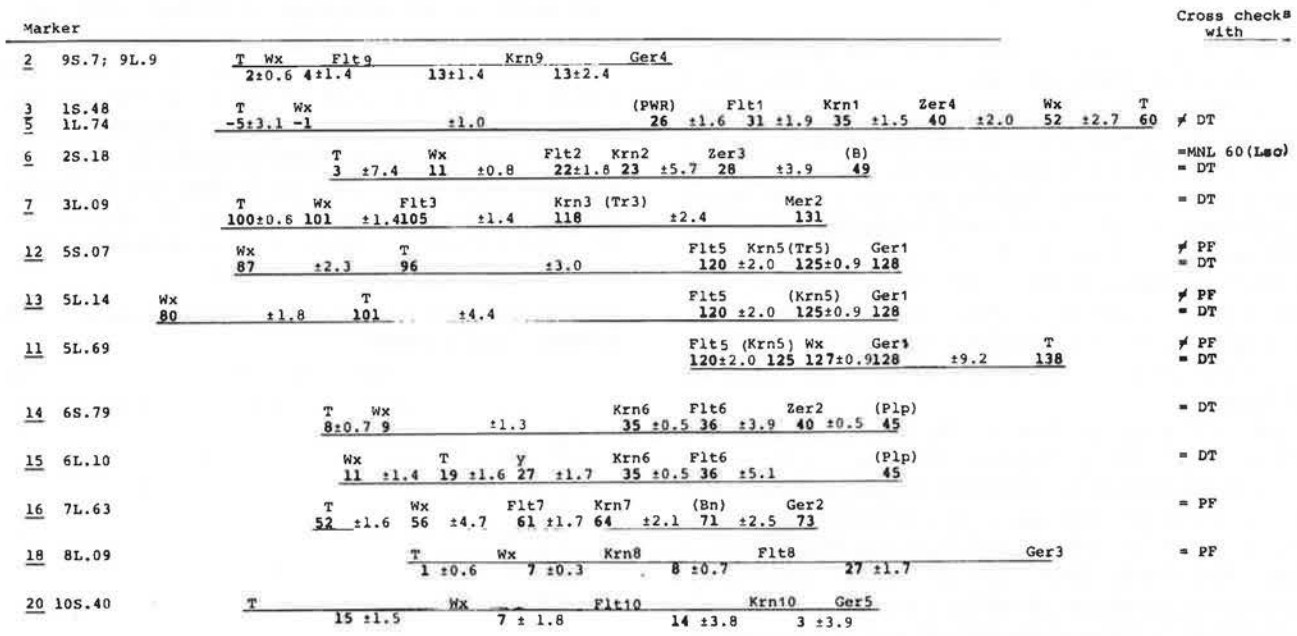
In MNL 58:38-46 the authors detected kernel row number factors *Krn* in all chromosomes except 4, which was not tested, and 10, which was not significant. In MNL 59:23-24 we gathered evidence of one more in 4S. In MNL 60:33-34 we pointed out that probably in 1 and 4 there was a pair of *Krn* factors. In the preceding work with Português Fasciado (PF) we found a *Krn* in 4L, and confirmed a few others already known. The new data also showed that fasciation in the sample tested was detected nearly as frequently as *Krn*. W.C. Galinat (in D.B. Walden, ed., Maize Breeding and Genetics) in the item on two-ranked versus many-ranked, reports *tr* in chromosomes 1, 2, 3, 6, 7, 8, 9, and 10. In the item on single versus paired female

Table 1. This table substitutes for Table 3 and 5 from MNL 58:38-46, p values and their standard errors are calculated by the allometric method.

			TU TKrn TFlt	WxU WxKrn WxFlt	UFlt KrnU KrnFlt				TU TKrn TFlt	WxU WxKrn WxFlt	UFlt KrnU KrnFlt		
		WxT						WxT					
2	9S.07	37.5	48.0	45.5	47.6			2.46	2.69	2.70	2.61		
	9L.09		41.8	41.6	33.2				3.24	2.96	2.41		
			41.3	44.6	48.5	PWRT	43.5		2.29	3.23	3.40	PWRT	3.32
						PWRWx	17.6					PWRWx	2.58
3	1S.48	25.9	46.1	46.1	46.4	PWRU	49.0	2.94	2.40	2.40	2.59	PWRU	3.05
			36.8	46.4	35.7	PWRKrn	47.6		2.04	3.59	3.51	PWRKrn	0.06
			41.6	41.7	47.4	PWRFlt	47.0		2.63	3.14	0.96	PWRFlt	2.10
						PWRT	42.5					PWRT	2.54
5	1L.74	8.7	38.7	41.4	47.1	PWRU	44.2	2.69	2.21	0.07	2.33	PWRU	2.36
			34.9	44.6	37.1	PWRKrn	49.2		3.44	2.93	3.39	PWRKrn	2.43
			39.5	39.8	36.3	PWRFlt	44.1		3.13	2.21	2.96	PWRFlt	2.98
						PWRWx	43.4					PWRWx	2.08
6	2S.18	10.8	41.6	40.4	45.6	BU	43.1	1.93	2.87	2.81	2.61	BU	2.10
			34.6	42.6	46.5	BKrn	45.1		2.04	3.01	2.58	BKrn	1.53
			19.2	40.3	39.1	BFlt	49.1		1.54	2.90	2.93	BFlt	3.36
						BT	49.1					BT	2.68
7	3L.09	11.6	45.5	45.0	43.8	BWx	47.4	2.45	2.19	3.06	0.85	BWx	3.83
			39.0	42.3	44.0				2.39	2.64	2.19		
			30.4	30.0	38.3				0.06	1.79	3.28		
12	5S.07	8.0	44.0	41.8	47.2			2.65	3.41	3.20	2.80		
			43.7	38.1	49.2				1.20	2.21	3.04		
			44.8	42.5	46.0				2.59	2.86	3.36		
13	5L.14	15.9	48.9	42.7	46.0			2.40	1.56	2.95	3.29		
			33.0	30.9	49.6				2.08	2.91	3.00		
			49.1	46.2	44.8				2.97	3.13	2.74		
11	5L.69	17.4	33.0	42.1	46.1			2.21	2.10	3.22	2.18		
			44.0	46.2	31.9				2.90	2.47	3.57		
			41.7	42.8	46.1	PlpT	25.4		2.69	2.76	2.32	PlpT	2.93
						PlpWx	31.9					PlpWx	3.20
14	6S.79	14.5	40.9	41.1	49.7	PlpU	44.2	3.09	2.09	2.53	3.87	PlpU	2.44
			49.1	49.8	48.7	PlpKrn	48.2		2.59	3.09	0.02	PlpKrn	2.40
			32.7	33.7	47.8	PlpFlt	47.3		1.35	2.73	1.20	PlpFlt	2.97
						PlpT	25.4					PlpT	2.93
15	6L.10	20.7	24.4	32.3	47.0	PlpU	21.2	3.07	1.14	3.16	3.17	PlpU	2.49
			30.9	22.8	45.6	PlpKrn	50.0		3.01	3.09	2.77	PlpKrn	3.20
			28.1	26.4	32.1	PlpFlt	20.0		2.04	2.71	2.34	PlpFlt	10.00
						Plp Wx	31.9					PlpWx	3.19
16	7L.63	14.3	39.8	49.0	41.5	BnU	47.8	1.93	1.81	2.35	2.89	BnU	1.45
			41.2	42.0	32.7	BnKrn	46.3		2.85	0.01	0.05	BnKrn	3.39
			32.5	36.7	44.4	BnFlt	45.9		2.30	2.48	1.33	BnFlt	2.35
						BnT	36.9					BnT	1.82
18	8L.09	12.6	45.4	46.3	45.7	BnWx	44.1	1.55	2.57	1.83	2.96	BnWx	2.22
			21.5	24.5	46.3				2.63	1.90	1.83		
			30.3	34.4	40.0				2.54	1.15	1.01		
20	10S.40	10.8	44.2	49.0	45.6			2.89	2.31	3.10	2.56		
			46.8	45.5	43.5				1.98	2.49	2.72		
			31.0	42.3	46.9				3.01	2.62	2.53		

16 y Wx 10.4 y T 7.5 y Krn 8.4 y Plp 4.6 y Wx 2.71 y T 3.50 yKrn 3.13 y Plp 3.30

Table 2. This table substitutes for the lower part of Table 6 in MNL 58:38-46. The difference is that the p values and their standard errors were calculated by the allometric genetics methods described in this issue. Comparisons with other data are indicated in the last column (PF, with Português Fasciado, DT with Durango teosinte). Symbols within parenthesis indicates the reference points onto which the other measurements were combined.



spikelets he reports *pd* in 3, 4, 7, 8 and 10. With the data from PF and Durango teosinte it dawned on us that we had unconsciously done with cultivated maize what had been done with wild maize, that is, teosinte. So *Krn* is probably allelic to *tr* and its symbol should turn to *Tr. Fas*, fasciated, is conceivably allelic to *pd* and would turn to *Pd*. With these data we arrive at 12 pairs of alleles for kernel row control in the species, linked to *Fas* in 4, 5 and 8 and to *pd* in 3, 6, 8 and 10 in total 3, 4, 5, 6, 8 and 10. See prior report with reanalysis of Langham's Durango teosinte data. Botanists have considered these types of data as incongruous. If we accept that wild maize was domesticated independently in three or four places, or if, not fully domesticated, it was transported into the presence of other races of teosinte before the isolating knobs were fully formed the story becomes congruous. Teosinte didn't transfer the knobs to maize, both developed the knobs as an isolation mechanism within each to eliminate cross pollinated individuals.

Looking at a map of the knobs in the species one is struck by two very marked characteristics. The knobs are wide apart or they are in three clusters of two to four knobs, in #6 three, L1, L2, L3, in #8 L1, L2, and in abnormal #10, four unidentified knobs.

It is as if the knobs inhibit the formation of adjacent knobs in one isolated race, and so for instance in #6, L1, L2 and L3 would be from three different domestications. B. McClintock (in D.B. Walden, ed.) calls attention to the distribution of a small knob at position three, L3 in #6, which with one in 7L she defines as the "Andean Complex". That takes care of three of the six lineages proposed by P.C. Mangelsdorf in Corn, Its Origin, Evolution and Improvement. These lineages are Chullpi, Kculli, and Confite Morocho. So, for L3 in #6 the question is settled. There remain the lineages Palomero Toluquenõ, Chapalote-Nal Tel, and Pira Naranja and the other knobs.

The development of the allometric method to calculate p values, shown in prior reports in this issue, led us to recalculate the data presented in MNL 58:38-46. The new results are presented in Tables 1 and 2.

Discussing the results from Table 2, we will make reference to the prior work on Durango teosinte (DT) and Português Fasciado (PF). The linkages detected for *Krn1* in #1 seem for a locus different from those detected in DT which then should be named *Tr11* and *Pd11*. For #2 there is agreement with DT, and with the work mapping *Lsc* which gave (*Krn2* 10) (*lte1* 30) (*Lsc* 35) (*B* 49). For #3 there is agreement with *Tr3* from DT. For #5 we verify that for 5S.07 the sequence is *Wx* T and there are different loci which then would be *Krn15* and *Fas15*, it agrees roughly with DT. For #7 there is agreement with PF. For #8, best fit would be the order *Wx* T for 8L.35 in PF and not as is shown in the Table.

Turning again to PF data 4L.33 could check with *Ga-S Krn4* in MNL 59:23-24, and 4L.90 involves new loci, presumed *Krn14* and *Fas14*, but with very small families. For #9 there is a reference to *tr* in the literature. As there is a reference for *tr pd* in the literature we include our sequence *Flt10 Krn10 Ger5* which was not presented in the first old report.

It goes without saying that much of this summing up is quite tentative but we think it will encourage more precise work with much more powerful tools for this kind of factors.

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The prolamin polypeptides of maize and related grasses

Seven tropical maize inbred lines, four teosinte varieties (*Zea luxurians, Zea mays mexicana, Zea mays parviglumis* and *Zea diploperennis*), *Tripsacum dactyloides* and *Coix lacryma-jobi* had their alcohol soluble proteins analysed by SDS-PAGE and isoelectric focusing (IEF) on agarose gels. On SDS-PAGE the prolamins of the maize inbred lines, teosinte varieties and *Tripsacum* were separated into the well-known four major size classes with apparent molecular weight of 22, 19, 15 and 10 kD. The prolamin of *Coix* presented a band with apparent molecular weight of 22 kD, along with a band of higher molecular weight, and an intermediary band between the 15 and 10 kD classes.

The IEF analysis showed distinct prolamin profiles ("fingerprints") for all samples analysed. In the seven maize inbred lines a total of 25 different polypeptides were identified. The polypeptides were numbered according to their distance to the cathode end of the gels (Wilson, Cereal Chem. 61:198-200, 1984). The most basic polypeptide found among the maize inbred lines was 10, and the most acidic was 47.5. Polypeptides 25.5, 31 and 35 were present in all the inbred lines.

A great heterogeneity was observed among the IEF pattern of *Coix, Tripsacum* and the teosinte varieties. The most basic band identified was 7.5. That band was present in *Tripsacum* and *Zea diploperennis*. The most acidic band was found in *Zea mays parviglumis* and was designated 58. Both bands, 7.5 and 58, were not present in the maize inbred lines analysed. Polypeptides 31 and 35, which were identified in all the seven maize inbred lines, were also found in *Tripsacum* and teosinte. Those polypeptides could be the product of genes that were conserved during evolution. This, however, should be confirmed by molecular analyses of those genes.

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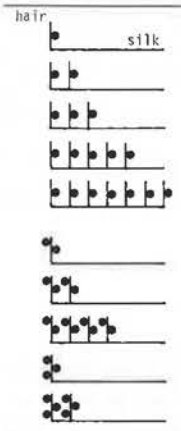
Fertilization by low numbers of pollen grains

Zea mays is recognised as a prolific pollen producer. It has been estimated that about 25,000 pollen grains are produced by one plant for each kernel on an ordinary ear with 1000 kernels. Allowing for dispersal, it is possible that at least 170 grains are available per silk during the course of pollen shedding (T.A. Kiesselbach, The Structure and Reproduction of Corn, Univ. Nebraska Press, 1980). The availability of large numbers of pollen grains competing for a limited number of ovules provides a selective advantage for the more vigorous pollen grains (Mulcahy, Science 206:20-23, 1979). Goss (Bot. Rev. 34:333-358) recognised it is difficult to estimate pollen viability by the application of single grains to silks.

During an investigation into the possibility of transferring DNA to individual pollen grains, it was necessary to estimate the minimum number of pollen grains required on a silk to effect fertilization. Detasseled maize plants (lines 256 and γ25) were kept in a glasshouse separate from the same lines of plants which produced the pollen. The tassels of the pollen plants were shaken each morning to remove old pollen. Two hours later (ca. 11 a.m.) the freshly shed pollen was collected. Detasseled plants were transferred to the laboratory. Individual pollen grains were picked up on a pointed scalpel blade and transferred to silks exposed under a dissecting microscope. Detasseled plants exposed to similar conditions in the laboratory without application of pollen grains produced no seed.

Repeated application of pollen grains to parts of the silk other than the hairs did not produce any fertilization. When individual pollen grains were applied to the hairs of silks, varying levels of fertilization occurred depending on the number of grains present (Table 1).

Table 1. Application of individual pollen grains to the hairs of silks of maize plants.

	No. grains /hair	No. hairs /silk	No. silks treated	% seed set
	1	1	127	8
	1	2	65	25
	1	3	43	72
	1	5	33	34
	1	7	22	77
	2	1	61	14
	2	2	38	34
	2	4	6	83
	3	1	43	24
	3	2	34	72

The application of one to three pollen grains on an individual hair of a single silk produced a low level of fertilization (8-24%). The addition of one, two or three grains to two or more hairs on a single silk produced increasing seed set (25-83%). These preliminary results suggest satisfactory levels of fertilization can be achieved with relatively low numbers of pollen grains applied to the bases of silks. It is not known if a minimum number of grains is required on each silk for efficient fertilization due to reduced pollen viability or if this number is necessary to stimulate synergistic pollen tube growth.

J.C. Waldron

Pollen transformation

A transformation system utilizing the normal fertilization cycle of maize is desirable since the protoplast system has limitations in not regenerating whole plants. Pollen seems to be a suitable vehicle with which to transfer foreign DNA into maize plants. Hess et al. (Z. Pflanzenphysiol. 74:52-63, 1974) consider pollen of *Petunia* and *Nicotiana* can take up foreign DNA during the germination phase and transfer it to the egg during fertilization. Maize pollen can be germinated in vitro and transferred to silks to effect fertilization (Raman et al., J. Hered. 71:311-314, 1980). Recently, Ohta (P.N.A.S. 83:715-719, 1986) obtained genetic evidence for the successful transformation of maize using pollen to transfer exogenous DNA to the embryo and endosperm.

The method used to attempt to transfer cloned DNA into maize plants was to germinate maize pollen from

alcohol dehydrogenase null plants in the presence of a plasmid containing *Adh* genes, then transfer pollen to the silks of detasseled, *Adh* null plants. A modification of the Brewbaker and Kwack medium (Amer. J. Bot. 50:859-865, 1963) was used to germinate maize pollen and maintain pollen tubes intact for at least two hours. The medium consisted of 15% sucrose, 0.03% $Ca(NO_3)_2$, 0.01% H_3BO_3; 0.02% $MgSO_4$; 0.01% KNO_3; 0.01% Tris; 0.7% Agarose, pH 6.5. Small pieces of dialysis tubing were placed on the surface of the solid medium some time before the pollen was applied. The dialysis tubing prevented germinated pollen grains from adhering to the agar medium and allowed easy transfer to the silks with a camel hair brush. DNA, as a supercoiled plasmid, contained either the *Adh1-1F* or *Adh1-1S* allele, each in combination with nopaline synthase as another marker gene. DNA was applied to the surface of the dialysis tubing at concentrations between 50-200 μg/ml. The effect of linearizing the plasmid DNA was also tested. Fresh pollen from *Adh* null plants (#256 = *Adh1-0, Adh2-0*; γ25 = *Adh1-0, Adh2+*) was sprinkled on the surface of the DNA solution over the dialysis tubing. After a minimum of 15 minutes germination the pollen was transferred to the silks of detasseled, *Adh* null plants in a "pollen free" glasshouse. The resulting seed was grown to maturity and tested enzymically for ADH activity in small scutellar slices.

The time of pollen germination on artificial medium before transfer to silks affected the resulting seed set:

Time of germination	Average no. seeds/ear
15 minutes	13.0
30	7.0
60	0.3
90	0.5
120	0.1

Shorter germination times produced more seed but the pollen was in contact with DNA for a shorter time. The possibility of seed being produced by non-germinated pollen grains cannot be excluded. However, counts suggested that generally if pollen grains did not germinate within 30 minutes on artificial medium they failed to germinate.

Another method used to determine if fertilization was effected by germinated pollen was to fix silks (3 volumes ethanol: 1 volume acetic acid) with the attached pollen and stain with aniline blue. Aniline blue stains specifically for callose in the pollen tubes and allows the course of the tubes to be followed down the silks to the ovules. Using this method, pre-germinated pollen grains could be seen to be attached to the silks and penetrate down to the ovule.

The site at which DNA is "taken up" by germinating pollen is uncertain. Hess et al. (Z. Pflanzenphysiol. 74:371-376, 1974) believe the tips of *Petunia* pollen tubes are thin and permeable and permit the uptake of large molecules. Experiments were conducted to try to detect the site of uptake of DNA in germinated maize pollen. Staining with the DNA specific fluorochromes, DAPI and mithramycin (Coleman et al., J. Histochem. Cytochem. 29:959-968, 1981), failed to detect any differences in DNA levels in treated and untreated pollen. However, the stains were useful in following the path of migration of pollen nuclei in the tubes of fixed material during germination. Pollen grains were also germinated in the presence of ^{35}S labelled DNA, fixed and autoradiographed. A very small percentage of pollen tubes showed the presence of silver grains which represent DNA molecules, down the length of the pollen tube. Whether the silver grains were located inside or outside the pollen tubes was not known.

Approximately 1500 seeds were tested for ADH activity after DNA treatment of *Adh* null pollen. Two seeds from *Adh1-1S* treated pollen showed possible *Adh1-1S* activity in one *Adh* null line, and another from *Adh1-1F* treated pollen showed *Adh1-1F* activity in a different *Adh* null line. After germination the three seedlings showed weak growth and did not survive. A number of seedlings produced from germinated pollen grains showed weak growth, and embryo rescue from fertilized ovules was carried out to overcome the problem. Several hundred small plants were regenerated in tissue culture and the roots tested for ADH activity. None of the plants showed ADH activity.

Although *Adh* genes may have been transferred into three maize plants via pollen, this method, which utilizes the "passive" uptake of DNA by germinating pollen, does not appear to be practical to obtain a reproducible, high frequency of transformation. Sanford et al. (Theor. Appl. Genet. 69:571-574, 1985) were also unable to obtain satisfactory frequencies of pollen mediated transformation in maize and tomato. Recent work by Matoušek and Tupý (J. Plant Physiol. 119:169-178, 1985) showed that pollen from a number of plants, including maize, released nucleases when germinated on artificial media. The combined effects of nucleases degrading the *Adh* containing DNA and the low seed set produced by pre-germinated pollen are some of the likely causes of the low frequency of pollen mediated transformation in maize.

An approach aimed at mechanically delivering DNA efficiently into cells is now being attempted to obtain transformation in maize.

J.C. Waldron

Stability of alleles of *Rp* (resistance to *Puccinia sorghi*)

There are 5-6 loci in maize known to confer resistance to rust disease caused by *P. sorghi*. The *Rp* locus maps at the tip of the short arm of chromosome 10 and has 14 alleles, *Rp-a*—*Rp-m*. Each allele can be recognised by the appropriate race of the pathogen and by reaction type, e.g.:

	Race 1	Race 2
Rp-d	- (;)*	+(4)
Rp-g	- (o)	- (o)
Rp-m	+(4)	- (1)

*The symbol in brackets describes the reaction using the terminology developed for wheat where ; (fleck) or O (immune) are the resistant reactions and 4 is fully susceptible.

Our original experiments were designed to tag the *Rp* gene with a known controlling element in order to provide a mutant accessible to molecular analysis. The *Rp-g* allele was the first target selected because the immune or (o) type reaction is the most clear cut resistance phenotype. In initial experiments with transmission through either pollen or egg the *Rp-g* allele gave rise to *Rp-g'* variants that were fully susceptible and occurred at about 7/1000 seedlings screened. Recovered *Rp-g'* alleles were stable and did not revert back to the resistant phenotype in any of 11,716 seedlings tested. Seven independently occurring *Rp-g'* alleles combined in all 21 possible heterozygous

combinations showed no complementation. The frequency was unaltered by the presence of any known controlling element system and presumably reflects some inherent instability of or event at the *Rp-g* allele. The frequency was clearly several orders higher than would be expected for an insertion event and ruled out the use of this *Rp-g* allele as a target for gene tagging. A subsequent examination of other alleles indicated that this was not an isolated event.

Allele	No. seedlings	No. susceptibles	Frequency
Rp-g	55,044	372	0.0068
c-k	1,214	10	0.0082
a	344	1	0.0029
b	2,913	3	0.001
c	2,807	2	0.0007
td	1,985	3	0.0015
f	1,853	9	0.0049
d	25,006	5	0.00016
m	11,206	0	0.0

The stabilities of *Rp* alleles fall into four frequency classes. The *Rp-m* allele shows the highest stability with no clear case of an *Rp-m'* susceptible variant being recovered from 11,206 seedlings scored. However, the resistance reaction is a type (1) or even (2) and this is difficult to score under some conditions.

The *Rp-d* allele, the next stable allele, was used as a target for tagging with the *Ac-Ds* system. If the background stability of *Rp-d* is about 1-2/10,000 and the expectation for controlling element insertion is assumed to be in the range 10^{-4} to 10^{-5} then in a population of 2-300,000 seedlings one would expect to recover 40-60 *Rp-d'* variants due to the background instability of the *Rp-d* allele and 1 or 2 susceptible seedlings due to insertional inactivation by *Ac-Ds*. These insertional events can only be distinguished if they are unstable and revert either to resistance or some other recognizable phenotype.
Cross: *Rp-d/Rp-d, Ac/-* × *Rp-m/Rp-m*

	No. seedlings	
	Rp-d/Rp-m	*Rp-d'/Rp-m*
Exp. 1 (*Ac* at *P-vv*)	121,078	31
Exp. 2 (*Ac* at *wx-m9*)	50,405	11

Characterization of the *Rp-d'* variants:

Class I -fully susceptible type (4)	32
Class II -intermediate type (1-2)	2
Class III-High necrosis type (; NN)	7
Total	41

Forty of the 41 variants were recovered and are being tested for stability in the presence and absence of *Ac*. Classes II and III were not observed in the control (no *Ac*) population of 25,006 seedlings. One of the Class II variants, *Rp-d'-5*, is an unstable phenotype giving rise to fully susceptible type 4 seedlings at a frequency of 0.4% (13/2725). This frequency of instability is in the range expected for a *Ds* excision event but further work is required to demonstrate that it is *Ac* dependent.

Analysis of the Class III high necrosis mutants is only preliminary. However, they appear to be very similar to barley *ml-0* mutants and to the lesion mutants described by Neuffer. *Rp-d'-21*, a class III variant, reacts to all races of the pathogen tested and this high necrotic reaction is induced spontaneously under low temperature conditions. Two of Neuffer's *Les* mutants (*Les*-1451,Les*-1453*) are located on the short arm of chromosome 10 and may be allelic to *Rp*. Class I and III variants could be explained if the *Rp* gene contained at least two parts, one conferring specificity (S) and the other triggering the hypersensitive response (HR). Mutations in HR would be incapable of response and phenotypically susceptible, while mutants in the S part would recognise all races of pathogen (as for *ml-o* and *Rp-d'-21*) and under environmental extremes may lack control and spontaneously give rise to necrotic lesions. Such a model would explain the existence of the estimated 60-70 lesion loci in maize since they would represent the evolutionary library of resistance genes that function by initiation of the hypersensitive response.

Tony Pryor

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The effect of proline on the regeneration of maize callus lines

The problem of the regeneration of plants from maize anther-culture-derived callus lines either by organogenesis or by somatic embryogenesis has been perplexing. In the last few years a number of maize callus lines had been selected and maintained by subculturing. During this period it was observed that most of them lost their capacity of proliferation after one or two years. However, several promising lines did show persistent vigor and prolificity.

In the last year, two of these continually grown callus lines, 81-85 and SAN1, were subjected to a regeneration test with the application of l-proline. These lines were chosen because they always provided abundant materials. According to the recommendations of Green et al. (Advances in Gene Technology: Molecular Genetics of Plants and Animals, 1983) and Rapela (J. Plant Physiol., 1985) varying amounts of the amino acid were employed. After repeated experiments, it was consistently revealed that no apparent difference occurred between the calluses grown on proline medium and those grown on control medium without proline. Hence, it appears tenable to say that the positive response of the maize calluses reported by Green et al. and Rapela to the proline regeneration medium is of genetic control. Our callus lines have a different genetic background from that of theirs.

Y.C. Ting and Stephen Schneider

Continued study on the meiotic chromosome behavior and fertility of anther-culture-derived plants

In 1978, Ting et al. (Acta Genetica Sinica) reported that H2 plants (2nd generation of pollen plants) from the intercrossing of sister dihaploid pollen plants consistently showed chromosome stickiness during the first meiotic divisions of the microsporocytes in the maize strain Lai-Bin-Bai. On the other hand, in the H2 plants from the selfing of pollen plants of maize strain Gui-Dan-12, meiotic chromosome behavior in the microsporocytes appeared normal in the first division. In 1985, Ting (Maydica) again studied chromosome behavior in pollen plants of the maize

strain Dan-San-91. In the first meiotic divisions of the microsporocytes of haploids, aneuploids and dihaploids of the H1 generation, chromosome fusions (stickiness) were always present. In view of the above inconsistent observations it was deemed necessary to make a further study on the meiotic chromosome behavior of maize pollen plants and their progenies.

In the summer of 1986, microsporocytes of three plants each of the H1 and H2 progenies of pollen plants of maize Dan-San-91 were collected and fixed according to the standard aceto-carmine squash technique. Upon microscopic examination it was found that, at the first meiotic prophase, chromosome fusions persisted from early leptotene-zygotene stage to metaphase I. Laggards at anaphase I and II were also observed, but the percent of cells having this kind of irregularity was very small. Both of the above appeared in all the plants studied. Nevertheless, the other aspects of meiotic chromosome behavior appeared normal. Regarding fertility, all of the plants had practically full seedsets.

Y.C. Ting and Stephen Schneider

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Partitioning the sources of abscisic acid found in developing embryos

Last year we reported that there were two separate sites of synthesis for abscisic acid (ABA) found in maize kernels, i.e., "in situ ABA" is synthesized within the kernel while "maternal ABA" is synthesized in the maternal plant and transported into the developing kernel. It now appears that there may be three sources, since "maternal ABA" may have two components. Cob tissue cultured in vitro synthesizes ABA which appears to be transported into ABA-deficient embryos. Thus, "maternal ABA" (synthesized in distal regions of the plant), "cob ABA" (synthesized in the cob tissue) and "in situ ABA" components contribute to the total ABA found in the kernel.

Coupling genetic and chemical inhibitors of ABA synthesis with cultural manipulation of seed development has enabled us to partition ABA into various combinations of component sources. We believe the carotenoid-deficient mutants *vp5*, *vp7(=ps)*, *w3* and the persistent herbicide fluridone completely inhibit ABA synthesis in the kernel if applied prior to 10 days after pollination (DAP). Due to the rapid metabolism of ABA, "maternal ABA" that may have been present in kernel blocks harvested at 5 DAP and cultured in vitro for 10 days should be completely degraded, or nearly so. These assumptions provide the logical basis for our partitioning methodology shown in Table 1.

Table 1. Sources of ABA found in maize kernels.

Plant Material	Source of ABA	
	In vivo	In vitro
Wild Type	Maternal	
	Cob	Cob
	in situ	in situ
ABA-deficient Mutants	Maternal	
	Cob	Cob
Fluridone-treated	Maternal	Residual
	Cob	

Preliminary data for this partitioning model are shown in Table 2. The field grown vs. cultured values are not directly comparable. The kernel blocks were cultured at a constant 30C which seemed to accelerate development, and their 15 DAP ABA levels are comparable to 18 DAP ABA in field grown embryos. However, comparisons within culture systems suggest that over 50% of the ABA in 15 DAP field grown embryos was of maternal origin. They also show that the cob contribution is appreciably less than that of the maternal plant.

Table 2. Abscisic acid concentration [pMo] ABA embryo^{-1} ±SE in 15 DAP embryos from field grown and in vitro cultured wild type[a] (WT), fluridone treated[b] (WT-F) and homozygous carotenoid-deficient[c] (MUT) kernels of maize.

Phenotype	Field Grown	In Vitro Cultured
WT	0.1422±0.0571	0.5236±0.1335
MUT	0.0864±0.0312	0.0854±0.0256
WT-F	0.0738±0.0201	0.0057±0.0008

[a] Wild type means and standard errors were calculated from 15 pooled sample means (4 assays/sample) of Tx5855, w3·Tx5855, vp5$^+$·Tx5855 and vp7$^+$·Tx5855 field grown and 8 pooled sample means of Tx5855 and w3$^+$·Tx5855 cultured kernels.

[b] Tx5855 kernels were sprayed with fluridone (100 mgl^{-1}) at 9 DAP in the field or cultured with fluridone (100 mgl^{-1}) added to the medium. Five field grown and 3 cultured samples were assayed.

[c] Eight w3·Tx5855, vp5·Tx5855 and vp7·Tx5855 samples were pooled for field data. Cultured data were from 6 w3·Tx5855 samples.

J.D. Smith, B.G. Cobb, C.W. Magill, D.J. Hole and C.A. Blakey

Identification of slow rusting resistance to *Puccinia polysora* Underw. in inbreds and single crosses

Ephiphytotics of southern rust occurred in the southern USA in 1972, 1973, and 1974 raising concern over the relative susceptibility of the maize germplasm in the United States to *Puccinia polysora* Underw. Slow rusting is a common form of resistance to many rust diseases, but slow rusting has not been evaluated in the maize/*P. polysora* interaction. For this reason, tests were designed to evaluate the slow rusting character of 23 single crosses and 33 inbreds in 1983 and 1984. The area under the disease progress curve (AUDPC) was calculated for each entry using weekly assessments of pustule density. Individual weekly assessments of pustule density were also analyzed to compare the relative effectiveness of the two methods for identification of slow rusting. Significant differences were observed among both inbreds and single crosses for AUDPC and for pustule density. The maize inbreds and single crosses evaluated displayed considerable variation for the slow rusting trait. Rank correlations between years were higher for AUDPC than pustule density, although rank correlations over years between pustule density and AUDPC were all high. The indication is that weekly assessments, if correctly timed, are as effective for identifying slow rusting as AUDPC, although they do not provide the details that can be gained by the multiple assessments used in calculating AUDPC.

B.A. Bailey, W. Schuh, R.A. Fredericksen, A.J. Bockholt and J.D. Smith

Identification of linkage groups controlling slow rusting in the *Puccinia polysora*/maize interaction using reciprocal translocations

A partial series of waxy reciprocal translocations involving chromosome 9 and one of each of the remaining chromosomes was used to identify linkage groups controlling slow rusting in the *Puccinia polysora*/maize interaction. The translocations were in a variable background and two lines of descent (LOD), one more resistant than the other, were evaluated for each inbred/translocation combination. The resistant LOD were analyzed separately from the more susceptible LOD. The inbreds B37R (resistant), Mo17 (slow rusting), B73 (moderately rusting), and Tx5855 (fast rusting) were evaluated in separate field tests in 1986 using a modification of Anderson's procedures (C.R. Burnham, Discussions in Cytogenetics, pp. 110-111). Area under the disease progress curve was calculated from 4 individual observations of pustule density on 10 plants per plot in a 5 block randomized complete block design test for each of the 2 LOD groups. Factors were translocation and endosperm character.

The complete resistance of B37R, which carries the *Rpp9* gene, was linked to chromosome 10 in both LOD tests. It was previously known that the *Rpp9* gene was linked to chromosome 10 (A.J. Ullstrup, Phytopathology 55:425-428), and B37R served as a positive check. The slow rusting character of B73 was almost entirely explained by linkage to chromosome 4 although chromosome 8 was also indicated important in the susceptible LOD test. The resistance of Mo17 was linked to chromosome 4 and 7 in the susceptible LOD test but not in the resistant LOD test. It was apparent that identification of linkage groups was impaired due to high background levels of resistance in the resistant LOD test. Chromosome 9 was indicated to be important in all the susceptible LOD tests except Mo17. The overall rate of epidemic progression varied between tests and seemed to affect the expression of resistance linked to chromosome 9. Hybrid vigor may also play a role in the resistance linked to chromosome 9 since in each case the normal kernel plots were held heterozygous for large sections of 2 chromosomes while the waxy kernels were forced toward homozygosity. In several cases resistance was linked to the translocation in the resistant LOD test, the most consistent of which were *wx* T5-9 and *wx* T6-9.

It is notable that resistance to *Puccinia sorghi* (W.A. Russell and A.L. Hooker, Crop Science 2:477-480), and *Helminthosporium turcicum* (M.T. Jenkins et al., Crop Science 1:450-455) have been linked to chromosome 4. Chromosome 10 also carries a complex group of complete resistance genes to *P. sorghi*. The consistent association of resistance genes and linkage groups suggests similar gene groups may be controlling both *P. polysora* and *P. sorghi*.

B.A. Bailey, J.D. Smith and R.F. Fredericksen

Morphological stages of embryo development in Tx5855

Our continuing studies on the physiological and biochemical characterization of maize embryogenesis have depended upon anatomical studies of Abbe and Stein (1954). The major anatomical stages were conveniently identified by the number of leaf primordia which had differentiated in the developing embryo. For instance Stage 1 is characterized by the differentiation of one leaf primordium, Stage 2 by 2 primordia, etc. Abbe and Stein completed their studies using inbred line A188 grown in Minnesota. The coleoptilar stage developed in 12 to 14 days, Stage 1 in 14 and 18 days, Stage 2 in 18 to 22 days, Stage 3 in 22 to 28 days, Stage 4 in 28 to 37 days, Stage 5 in 37 to 50 days and Stage 6 in greater than 50 days. It was evident from our earliest studies that some modification of the developmental time scale would be required since only 30 days after pollination are required for kernel development in several inbred lines adapted to our growing region (Texas).

Inbred line Tx5855 was pollinated and bagged in the field. Kernels and embryos at various times after pollination were collected and immediately fixed in Craft's II fixative (Sass, 1940). Tissues were dehydrated in alcohol, embedded in paraffin, and the entire kernel or embryo was serial sectioned (6 microns thick). Sections were cleared and stained with safranin and fast green. The coleoptilar stage is evident at 9 days, Stage 4 at 15 days and Stage 6 at 21 days. In comparing A188 and Tx5855, the developmental time scale for Tx5855 is earlier by 4 days at the coleoptilar stage, earlier by 13 days at Stage 4, and earlier by 29 days at Stage 6. Thus the acceleration in Tx5855 embryo development is less pronounced at the younger stages and more evident at older ages.

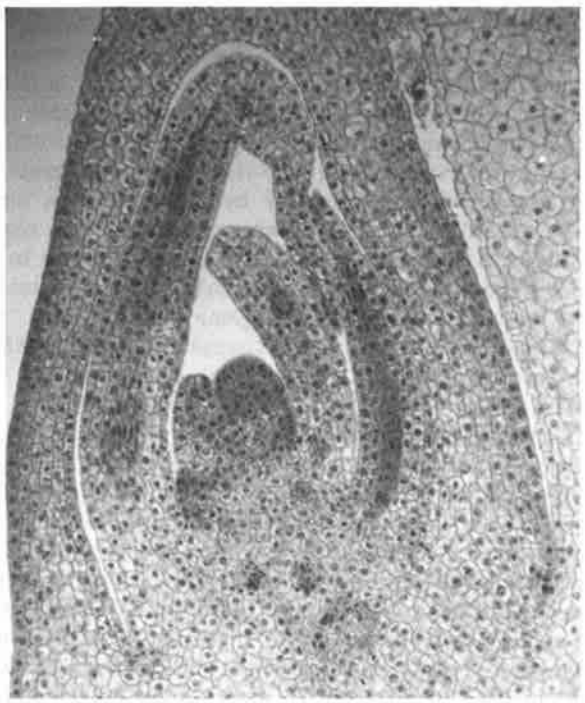

Figure 1. Sagittal section of Tx5855 embryo, 15 days after pollination. Note the development of 4 leaf primordia.

In relation to our previous studies on maize embryogenesis in Tx5855 (Fong et al., Plant Physiol. 73:899-901) the induction of carotenoid biosynthesis (fluridone sensitivity) is maximal at the coleoptilar stage, and the phytohormone abscisic acid is maximally effective in reversing vivipary or maintaining dormancy at Stage 4 of embryogenesis. The morphological and physiological characteristics of embryogenesis provide a convenient basis of comparison between studies done with different inbred lines or at different growing regions.

Franklin Fong and J.D. Smith

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Studies on *Ac*-derived mRNA

mRNA was isolated from various *Ac*-containing maize lines and tissues (seedlings, endosperm, adult plant) and hybridized with *Ac*-derived probes. A 3.5 kb band segregates with *Ac*; other hybridizing bands are detected also in *Ac*-free material. Without exception, the 3.5 kb band is seen on Northern blots when the plant genome contains *Ac*. The expression level varies only slightly between different plant tissues.

cDNA libraries were prepared and screened with *Ac*. Three overlapping clones were isolated which are probably derived from the 3.5 kb transcript, and sequenced. They span together 3.1 kb on the *Ac* sequence, not including the 4 introns. A long untranslated leader precedes an ORF 807 amino acids in length. The first 2 ATG's to open this frame are not enclosed by Kozak's consensus sequence. To elucidate the ability of the translation apparatus to accept this first ATG as a start signal, the cDNA was transcribed in vitro and the products translated in vitro in two systems. The products were slightly larger than expected when the first ATG was used as the start. In addition, the N-terminal end of the protein was sequenced and by this means it was clearly demonstrated that the first ATG is used as a start site in vitro. The 5'-end of the transcript could not be isolated as cDNA. Instead, a 1 kb long uniformly labeled single-stranded DNA fragment was used as a probe in a S1-protection assay.

This probe overlaps terminally 158 bases with the 5'-end of the cDNA, its 3'-end extends beyond the *Ac* element. Upon S1 digestion a cluster of bands spanning a range of nearly 80 bases is detected. There appear to be two major start sites, 334 and 358 bases distant from the end of *Ac*. To exclude the possibility of the existence of another short exon close to the 5'-end of *Ac*, a 100 bases long M13 probe starting 54 bases upstream of the more 5' located major transcription start site was constructed. This probe does not hybridize on a Northern blot with the 3.5 kb band, whereas a similar probe reaching 120 bases farther downstream and thereby overlapping 65 bases with the more 5' located major start site clearly hybridizes with this 3.5 band. The detection limit under these hybridization conditions was shown to be better than 20 bases. First results from primer extension experiments seem to confirm the S1 results.

Reinhard Kunze, Ursula Stochaj, Ulrike Courage, Jürgen Laufs and Peter Starlinger

An initial functional analysis of *Ac* in tobacco

We have developed a method to detect the frequency with which *Ac* excises from a neomycin phosphotransferase II (NPTII) gene (Baker et al., this issue). This system is now being used to determine which sequences within *Ac* are required for the excision process. *Ac* derivatives were constructed in vitro and inserted at the same position within the NPTII gene as described previously for *Ac*. These experiments differed slightly from those of Baker et al. in that the T-DNA of the Ti-plasmid also contained a hygromycin resistance gene expressed in tobacco protoplasts, which rendered these resistant to the antibiotic hygromycin. Therefore, in these experiments, hygromycin resistance indicated the T-DNA was successfully transferred to tobacco, and kanamycin resistance indicated that *Ac* had excised from the NPTII gene.

The *Ac* derivatives tested so far are shown in Figure 1. Whether or not they are capable of excision is also indicated. These data, together with our knowledge of the only *Ac* transcript so far detected (Kunze et al., this issue) allow us to make certain predictions of how *Ac* may be organized.

Four *Ac* derivatives (in plasmids pKU36, pKU32, pKU33 and pKU31) which contain deletions within the long 5' untranslated region of the *Ac* transcript are all capable of excision. At least 400 bp of the leader (in pKU31) are unnecessary for transposase expression and for excision. However, the *Ac* derivative present in pKU30 is incapable of excision. This derivative contains *Bgl*II linkers inserted at the *Eag*I site within the leader. It is not clear why the insertion of *Bgl*II linkers in pKU30 prevents transposition while the insertion of the same linker at the endpoints of the deletions indicated above does not. It is known that in pKU30 there is more than one *Bgl*II linker in tandem as this has created a *Pst*I site, but this is also true of pKU31 and pKU32. We are presently determining the nucleotide sequence of these derivatives to try to answer these questions.

Although deletions within the leader of the *Ac* transcript did not prevent excision, deletions removing part of the long open reading frame did. This was indicated by the *Ac* derivatives present in pKU35 and pKU9. In pKU9 the deletion has removed the 5' end of the open reading frame including the first two potential ATG initiation codons within the long open reading frame. This further suggests that translation of the open reading frame initiates at one of these two ATG codons, and not at one farther downstream (see Kunze et al., this volume).

The sequences deleted from the *Ac* derivatives present in plasmids pKU37, pKU19 and pKU29 are not within the *Ac* encoded transcript. These three derivatives cannot undergo self-catalyzed excision. The derivative present in pKU37 has lost the terminal sequences of *Ac* which are probably necessary for recognition of the ends of *Ac* by the transposase prior to excision. The sequences deleted from pKU19 and pKU29 may be required for recognition of the end of *Ac* by the transposase, for expression of the transposase (e.g., a promoter sequence) or for both of these. Experiments are underway to distinguish these possibilities. In addition, we are constructing further deletions to characterize more exactly the sequences required at the ends of *Ac* for recognition by the transposase, and are constructing point mutations within the open reading frame to try to determine which areas of this are required for transposase expression.

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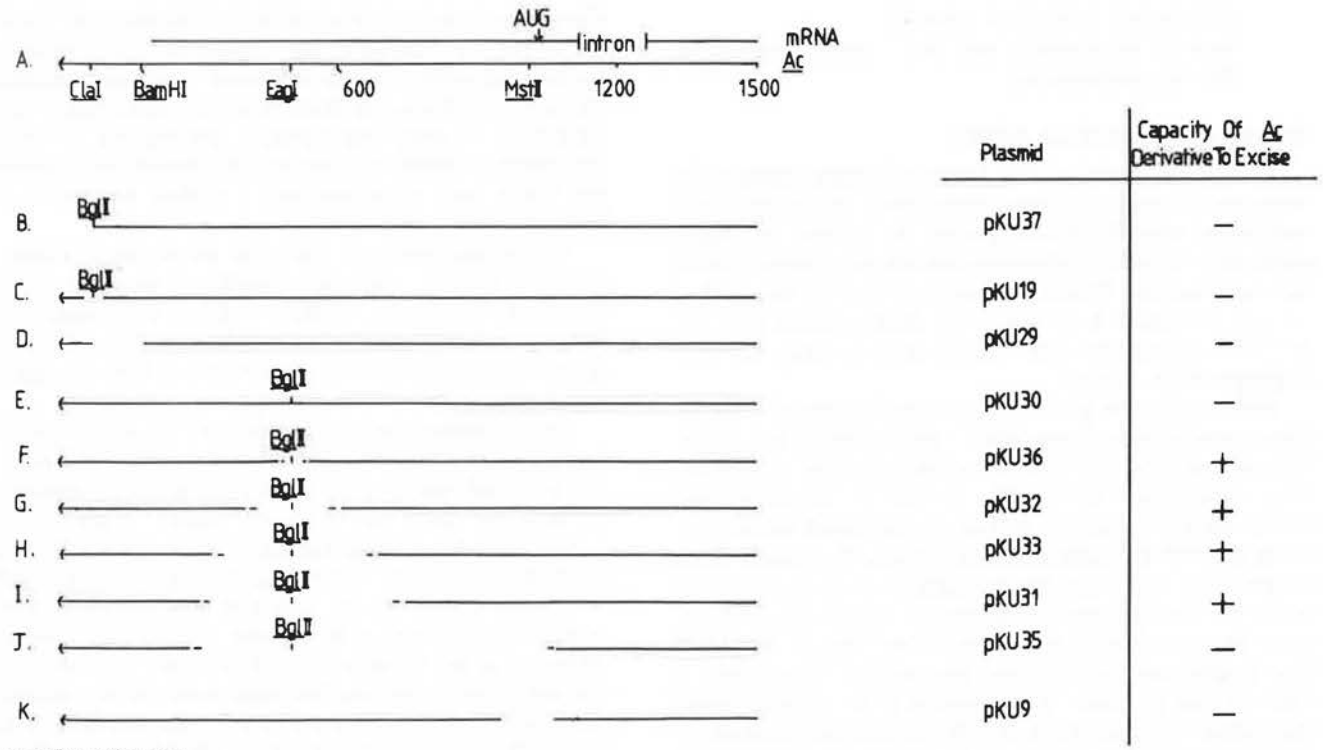

Legend to Figure 1

A. The 1500 bp of *Ac* from the end containing the *Bam*HI restriction site. Important restriction sites are indicated. The upper line indicates the mRNA positioned above the region which encodes it, and the gap in the upper line denotes the first intron. The AUG marked is the first potential start codon within the mRNA (see Kunze et al., this issue).

B. A Bal31 mediated deletion from the *Cla*I restriction site. This removed the terminal 92 bp. After Bal31 digestion a *Bgl*II linker was inserted.

C. Constructed as B. Removed 38 bp around *Cla*I. The terminal inverted repeat is intact. After Bal31 digestions a *Bgl*II linker was inserted.

D. Deletion of the 105 bp *Cla*I-*Bam*HI fragment.

E. Insertion of *Bgl*II linkers at the *Eag*I site. *Eag*I is an isoschizomer of *Xma*III. There are three recognition sequences within 40 bp of one another. It is not known into which one the *Bgl*II linkers are inserted.

F-J. Bal31 mediated deletions from the *Bgl*II linker described in E. The deletion sizes were estimated after agarose gel electrophoresis to be: F, 50 bp; G, 200 bp; H, 250 bp; I, 350 bp; J, 400 bp. *Bgl*II linkers were inserted at the end points of these deletions.

K. A 120 bp Bal31 mediated deletion from the *Mst*II site. Dash (—) denotes that these derivatives produced less than 1% of the number of Km-resistant calli found with intact *Ac*. The number of hygromycin-resistant calli was approximately equal after transformation with each derivative.

Transcription of *Adh1-2F11::Ds2*

We studied the transcription of the *Adh1-2F11::Ds2* allele. In this allele a *Ds2* element (described in MNL 1986 by A. Merckelbach and P. Starlinger) is inserted in the fourth exon of *Adh1*.

In Northern blot hybridizations with an *Adh1*-specific probe two bands light up:

1. A 3 kb transcript that has the combined length of wildtype *Adh1* RNA plus the length of the insertion.
2. A transcript of about 1.6 kb (described in MNL 1983 by S. Hake and M. Freeling).

Only the 3 kb band is detected with a *Ds2*-specific probe. The 1.6 kb RNA cannot be a transcript of an *Adh1* allele created by the excision of the *Ds2* element as it was also seen in *Ac*-free maize lines.

By Southern blot hybridizations it was confirmed that the *Ds2* element still resides at the *Adh1*-locus. For further analysis of the 1.6 kb RNA we prepared a cDNA library and screened for clones that hybridize to *Adh1* but not to *Ds2* probes. Recombinant phages were partially sequenced around the insertion site of the *Ds2* element in *Adh1*.

The cDNA clones sequenced are derived from RNA molecules that have lost 132 bp of the 5′ part of exon 4 including the whole *Ds2* sequence. This mRNA molecule must be the result of a splicing of the normal 5′ donor splice site at the beginning of intron 3 to a cryptic 3′ acceptor splice site in exon 4. The acceptor splice site used is not visibly inferior to the one terminating intron 3.

Is this cryptic splice site also used in the wildtype or is it activated only in the *2F11* allele by the *Ds2*-element? To test this we did Northern blot experiments with a synthetic oligonucleotide that spans the abnormal splice junction and thus can hybridize only to the aberrantly spliced 1.6 kb RNA molecules. This oligonucleotide probe detected a 1.6 kb mRNA in *Adh1-2F11*, but not in wildtype material.

Some other abnormal transcripts of the *Adh1-2F11* allele were detected. One group of cDNA clones was derived from RNA molecules that were terminated and polyadenylated at two different sites in intron 3. In addition one cDNA clone that had lost the *Ds2* element and part of exon 4 by aberrant splicing was terminated in intron 6.

This analysis shows that the insertion of a *Ds2* element can drastically alter transcription termination and the splicing pattern at a distance to the insertion site.

Rüdiger Simon and Peter Starlinger

Phenotypic assay for excision of the maize controlling element, *Ac*, in tobacco

The maize controlling element *Ac* was previously shown to transpose in tobacco cells (Baker et al., Proc. Natl. Acad. Sci. USA. 83:4484, 1986). We report here a phenotypic assay designed to detect excision of *Ac* from a selectable marker gene, neomycin phosphotransferase II (NPTII). An NPTII gene which expresses Km resistance in tobacco cells, and contains a unique restriction site in the transcriptional leader, was constructed in plasmid pKU2. *Ac*, or an internally deleted defective *Ac* element, was inserted into the leader of this gene in plasmids pKU3 and pKU4, respectively. These insertions inactivate the NPTII gene. These three plasmids were inserted into the T-DNA of the *Agrobacterium tumefaciens* Ti-plasmid pGV3850, and then transferred to regenerating tobacco protoplasts. The transformed cells were selected with 100 or 200 mg/l kanamycin (Km). Protoplasts transformed with pGV3850::pKU3 formed approximately 22% (100 mg/l Km) or 30% (200 mg/l Km) as many Km^r calli as protoplasts transformed with pGV3850::pKU2. Protoplasts transformed with pGV3850::pKU4 formed approximately 4% (100 mg/l Km) or 0% (200 mg/l Km) of the number of Km^r calli obtained after transformation with pGV3850::pKU2 (see Table 1). Southern blot analysis of five of the rare Km^r calli transformed with pGV3850::pKU4 and selected on 100 mg/l Km showed no evidence of excision of the transposon sequences, and were produced by an unknown mechanism. However, similar analysis of seven Km^r calli formed after transformation with pGV3850::pKU3 revealed that in all cases *Ac* had excised, restoring the structure of the NPTII gene.

This assay is being used to perform functional analysis of *Ac* (see Coupland et al., this issue). Several features of the phenotypic assay can be employed in the design of appropriate plasmid vectors using the *Ac/Ds* family of elements as mutagens and gene tags.

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Multiple transacting factors may be involved in the regulation of the sucrose synthase gene

The shrunken gene on chromosome 9 is regulated during plant development and exhibits positive and negative control mechanisms (Springer et al., Mol. Gen. Genet., in press). It is therefore a good candidate for studying plant gene regulation.

We have started to identify regulatory proteins which interact with the promoter of the shrunken gene. In the first step we had to adapt a nuclei isolation procedure to different tissues of the maize plant including immature kernels (20 days after pollination), primary roots, shoots and mature leaves. Low salt (250-300 mM) protein extracts from the isolated nuclei were prepared and tested in gel retardation experiments for specific protein DNA interactions. With nuclear protein extracts prepared from immature kernels we see several interactions with small radioactively labeled DNA fragments from the promoter region of the shrunken gene. The strongest interactions are found with several overlapping DNA fragments containing the sequences between -234 and -77. Another DNA protein complex is formed with DNA fragments spanning the sequences between -600 and -577. A weak interaction is found between -77 and +41 with the fragment containing the TATA box and the transcription start. The complexes are stable in the presence of 1000-2000 fold excess of unspecific competitor DNA but formation of visible complexes is abolished in the presence of a low

Table 1

Number of kanamycin-resistant colonies derived from tobacco protoplasts infected with *Agrobacterium tumefaciens* strains.
A. Total number of colonies that grew after selection with kanamycin as indicated. For experiments 2, 4, 5, 6, 7 and 8 colonies were counted directly from bead culture after 5 or 6 weeks of selection. For experiments 1 and 3 colonies were counted that grew after transfer of callus colony from bead culture to solidified MS agar medium containing 100 ug/ml Km. In experiments 4 and 5 protoplasts infected with pGV3850::pKU4 did yield Km^r calli (8 calli, experiment 4; 2 calli, experiment 5) when 2.5×10^5 and 2.2×10^5 protoplasts, respectively, were plated. These figures represent 1.0% and 0.3% of the number of pGV3850::pKU2 transformants in experiments 4 and 5, respectively.
B. The number of kanamycin resistant transformants after transformation with indicated *A. tumefaciens* strain expressed as a percentage of the pGV3850::pKU2 value normalized to 100% in each experiment.
***: Number of protoplasts infected with indicated strain of *Agrobacterium*. Number of cells surviving was estimated to be 30-50% of the starting number of protoplasts.**

			Agrobacterium strain									
Experiment	Number of infected protoplasts*	Km	pGV3850::pKU2		pGV3850::pKU3		pGV3850::pKU4		pGV3850::pKU27		pGV3850::pKU11	
			A	B	A	B	A	B	A	B	A	B
1	2.1×10^4	100	185	(100%)	50	(27%)	17	(9%)	-		-	
2	2.1×10^4	100	78	(100%)	16	(21%)	0	(0)	-		-	
3	2.1×10^4	100	140	(100%)	27	(19%)	4	(2.5%)	-		-	
4	3.15×10^4	200	274	(100%)	70	(25.5%)	0	(0)	146	(53%)	0	(0)
5	5.60×10^4	200	224	(100%)	91	(40%)	0		-		-	
6	1.3×10^5	200	140	(100%)	51	(36.4%)	0	(0)	-		-	
7	1.3×10^5	200	120	(100%)	42	(35%)	0	(0)	-		-	
8	1.3×10^5	200	197	(100%)	26	(13%)	0	(0)	-		-	

excess of homologous DNA fragments. At the moment we are in the process of footprinting this interaction on the nucleotide level.

Wolfgang Werr, Regina Bellmann, Boris Springer, Hans-Jürgen Joos and Peter Starlinger

Chromatin structure of the sucrose synthase gene

We have tried to study chromatin structure in the 5′ upstream region of the sucrose synthase gene. This gene is active in endosperm, inducible by anaerobiosis in roots and shoots, and hardly expressed in leaves (Springer et al., Mol. Gen. Genet., in press). We have tested for the presence of DNaseI hypersensitive sites.

Nuclei were isolated essentially as described by Rowland and Strommer (PNAS 82:2875-2879, 1985) with modifications for tissues other than roots. Though contaminated with cell wall fragments they contained high molecular weight DNA. The chromatin could be degraded by micrococcal nuclease or MPE (Fe^{2+}) to yield the typical nucleosomal ladder of about 180bp repeat length.

The nuclei are active in run-off transcription. By this method it could be shown that anaerobiosis induces sucrose synthase at the transcriptional level. The maximum levels of initiation occur after a few hours submersion of the seedlings in water roughly as for *Adh1* (Rowland and Strommer, Mol. Cell. Biol. 6:3368-3372, 1986).

In nuclei isolated from kernels a set of DNaseI-hypersensitive sites could be identified which extends more than 1 kb into the 5′ region. The major sites (+/- 50 bp) are located relative to the transcription start at positions +50, -25, -180, -280. The same major sites are seen in nuclei from isolated endosperm. No sites were detectable in naked genomic DNA. The same major sites in front of the transcription start are found both in aerobic and anaerobic (6h) roots.

Wolf-Bernd Frommer, Philipp Franken and Peter Starlinger

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Mutator-induced PSII photosynthesis mutant is allelic to *hcf3*

A mutation isolated from among M2 progeny mutagenized with Robertson's Mutator (MNL 60:46) has been found to be allelic to the EMS-induced mutation responsible for the photosynthetic mutant *hcf3* (Leto and Miles, Pl. Physiol. 66:18). *hcf3* has been described as "one of the most extensively characterized PSII-deficient mutants in higher plants" (Somerville, Ann. Rev. Pl. Physiol. 37:483). It exhibits a high level of chlorophyll fluorescence, abnormal fluorescence induction kinetics and lacks electron transport activity through PSII. Polypeptides associated with PSII are severely reduced or missing when membrane proteins are examined by LDS-polyacrylamide gel electrophoresis. The *hcf3* lesion has been located on chromosome 1S but no information about the nucleotide sequence of the gene or about its product is available.

The isolation of a mutation allelic to *hcf3* which is tagged with the *Mu* transposon opens the door to the cloning and characterization of the gene and its translation product. Ultimately this may lead to a better understanding of the nature of the mutation in particular and of the structure and function of PSII in general. We are currently considering the most appropriate method of cloning the *Mu*-tagged gene.

Allelism was tested by crossing pollen from M2 progeny of the *Mu* mutagenesis onto the ears of progeny of the original *hcf3* isolate. Eight of 50 progeny of this cross exhibited high fluorescence and the expected fluorescence induction kinetics. The genotypes of both parents of this cross were confirmed. Three other crosses between siblings of this parent and plants bearing *hcf3* in other backgrounds also produced progeny with *hcf3* characteristics. For these crosses, only the *Mu* parents' genotypes were confirmed.

Bill Cook and Don Miles

***Mutator*-induced mutation on 8L affects the chloroplast cytochrome b_6/f complex**

A mutation affecting the cytochrome b_6/f complex which was isolated from among M2 progeny mutagenized with Robertson's Mutator was uncovered by TB-8Lc. The mutation, 1113-3 (a lab designation), is a nuclear lesion which results in the loss of the cytochrome b_6/f complex of photosynthetic electron transport.

Mutant plants have an easily detected yellow-green phenotype and exhibit abnormal chlorophyll fluorescence and fluorescence induction kinetics. Staining of thylakoid polypeptides with 3,3′,5,5′-tetramethylbenzidine (TMBZ) following separation by LDS-polyacrylamide gel electrophoresis reveals the loss of cytochromes b_6 and f from the mutants. They are also significantly reduced in electron transport activity through the complex.

TB crosses were made and harvested by Dan England, for which we are grateful. We identified 6 of 90 progeny which were yellow-green, had abnormal chlorophyll fluorescence and the expected induction kinetics and also lacked cytochromes b_6 and f on TMBZ stained gels.

Concurrent tests of allelism with *hcf2* and *hcf6* (located on 1L and 1S respectively) were reassuringly negative.

Bill Cook, Marj Hunt and Don Miles

Location and designation of duplicate factors for orange pericarp, *orp1* and *orp2*

Duplicate factors conditioning orange pigment in the pericarp overlying doubly homozygous (*orp1 orp1, orp2 orp2*) kernels, reported last year (MNL 60:55), have now been located to chromosomes 4 and 10 respectively. The first of these genes was located on chromosome 4 using a modification of the method described by Kindiger and Beckett (MNL 60:43). This was done by first crossing a stock homozygous for one factor and heterozygous for the other (from an ear showing 3:1 segregation instead of 15:1) by the full B-A chromosome set. Heterozygosity for one factor was required because the double homozygote does not survive to flowering. This material was planted and hypoploids were identified for each translocated arm. Hypoploids plus a few normal plants in each case were selfed and the resulting ears were examined for 15:1 vs. 3:1 segregation for orange pericarp. The former would occur in the double heterozygote +/*orp1*, +/*orp2* hypo/( ) where the mutant was not on the hypoploid arm. The latter would occur when the factor was located on the hypoploid arm *orp1*/( ), +/*orp2*. Hypoploid selfs for all but TB-4Sa and TB-4Lf gave the expected 15:1 segregation (TB-10Sc

and TB-10L19 were not tested). Two families of TB-4Sa gave 18 semisterile ears (hypoploids) of which 11 had an apparent 3 normal : 1 orange segregation (the other 7, as well as the nonspecified ears below, did not segregate), and 8 normal ears of which 4 had a surprising 61 normal : 1 *orp* segregation. One family of TB-4Lf gave 6 semisterile ears of which 1 had a 3:1 segregation, and 9 normal ears of which 4 had a 15:1 segregation (a summary of counts of some of these ears appears in Table 1). The difference in

Table 1. Proportion of *orp* kernels on selfed ears from normal and hypoploid plants from the cross of *orp1 orp1* + *orp2* or + *orp1, orp2 orp2* by the B-A translocations TB-4Sa and TB-4Lf.

TRANSLOCATION	Ears	Total Kernels	Normal	orp	N/orp
TB-4Sa	2N	984	968	16	60.5
	7SS	923	688	235	2.9
TB-4Lf	1N	470	443	27	16.4
	1SS	105	83	22	3.8

ratios on the normal ears (4S, 60:1 and 4L, 15:1) from plants that probably were hyperploid for their respective arms proves that one member of the duplicate pair is actually located on 4S and not on 4L.

Fortunate circumstances provided the location of the other member on chromosome 10 even though the B-A crosses for 10S and 10L failed to give segregating ears because a homozygous normal female was selected. In several of the selfed ears from the other B-A crosses a segregation for aleurone color due to *R* occurred. Classification of orange pericarp on colored kernels is difficult but possible by examining the pericarp overlying the embryo. An examination of selfed ears from normal plants segregating 3:1 for aleurone color and 15:1 for orange pericarp revealed an excess of orange kernels in the colorless (*rr*) class (Table 2). This indicates that the selfed plants were

Table 2. F2 phenotypes for selfed ears segregating 3:1 for aleurone color and 15:1 for orange pericarp. N = normal, mostly hyperploid TB-9Sb. SS = semi-sterile ear of hypoploid TB-9Sb. Segregation of 15:1 on SS ears proves *orp* is not on chromosome 9.

EAR TYPE	Kernels	Orp R	orp R	Orp r	orp r
N	437	322	9	90	16
N	426	316	8	82	23
N	347	249	8	77	13
SS	222	149	1	65	7
SS	201	143	8	45	5
SS	178	134	2	34	8
TOTAL	1811	1310	36	393	72

orp r/*Orp R* with *r* and *orp* linked in coupling, and provides F2 data from which an *orp r* map distance may be derived. The segregation ratio of *R:r* was 2.9:1 and for *Orp:orp* was 15.8:1, which is not significantly different from the expected 3:1 and 15:1. The map distance calculated by maximum likelihood is 19 ± 3 centimorgans.

In view of these findings we have designated the duplicate factor on 4S as *orp1* and the one on 10 linked to *r* as *orp2*.

M.G. Neuffer, J.B. Beckett and Allen Wright

Gene expression in NCS2 mutant plants

The maternally inherited Nonchromosomal stripe (NCS) mutants in maize are characterized by variable leaf striping, poor growth, and decreased yields (Shumway and Bauman, Genetics 55:33; Coe, Maydica 28:151). It has been shown that the phenotypically distinguishable NCS2 and NCS3 mutants have distinct alterations in their mitochondrial DNA (mtDNA) restriction enzyme profiles, relative to their common progenitor cytoplasm, cms-T (Newton and Coe, PNAS 83:7363). To determine whether the observed DNA alterations in the NCS2 mutant are functionally significant, we used several approaches. Proteins synthesized by mitochondria isolated from mutants and from related plants with normal growth have been compared. NCS2 mitochondria synthesize very reduced amounts of a single polypeptide (approximately 24 kD).

Clones containing previously characterized plant mitochondrial genes were kindly provided by Sam Levings, Chris Leaver, Dave Stern and Axel Brennicke. They were used to test whether any of these genes had altered structure or expression in NCS2. Southern blot analysis with labeled probes for cytochrome oxidase subunits I, II and III; ATPase subunits 6, 9 and alpha; cytochrome b; URF1 and ribosomal RNA genes demonstrated that the DNA regions with these genes (or homologous sequences) appear to be the same in NCS2 mutant and related normal plants. Northern blot analysis of mitochondrial RNAs, using these same probes, showed similar transcripts were produced in NCS2 and related non-mutants.

An 11kb NCS2-specific *Hin*dIII fragment was cloned and tested by Southern and Northern blot analysis. It detects the NCS2 mtDNA alterations observed with several different restriction enzymes (it is not homologous to the NCS3 alterations.) This probe hybridizes to an aberrant set of transcripts in NCS2 mitochondria, relative to normal mitochondria.

Thus, in NCS2 mutants, the synthesis of one mitochondrial protein is reduced and one set of transcripts, homologous to the NCS2-altered region of mtDNA, is also aberrant.

Heidi Feiler and Kathleen Newton

NCS2 mutants: ultrastructural observations

NCS2 mutant plants are characterized by pale green stripes on leaves and defective kernel sectors on ears. Specific mitochondrial DNA changes were found to correlate with the phenotypic alterations (Newton and Coe, PNAS 83:7363). Furthermore, specific differences in mitochondrial gene expression are found in mutant plant material (Feiler and Newton, MNL 1987). We were interested in determining whether ultrastructural alterations could be observed in the mutant tissues and organelles. For this initial study, a pale green sector on an NCS2 leaf was compared with a section of a leaf from a normal plant. Both plants were field-grown and leaf samples were chosen at comparable stages of development.

Light and electron microscopic examinations revealed an altered general morphology of affected NCS2 tissue. At the lower magnifications of the light microscope, one can observe abrupt shifts from normal to affected cells. A conspicuous asymmetry in the vascular bundles and bundle sheaths is seen, especially under low magnification of the resin prepared material. This distortion could reflect the actual situation in the plant or it may result from the cells being more fragile and subject to damage by the rather harsh preparation procedures. At higher magnifications (30,000-fold) the internal organization of the organelles can be compared. In mutant cells, the

mitochondria have lost most of their internal membrane (cristae) structure. The inner and outer mitochondrial membranes appear to be more closely appressed. Ribosomes appear to be present and many DNA-like fibrils are also observed in the mutant mitochondrial matrix.

Chloroplasts are also affected in pale green sectors of NCS2 plants. Thylakoid membranes are present but there is some loss of stacking and a reduction in stromal constituents. In the bundle sheath cells, normal starch grains are absent and appear to be replaced by osmiophilic bodies.

The maternally inherited NCS2 mutants have altered mitochondria and chloroplasts in affected tissues. The correlation between phenotype and the mitochondrial genome is well established for this mutant. The effect on the chloroplast is presumed to be a physiological effect. It is not yet known whether the mitochondrial ultrastructural alterations are a result of a direct effect on mitochondrial biogenesis or of an indirect physiological effect.

Deborah Thompson and Kathleen Newton

Further genetic study on involvement of the *P* locus in silk browning

C.S. Levings and C.W. Stuber (Genetics, 1971) showed that browning of cut ends of silks of inbred lines T61 and NC232 is under monogenic control in crosses and backcrosses with Kys and NC236. In MNL 60:50 and 59:40, we suggested the control of silk browning by the *P* locus, based on the observation that the cob color of *P* locus and silk browning were not separated by genetic recombination. White cob and browning inbreds that we tested further are the following: K44, K166, Ky228, Mo1W, Mo14W, 79-R1141, 79-R1193, Mo22, and K64. Interestingly, one plant of 15 F2 progeny was nonbrowning, red cobbed from the cross of browning, red cobbed W23 to 79-R1193, one of the white cob and browning inbreds. This indicates the possibility that the phenotypic expressivity of silk browning, mediated by the *P* locus, could be dependent on genetic background or be labile to physiological conditions. We are conducting further genetic examination of those possibilities.

	F1		F2	
Cross	B	NB	B	NB
K44/79-R1141	21	1	36	3
K44/79-R1193	26	0	..	..
K44/Mo14W	21	0	30	0
K44/Ky228	23	0	32	1
K44/K166	16	0	-	-
79-R1141/79-R1193	10	0	15	0
79-R1141/Mo14W	-	-	20	0
79-R1141/Mo1W	4	0	43	0
79-R1141/Ky228	22	0	36	0
Mo22/Ky228	22	0	-	-
79-R1193/Mo22	10	0	-	-
79-R1193/Mo14W	23	0	47	0
79-R1193/Ky228	17	0	43	2
79-R1193/K166	24	0	38	0
Mo1W/79-R1193	20	0	44	0
Mo1W/K166	22	0	44	0
Mo1W/Ky228	17	0	33	0
Mo1W/Mo14W	18	0	43	0
K166/79-R1141	21	0	41	0
K166/Mo22	20	0	41	0
K166/Mo14W	22	0	43	0
K166/Mo1W	23	0	30	0

B: silk browning
NB: silk nonbrowning

To further examine whether browning of silks in those white cobbed inbreds results from possible involvement of a duplicate factor separable from the *P* locus or from allelic relationship, we intercrossed silk browning, white cob inbreds and selfed the progeny.

All but one of the F1 progenies, and all but three of the F2 progenies, showed only browning silks. Since silk browning is a dominant character, 1 nonbrowning plant out of 21 F1 progeny of K44/79-R1141 could be due to seed contamination or to low expressivity resulting from physiological conditions. The same explanations could apply to nonbrowning plants of F2 progeny of K44/79-R1141, 79-R1193/Ky228, and K44/Ky228 (3 out of 36, 2 out of 47, and 1 out of 23, respectively). All F2 progeny of 79-R1141/Ky228 and 79-R1141/79-R1193 showed silk browning, but F2 data for K44/79-1193 and Ky228/79-R1141 are not available. These observations favor the conclusion that the determinant of browning of cut ends of silks, which was not separable from the *P* locus by genetic recombination, is of allelic relationship, even though larger samples are needed for more tests.

Chang-deok Han and Ed Coe

Survey for inbreds carrying homozygous *whp*

Several inbreds had been tested for *whp* constitution (Coe, MNL 56:49). All of the inbreds (Ky21, Mo20W, Ky27, L289, and Mo17) have been identified to carry dominant *Whp* (yellow pollen with *c2 c2*). To further test for *whp*, 32 inbred lines were crossed onto and backcrossed with *C2/c2 whp/whp* plants of K55, which is an original source of the *whp* factor. Colorless (*c2 c2*) seeds from the backcrosses were planted for observation of pollen color. The inbreds we tested are the following: K41, K63, K64, K150, K155, K302, K303, K304, K306, K814, K816, FR802, FR807, FR809, FR810, FR29, FR35, FRMo17WC, W77-R3027, 78-S159, 79-R1130WC, 79-R1141, 79-R1193, Mo14W, Mo1W, Mo16W, Ky228, A619, Oh51a, B73Ht, and CI66. Only one of the inbreds, FR810, shed white pollen in the *c2 c2* progeny (15 plants). This inbred consequently is either homozygous for *whp*, or at least carries the factor. Because FR810 carries *C2*, however, its pollen is yellow.

Chang-deok Han and Ed Coe

Flavonoid 3-hydroxylase in aleurone

Flavonoid 3-hydroxylase has been demonstrated using freshly peeled intact aleurone tissue as the source of the enzyme in the reaction mixture. The reaction requires α-ketoglutarate, ascorbic acid and Fe^{2+}. Eriodictyol serves as a substrate, resulting in the formation of dihydroquercetin, which can be chemically oxidized to quercetin. Quercetin has been identified by thin layer and high performance liquid chromatography. Thus far it has not been possible to obtain catalytic activity free of the intact aleurone tissue, nor has it been possible thus far to obtain any information as to what gene(s) influence hydroxylation.

Russell L. Larson

Barren-stalk-fastigiate, *baf*, chromosome 9S

Material segregating for a recessive barren-stalk was provided by C.R. Burnham. Expression in segregating progenies is variable for barren-stalk, most plants having neither an ear nor the characteristic "ear notch" in the

culm, but occasional ears do develop and produce seed. The ear shanks are modified, having a lengthened internode between the node and the prophyll and a vertical, flattened shank that is often fused with the accompanying internode. The internode tends to curve into a distorted arc and often cracks horizontally. The tassels remain bundled vertically, the branches failing to drop to a horizontal position; the branches show elongated, spindly form reminiscent of broom corn sorghums; the pulvinus does not develop or does not expand, leaving the bundle of branches unspread. The symbol *baf*, barren-stalk-fastigiate, is assigned.

TB-9Sb uncovers this factor (listed as *ba*-s* in MNL 59:40), so it is distal to *wx* and *w11*. An F1 of *sh bz wx/baf* was testcrossed to *sh bz wx*, and recombinants were selected, self-pollinated and progeny tested for *baf*. Six + + *wx* individuals did not segregate for *baf* (the numbers of plants observed, however, were very small, totalling 30 plants for all six progenies; nonetheless this shows that recombination with *bz* is higher than with *wx*). Seven *sh bz* + individuals segregated for *baf*, and one did not (among 11 progeny). Two *sh* + + individuals both segregated for *baf*. Since *baf* is distal to *wx*, the order is clear: *sh bz baf w11 wx*. If the one *sh bz* + individual that did not segregate is correctly classified, it is the only crossover between *baf* and *wx* in this small experiment, suggesting that *baf* may be very close to *w11*.

Ed Coe and J.B. Beckett

Linkage data for luteus-7, chromosome 9S

Among 562 F2 plants from *sh bz wx/l7*, no *sh l7* or *bz l7* progeny were observed. Counts involving *wx* were 276 + +, 173 + *l7*, 110 *wx* + and 3 *wx l7*. The estimated distance for *L7* to *Wx* is 14.4 map units; for *Sh* to *L7*, less than 10.1; for *Bz* to *L7*, less than 9.9.

Ed Coe

v30 (was *v*-8587*) linkage data, chromosome 9L

The virescent known as *v*-8587* can be designated *v30*, as it is uncovered by TB-9La, is not allelic to *v1* or *ar*, and has a map location well away from either of these virescents. Testcross data were obtained for the cross of + + *Wc Bf/bk2 v30* + + x *bk2 v30* + +.

P	+ + Wc Bf	46		
P	bk2 v30 + +	52		
1	+ v30 + +	2	Bk2 - V30	4.6 ± 1.7
1	bk2 + Wc Bf	3		
2	+ + + +	15	V30 - Wc	19.0 ± 3.2
2	bk2 v30 Wc Bf	11		
3	+ + + Bf	11	Wc - Bf	15.7 ± 2.9
3	bk2 v30 Wc +	9		
1-3	+ v30 + Bf	1		
2-3	+ + + Bf	2		
1-2-3	bk2 + + Bf	1		
	Total	153		

The order of the factors, *bk2 v30 Wc Bf*, appears to be reliably defined. The numerical data may be subject to some modification after a few retests; in particular, conditions were poor in the field and classification of *v30*, while generally reliable, was occasionally uncertain and has been assumed for some of the above testcross plants.

Ed Coe

Anthocyaninless-lethal, *anl1*, chromosome 5S

The new anthocyanin factor(s) reported by Coe and Neuffer in MNL 60:54 on 5S resolve to a single group of allelic mutants. Six occurrences have been identified in progenies from EMS treatments. Allelism tests with *a2* are negative, as are tests with *ps* and *vp2*. All are homozygous inviable and fail to germinate, though the embryo appears normal and in rare instances poor seedlings arise but die. Small kernels are typical of four of the occurrences more or less regularly; there is sufficient variation in this to indicate that it is an expression subject to modifiers. Low ratios are also common, but not constant; tests for involvement of the gametophyte factors on chromosome 5 have not been conducted. Two of the occurrences have a pale-aleurone expression rather than colorless; one of the two expresses the color in irregular spots, while the other is uniformly pale. The individual occurrences have characteristics as follows:

anl1-p1634	pale aleurone; rare seedlings, die
anl1-1643	colorless; rare seedlings, die
anl1-1645	colorless; small kernels
anl1-1671	colorless; small kernels
anl1-p1673	colorless; pale spotted
anl1-1685	colorless; small kernels
anl1-1691	colorless; small kernels

Among progeny from crosses with markers on chromosome 5, two of the colorless types have become pale in expression; modifiers may influence the color development, or may confer the missing function with low activity.

Linkage analysis places *anl1* distal to *a2*. Specifically, colored nonbrittle kernels from testcrosses of + *a2 bm bt pr/anl1* + + + + x *a2 bm bt pr* were selfed and classified. Of 28 kernels, 3 carried a recombinant + + + + + strand and the remaining 25 carried *anl1* + + + +, demonstrating that *anl1* is distal to *a2* and roughly 10 units away. F2 data are also consistent with the position and the distance.

Ed Coe

Location of *nec2* on chromosome 1S

Mapping studies involving the seedling necrotic mutant, *nec2*, with *dek1*, *zb4* and *p1* have placed *nec2* approximately 7 units proximal to *dek1* on chromosome 1S. Seeds from the testcross *zb4 p1-ww* + x (+ *p1-wr nec2*)/(*zb4 p1-ww* +) were planted, scored for *zb4* and selfed. Each selfed ear was then scored for cob color and a seed sample was planted in the sandbench and scored for *nec2*. The following data place *nec2* approximately 9 units proximal to *p1*.

Reg	Genotype	No.	Totals	Expected
0	+ + nec2	59		
	zb4 p-ww +	52	111	110.6
1	zb4 + nec2	1		
	+ p-ww +	4	5	5.4
2	zb4 p-ww nec2	3		
	+ + +	8	11	11.4
1,2	+ p-ww nec2	1		
	zb4 + +	0	1	0.6

zb4 --- p-ww 0.0469 +/- 0.0187
zb4 --- nec2 0.1250 +/- 0.0292
p-ww --- nec2 0.0938 +/- 0.0258

Seeds from the testcross, + + x (*dek1* +)/(+ *nec2*), were planted and selfed. The resulting ears were scored for *dek1*, a seed sample was planted and the seedlings were scored for *nec2*. The following data were obtained.

Genotype		No.	
dek1	+	52	
+	nec2	45	
			dek1 --- nec2 0.067 +/- 0.025
+	+	3	
dek1	nec2	4	

As in any testcross involving two lethal mutations, the double mutant class may be under-represented since it requires a crossover in the testcross individual in order to score the presence of both mutants. This appears not to be a major factor in this analysis as both crossover classes were roughly equal and the resulting *dek1* to *pl* distance of 2 units is in close agreement to the established distance. Nevertheless, the linkage distance may be greater than 7 units.

As reported by Beckett (MNL 49:130), TB-1Sb uncovers *nec2* and, thus, the breakpoint of TB-1Sb lies between *nec2* and *as1*.

Dave Hoisington

Computer programs for use in linkage analysis

During the last couple of years, I have written several computer programs to aid in the analysis of various types of linkage data. All of the programs use maximum likelihood equations to determine recombinational distances. I am in the slow process of consolidating several together into a few generalized programs and plan to port these to at least IBM compatible systems. Rather than an in-depth discussion of each, a brief description for the currently available programs follows. Please contact me if you are interested in obtaining copies of any or all of these.

Linkage—a program that analyzes multi-point (up to 10) backcross data. Output includes total and percent of each gene class; genotype, number, total, and expected total for each crossover class; and paired recombinational values.

ml—a computerized version of Allard's maximum likelihood equations. Most of the equations have been entered, although any additional equations can be added rather simply. Once the desired equation is selected and the number in each phenotypic or genotypic class is entered, the recombinational value and its standard error are calculated and displayed.

ml-rfp—a program to analyze restriction fragment length polymorphism data. The current version accepts segregation data for dominant, recessive, and co-dominant traits, in backcross and F2 populations. Any combination of traits and populations can be analyzed. Also, any locus or subset of loci can be analyzed. Output includes the segregation of each locus within the population, as well as its segregation and recombination value with all other loci being analyzed.

Dave Hoisington

Les10, a new lesion mutant located near *v4* on chromosome 2L

Linkage analysis of *Les*-A607*, a dominant lesion mutant kindly provided by Jerry Kermicle, has placed this mutant near *v4* on chromosome 2L. Since this mutant has a different phenotype from both *Les4* and *Les*-1378* (see MNL 60:50-51 for phenotypic description) and shows linkage to both T2-9b and T2-9d, whereas both *Les4* and *Les*-1378* show linkage to only T2-9d (see following data), the symbol *Les10* is proposed for *Les*-A607*.

Translocation Backcross Data - only those crosses resulting in linkage or involved in the same chromosome are presented.

Mutant	wxT	# Pl	MWx	NWx	Mwx	Nwx	Chi-square (1:1:1:1)	% Rec.
Les10	2-9b	254	102	32	32	88	62.70	25
	2-9d	436	157	74	71	134	49.18	33
Les4	2-9b	145	30	38	37	40	1.06	52
	2-9d	169	75	14	6	74	99.61	12
Les*-1378	2-9b	115	27	30	30	28	0.23	52
	2-9d	83	42	1	5	35	61.59	7

The following two testcrosses place *Les10* near *v4*. Data for the testcross, *lg1 gl2 wt1* + x (+ + + *Les10*)/(*lg1 gl2 wt1* +), establish that *Les10* is 15 units proximal to *wt1*.

Table 1. *lg1, gl2, wt1, Les10* linkage.

Reg	Genotype				No.	Totals	Expected
0	+	+	+	Les10	24		
	lg1	gl2	wt1	+	21	45	46.8
1	lg1	+	+	Les10	5		
	+	gl2	wt1	+	4	9	8.4
2	lg1	gl2	+	Les10	5		
	+	+	wt1	+	7	12	10.1
3	lg1	gl2	wt1	Les10	3		
	+	+	+	+	6	9	8.4
1,2	+	gl2	+	Les10	1		
	lg1	+	wt1	+	0	1	1.8
1,3	+	gl2	wt1	Les10	0		
	lg1	+	+	+	2	2	1.5
2,3	+	+	wt1	Les10	1		
	lg1	gl2	+	+	0	1	1.8
1,2,3	lg1	+	wt1	Les10	0		
	+	gl2	+	+	0	0	0.3

lg1	--- gl2	0.1519 +/- 0.0404
lg1	--- wt1	0.3038 +/- 0.0517
lg1	--- Les10	0.3797 +/- 0.0546
gl2	--- wt1	0.1772 +/- 0.0430
gl2	--- Les10	0.3038 +/- 0.0517
wt1	--- Les10	0.1519 +/- 0.0404

Table 2. *Bl, ts1, Les10* linkage.

Rgn	Genotype			No.	Totals	Expected
0	+	+	Les10	35		
	Bl	ts1	+	36	71	71.4
1	Bl	+	Les10	11		
	+	ts1	+	5	16	15.6
2	Bl	ts1	Les10	5		
	+	+	+	2	7	6.6
1,2	+	ts1	Les10	0		
	Bl	+	+	1	1	1.4

Bl	--- ts1	0.1789 +/- 0.0393
Bl	--- Les10	0.2421 +/- 0.0439
ts1	--- Les10	0.0842 +/- 0.0285

Testcross data for the cross, *b1 ts1* + x (*b1* + *Les10*)/*B1 ts1* +) place *Les10* roughly 8 units proximal to *ts1*, which would be near *v4*. Additional tests with *v4* are in progress.

Dave Hoisington

Publicly available RFLP clones

Since last summer, we have been actively isolating a set of publicly available RFLP clones for maize. We were graciously provided a genomic library by Tim Helentjaris containing over 700 unscreened clones. Each of these clones is being screened against Southern blots of *Eco*RI, *Eco*RV, and *Hin*dIII digests of genomic DNA from the inbreds Tx303 and CO159 and the F1, Tx303/CO159. So far we have identified over 75 clones that detect polymorphic loci between these two inbreds. We have mapped approximately half of these in an F2 involving these same two inbreds to produce a "rough" RFLP map. In addition to our own clones, we have also probed the F2 blots with a select group of NPI clones and other cloned loci in order to establish linkages with already existing loci. Tim Helentjaris has also provided linkage data of their loci in order for us to use our data with theirs to produce a combined RFLP map. The current map with both NPI and UMC loci is presented with the working maps toward the end of this newsletter. Any of the clones we have isolated are available upon request.

On the technical side, we have resorted to isolating the insert from each clone prior to hybridizations. The presence of several "contaminating bands" in one of the inbreds and several of the F2 samples made interpretation of the blots difficult. These bands appear to be present in the original leaf material used for DNA isolation and hybridized to the plasmid (puC 8) alone. There appears to be a dichotomy among RFLP researchers for those who have problems and those who do not. We felt that even the possibility of additional bands could lead to future problems and have decided to take the extra time to isolate inserts. Basically our procedure involves digesting a miniplasmid prep with the appropriate enzyme, electrophoresis in 1% DNA grade agarose, and excising the insert band. Labelling of the isolated insert is achieved through oligolabelling of an aliquot of the diluted gel slice directly. So far, all of the clones from the genomic library have been labelled successfully. Copies of our procedures are available.

Dave Hoisington and Jack Gardiner

Toward unambiguous designations for loci defined by restriction fragment polymorphisms

The definition of polymorphisms with probes, rapidly becoming a major tool for mapping and other purposes, raises some potential ambiguities in terminology for the gene as a functional unit. We would like to propose a conservative, yet efficient and informative, symbolization of RFP loci that is consistent with current nomenclatural standards and that will minimize ambiguities, especially for probes derived from functionally defined units. We hope this proposal responds to some of the other concerns raised in the 1986 Maize Conference and reflects to a suitable degree the suggestions and comments of Cooperators who have discussed the need for a systematic nomenclature.

The ambiguity to be avoided is that of specifying a polymorphic locus as the structural gene or coding sequence when it is not. Probes are rapidly being obtained for functional products, e.g., enzymes for which no "Mendelizing" variation in the product has been established. For example, a genomic clone for the second sucrose synthase gene has been used to map a new locus through polymorphisms probed by the clone (D.R. McCarty et al., PNAS, 1986); an impressive number of other loci, probed by homologous or heterologous probes, is presented by T. Helentjaris and collaborators in a new report in this issue of the News Letter. Taken at face value, the assumption is easily made that the polymorphism (which is the genetically mapped property) is co-sequential with the functional gene; but it is much more probable that the polymorphism is actually in an intron or in an adjacent, noncoding sequence. While current mapping resolution makes the distinction moot for the time being, we suggest that, in the interest of accuracy, the polymorphism be defined separately from the functional unit.

Our nomenclatural suggestion is that, following the practice of T. Helentjaris in the maps to date, a polymorphic locus be defined by *a number that is uniquely applied to the segregating variation, specifically mapped relative to other factors.*

For the 3-letter designator, a reconsideration is in order. In MNL 60 the interim symbol RFP, with the number appended to it, was used for purposes of indexing, especially toward consolidating the loci under one source symbol. Unfortunately the use of one 3-letter symbol for all polymorphisms may lead to overlapping numbers, and would require a clearing-house system. A simple alternative is for each laboratory to choose a *distinctive 3-letter symbol for the source of the mapping study*, e.g., NPI, UMC, PIO, etc. Thus the loci will be *NPI1, NPI2*, etc., *UMC1, UMC2*, etc., with the number immediately following the 3-letter symbol without a hyphen, consistently with the current standards of nomenclature.

For loci that are defined by a probe for a functional product, *a unique number for the locus should again be chosen by the lab that defines and maps the polymorphism.* The product may, if desired, be specified by a hyphenated addition to the numbered symbol. Thus, the polymorphic locus defined by a probe for the small subunit of rubisco could be, for example, *NPI227-ssu*. In our current standards of nomenclature a hyphenated addition specifies allelic variations at a locus, and the particular RFP morph could be specified efficiently by numbers or letters, as with isozymes, with or without the functional specification.

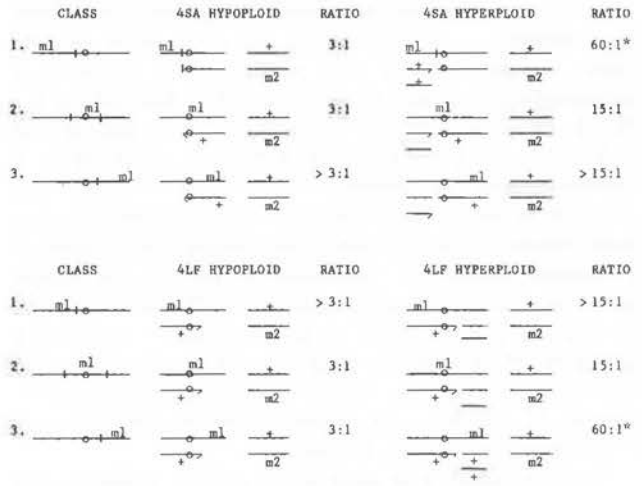
This proposal is used in the working maps presented with this issue of the News Letter, and we invite comments, suggestions or critiques.

Ed Coe and Dave Hoisington

Hyperploid and hypoploid selfs provide accurate arm location of duplicate factors

Location of *orp1* on chromosome 4S demonstrates a useful aspect of the selfed-hypoploid test for locating duplicate factors. If mutant alleles of both members of the duplicate pair are present in an ear stock and are crossed by both short arm and long arm B-A translocations for the chromosome on which one member of the pair is located, then depending upon whether the mutant tested is (1) distal to the breakpoint on the short arm; (2) between the breakpoints on both arms; or (3) distal to the long arm

breakpoints, the following types, as reflected by normal to orange ratios, should occur:



*Approximation only: depends on a number of factors.

The diagnostic ratios are: 1) 3:1 ratios for all the segregating hypoploid selfs, which place the mutant on the correct chromosome; 2) 15:1 ratios for all the segregating hyperploid selfs for both translocations, which would place the mutant between the breakpoints of both arms; and (3) the 60:1 vs. the 15:1 ratios for the segregating hyperploid selfs of the respective B-A translocation, which would place the mutant on the correct arm.

M.G. Neuffer and J.B. Beckett

Designation of new recessive mutants

Over the years we have tested a large number of recessive mutants for location to chromosome arm using the B-A chromosome set. Initially mutants were placed after a single test but it was learned that a single positive test is not always correct; hence, a series of confirmation tests were conducted. There is now a group of 666 recessive mutants that have been located and confirmed to chromosome arm. From this group those with no counterpart on the same arm among the current gene list have now been given a name and symbol. These are listed by chromosome arm with a brief description where appropriate.

M.G. Neuffer and J.B. Beckett

Recessive Seedling Mutants

Gene Symbol	Name	Lab Symbol	Description
		CHROMOSOME 1S	
116	luteus	1*-515	yellow seedling bleaches to paler yellow in patches
pg15	pale green	ppg*-340B	pale green seedling bleaches to near white in patches
v25	virescent	v*-17	greenish white seedling, greens from the base upward
		CHROMOSOME 1L	
hcf13	high chloro-phyll fluorescence	hcf*-1097B	
ij2	iojap	ij*-8	white stripes; green with white margins
117	luteus	1*-544	yellow seedling with lighter crossbands
pg16	pale green	pg*-219	pale green seedling
py2	pigmy	py*-521A	
spc2	speckled	spc*-262A	green seedling with light green speckles
zb7	zebra	zb*-101	lighter green crossbands, glossy
		CHROMOSOME 2S	
nec4	necrotic	nec*-516B	general necrosis beginning tip of 2nd leaf, spreads to entire seedling
v26	virescent	v*-453	yellowish white with green tip and midrib
		CHROMOSOME 2L	
118	luteus	1*-1940	yellow seedling
spt1	spotted	spt*-464	pale green with dark green spots
v24	virescent	v*-424	pale yellow green to pale green
		CHROMOSOME 3S	
gl19	glossy	gl*-169	glossy seedling; lethal
		CHROMOSOME 3L	
spc3	speckled	pg*-553C	speckled pale and dark green seedling
wlu1	white luteus	wl*-28	pale yellow seedling
		CHROMOSOME 4S	
spt2	spotted	pg spt*-1269A	pale green with dark green spots
wt2	white tip	cb*-10	white tip and crossbands on first 2 leaves
		CHROMOSOME 4L	
nec5	necrotic	nec*-642A	necrotic seedling-dark brown exudate
		CHROMOSOME 5S	
nec6	necrotic	nec*-493	tan necrotic seedling
		CHROMOSOME 5L	
grt1	green tip	grt*-1308B	pale yellow seedling with green leaf tip
nec7	necrotic	nec*-756B	necrotic seedling appears first in crossbands
ppg1	pale pale green	cb*-199A	white with faint green; white necrotic crossbands
wgs1	white green sectors	sct*-206B	white with green sectors
		CHROMOSOME 6L	
gs3	green stripe	gs*-268	light green midvein; like gs2
		CHROMOSOME 7L	
v27	virescent	v*-590A	pale yellow to normal green
wlu2	white luteus	wl*-543A	pale yellow seedling
		CHROMOSOME 8L	
v21	virescent	v*-25	light green seedling; greens first at leaf tip, margins, and midrib
wlu3	white luteus	wl* 203A	pale yellow seedling
		CHROMOSOME 9S	
v28	virescent	v*-27	pale yellow green to pale green
		CHROMOSOME 9L	
wlu4	white luteus	wl*-41A	pale yellow seedling
		CHROMOSOME 10S	
gl21	glossy	gl*-478B	glossy seedling
119	luteus	1*-425	yellow seedling;
		CHROMOSOME 10L	
113	luteus	1*-59A	yellow seedling
v29	virescent	v*-418	light green with grainy appearance

Designation of new dominant mutants

We have tested many dominant mutants for location to chromosome using the waxy translocation set. These are listed here by chromosome with gene symbol, name, and original temporary laboratory symbol and number.

Gene Symbol	Name	Lab Symbol	Description
		CHROMOSOME 1S	
Les2	lesion	Les*-845A	tiny white necrotic spots 1 cm srl
Les5	lesion	Les* 1449	like Les2, 20 cm srl
		CHROMOSOME 1L	
Les7	lesion	Les*-1461	many tiny yellow chlorotic spots
Msc1	mosaic	Msc*-791A	aleurone mosaic
Tlr1	tillered	Tlr*-1590	heterozygote has extra tillers
		CHROMOSOME 2S	
Les1	lesion	Les*-843	large necrotic lesions beginning at 2-leaf stage
		CHROMOSOME 2L	
Les4	lesion	Les*-1375	
		CHROMOSOME 3S	
Wrk1	wrinkled	Wr*-1020	wrinkled small kernel
		CHROMOSOME 3L	
Spc1	speckled	Spc*-1376	brownish speckles on leaf blade--near flowering stage, midrib and leaves limp
		CHROMOSOME 4S	
Ysk1	yellow streak	Ysk*-844	yellowing of mid-vein areas, strong anthocyanin on leaf tip and margins
		CHROMOSOME 4L	
Ms41	male sterile	Ms*-1995	shrivelled anthers not extruded; (like ms1)
		CHROMOSOME 5S	
Msc2	mosaic	Msc*-1124B	aleurone mosaic of colored and colorless with ACR
Rgd2	ragged	Rgd*-1445	leaves narrow and distorted, extra tillers
		CHROMOSOME 5L	
Hsf1	hairy sheath striped	Hsf*-1595	excess hairs on sheath, leaf margins etc. Homozygote has abnormal growth on leaf margins
		CHROMOSOME 7	
Les9	lesion	Les*-2008	
		CHROMOSOME 8	
Clt1	clumped tassel	Clt*-985	plant short, 1/2 normal height, tassel and ear with compressed spikelets
Bif1	barren in-fluorescence	Bif*-1440	few to most spikelets missing from ear and tassel
Sdw1	semidwarf	Sdw*-1592	3/4 normal height erect leaves

Gene Symbol	Name	Lab Symbol	Description
		CHROMOSOME 9	
Zb8 (was Atc1)	zebra	C1*-1443	yellow green crossbands, strong anthocyanin in leaf tip and blade
G6	golden	G*-1585	lighter yellowish sheath
Les8	lesion	Les*-2005	
		CHROMOSOME 10S	
Les6	lesion	Les*-1451	
		CHROMOSOME 10L	
Vsr1	virescent striped	Vsr*-1486	virescent seedling, greens to normal, with many white and yellow green stripes

M.G. Neuffer, D.A. Hoisington, and R. McK. Bird

Location of dominant golden sheath on chromosome 9S

Golden sheath, *G6*, was located on chromosome number 9 using the T *wx* series. The backcross data showed *G Wx* linkage for several translocations, as listed.

G 1585	BRK PT	#	Pl	MWx	NWx	Mwx	Nwx	1:1 CS	% CO	CS
1-9c	S.48 L.22	80	39	10	8	23		24.42	23	0.32
1-9(4995)	L.19 S.20	88	27	13	18	30		7.90	35	0.24
1-9(8389)	L.74 L.13	66	34	15	4	13		12.13	29	0.31
2-9b	S.18 L.22	84	26	6	17	35		18.73	27	1.94
2-9d	L.83 L.27	90	38	14	7	31		26.23	23	0.89
3-9c	L.09 L.12	79	37	18	6	18		12.56	30	0.47
4-9g	S.27 L.27									
4-9(5657)	L.33 S.25									
4-9b	L.90 L.29	119	44	18	22	35		13.87	34	1.22
5-9c	S.07 L.10	89	38	12	12	27		19.29	27	0.51
5-9a	L.69 S.17	105	25	26	18	36		6.02	42	3.36
6-9a	S.79 L.40	188	71	21	18	78		64.67	21	0.47
6-9b	L.10 S.37	201	63	31	36	71		22.34	33	0.01
7-9(4363)	.00 .00									
7-9a	L.63 S.07	86	22	22	19	23		0.38	48	0.20
8-9d	L.09 S.16	87	34	18	10	25		11.35	32	0.35
8-9(6673)	L.35 S.31	96	42	11	10	33		30.43	22	0.09
9-10b	S.13 S.40	105	31	16	18	40		13.13	32	0.11

The closest linkage was with T6-9a and T8-9(6673), with breakpoints at S.79, L.40 and L.09, S.16, respectively. Subsequent crosses of *G Wx/g wx* x *g wx*, in which no translocation was involved, gave 54 *G Wx*, 13 *g Wx*, 12 *G wx*, 60 *g wx* for a total of 25 crossovers among 139 progeny tested, for a map distance of 18 cM. Progeny from crosses of golden plants by B-A translocations TB-9Sb and TB-9Lc gave both golden and green hypoploids, and TB-9Lc hyperploids gave both golden and green plants; however, TB-9Sb hyperploids were all green, indicating that *+/+/G6* is green and that *G6* is located beyond the breakpoint of TB-9Sb. The above information places *G6* on the short arm of 9, approximately 18 cM distal to *wx*.

M.G. Neuffer

Location of dominant male sterile on chromosome 4L

Male sterile, *Ms41*, was located on chromosome 4 using the T *wx* series. The backcross data listed show close *Ms Wx* linkage for T4-9b, less close for T4-9(5657) and very little for T4-9g. These data place *Ms41* on the long arm near, and possibly distal to, the breakpoint of T4-9b.

Ms_1995	BRK PT	#	Pl	MWx	NWx	Mwx	Nwx	1:1 CS	% CO	CS
1-9c	S.48 L.22	103	24	28	24	27		0.48	50	0.47
1-9(4995)	L.19 S.20	109	28	27	23	31		1.20	46	0.46
1-9(8389)	L.74 L.13									
2-9b	S.18 L.22	104	27	27	15	35		8.00	40	4.31
2-9d	L.83 L.27	156	35	49	26	46		7.89	48	7.67
3-9c	L.09 L.12	105	15	37	23	30		10.23	57	8.26
4-9g	S.27 L.27	88	23	22	22	21		0.05	50	0.05
4-9(5657)	L.33 S.25	78	29	19	7	23		10.62	33	2.19
4-9b	L.90 L.29	100	45	3	1	51		84.83	4	1.22
5-9c	S.07 L.10	88	20	34	12	22		6.57	52	6.40
5-9a	L.69 S.17	129	33	31	27	38		1.92	45	0.62
6-9a	S.79 L.40	102	24	28	23	27		0.63	50	0.63
6-9b	L.10 S.37	95	29	20	26	20		2.44	48	2.34
7-9(4363)	.00 .00	174	37	60	40	37		5.57	57	1.72
7-9a	L.63 S.07	103	21	27	18	37		7.31	44	5.76
8-9d	L.09 S.16	88	14	37	18	19		10.40	63	5.23
8-9(6673)	L.35 S.31	188	52	50	38	48		1.20	47	0.44
9-10b	S.13 S.40	107	25	27	29	26		0.24	52	0.01

M.G. Neuffer

Chromosome 8 linkage studies

The accompanying tables contain linkage data for *de*-1386A* with *Bif1, pro1, v16, ms8* and *j1*. The data further support the order *Bif1-pro1-v16-ms8-j1* (MNL 58:77). All listed tests are Repulsion Backcross type. All paired recombination values were determined by the maximum likelihood method.

INDIVIDUAL TOTALS

prol	ms8	j1	de	TOTAL
61	97	101	126	220
(27.7%)	(44.1%)	(45.9%)	(57.3%)	

RECOMBINATION CLASSES

Rgn	Genotype	#	Totals	Expected
0	+ + + de	88		
	prol ms8 j1 +	35	123	119.2
1	prol + + de	17		
	+ ms8 j1 +	40	57	58.0
2	prol ms8 + de	1		
	+ + j1 +	12	13	16.0
3	prol ms8 j1 de	5		
	+ + + +	3	8	11.3
1,2	+ ms8 + de	7		
	prol + j1 +	1	8	7.8
1,3	+ ms8 j1 de	6		
	prol + + +	0	6	5.5
2,3	+ + j1 de	2		
	prol ms8 + +	2	4	1.5
1,2,3	prol + j1 de	0		
	+ ms8 + +	1	1	0.7

PAIRED RECOMBINATION VALUES

prol ---	ms8	0.3273 +/- 0.0316
prol ---	j1	0.3636 +/ 0.0324
prol ---	de	0 3591 +/- 0.0323
ms8 ---	j1	0.1182 +/- 0.0218
ms8 ---	de	0.1591 +/- 0.0247
j1 ---	de	0.0864 +/- 0.0189

INDIVIDUAL TOTALS

Bif	prol	v16	de	TOTAL
79	71	75	76	157
(50.3%)	(45.2%)	(47.8%)	(48.4%)	

RECOMBINATION CLASSES

Rgn	Genotype	#	Totals	Expected
0	+ + + de	25		
	Bif prol v16 +	17	42	49.1
1	Bif + + de	10		
	+ prol v16 +	9	19	17.9
2	Bif prol + de	22		
	+ + v16 +	26	48	39.5
3	Bif prol v16 de	10		
	+ + + +	7	17	14 6
1,2	+ prol + de	4		
	Bif + v16 +	8	12	14.4
1,3	+ prol v16 de	2		
	Bif + + +	7	9	5.3
2,3	+ + v16 de	3		
	Bif prol + +	5	8	11.8
1,2,3	Bif + v16 de	0		
	+ prol + +	2	2	4.3

PAIRED RECOMBINATION VALUES

Bif ---	prol	0.2675 +/- 0.0353
Bif ---	v16	0.5350 +/- 0.0398
Bif ---	de	0.5478 +/- 0.0397
prol ---	v16	0.4459 +/- 0.0397
prol ---	de	0.5478 +/- 0.0397
v16 ---	de	0.2293 +/- 0.0336

Dan J. England and M.G. Neuffer

Mutable cytochrome f/b6 mutant isolated in *Spm* background

We report the isolation of a mutable nuclear pale green recessive maize mutant, *pg*-m1998*, from a line with autonomous *Spm*. Leaf chlorophyll fluorescence induction kinetics indicated that the mutant is blocked on the reducing side of photosystem II. Analysis of thylakoid polypeptides by lithium dodecylsulfate polyacrylamide electrophoresis, followed by sequential staining for heme and polypeptides, revealed that the mutant had greatly reduced amounts of a polypeptide that stained positively for heme and corresponded to cytochrome f (35-37 kD apparent molecular weight). No cytochrome b6 was detected by heme-staining. No other differences were noted in the polypeptide composition by these criteria.

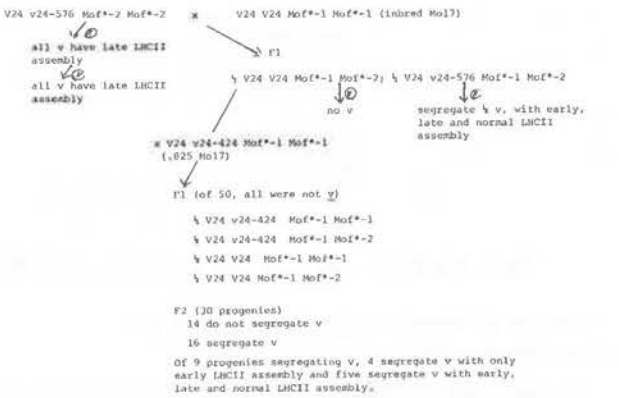
Western blot analyses confirmed that the mutant had trace levels of cytochrome f and also showed that the mutant had reduced (50-70%) levels of subunit 4 and Rieske FeS polypeptide. We had no specific antisera for cytochrome b6. The antisera were kindly provided by W. Taylor, Berkeley, CA. Of the four polypeptides in the cytochrome f/b6 complex, only one, the Rieske FeS polypeptide, is nuclear encoded. Two nonallelic nuclear mutations (*hcf2, hcf6*) have been reported in maize that inhibit assembly of this complex (Metz et al., Plant Physiol. 73:452-459, 1983). We plan to test allelism of the new locus with *hcf2* and *hcf6*. The recent cloning of the spinach structural gene for the *pg*-m1998* Rieske FeS polypeptide should permit assessment of linkage with this locus (loci) if the maize and spinach gene(s) share adequate homology. Work is in progress to test whether *pg*-m1998* is under *Spm* control, and to ascertain whether the other heme protein of thylakoids, cytochrome b559, is affected by the mutation.

Leonard Rosenkrans, Mary Polacco and Craig Echt

*Mof** is unmasked by two *v24* mutations

We have been studying a locus that affects the assembly timing of the major chlorophyll protein complex, LHCII, an antenna complex for photosystem II. Previous work (MNL 60:44) has shown that inbred Mo17 carries an allele (*Mof*-1*) that allows premature assembly of LHCII in *v24-576* (previously designated *v*-576*) material. A second allele, *Mof*-2*, segregated in the original material segregating *v24-576*, and rendered LHCII assembly abnormally late. Heterozygous material, *Mof*-1 Mof*-2*, had apparently normal assembly timing of LHCII. The effect of *Mof** on LHCII assembly is only observed in virescent material and not in normal siblings.

To test whether *Mof*-2* is unmasked by other *v24* alleles, the F2 progenies were examined for a cross between *V24 V24 Mof*-1 Mof*-2* and *V24 v24-424 Mof*-1 Mof*-1* (see accompanying figure for pedigree of parental genotypes and for information that substantiates genotype assignment; *v24-424* was previously designated *v*-424*). The original material segregating *v24-424* has shown no evidence of the *Mof*-2* trait. Our backcrossing program for *V24* alleles has until recently only involved Mo17 (*Mof*-1 Mof*-1*). Within each progeny segregating virescent seedlings, 16-25 *v* seedlings were tested for *Mof*-1* or *Mof*-2* by combined criteria of leaf chlorophyll induction kinetics and initial fluorescence yield (described



in MNL 60:44). Confirmation of the LHCII assembly timing with fluorescence was obtained for two progenies by analyses of thylakoid polypeptides. The data show that *Mof*-2* can also act on *v24-424*. Four of nine progenies segregated only *v* seedlings with early LHCII assembly (*Mof*-1 Mof*-1*) while the remaining five segregated *v* seedlings with early, normal and late LHCII assembly (*Mof*-1 Mof*-2* selfed).

In progress: We are testing (1) whether *Mof** is linked to any *cab* loci that encode apo-proteins of LHCII, (2) whether *Mof** can affect timing of LHCII assembly of other *v* mutations (*v, v3, v5, v12, v16*), and (3) whether any inbred lines carry *Mof*-2*.

Mary Polacco

Inter-regional maize inbred evaluation report

Recently released maize inbred lines were evaluated in five maturity groups (Table 1) for agronomic characteris-

Table 1. Inbreds and maturity groups in the inter-regional maize evaluation.

Number	100-300	400-600	700-800	900-1000	1100-1200
1	CM105 *	A619 *	B73 *	CI66 *	SC76 *
2	CO109 *	A632 *	Mo17 *	FR802W *	SC213 *
3	A661	B85	N28Ht *	FR805W *	T232 *
4	A665	A634	B68	Ga209	GT112RF
5	A668	A635	B75	Mo17	NC246
6	A671	A659	B76	T145	NC248
7	ND100	A670	B77	T147	SC12
8	ND240	Mo42	B79	T151	SC43
9	ND241	AY499	B84	T153	SC55
10	ND245	NY552	Mo14W	T155	Mp496
11	ND246	NY378	Mo20W	T159	Tx601
12	ND300	NY821LERF	Mo40	T250	
13	ND301	NYD410	Mo42	T254	
14	ND376	NYRW3	N132	T256	
15	ND408	NYRW20	N139	T258	
16	ND474	NYRW23	N152	Ar258	
17	Pa326	Pa405	Oh509A	Ar262	
18	Pa329	FR19	Oh514	Ar266	
19	Pa373	CH9	Pa91	Tx29A	
20	Pa374	CH581-13	Pa762	Tx61M	
21	CK52	CH586-12	Pa871	Tx403	
22	CK64	CH591-36	Pa872	Tx5855	
23	CK69	CH592-46	FR16	Tx6252	
24	CK75	CH593-9	FR20		
25	CG11	CH606-11	FR21		
26	CG12	CH663-8	H80		
27	CG13	B87	H84		
28	CG14	Ms71	H93		
29	CG15	Ms75	H98		
30	CG16	Ms76	H100		
31	CG17	Ms200	H102		
32	CG18	H95	H103		
33	CL1	H99			
34	Ms72	W64A *			
35	Ms74	W548			
36	W117Ht *	W552C			
37		W562			
38		W570			
39		CH753-4			
40		CH871-28			

* Check entry.

tics and reaction to various diseases and insects. Agronomic data reported include yield, stand, root lodging, stalk lodging, usable ears, plant height, ear height, grain moisture, days-to-tassel, days-to-silk, ear row number, ear length, ear diameter, 300-kernel weight, and stalk crushing strength. Disease reactions include bacterial wilts, ear rots, fungal leaf diseases and rusts, smuts, stalk rots, and viruses. Insect data includes reactions to first and second generations of the European corn borer, corn earworm, fall armyworm, and the southwestern corn borer.

The results have been published as Missouri Special Report no. 325 and should be a valuable reference for maize researchers who work with inbred lines. Copies can be ordered from Extension Publications, University of Missouri, 115 South Fifth Street, Columbia, MO 65211 at a postage paid cost of $5 per copy. Be sure to indicate that you want SR325.

L.L. Darrah

CORVALLIS, OREGON
Oregon State University

A modified slot blot technique for use with nylon membranes

In this report, we present refinements to a previously published technique for slot blotting, a method we have used for detecting and measuring quantitative polymorphisms in maize DNA sequences. We have found this technique to be useful for studying copy number variation of repeated sequences among various maize varieties (Rivin et al., Genetics 113:1009, 1986) and for examining very low copy numbers of Robertson's Mutator transposon-homologous sequences in non-Mutator maize lines (Chandler et al., Genetics 114:1007, 1986). A detailed protocol for the technique and data analysis has been published (Rivin, Meth. Enz. 188:75-86, 1986).

We have modified our procedure by adapting the method of Reed and Mann (NAR 13:7207, 1985) for Southern blotting onto positively charged nylon membranes. There are two major differences in the modified method: denatured DNA samples are not neutralized prior to loading onto the filters and baking filters in vacuo prior to hybridization is eliminated. These changes represent a great savings in time and ease of handling and the resulting blots give very sharp signals that are stronger than with the original method.

Modified slot blot method:

1. Preparation of the apparatus: The slot blot apparatus is soaked in a 0.4 M NaOH solution prior to use. A positively charged nylon filter (we use Genatran) is wetted in distilled water and then briefly rinsed in 0.4 M NaOH. The apparatus is assembled as described previously.
2. Preparation of DNA samples: The DNA samples to be loaded are denatured by adding NaOH to a final concentration of 0.2 M and heated at 95C for 2 minutes.
3. Loading samples: The denatured samples are cooled on ice and vortexed before loading into the slots. After the entire sample has blotted, the well is rinsed through with 200 microliters of 0.4 M NaOH.
4. Handling the filter: After the apparatus is disassembled, the membrane is soaked for 5 minutes in 5 X SSC. It is then ready for prehybridization. The filters do not need to be baked. They can be stored after air drying.

Donna Hazelwood and Carol Rivin

Mutator-homologous sequences in normal lines and in somaclonal variants

Genetically stable maize lines and varieties have been shown to have sequences homologous to Robertson's Mutator transposons, including apparently intact transposon structures (Chandler et al., Genetics 114:1007, 1986). We have found three kinds of *Mu*-homologous sequences in normal maize: (1) *Mu1* terminal sequences not associated with internal sequences, (2) "endogenous elements"—structures very similar to *Mu1* and *Mu2* (also known as *Mu1.7*) are found in many, but not all normal maize lines we have examined, and (3) "endogenous sequences"—a sequence similar to an internal sequence of *Mu2* is found in every maize line we have checked. We have also found it in teosinte, *Zea diploperennis* and *Zea mexicana* samples. Both endogenous elements and sequences appear to be very stable in normal genetic backgrounds. We have found no differences in genomic restriction patterns among individuals of the line W22 (which carries a *Mu2*-like element as well as the endogenous sequence) or between W23 and the Golden Glow population from which it was derived.

The "endogenous sequence" from W23 has been cloned and sequenced. It is highly homologous to *Mu2*, but it has no *Mu* termini and is missing 401 bp of internal sequence. It does not have a transposable element structure. In all maize lines and relatives examined, the endogenous sequence is flanked by the same non-Mutator sequence. The endogenous sequence has been mapped to chromosome 2 (T. Helentjaris, pers. comm.).

We are interested in the possibility that cryptic transposons may become activated during growth in tissue culture or during regeneration and lead to the production of new phenotypes or somaclonal variants. To investigate this possibility, we have examined the *Mu*-homologous sequences in the inbred line W182BN and in eleven somaclonal variants that were derived from it. Both the inbred and the somaclones were given to us by Elizabeth Earle.

DNA from these sources was cut with a variety of restriction enzymes, electrophoresed in adjacent lanes of agarose gels and Southern blotted. The blots were probed with clones from internal and terminal portions of *Mu1* (see Chandler et al., 1986) and with an internal sequence unique to *Mu2*. The latter clone was given to us by Loverine Taylor. We found that W182BN carries a sequence with a structure like *Mu2* and two different endogenous sequences. These two sequences differ in their degree of homology to *Mu2*, but they are each flanked on one side by the same non-Mutator sequence.

When the DNA samples are cut by enzymes that cleave inside the *Mu*-homologous regions of the three sequences, we see no difference between the inbred lines and the somaclonal derivatives, indicating that tissue culture and regeneration has not changed the structure or the DNA modification pattern of these sequences. However, when cut with restriction enzymes that cut outside *Mu*-homologous regions, one of the somaclonal lines shows the loss of a restriction band and the gain of a new band. One

of the endogenous sequences rather than the intact *Mu2* -like sequence appears to be involved in this rearrangement. We are continuing to map this change to determine what kind of rearrangement has occurred in this somaclone and what role the *Mu*-homologous sequence may have played in creating it.

Carol Rivin, Chee Harn,
Vicki Chandler[1] and Luther Talbert[1]

[1]University of Oregon

ABA and a developmental switch in embryogeny

Developing maize embryos have the capacity to either mature or germinate. Many lines of evidence point to the hormone abscisic acid (ABA) as playing an important role in directing the embryo into maturation. We have been examining embryo maturation and germination in wildtype maize and in viviparous mutants both in planta and in culture to try to dissect the regulatory pathways of this developmental switch and to learn how ABA may control them. We have looked at the polypeptides produced at various stages of embryo development and correlated the appearance of these with the changing levels of embryo ABA. We have also cultured dissected embryos on medium with or without ABA and looked at the polypeptides produced at various stages.

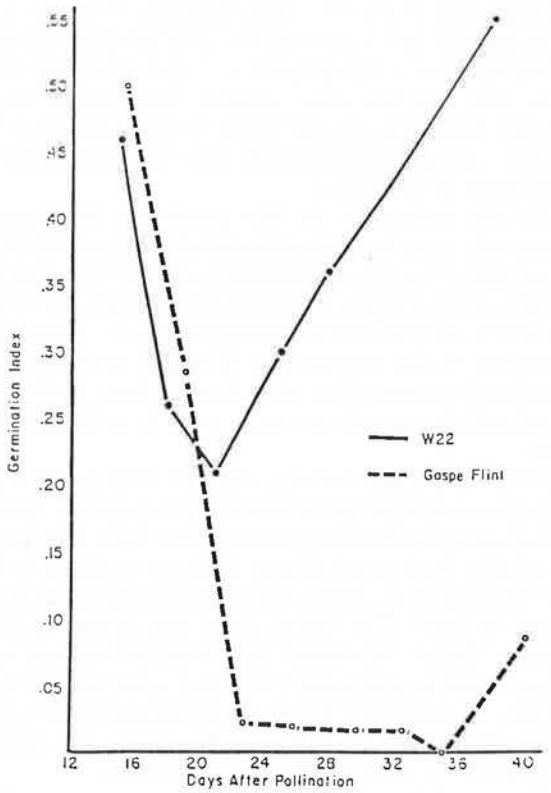


Figure 1. Germination index for embryos of the wild type lines W22 and Gaspé Flint. The embryos were dissected from ears harvested at the indicated days after pollination and then placed in a nutrient medium containing no hormones for five days. The germination index is a weighted value of % embryos germinating on each day in culture. It is large when embryos germinate quickly. W22 embryos at 18-25 days show a lag time in culture before germinating. Very few Gaspé Flint embryos 20-35 DAP germinate at all in five days of culturing.

We have found ABA regulated proteins in wildtype maize are first apparent about 18 days after pollination (DAP) (stage 3), a stage when embryo ABA levels become quite high. Synthesis of these proteins can be stimulated in embryos as young as 10 DAP (stage 2) by culturing on 10^{-5} M ABA. The mutants *vp2* and *vp5* do not synthesize normal levels of embryo ABA nor do they accumulate these maturation proteins. These mutants germinate precociously on the ear. If cultured with ABA, however, they are inhibited from germinating and the normal spectrum of maturation polypeptides is produced.

When developing embryos are cultured without ABA, they germinate within a few days. The rate of germination varies during early and mid-development. W22 embryos dissected out 18 to 25 DAP and cultured without ABA show a lag of a few days before they stop making maturation proteins and begin to germinate, although the ABA content of these embryos drops very quickly in culture. As shown in Figure 1, embryos of the variety Gaspé Flint behave very differently. At about 18 DAP the germination rate also drops in these embryos, in fact it becomes practically zero, but they do not regain the capacity to germinate precociously until about 40 DAP, long after the endogenous ABA level has declined. If these embryos are prematurely dried and reimbibed, however, they germinate quite synchronously within 24 hours.

The non-germinating embryos of Gaspé Flint are actively synthesizing the proteins characteristic of matu-

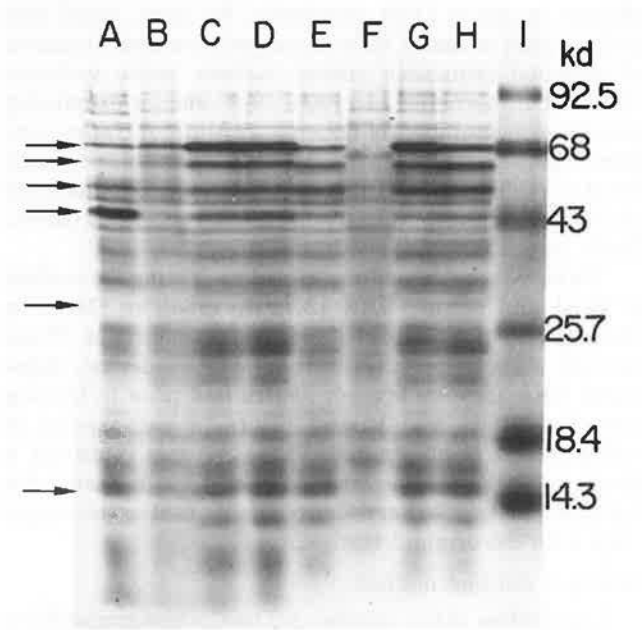


Figure 2. Gaspé Flint embryos dissected at 25 and 30 days after pollination (DAP) were initially labelled for 6 hours with ^{3}H-leucine or were labelled after culturing 5 days in basal media with or without 10^{-5} M ABA. Lanes A-D, 25 DAP; E-H, 30 DAP.

- **A, E: labelled after dessication.**
- **B, F: basal media without hormones - embryos germinating after 5 days in culture.**
- **C, G: basal media without hormones - embryos not germinating after 5 days in culture.**
- **D, H: basal media with 10^{-5} M ABA.**
- **I: protein standards.**

Fluorography shows that synthesis, as well as accumulation, of proteins marked with arrows is similar in lanes C and D and in lanes G and H.

ration phase, although the level of ABA in these embryos is negligible. Addition of ABA to the medium does not cause any change in the accumulation of these proteins (Figure 2). It appears that in W22 embryos the induction of maturation proteins and the inhibition of germination caused by ABA is lost quickly when the hormone is removed, but in Gaspé Flint embryos the developmental program initiated by ABA may be stable for a very long period of time.

Chris Holmes-Baker, Timothy Grudt and Carol Rivin

FREIBURG, WEST GERMANY
University of Freiburg

Binding of nuclear factors to upstream regions of zein genes

The tissue and developmental specific expression of zein genes is highly regulated. It can be assumed that at least part of this regulation is achieved by *trans*-acting factors. This assumption is supported by genetic data with mutants such as opaque that affect zein protein production.

In this context we have started to look for the specific interactions of nuclear proteins with sequences lying upstream of cloned zein genes. In the case of pMS1, a clone containing a gene coding for a 19,000 dalton protein, we have shown by nitrocellulose filter binding and gel retention techniques several specific binding sites (see scheme). We have characterized one of these sites (at position -300) by footprint analysis and have shown a 22 bp protein binding site, which contained a 15 bp sequence that is found in all zein genes analyzed so far. Interestingly, this binding site can only be seen with nuclear extracts from endosperm tissue where the zein genes are specifically expressed. The other potential binding sites are being further characterized.

Scheme: Flanking sequence of pMS1 (see MNL 58:88-89, 1984). The open triangle points to the binding site at -300 and the closed triangles point to further uncharacterized binding sites.

We conclude that the extensive flanking regions of zein genes are important for the regulation of the zein gene system, and more specifically for the interaction with the gene products of regulatory genes.

The first part of this work will be published in the January issue of the EMBO Journal.

U. Maier, J.W.S. Brown and G. Feix

GAINESVILLE, FLORIDA
University of Florida and USDA-ARS

On gene symbolization for the second sucrose synthase enzyme

Two sucrose synthase (SS) isozymes in maize have been known for the past several years (Chourey and Nelson, Biochem. Genet. 14:1041, 1976, and Genetics 91:317, 1979). The *Sh*-locus-encoded enzyme is unique with respect to its abundant levels in the *Sh* endosperm. The genetic basis for the second sucrose synthase isozyme, which was first uncovered through the analysis of *sh* mutants, was however not firmly established initially. Although a hypothesis that the second enzyme is due to a second gene (i.e., non-allelic to *Sh*) was favoured, an alternative idea that it was due to leaky expression of the *sh* allele in *sh* genotype could not be ruled out unequivocally. In this regard, the SS analyses of the *sh*-deletion strain (*sh bz-m4*) were quite useful, since the deletion stock proved to be similar to several spontaneous *sh* mutants, providing the first conclusive evidence that a second SS gene was present in the maize genome (Chourey, MGG 184:372, 1981). In addition, it was also possible to demonstrate reproducibly that there were two SS proteins in *Sh* endosperm, thus demonstrating that both genes are expressed in homozygous *Sh* endosperm (MGG 184:372, 1981). McCormick et al. (MGG 187:494, 1982) showed molecular similarity between the two SS encoding genes. Although no formal attempt was made to name the second gene, the most obvious and practical symbol, *Sucrose synthase 2* (*Ss2*) for the gene and SS2 for its enzyme, have been used in the past several publications originating from this laboratory on the implicit assumption that the *Sh* encodes the SS1 enzyme. During the 1981-1986 period, at least 4-5 presentations on various aspects of the *Ss2* gene have also been made at the Annual Maize Genetics meetings. Interestingly, no concerns or comments to express change in the *Ss2* symbol were raised at these meetings, the best possible forum for the discussion of such matters. A recent publication, however (McCarty et al., PNAS 83:9099, 1986), uses an entirely new name, *constitutive sucrose synthase (Css)*, instead of the previous designation, *Ss2*. A casual rejection of the *Ss2* symbol is surprisingly uncorroborated by any statements to justify the action. Ironically, McCarty et al. used the *Ss2* symbol to discuss the present data at the 29th Maize Genetics meetings (1986). The main intent of this communication is to point out that the *Css* symbol for the *Ss2* gene is inappropriate for the following reasons:

(1) *Ss2* expression, in contrast to McCarty et al.'s claim, is *not constitutive* in maize plants. Although the statement is made that "Css *protein* has been found in all maize tissues so far studied", (my underlining), an assertion of such critical importance is surprisingly unsubstantiated either by data or by reference to previous publications. This information is critical, since a previous extensive study of starch synthesis enzymes in endosperm and pollen has detected no SS activity in mature or immature pollen (Bryce and Nelson, Plant Phys. 63:312, 1979). I have similarly not detected any SS activity in pollen derived from plants of *Sh* or *sh* genotype (unpubl. data). The *Css* symbol would thus need to specify the tissues where the gene is expressed constitutively.

(2) In principle, gene symbolization on the basis of regulatory properties is unprecedented in maize. Usually, one needs a name for the gene before it is analyzed. This may explain why some of the genes, particularly those which are better characterized for their regulatory properties (e.g., *Adh1* and *Adh2*) than the SS encoding genes, are symbolized on a neutral basis such as the order of their discovery. Furthermore, I believe, we know very little about the regulatory properties of *Sh* and *Ss2* at the present time and it is extremely premature to label one as

Css. The successful use of Western blot analysis on denaturing gels to examine SS specific proteins and the recovery of gene specific probes to analyze transcripts will, however, soon fulfill this deficiency. Neutral symbols such as *Sh* and *Ss2* allow such analyses to be done independently of their regulatory properties.

(3) Traditionally, constitutive refers to some constant level of expression at the protein/enzyme level, although it could include expression at any point in the path of gene expression. The fact that one can now incisively analyze various steps in gene expression and define points of blockage, demands that greater care be taken in gene symbolization. It is entirely possible that *Ss2* could be constitutive at the transcription level but highly regulated at the protein level (an on-going project in my lab). Indeed, SS2 protein as well as the enzyme levels in leaf tissue of one-week and nearly four-week-old seedlings of the *sh* genotype vary by more than an order of magnitude (unpubl. data); transcription analyses are presently in progress. The point is that the designation "constitutive" in gene symbolization, as in the case of the *Css* symbol, sets an undesirable precedent and appears ambiguous due to the lack of information regarding the level of analysis or specifically, the level at which the gene is constitutively expressed.

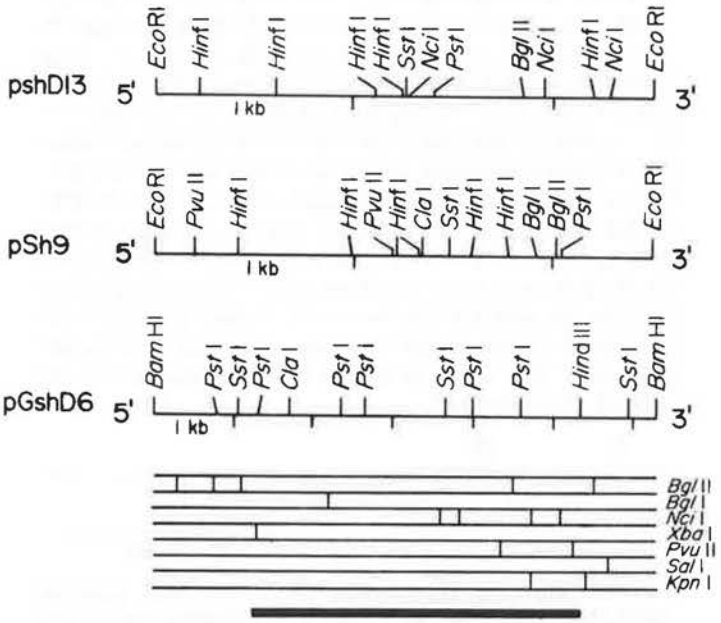
In summary, although gene symbolization for SS enzymes is a bit problematic due to certain unusual constraints (e.g., the *Sh* symbol cannot be changed to associate with the first of the two sucrose synthases - the SS1) the symbol *Css* makes matters worse. The most important consideration, however, is that *Ss2* expression in the maize plant is not constitutive, and thus the usage of *Css* is technically incorrect and highly misleading.

Prem Chourey

Cloning, immunoselection and characterization of cDNA clones of the two non-allelic sucrose synthase genes

cDNA clones of the two non-allelic sucrose synthase (SS) genes, *Ss2* and *Sh*, have been isolated from λgt11 expression libraries derived from immature kernel poly(A)$^+$ RNA of the *sh bz-m4* (*sh*-deletion) and *Sh/Sh* genotypes respectively. Recombinant clones containing the longest *Ss2* (λshD13) and *Sh* (λSh9) cDNAs, each of approximately 2.5 kb size, were characterized and comparatively analysed. Proteins were analysed on polyacrylamide gels to identify the chimeric expression of the SS cDNAs fused to β-galactosidase gene in *E. coli* λlysogens. The SS and β-galactosidase epitopes were recognized on Western blots using antisera against both proteins. Although the λSh9 expresses as a sucrose synthase-1 (SS1)-β-galactosidase fusion protein (~200 kD: 115 kD β-galactosidase and 92 kD SS) in λlysogens, the λshD13 failed to form such a chimeric protein and instead showed a ~70 kD SS2 polypeptide. Another *Ss2* cDNA clone, λshD12, which is ~50 bp shorter at both ends than λshD13, also did not form any fusion protein and only ~70 kD SS2 polypeptide could be seen. The reason for the lack of fusion protein in λshD13 and λshD12 lysogens is not known.

The following evidence indicates that the clone pshD13 (λshD13 cDNA insert subcloned in pUC19) contains the *Ss2* cDNA sequence: 1. The pshD13 was immunoselected from the cDNA expression library of the *sh bz-m4* stock which is known to have a deletion at the *Sh* locus (Burr and Burr, Genetics 98:143, 1981; Dooner, CSHS Quant. Biol. 45:457, 1981). The residual activity in the endosperm of this genotype is due to the *Ss2* locus (Chourey, MGG 184:372, 1981). 2. The pshD13 hybridized to endosperm poly(A)$^+$ RNA of the *sh*-deletion genotype as a single sharp band (~2900 b) which is also present in the *Sh* genotype. A second weak hybridizing band in the *Sh* genotype (2750 b) is considered due to cross-homology with *Sh* transcript as it comigrates with the band seen in the *Sh* poly(A)$^+$ RNA using *Sh* cDNA as a probe. 3. pshD13 showed homology to the restriction fragments of *Sh* cDNA and *Sh* genomic clone (Werr et al., EMBO J. 4:1373, 1985). 4. An identical pattern of hybridization was observed when pshD13 was used as a probe on different restriction enzyme digests of *sh*-deletion and *Sh* genomic DNA. One such 6.3 kb *Bam*HI hybridizing fragment cloned in EMBL4 from the *sh*-deletion stock, pGshD6, also showed different enzyme cleavage sites (Fig. 1) as compared to 16.3 kb *Bam*HI *Sh* genomic clone (Werr et al., EMBO J. 4:1373, 1985).



Restriction enzyme cleavage site maps of sucrose synthase cDNA clones, pshD13 and pSh9 and of the *Ss2* genomic clone, pGshD6. The line drawings below pGshD6 map show additional restriction enzyme sites on pGshD6. A solid bar shows the region which hybridizes to pshD13 and pSh9.

The pSh9 (λSh9 cDNA insert subcloned in pUC19) can be characterized as *Sh* cDNA clone by the following criteria: 1. It did not hybridize to poly(A)$^+$ RNA of the *sh*-deletion genotype at the *Sh* transcript position. 2. pSh9 did not hybridize to genomic DNA from the *sh*-deletion strain at the *Sh* position. 3. pSh9 contains the expected restriction sites of the *Sh* cDNA (Werr et al., EMBO J. 4:1373, 1985). 4. It shows homology to *Ss2* cDNA.

The restriction enzyme cleavage site maps of pSh9 and pshD13 are very different (Fig. 1). There are unique restriction sites in both clones, i.e., *Nci*I in pshD13 and *Pvu*II, *Cla*I, and *Bgl*I in pSh9. Among the common restriction sites, the *Sst*I and *Bgl*III are also located ~550 bp apart in *Sst*I-*Bgl*III fragments of the two cDNAs. Southern

cross-hybridization studies also revealed more homology around this region based on the intensity of hybridizing fragments. The sequences 5′ to *Sst*I restriction site on two cDNA clones are diverged.

Genetic mapping analysis using the first *Ss2-null* mutant isolated among the *Sh*-revertants upon *Ds* excision from *sh-m5933* allele indicates a tight linkage between *Sh* and *Ss2* on chromosome nine (Chourey et al., CSH meeting p. 65, 1986; and Chourey et al., manuscript in preparation). The recent molecular mapping data, obtained in collaboration with Ben Burr (Brookhaven Lab) using B-A translocation stocks and the *Ss2* cDNA clone (pshD13) as a probe, suggest that the *Ss2* locus is close to the bronze (*bz*) locus on chromosome nine (Gupta et al., manuscript in preparation).

M. Gupta, P.S. Chourey and P.E. Still

Transcriptional analysis of the mitochondrial gene URF13-T in T cytoplasm

The unique mitochondrial gene, URF13-T, in Texas male-sterile cytoplasm maize, has a complex transcriptional pattern. This is related to its association with a repeated region which is 5′ to both URF13-T and *atp6*, and to processing of the transcripts (Dewey et al., Cell 44:439, 1986). By using small DNA probes (31-630 bp), we have Northern-walked from a region -1215 through the coding region of URF13-T and a co-transcribed gene, ORF25. The first five probes also represent a region of -1590 to -444 5′ to *atp6*. Other *atp6*-specific probes were used to cover the remaining 5′ region and the coding region for this gene.

The majority of the transcripts for both *atp6* and URF13-T appear to initiate within the repeated region and undergo RNA processing events. No differences were detected in the transcript pattern between sterile and restored lines using *atp6*-specific probes, however, differences between N and T cytoplasms were seen. A unique transcript of 1.55 kb occurred in T cytoplasms from five different nuclear backgrounds. This transcript may be related to differences in DNA sequence seen between -580 and -562 of *atp6*. There are two small insertions in T, of 4 and 5 base pairs, at -580 to -577 and -566 to -562, respectively, relative to the sequence in N cytoplasms. One insertion disrupts an *Alu*I restriction site, allowing for an easy assay of many genotypes. The insert(s) is present in all T cytoplasms surveyed and absent in all N, C, and S cytoplasms we have examined.

The effect of dominant nuclear restorer genes on URF13-T has been described by Dewey et al. as differential processing of transcripts, seen with a probe representing the region +21 to +41. We find that a differential effect of restorers is detected on transcripts using a probe within the repeated region, representing positions -202 to -81 as well as in the region +2 to +200. The region from -80 to +4 is characteristic of an intron region, as a discrete transcript (1.6 kb) is missing from both T and T-restored mitochondria that is seen with flanking probes in T-restored mitochondria. A 1.5 kb transcript is seen in both T and T-restored mitochondria with probes covering the region +200 through the coding region. This transcript appears to be the result of an RNA processing event, whereas the 1.6 kb transcript could be the result of RNA splicing, which is unique to T-restored mitochondria. The current model to explain the effect of the nuclear restorer genes on the expression of URF13-T is that splicing and processing of larger transcripts (2.0 and 1.8 kb) decrease their relative copy number, lowering the effective number of transcripts that could encode the entire open reading frame of URF13-T. Antisera to this gene have detected a protein of Mr 13,000 which is unique to T cytoplasms (Wise et al., MNL, this volume). Synthesis of this protein is reduced in T-restored mitochondria (Forde and Leaver, Proc. Natl. Acad. Sci. 77: 418, 1980).

John C. Kennell, Roger P. Wise and Daryl R. Pring

Genomic configurations of T, Wf9(N), and A188(N) mtDNAs adjacent to URF13-T

Several major mtDNA rearrangements characterize the genomic configurations that distinguish N cytoplasms from T cytoplasms. One rearrangement is the duplication of ca. 5 kb of a single copy region of N cytoplasm mtDNA which is 5′ to the gene *atp6* (Dewey et al., Cell 44:439, 1986; Wise et al., Proc. Natl. Acad. Sci., submitted). The duplicated region in T places the *atp6* transcription start signals 5′ to URF13-T and ORF25. To identify the presumed progenitor region in Wf9(N) mtDNA contiguous to the repeated region of Wf9(T), a 1.7 kb *Bam*HI clone, representing the left junction fragment of the repeat in T, was used as probe on *Sst*II, *Sma*I, and *Xho*I digests of Wf9(N) and A188(N) mtDNAs, and mapped by the coordinate map of Lonsdale et al. (MNL 60:170). A 14 kb *Xho*I fragment left of the repeat in T was also used as a probe. Sequences of the two clones mapped to approximate coordinates 231-244. Thus the unique configuration of contiguous T sequences includes regions which are 240 kb apart on the Wf9(N) map. Interestingly, the 5 kb repeat and the adjacent sequences in T are very close to the recombinationally active 5.2 kb repeats of N cytoplasm; the 244 coordinate is within 2 kb of the N repeat, and the right edge of the 5 kb repeat of T is within 8 kb of the second copy of the N repeat. The 14 kb clone contained sequences which are represented in two regions of N cytoplasm. A188(N) and Wf9(N) mtDNAs were distinguishable with both the 1.7 and 14 kb clones.

Sequences adjacent to ORF25 vary within N cytoplasms, and we have found that A188(N) is more similar to T than is Wf9(N) in this region. Fifteen probes, covering ca. 11 kb from T cytoplasm, were hybridized to digests of Wf9(T), Wf9(N), A188(T), and A188(N) mtDNAs, using at least three endonucleases. A188(N) is colinear with T from within ORF25 to more than 3.5 kb 3′ to the gene. This region appears to be repeated in T cytoplasm. In contrast, at least 3.5 kb of sequences adjacent to ORF25 were not present in Wf9(N) mtDNA. At least part of this sequence is transcribed in T and A188(N). The colinear region of T and A188(N) diverged within a 1.16 kb *Hin*dIII-*Xho*I fragment at the right hand end of the 6.7 kb *Xho*I fragment. It is apparent that A188(N) and T cytoplasm mtDNAs share genomic configuration through this region, and that Wf9(N) is clearly divergent. The data do not imply that A188(N) can be considered as a progenitor to T, but it is evident that Wf9(N) does not carry sequences which are represented and transcribed in A188(N) and T. It is interesting to note that A188(N) and T also share the 2.1 kb minilinear DNA, while Wf9(N) has a larger 2.3 kb counterpart.

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URF13-T codes for a 13kd polypeptide

A 345 bp open reading frame of T cytoplasm mtDNA codes for a predicted polypeptide of 13 kd (Dewey et al., Cell 44:439, 1986). A polypeptide of this size is produced by amino acid incorporation by isolated T mitochondria (Forde and Leaver, Proc. Natl. Acad. Sci. 77:418, 1980). Plants regenerated from callus tissue culture of T cytoplasm, exhibiting male fertility and resistance to the toxins of race T of *Cochliobolus heterostrophus (Bipolaris maydis)* (Gengenbach et al., Proc. Natl. Acad. Sci. 74:5113, 1977), have this gene deleted, or in the case of a mutant designated T-4, the gene has a G to A transition adjacent to a 5 bp insertion (Wise et al., MNL 60:63, 1986; Proc. Natl. Acad. Sci., submitted). This insertion event places a TGA stop codon in frame 4 bp from the insertion, truncating the predicted polypeptide at 8.3 kd. Transcription of this region is unaltered in the T-4 mutant compared to T cytoplasm, suggesting that T-4 may synthesize an 8.3 kd polypeptide. ^{35}S-methionine incorporation by isolated mitochondria showed that T synthesized a prominent 13 kd polypeptide, which was absent in N, T-4, or T-7 (a deletion mutant). A unique polypeptide migrating at approximately 8 kd was synthesized by T-4. A highly immunogenic region of URF13-T was selected for the synthesis of a 17 amino acid polypeptide, designated PEP17. Polyspecific antibody to PEP17 was raised, and immunoprecipitation of native polypeptides from mitochondrial amino acid incorporation revealed precipitation of the 13 kd polypeptide, indicating that it is a gene product of URF13-T. A polypeptide of approximately 7.2 kd, similar to the size of a polypeptide identified as subunit 9 of ATPase, was also precipitated by the antibody from mitochondria of T but not T-7 or N, suggesting that the 13 kd protein may be part of a complex with the *atp9* product.

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Characterization of accumulated compounds in recessive *r-r*

Earlier studies with single and double recessive mutants of anthocyanin biosynthesis have led to the elucidation of specific roles of the genes in the gene action sequence analysed by Reddy and Coe (1962). From aleurone extracts of *r-r* one of the isolated compounds gave red color with concentrated H_2SO_4 as for a chalcone, and pinkish with alcoholic $FeCl_3$ as for presence of a phenolic hydroxyl group. Under the UV light the spot showed yellow fluorescence. The UV data in MeOH, showing λ_{max} 239sh (log E 4.18), 266 (log E 3.99), 319sh (log E 4.20), 379 (log E 4.44), suggested the characteristics of a chalcone. These observations were further confirmed by co-paper chromatography, superimposable UV and IR with an authentic sample of 2′, 4′, 3,4-tetrahydroxy chalcone (butein). Based on the preliminary data it was concluded that the isolated compound was butein.

Another compound from the same extract gave positive orange color for presence of the flavonoid skeleton with the Shinoda test, and dark coloration with alcoholic $FeCl_3$ for a phenolic hydroxyl group; under the UV light the spot showed yellow fluorescence. The UV data in MeOH, 248 (log E 4.30), 262sh (log E 4.14), 307sh (log E 4.15), 319 (log E 4.24), 362 (log E 4.43), suggested the characteristics of a flavonol. These observations were further confirmed by co-paper chromatography and superimposable UV and IR with an authentic sample of 3,7,3′,4′ tetra hydroxy flavonol (fisetin). Based on the data it was concluded that the isolated compound was fisetin. Based on the results that recessive *c2* accumulates a C9 compound and the present observation that recessive *r-r* accumulates C15 compounds, chalcone and flavonol, further confirm the position of *R* after *C2* in the gene action sequence. These observations have provided insight into the control of specific gene-product relationships and also the position of genes in the control of anthocyanin biosynthesis in maize.

V. Satyanarayana and G.M. Reddy

The analysis of aleurone protein patterns of *C-I* and pigment inhibition

The soluble aleurone proteins were analyzed by disc gel electrophoresis to study the protein patterns of *C-I, Pr, pr, bz1, bz2* and their F1s. A total of 17 bands were observed in all genotypes. The extracts of *C-I* exhibited 12 bands, *Pr,* 11; *pr,* 13; *bz1,* 10; *bz2,* 13. Band-5 (Rm 0.23), which was present in *C-I,* was absent in all the parents, suggesting that this band may be associated with the inhibition of pigment. All the F1s exhibited a characteristic banding pattern with five common bands, 3 (Rm 0.16), 5 (Rm 0.23), 8 (Rm 0.40), 13 (Rm 0.65) and 15 (Rm 0.78). In all the F1s a specific protein band of *C-I,* i.e. band-5, was present with Rm 0.23. It may be suggested that this band may be associated in the control of precursors/substrates required by other genes for anthocyanin biosynthesis in the aleurone tissue of maize.

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

Initiation and maintenance of suspension cultures

Friable calli derived from seedling roots of three genetic stocks, A188 *Pr* and *r-r*, were transferred into liquid MS basal media containing 0.5-4 mg/l 2,4-dichlorophenoxy acetic acid (2,4-D), 2,4,5-trichlorophenoxy acetic acid (2,4,5-T) and 2,4,5-trichloropropionic acid (2,4,5-P) to establish fine cell suspensions for studying growth rate and effect of hormones and genotypes. After a week, the suspension mostly consisted of cell aggregates (2mm) and few single cells (5-10%). Of the four concentrations of 2,4-D tested, 2 mg/l exhibited good separation of the callus mass into fine suspensions of small aggregates and single cells (20-40%); whereas the degree of separation was less in other concentrations as seen by larger aggregates (2mm) and frequency of single cells was low (4-5%). The two analogues of 2,4-D, i.e. 2,4,5-T and 2,4,5-P, were not effective in inducing a fine suspension compared to 2,4-D as evidenced by fresh weights (128, 10.2 and 20.1 mg) and dry weights (15.5, 2.3 and 4.31 mg). Differences were observed, however, in the size of cell aggregates. 2,4,5-P was found to be superior over 2,4,5-T in having small cell aggregates (2-4%) whereas in the former, only aggregates were observed. Larger aggregates had revealed loosely

connected smaller cells. The cells were of mostly parenchymatous type and revealed cytoplasmic strands. As subculture proceeded, the rounded cells predominated and formed into aggregates, whereas the elongated cells remained undivided, suggesting association of cell types in differentiation. A188 exhibited good cell suspension compared to other genotypes.

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In vitro selection for methomyl resistance in cms-T

The carbamate insecticide Lannate (active ingredient methomyl) mimics the cytoplasm-specific action of *Helminthosporium maydis* race T toxin (HmT toxin) on cms-T plants (Humaydan and Scott, Hort. Sci. 12:312, 1977) and isolated mitochondria (Koeppe and Malone, Science 201:1227, 1978). In vitro selection for resistance to *H. maydis* race T toxin has been successful (Brettell et al., Theor. Appl. Genet 58:55, 1980; Gengenbach et al., Proc. Nat. Acad. Sci. 74:5113, 1977), suggesting that methomyl may also be a useful selective agent. The chemical is more readily available than the purified fungal toxin and is also water soluble, making it convenient to use for in vitro selection. We obtained over 100 maize plants which were resistant to methomyl, and whose progeny also show resistance.

Callus was established from immature embryos derived from cms-T versions of the inbreds W182BN, P39, MDM-1, and of the F1 hybrids of SW-1, IL766A-1 and IL766A-2 with W182BN-N (a non-restoring pollen parent, used here to improve regeneration). All lines except W182BN are sweet maize. Embryos were obtained from fully male sterile plants except for IL766A-2 and MDM-1, which were either unrated or partially fertile. The maternal parents all show methomyl sensitivity regardless of fertility restoration.

Callus was increased over a period of 3 months before selection was initiated. Meanwhile, selection conditions were defined for different genotypes. Addition of 0.6 mM methomyl to modified Murashige-Skoog medium with 5mg/l 2,4-D, 4% sucrose caused about 20% inhibition of fresh weight increase for W182BN and W182BN x IL766A-2. Callus plated on 0.65mM methomyl showed intermediate inhibition of growth and on 0.7mM methomyl showed 80 to 100% inhibition. The other genotypes were cultured on Duncan's Medium D (Duncan et al., Planta 165:322, 1985). Inhibition of fresh weight increase on this medium containing 0.6mM, 0.65mM, or 0.7mM methomyl varied between 50 to 95%, depending on the genotype.

Over 2800 callus pieces were used in selection experiments, with half of these pieces serving as controls not exposed to methomyl. Callus which grew and remained regenerable in appearance was subcultured monthly on selection medium or, for the controls, on maintenance medium. Both gradual and high selective pressures with media containing methomyl were used. Regeneration was started after 3, 4, 5, 6, or 10 subcultures by transfer to MS medium with 10% sucrose and no 2,4-D; in some cases 1mM methomyl was included. Regenerated plantlets were tested for resistance to methomyl at the 3-5 leaf stage by direct swabbing of 0.3M methomyl onto the distal sections of a leaf. In this assay, leaf necrosis was visible after 1-3 days on the treated leaves of cms-T plants while no effects were seen on treated leaves of plants with other cytoplasms (N, C, S). Regenerants showing no necrosis were scored as resistant.

Presently, 288 plants regenerated after 3 to 5 selection cycles have been examined. Of the regenerants from callus exposed to methomyl during subculture, 76% (117/153) showed methomyl resistance; of these, 97 plants were fully male-fertile, 16 had unrated or abnormal tassels, and 4 were male sterile. Only 2% (3/135) of the regenerated controls showed resistance: these were all derived from IL766A-2, which yielded the most control regenerants (100). Several fertile, methomyl sensitive regenerants have also been obtained from sterile starting material subcultured on methomyl. Almost all (40/41) plants regenerated in the presence of methomyl were resistant, while only 69% (77/112) of the plants were resistant after regeneration in the absence of the insecticide. Control callus was generally not capable of plant regeneration on medium containing methomyl. This suggests that high methomyl concentrations in the regeneration medium provide an effective roguing of sensitive material. The influence of time in culture, type of selection pressure, and of genotype on the proportion of resistant and sensitive plants is currently being examined.

The resistant plants varied in their response to injection of HmT toxin into the whorls. Of the 82 resistant plants examined, 39% showed little or no response to toxin and 61% showed intermediate to high sensitivity. The regenerated controls for the corresponding cultures showed 100% sensitivity. It is also interesting that gain in fresh weight of callus was not a clear indication of resistance. A resistant plant was obtained from material on methomyl which showed no weight increase during the last selection cycle before regeneration, and a mix of resistant and sensitive plants resulted from callus which grew even better on methomyl than healthy control tissue.

We are currently doing seed increases in the greenhouse and in the winter nursery in preparation for inheritance studies. Considering the results of the HmT toxin resistance work, it is likely that the shift to methomyl resistance and male-fertility involves alterations in the mitochondria. Progeny of resistant selections show resistance, both in seedling tests with methomyl and in field-grown material subjected to biweekly sprayings of Lannate.

The apparent link of methomyl resistance with male-fertility (seen also in material selected for resistance to the HmT toxin) may limit the usefulness of such methomyl-resistant plants for hybrid seed production. However, selection for methomyl resistance offers a method for rapid cytotype conversion from male-sterile to male-fertile; it eliminates the need for repeated backcrosses after conventional fertility restoration. Also, methomyl resistance could be easily incorporated into lines where fertility changes are less important than the maintenance of specific cytoplasm-nuclear background combinations. One such example is T-Rf sweet corn. In addition, apparent differences in structural requirements and effective dosages of the toxin and insecticide leave open the possibility that different mechanisms of action may be involved. Molecular analysis of methomyl resistant mutants offers

distinct potential for providing additional understanding of the mitochondrial genomes and cytoplasmic male sterility.

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Continued study of a defective WF9 cytoplasm, *"wsp"*

The *wsp* and related progenies described in MNL 60:69-70 were regrown in 1986. Nuclear/cytoplasmic interactions and segregation ratios were essentially the same as in 1985. Thus it appears that expression of the *wsp* phenotype, the result of a cytoplasmic/nuclear genetic interaction, is reasonably stable. Tables 1 and 2 summarize the two-year results among families (Table 1), and within those families that showed segregation (Table 2).

Table 1. Classification of families for presence of *wsp* phenotype, 1985 and 1986.

Pedigree	Cyt.	Family	%wsp,1985	%wsp,1986
(SK2wsp x WF9)Ⓧ	wsp	367198	6	7
		367194	6	5
(SK2 x WF9)Ⓧ	SK2	367196	0	0.3
		367192	1	0.3
(SK2wsp x WF9)WF9	wsp	463091/90	32	22
		463083/82	3	13
(SK2 x WF9)WF9	SK2	463089/90	0	0
		463081/82	0	0
(SK2wsp x WF9)SK2	wsp	463087/86	0	0
		463079/78	0	0
(SK2 x WF9)SK2	SK2	463085/86	0	0
		463077/78	0	0
SK2wsp x WF9	wsp	367198/197	-	0
SK2 x WF9	SK2	367196/197	-	0
WF9	WF9	463090	19	27
		463082	1	0
SK2	SK2	463086	0	0
		463078	0	0

Table 2. Classification within families for *wsp* phenotype, 1985 and 1986.

Pedigree	Cyt.	Ear-row	%wsp,1985	%wsp,1986
(SK2wsp x WF9)Ⓧ	wsp	104	7	9
		103	13	7
		102	0	4
		98	4	3
		97	13	9
		96	1	3
(SK2wsp x WF9)WF9	wsp	90-A8	11	19
		-A6	63	26
		-7	21	24
		-5	29	11
		-3	38	27
		82-A8	0	0
		-A4	6	-
		-2	5	25
		-1	5	7
WF9	WF9	90-A8	0	0
		-A6	44	56
		-7	0	0
		-5	0	0
		-3	45	56
		82-A8	4	0
		-A4	0	0
		-2	0	0
		1	0	0

Additionally, ear-rows were grown and observed of seed from open-pollinated ears borne on plants classified as *wsp* in the 1985 observation planting. Each ear-row generally contained several plants of *wsp* phenotype when its pedigree indicated *wsp* cytoplasm, but not when the pedigree indicated SK2 cytoplasm. When pedigrees indicated WF9 cytoplasm (all such ear-rows came from open-pollinated ears of WF9) about half the ear-rows contained plants of *wsp* phenotype.

These results agree with the hypotheses that: (1) *wsp* is stably inherited through the seed parent, even after being hidden for many generations by a "repressor" nuclear genotype, (2) the small number of plants with SK2 cytoplasm that were classified as *wsp* in 1985 were probably misclassified, and (3) WF9 (in at least some progenies) is capable of producing new cytoplasmic defectives of *wsp* phenotype. Appropriate backcrosses have been initiated to facilitate study of the persistence and inheritance of the recovered *wsp* selections.

Work also continues to investigate the possibility that the *wsp* cytoplasmic genotype may be lost as well as created. To date, evidence indicates that once created, it persists, although its expression in uniform nuclear "expressor" genotypes is highly variable from plant to plant. One WF9 backcross ear-row (82-A8 in Table 2) has not yet shown any *wsp* plants in observations of about 40 plants; progeny from that ear-row will be further tested to see if the *wsp* cytoplasmic genotype is indeed gone, or only not yet expressed.

Donald N. Duvick

Defective cytoplasms from teosinte

B73 has been backcrossed as male into the cytoplasm of several accessions of most of the described races of teosinte, as well as of *Z. diploperennis*. Most of the backcross lines are phenotypically indistinguishable from B73 after 4 to 5 backcrosses have been made. But B73 in the cytoplasm of 2 accessions of Jutiapa teosinte, in an old accession of "florida" teosinte (probably *Zea luxurians*), and in a new accession of *Z. luxurians* is small and slow growing and shows poor ear and kernel development. Hybrids of these strains (used as seed parent) with other maize genotypes are also weak, late in maturity and have poor ear and kernel development. Interestingly, B73 in *Z. diploperennis* cytoplasm is not reduced in vigor; B73 backcross lines in *diploperennis* cytoplasm appear to be identical in vigor and maturity to B73 in its own cytoplasm.

Donald N. Duvick

Mapping the *Css* gene relative to the genes for *Sh1* and *Wx*

In the process of constructing a maize RFLP map, we have recently positioned the *Css* (sucrose synthase-2) gene relative to the genes for *Sh1* and *Wx1*. We find that *Css* maps 32 ± 4 cM from *Sh1* and 11 ± 2 cM from *Wx1* (Figure 1). This location is in close agreement with that reported by McCarty et al. (MNL 60:60, 1986) using different RFLP markers and in a different F2 population.

Figure 1. The region of chromosome 9 containing the genes for *Sh1*, *Wx1* and *Css*.

Our initial experiments were designed to assess polymorphism around these loci in our F2 population. We nick translated probes prepared from the *Bam*HI insert from the *Css* clone p21.2 (McCarty et al., MNL 60:58, 1986), the *Eco*RI insert of the *Sh1* clone p17.6 (Sheldon et al., MGG 190:421, 1983), and *Sal*I subfragment #1 from the *Wx1* clone pWx5 (Wessler and Varagona, PNAS 82:4177, 1985) and hybridized them against DNAs isolated from the inbred parents B73 and Mo17 digested with various restriction enzymes. As expected, usable polymorphisms were found for each probe. Next, linkage data were obtained by hybridizing each probe to Southern blots containing DNAs from B73, Mo17, B73 x Mo17, and 112 F2 plants. F2 plants were scored as B73-like, Mo17-like, or F1-like for each probe. Map position was determined using the maximum likelihood method. Genetic distances observed were *Sh1-Wx1* 24 ± 3 cM, *Sh1-Css* 32 ± 4 cM, and *Wx1-Css* 11 ± 2 cM.

These mapping experiments demonstrate the utility of RFLPs in genetic studies. The chromosomal location of *Css* was determined even though no phenotypic mutant had been identified. In addition, we have shown that the F2 population B73 x Mo17)X is recombinationally equivalent to the one used by Helentjaris et al. (MNL 60:118, 1986), at least in this region. This finding should make it easier to correlate the RFLP maps derived in different laboratories.

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Kinetic parameters of RNA synthesis in isolated mitochondria of different genotypes

The analysis of mitochondrial RNA-synthesizing systems in different maize genotypes may be important for elucidation of the role of the mitochondrial genome in molecular-genetic mechanisms responsible for commercially essential traits of maize plants. In this connection it appears reasonable to investigate the kinetics of mtRNA synthesis by isolated mitochondria from maize plants differing in the level of adaptability. The mitochondria were isolated from 3-day-old etiolated maize seedlings by a standard method of differential centrifugation. Mitochondrial protein was determined by the Lowry method. The functional activity of the isolated mitochondria (rates of mitochondrial respiration in the 3d and 4th metabolic states, respiration control) was estimated during kinetic experiments by a polarographic method with a Clark electrode. The synthesis of mtRNA was registered with labelled ^{3}H-UTP (specific radioactivity was 560 TBq·mol^{-1}). DNA-dependent mtRNA synthesis was determined by incorporation of labelled ribonucleosidetriphosphate into acid-insoluble mitochondrial material. The mtRNA synthesis was highly sensitive to specific inhibitors of mitochondrial transcription (ethidium bromide, actinomycin D). The kinetic data were obtained at least from 3-4 experiments.

The kinetics of mtRNA synthesis was registered within the first 30 minutes after mitochondrial isolation since it was possible to eliminate in these conditions the changes in mitochondrial genome activity following uncoupling of oxidation and phosphorylation as a result of organelle aging.

Depending on the rate of mtDNA transcription, two groups of inbreds may be identified (Fig.): 1, W64A and A344 +/+ inbreds showing a low rate of mtRNA synthesis in their mitochondria and 2, Sg25 inbred and dwarf mutant A344 sin/sin demonstrating a high level. The first group of inbreds had higher kernel yield than the second. The inbreds of the first group differed in ecological plasticity: the W64A inbred exhibited high plasticity while the A344 +/+ exhibited low plasticity. These inbreds showed relatively constant variation of the kernel yield in a wide range of environment. The yield potential of the Sg25 and induced mutant A344 sin/sin was genetically limited, i.e. the yield gain significantly decreased after reaching 25 q/ha level. Therefore, the high level of mtRNA synthesis may be assumed to result from inhibiting effect of nuclear genes. The Sg25 inbred with high general combining ability produced crosses of an extensive type (bi = 0.24, s^2di = 3.45).

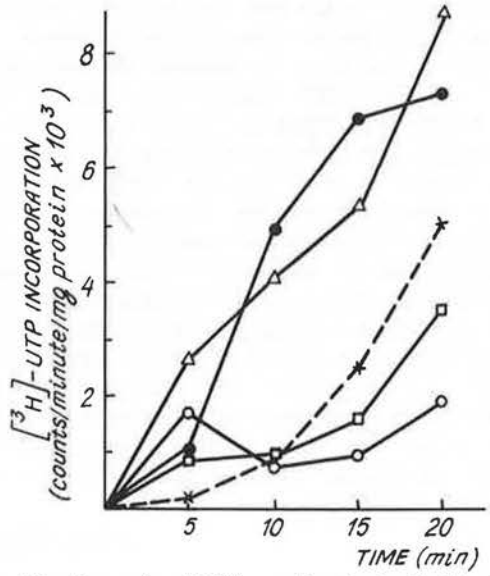

Figure. Kinetics of mtRNA synthesis in isolated mitochondria from A344 +/+ (O), A344 sin/sin (●), W64A (□), Sg25 (Δ) and W64A x Sg25 (X).

The W64A x Sg25 cross was intermediate according to a rate of mtDNA transcription. It showed: 1, low kernel yield potential, 2, high level of long-term mean kernel yield in rainfed conditions, 3, high stable ecological response (bi = 1.16, s^2di = 4.9). For W64A x V158 and W64A x A344, bi was 1.24 and 0.35, s^2di was 42.5 and 26.7, respectively.

We propose that the rate of mtDNA transcription is negatively associated with a genotype's ability to maintain the constant value of variation in the adaptation response to a wide range of environment.

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Evidence for transposable element activity in Nebraska Stiff Stalk Synthetic

I have crossed a number of lines to Nebraska Stiff Stalk Synthetic (NSSS) to obtain material which is better

adapted to the Nebraska environment. Among these lines were several stocks which were testers for transposable elements. NSSS was crossed as the female by lines carrying *bz2-m* or *a-m-1* and two or three ears of the resultant F1 were selfed. The *bz2-m* allele could be followed by the bronze phenotype while the *a-m-1* allele was followed by the linked *sh2* marker. In the case of the *a-m-1* allele, no dots or sectors of color were seen on shrunken kernels, indicating an absence of *Spm* activity. Only ears which were segregating 3:1 for colored, plump vs. colorless, shrunken kernels were considered. In the case of *bz2-m*, seven different crosses were done. The ears resulting from the self pollination of two of these crosses showed mutability of the bronze phenotype. The mutability is expressed as sectors and dots of color. This suggests the presence of an *Ac* like activity. With the small sample size, no conclusion can be drawn about the copy number of the element.

This *Ac* element could be resident in either the NSSS or the *bz2-m* line. In the first case, the cross to the tester served to indicate the presence of an active element while in the second case an active element was generated by outcrossing to the NSSS. Crosses with other *Ds*-induced alleles in different genetic backgrounds would be necessary to support one model over the other. It is possible that the *Ac* element is resident in the NSSS. Peterson and Salamini (Maydica 31:163, 1986) have shown the presence of the *Uq* and *Mrh* systems in Iowa Stiff Stalk Synthetic. Tests for the presence of five other transposable element systems failed to show any positive results but did not rule out the possibility that these elements existed in the material. NSSS, originally derived from Iowa Stiff Stalk Synthetic, is a composite of two sub-populations which were selected for high grain yield. The presence of an *Ac* element in this breeding population lends support to the idea that transposable elements provide diversity which can be used in selection.

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New cytological evidences for a basic number x = 5 in the genus *Zea*

In the last News Letter (MNL 60:77-79, 1986) we presented an introduction and discussion of our first results on cytological evidences for a basic number x = 5 in *Zea*. The same results were published afterwards in Theor. Appl. Genet., in press (1987). The aim of this article is to present new results that support this working hypothesis and add three new pieces in the puzzle of the x = 5 genomic relationship among taxa.

The new results consist of the analysis of the meiotic configurations of *Zea mays* ssp. *mexicana* and two F1 artificial interspecific hybrids (*Z. mays* ssp. *mays* x *Z. mays* ssp. *mexicana*, 2n = 20; *Z. perennis* x *Z. mays* ssp. *mexicana*, 2n = 30). In Table 1 the results of meiotic studies are detailed. *Z. mays* ssp. *mexicana* (2n = 20) and the F1 hybrid *Z. mays* ssp. *mays* x *Z. mays* ssp. *mexicana* (2n = 20), present a regular meiosis with the formation of 10 bivalents. In both tetraploid taxa secondary associations were found with means of 2.74 and 3.57 pairs of bivalents, respectively. In F1 hybrid *Z. perennis* x *Z. mays* ssp. *mexicana* (2n = 30) 5III + 5II + 5I were formed in ca. 47% of the 104 cells studied and the means were III = 5.27, II = 5.5 and I = 4.58 (Table 1).

In Figure 1 the most frequent meiotic configurations are presented. Our previous results on other taxa, with the purpose of making an integration, are also included in the same figure.

In the 2n = 30 hybrid (*Z.m.mx.* x *Z.per.*, Fig. 1) the 5III could be formed by autosyndetic pairing of A_1' and A_1'' genomes from *Z. perennis* and by allosyndetic pairing with the A_2' genome from *Z. mays* ssp. *mexicana*. The 5II could be formed by autosyndetic pairing between C_1 and C_2 homeologous genomes from *Z. perennis*. Finally the 5I

Table 1. Meiotic configuration in species and F_1 hybrids studied.

SPECIES or HYBRIDS	2n	Diakinesis-metaphase I configuration I	II	III	IV	%	Frequency of quiasmata ($\bar{X}$ ±SE)	Secondary associations of bivalents ($\bar{X}$ ±SE)	N° of cells studied
Z.mays ssp.mexicana	20	-	10	-	-	97.06	17.84 ±0.21	2.74±0.14	68
		2	9	-	-	2.94			
		$\bar{X}$ 0.059	9.97	-	-				
Z.mays ssp.mays x Z.mays ssp.mexicana	20	-	10	-	-	90.77	16.12 ±0.41	3.57±0.14	65
		2	9	-	-	9.23			
		$\bar{X}$ 0.18	9.91	-	-				
Z.perennis x Z.mays ssp.mexicana	30	8	8	2	-	1.92			
		7	7	3	-	13.46			
		4	7	4	-	7.69			
		6	6	4	-	17.31	---	---	104
		5	5	5	-	45.15			
		4	4	6	-	12.50			
		2	2	8	-	0.96			
		$\bar{X}$ 5.269	5.5	4.576	-				

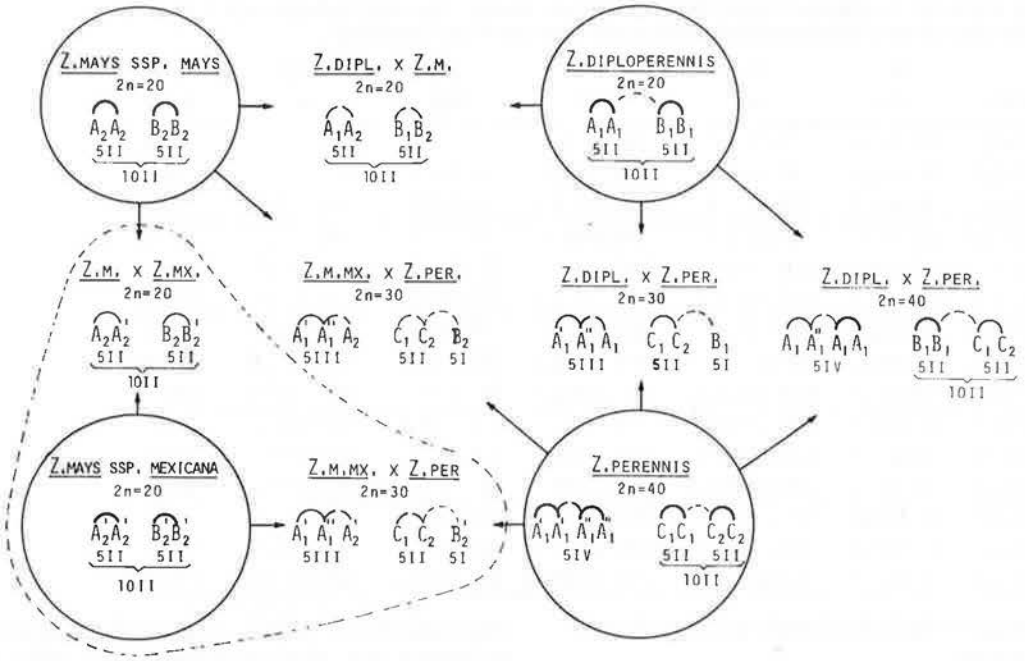

Figure 1. Chromosome numbers, most frequent meiotic configurations and hypothetical genomic constitution of species and hybrids of *Zea* assuming x = 5. The results presented here are surrounded with a broken line, the other ones were taken from Molina and Naranjo (MNL 60:78, 1986; Theor. Appl. Genet., 1987).

would belong to the B_2' genome from *Z. mays* ssp. *mexicana*. As in the hybrids with 2n = 30 previously studied (Molina & Naranjo, 1987), in this new hybrid there is a tendency of A, C and B genome separation in meiotic metaphase I, with the trivalents, bivalents and univalents grouped respectively, as indicated.

The case for an allopolyploid origin of 2n = 20 taxa and autoallopolyploid origin of 2n = 40 taxon, seems strong (Fig. 1). Until this moment our results suggest a minimum of three distinct original genomes (A, B and C) in order to explain the genome constitution and the cytogenetic relationships among the four species studied.

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Early expression of heterosis in diploperennial teosinte-maize hybrids

While we were working with hybrids between diploperennial teosinte and maize, the fast and vigorous development of the hybrid plants in relation to their parents, especially diploperennial teosinte, really took our attention. The hybrids between maize and its wild relatives are generally heterotic in the extreme. Heterosis expresses itself through different morphological and physiological effects and it is especially related to the kind of maize used in the crossings.

Our observations let us realize that a simple experiment, carried through in an early stage, could probably be used to foretell the heterosis expressed in inter-specific hybrids. It flashed into our minds that the initial strength of the plants could be measured through different parameters of measurement to be able to associate them, in the future, with traits of the mature plants. We designed an experiment consisting in the breeding, under similar environmental conditions, of hybrids between *Z. diploperennis* and a sweet maize variety (Evergreen). Seeds of each participant (parents and hybrids) were germinated in flower pots. Measurements were made at 10, 15, 20, 25 and 30 days after planting for the three participants and on 80 individual plants (four repetitions) for each stage of development. During all the experiment, the flower pots were sprinkled with a nutrient solution. The traits measured in each stage of development were the following: shoot length (SL); root length (RL); shoot fresh weight (SFW) and dry weight (SDW), and root fresh weight (RFW) and dry weight (RDW). The results obtained are shown in Table 1.

At the beginning, maize has greater development than the others for the different traits studied. As the initial growth of the plant mainly depends on the weight of the original seed, it can be concluded that they agree with the experimental values obtained; the initial dry weight of the seeds is very different; diploperennial teosinte is only 55.1 mg, hybrids 78.7 mg and maize 176.1 mg.

These great initial differences give advantage to maize, and because of this it was considered inconvenient to express the results in such a way that they could be comparable. For the last column of Table 1, we therefore calculated the total dry weight increment (TDWI) on the base of the initial dry weight of the seeds. The value of TDWI, for each stage of development, is the result of subtracting from the total dry weight (shoot + root) the initial dry weight.

During the first half of the experiment the hybrid plants are as heavy as maize plants, and they have a greater content in dry matter. Afterwards, fresh weight and dry weight values are higher in hybrids than in maize. TDWI is always higher in hybrids, whatever stage we consider. This fact lets us deduce an upper efficiency in the elaboration of dry matter and a higher production rate, as

Table 1. Seedling traits of *Z. diploperennis* (Zd), Evergreen maize (Eg) and hybrids (Zd x Eg) at different developmental stages (experimental results are expressed per seedling).

dap	Parent or cross	SL (cm)	RL (cm)	SFW (mg)	RFW (mg)	SDW (mg)	RDW (mg)	TDWI (mg)
	Zd	5.3+0.7	15.5+2.6	71.7+10.2	201.2+29.4	9.2+0.9	53.8+2.3	7.9
10	Eg	14.4+0.5	36.1+1.1.	753.2+79.4	1790.7+60.7	64.8+4.4	115.7+7.5	3.8
	Zd x Eg	14.9+1.2	26.8+1.9	486.0+72.3	1062.3+138.4	55.4+9.8	72.6+3.8	49.3
	Zd	8.1+0.6	22.7+0.6	168.4+22.7	354.4+48.7	21.1+1.8	68.5+4.0	34.5
15	Eg	23.4+1.9	47.4+3.1	1376.7+101.8	2499.1+94.1	117.8+8.7	137.4+7.3	78.5
	Zd x Eg	23.1+2.9	39.6+3.3	1177.1+178.6	1690.9+172.1	123.6+14.4	107.1+16.4	152.0
	Zd	11.7+0.4	29.9+3.3	378.0+1.8	707.4+104.1	57.8+5.1	89.4+5.7	92.1
20	Eg	35.7+1.1	50.0+6.8	2687.5+123.7	3063.2+446.1	272.1+23.2	179.2+27.0	274.6
	Zd x Eg	35.1+4.2	47.0+6.7	2638.8+168.1	3361.5+243.7	289.0+23.5	198.7+20.3	409.0
	Zd	16.6+1.7	35.0+1.4	803.2+185.8	1119.5+171.4	102.1+27.4	127.0+16.1	174.0
25	Eg	49.5+2.4	60.8+5.1	4234.2+342.6	3030.3+449.8	415.5+40.6	226.0+42.0	464.8
	Zd x Eg	48.2+2.6	48.8+6.0	5360.4+1048.0	4459.4+504.7	586.8+87.6	380.0+41.2	888.1
	Zd	21.6+0.5	36.5+1.0	1071.2+42.3	1254.5+211.5	168.1+11.7	146.8+21.6	259.8
30	Eg	52.7+3.5	61.0+4.2	4824.5+657.1	3973.5+984.4	586.8+57.0	295.4+55.3	705.5
	Zd x Eg	55.2+4.8	50.0+2.3	5924.6+722.8	5752.5+1479.5	823.6+118.4	589.4+170.5	1334.3

the initial dry weight of the hybrid seeds was less than 50 percent those of maize.

A remarkable fact is the slow growing of diploperennial teosinte, opposite to maize. In spite of it, heterosis comes out again when they greatly overcome the most vigorous parent. At the end of the experiment, hybrids almost make twice the amount of total dry weight per plant than maize. The results obtained have a special meaning for being clearly demonstrative of the wide possibilities that wild germplasm presents and which are possible to be used, to increase the biological efficiency of maize.

J.L. Magoja and I.G. Palacios

Effect of perennial teosinte introgression on maize tassel traits

Teosinte introgression in maize, as pointed out by several authors, has played a decisive role in the evolution of modern maize. The introgression degree in the different maizes from America is not similar and it is sometimes necessary for different reasons to recognize what races or varieties have teosinte introgression. To date, the recognition of teosinte introgression in maize has been fundamentally detected by studying certain morphological traits, especially the structure of the female inflorescence.

Considering that teosintes, especially those belonging to the *Luxuriantes* section (Doebley and Iltis, 1980), have distinctive traits in the tassel that make them completely different from maize, it flashed into our minds to find out if quantitative traits of the male inflorescence can be used to measure the effect of introgression. Nine quantitative traits of the male inflorescence were employed to compare two maize populations. One of them represents actually cultivated maizes and it is composed of a mixture of red flint commercial hybrids grown in Argentina. We called this population Normal (N). The other maize population introgressed by perennial teosinte (I) was obtained through the methodology previously communicated (MNL 60:82).

The traits that were sized, some of them used by Doebley and Iltis (1980) to work out the taxonomic classification of the genus *Zea*, were the following: tassel branch number (TBN); tassel branching axis length (TBAL); tassel central spike length (TCSL); lateral tassel branch internode length (LTBIL); distance between the two primary lateral veins of male spikelet glume (DVL); number of veins between primary lateral veins (VBL); total vein number (TV); total tassel branch length (TTBL) and tassel dry weight (TW).

Some of those same traits evaluated on perennial teosinte (*Z. perennis*) and its F2 and F3SM progenies (see MNL 59:70) are given in Table 1, to take them as a reference, and to note what are the changes that have taken place. Perennial teosinte has low TBN and low TBAL, when we compare it with maize. It also has short tassel internodes. In the F2 progeny (derived from hybrids with maize), those traits increase their value comparing to teosinte. In an F3 population descending from the F2, and selected for maizoid traits, TBN, TBAL and TCSL increase. F3SM was used as donor of perennial teosinte germplasm to obtain the introgressed population (see MNL 60:82).

Table 1. Tassel traits of perennial teosinte (Zp) and its progenies F2 and F3SM derived from crosses with maize.

Character	Zp	F_2	F_3SM
TBN	2.8 ± 1.2	7.8 ± 4.2	17.9 ± 6.6
TBAL(cm)	1.6 ± 0.4	5.1 ± 2.3	10.9 ± 2.4
TCSL(cm)	9.4 ± 1.9	—	19.5 ± 2.9
LTBIL(mm)	3.7 ± 0.3	4.7 ± 1.0	4.5 ± 0.9
DVL(mm)	2.4 ± 0.3	" "	" "
VBL	12.0 ± 1.1	"	"
TV	17.9 ± 1.7	—	" "

On a sample taken at random from each population (approx. 100 tassels from each one), determinations were carried out on N and I. Results are shown in Table 2. The introgressed population does not differ from the normal one in such traits as TBN, TBAL and TCSL. On the other hand it has shorter internodes, a greater number of veins

Table 2. Tassel traits of normal (N) and perennial teosinte introgressed (I) maize populations.

Character	N	I	Sig.(1)
TBN	22.6 ± 6.4	23.6 ± 9.2	NS
TBAL(cm)	13.5 ± 2.7	14.2 ± 3.4	NS
TCSL(cm)	21.8 ± 3.5	20.9 ± 4.4	NS
LTBIL(mm)	5.9 ± 0.9	4.9 ± 0.9	*
DVL(mm)	2.1 ± 0.2	2.2 ± 0.3	*
VBL	6.7 ± 1.0	9.5 ± 2.3	*
TV	13.8 ± 1.6	16.8 ± 2.5	*
TTBL(cm)	302.4 ± 92.7	356.5 ± 124.9	*
TW(g)	3.9 ± 1.3	6.1 ± 2.7	*

(1):NS: no significant; *: significant at 5% level.

in the male glumes, a greater total tassel branch length and heavier tassels. The higher weight of the tassels belonging to the I population is the result of the greater value of TTBL, as the correlation between TW and TTBL ($r = 0.73$) is highly significant. That higher weight also depends on the internode length, because having a higher number of spikelets increases the weight per unit. The correlation between TW and LTBIL is significant ($r = -0.39$). Probably the higher weight of the tassels also depends on their chemical composition (especially content of SiO_2), for which we shall next make the necessary chemical analyses.

Tassel weight takes our attention because perennial teosinte has small and slight tassels, but when its germplasm is melted with maize germplasm, we obtained heavier tassels (heavier than maize tassels). In each population, the tassel weight depends on the tassel branch number, the correlation value between these traits is significant ($r = 0.58$). Nevertheless, and as both populations do not differ in their tassel branch number, the weight mainly depends on TTBL and LTBIL. Tassel traits, as those previously pointed out, consequently let us detect teosinte introgression on maize. The shortening of internodes, the greater number of veins in male spikelet glumes and the greater weight of the tassels are particular signs of introgression.

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Potential use of diploperennial teosinte germplasm for maize improvement

In 1983 (MNL 60:82), we first presented the idea of using wild germplasm with the purpose of increasing variability in maize. Although we have progressed with great success in the project of introgression using perennial teosinte (see the accompanying article), as we have pointed out before we are also using *Zea diploperennis* as wild germplasm donor. With diploperennial teosinte we have never had the practical difficulties which were set out when we used the other perennial species: maize hybrids and the backcrosses are fertile, and the good viability of the kernels obtained let us derive a quantity of material with which we are actually working.

As summarized in Figure 1, interspecific hybrids between maize (as female parent) and diploperennial teosinte (as male parent), were carried out. The F1 hybrids were backcrossed by maize, obtaining the first backcross progeny (FBCP). Only one backcross was enough to get the greatest part of specific maize traits back. From the FBCP, we have begun a selection and recombination process to obtain an improved population that can be employed in the near future for practical purposes. During the growing season 1985/86, FBCP population was cultivated in three different locations of the province of Buenos Aires, among them, Pergamino. In this location, based on individual plants, a series of traits of agronomic importance were measured. The results obtained are given in Tables 1 and 2.

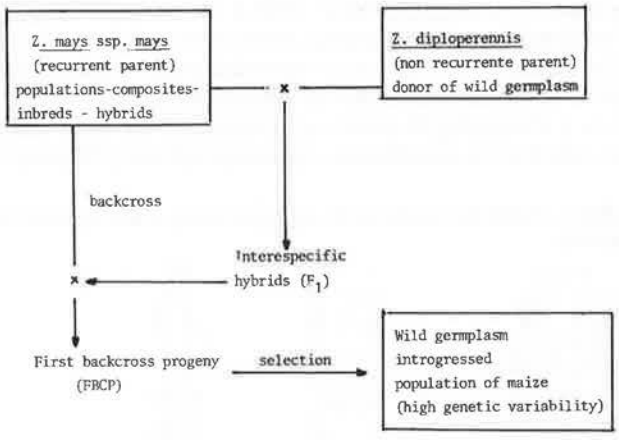

Figure 1. Method of diploperennial teosinte introgression in maize.

Table 1. Plant traits of FBCP (Pergamino 85/86).

Character	Mean ± SD	Range
Days to tassel	60.9 ± 4.4	56-76
Days to silking	71.8 ± 5.6	60-85
Days to pollen	67.8 ± 5.5	58-83
Protoandrous (days)	4.1 ± 2.0	1-8
Plant height (cm)	167.1 ± 15.0	135-200
Ear insertion height (cm)	111.4 ± 18.7	70-157
Number of tillers	2.6 ± 1.1	1-5
Number of ears per plant	5.6 ± 4.0	1-18

Table 2. Ear traits and plant yield of FBCP (Pergamino 85/86).

Character	Mean ± SD	Range
Ear diameter (cm)	2.7 ± 0.5	1.6-3.6
Ear lenght (cm)	10.9 ± 2.4	7-17.5
Number of kernel rows	9.9 ± 1.8	4-14
Kernel number per row	14.9 ± 5.4	6-26
Kernel number per ear	151.3 ± 70.2	44-300
Ear weight per plant (g)	122.7 ± 68.0	20-300
Kernel weight per plant (g)	79.0 ± 52.4	10.5-257.6
Weight of 50 kernels (g)	10.2 ± 2.1	6.4-18.9

In general, as in the case of the maize population introgressed with perennial teosinte, this one introgressed by diploperennial teosinte shows plants which join a good deal of agronomic traits. The plants of the FBCP are vigorous, extremely prolific, and show for the greatest part of the measured traits a high variability, which includes from little developed and poor-yielding plants, up to those combining prolificacy with a big ear size and high yield.

In this population the greatest part of the plants are chiefly prolific, and for this reason ears are generally small, but despite this fact there is a wide yield and ear size variability which can be used for selection.

The results obtained up to the present in the projects of wild germplasm introgression demonstrate that the possibilities of increasing genetic variability in maize are enormous. This is especially possible when the less closely related teosintes are used. The possibility that in the near future the yield of maize can be significantly increased through non-conventional improvement methods has a solid base. It has been pointed out by several authors that the great productive efficiency of modern maize could be a direct consequence of introgressive hybridization with teosinte. If the greatest part of modern maize variability has been produced or supported by natural teosinte introgression, it is easy to suppose that nowadays we dispose of the necessary genetic resources that need to be available in the search for better yields.

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Potential use of perennial teosinte germplasms for maize improvement

As it was previously reported (MNL 60:82), we are working on an introgression project of wild germplasm into maize, with the purpose of increasing heterosis and to produce greater variability in the cultivated species. Perennial teosinte (*Zea perennis*) has been used as donor of wild germplasm through advanced progenies derived from interspecific hybrids with Gaspé (see MNL 60:82). We have employed a great diversity of maize stocks as recurrent parents, especially inbreds, commercial hybrids and whole populations. As a result of controlled introgression carried out according to the scheme given in the last issue (MNL 60:82), we could obtain a perennial-teosinte-introgressed population of maize that constitutes the second back-cross progeny (SBCP).

Only two backcrosses with maize were necessary to recover the specific traits of the cultivated species. Nevertheless, the plants of this population still maintain as their main characteristic a high prolificacy, which is typical of the hybrid progenies between maize and perennial teosinte.

The SBCP was cultivated in several locations, to study its behaviour. It was subjected to an evaluation through the quantitation of some agronomic traits in two locations of the province of Buenos Aires (Pergamino and Santa Catalina). The results obtained are shown in Tables 1, 2 and 3. Generally the SBCP plants are extremely precocious.

Table 1. Plant traits of SBCP (Pergamino 85/86).

Character	Mean ± SD	Range
Days to tassel	45.9 ± 5.1	37-65
Days to silking	56.4 ± 7.6	47-76
Days to pollen	53.6 ± 6.4	44-71
Protoandrous (days)	2.9 ± 2.4	(-4)-9
Plant height (cm)	151.8 ± 19.6	115-210
Ear insertion height (cm)	88.8 ± 17.7	50-150
Number of tiller	2.7 ± 1.3	1-6
Number of ears per plant	3.4 ± 2.8	1-14

Table 2. Ear traits and plant yield of SBCP (Pergamino 85/86).

Character	Mean ± SD	Range
Ear diameter (cm)	3.6 ± 0.7	1.6-4.7
Ear lenght (cm)	14.9 ± 2.8	5.5-20
Number of kernel rows	12.6 ± 2.4	4-18
Kernel number per row	19.6 ± 8.1	2-38
Kernel number per ear	258.1 ±117.9	8-496
Ear weight per plant (g)	168.7 ±106.3	25-475
Kernel weight per plant (g)	118.5 ± 82.2	3-368
Weight of 50 kernels (g)	12.8 ± 3.1	3-19.2

Table 3. Relevant traits of SBCP cultivated in two localities (85/86).

Character	Locality: Pergamino (Average ± SD)	Locality: Santa Catalina (Average ± SD)
Number of ears per plant	3.4 ± 2.8	4.7 ± 3.4
Ear lenght (cm)	14.9 ± 2.8	12.9 ± 2.4
Ear diameter (cm)	3.6 ± 0.7	3.1 ± 0.3
Number of kernel rows	12.6 ± 2.4	11.7 ± 2.4
Kernel number per row	19.6 ± 8.1	23.6 ± 4.6
Wroght of 50 kernels (g)	12.8 ± 3.1	10.8 ± 1.9
Kernel weight per plant (g)	118.5 ± 82.2	109.7 ± 67.8

It really calls attention to the fact that, though they are short cycle plants, some of them have a luxuriant development joined to a high yielding capacity. Some plants' yield is really unusual in such early individuals. Protandrous value is low, though quite variable, as there can be chosen plants with protogynous (like teosinte) up to those with remarkable protandrous levels. Tillering still perpetuates in the SBCP plants, but tillers are really scarce and almost as vigorous as the principal stalk and able to flower at normal plant densities. Cropping the studied population with a density of 57,000 plants/ha does not constitute any obstacle to the expression of a high prolificacy. These plants' prolificacy is not only the result of the existence of several tillers, but of the developing capacity of several productive nodes per tiller. Ear size is not big, but is acceptable if we consider that there is a greater number of ears per tiller than in common maize.

The majority of all those traits studied points out a high variability among the SBCP plants for different traits combinations, and let us suppose that there is enough base to improve significantly the population, especially for its yield.

The average values resulting from the evaluation of different traits (see Tables 1 and 2) arise from computing in a sample taken at random, including little-developed plants with small ears and highly sterile, susceptible to insects and disease damage, up to those plants which join the greatest part of those traits which define the maize ideotype. These exceptional plants are able to produce almost half a kilo of kernels when we spread them at normal plant densities.

To date the results obtained show the potential usefulness of perennial teosinte germplasm in the genetic improvement of maize. This introgression of perennial teosinte genes or gene groups into the genetic background of modern maize can positively affect quantitative inheritance traits.

An enormous variability was generated in the SBCP as a result of introgression, and this can be explained on the basis of transgressive combinations. It is just where extreme combinations can be produced through unusual phenotypes, that a wide possibility of getting the superior individuals back is presented. Plants combining strong stalks and roots, simultaneous sex maturation, precocity and high yield do not appear in low frequency and they constitute the base of a selection process that we have already begun. Extreme individuals with exceptional yield have a potential productive capacity much greater than those maize plants actually cultivated.

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Relevant traits and heterosis of diploperennial teosinte-maize hybrids

During the growing season 1984/85, small populations of F1 individuals from the cross between diploperennial teosinte (*Zea diploperennis*) and a sweet corn variety (Evergreen), and both parents, were cultivated in separate plots. A series of traits was evaluated on a sample of F1 hybrid plants taken at random. Results are summarized in Table 1. Excepting those traits belonging to the cycle time, the rest show a considerable variation between F1 plants

Table 1. Relevant traits of *Z. diploperennis* x Evergreen F1 hybrids.

Character	Mean ± SD	Range		
Days to tassel	79.5 ± 4.0	67-83		
Days to silking	101.6 ± 5.3	93-113		
Days to pollen	94.9 ± 5.5	84-108		
Protoandrous (days)	6.7 ± 6.1	(-5)-23		
Tillers per plant	6.8 ± 2.5	2-13		
Productive nodes per tiller	5.1 ± 1.0	3-7.5		
Ears in the upper most node	6.2 ± 3.1	2-14		
Ears per tiller	29.4 ± 14.0	8.5-64.5		
Ears per plant	176.0 ±100.8	29-444		
Kernel row number	4.4 ± 1.1	2-6		
Plant yield (grams)	179.8 ± 86.2	28.8-370.9		
Tassel central spike type		Distichous (81%)	Polystichous (19%)	
Ear type		Distichous (74%)	Polystichous (26%)	
Female spikelets arrangement		Paired (93%)	Single (7%)	

derived from the same cross. It is possible that this is a consequence of the expression of different F1 genotypes.

Likewise the expression of specific traits such as tassel central spike type, ear type and female spikelet arrangement is not uniform, though with the predominance of one of the possible states of each trait. The results obtained point out that the inheritance of those traits by which maize is distinguished from teosinte cannot be attributed to simple genes unless, as is probable, a strong modification is caused by the genetic background of each particular hybrid. As was previously communicated (MNL 59:68) the expression of specific traits in other hybrids between diploperennial teosinte and maize was different. This fact let us suppose that each particular type of maize can have a different influence on the expression of specific traits in the hybrids obtained. The most conspicuous aspect in these hybrids is the enormous heterosis that they reveal, which is mainly expressed through a greater number of productive nodes, ears per tiller, ears per plant and plant yield, over the average of their parents (Table 2).

Table 2. Comparison of prolificacy traits and plant yield between *Z. diploperennis* x Evergreen F1 hybrids (Zd x Eg) and the parents.

Character	Zd	Eg	Zd x Eg	MP(1)	% heterosis(2)
Productive nodes	5.6	1.1	5.1	3.4	50
Ears in the uppermost node	1.0	1.0	6.2	1.0	520
Ears per tiller	7.0	1.1	29.4	4.1	617
Ears per plant	105.0	1.1	176.0	53.1	231
Plant yield	26.5	98.6	179.8	62.6	187

(1): mid parent value--MP= Zd + Eg/2

(2): per cent of heterosis= $(F_1 - MP/MP) \times 100$

Yield and prolificacy have a high heterotic expression. Highly significant increase of productivity per plant, not only relative to the parental average but to the higher yield parent, results.

This particular fact lets us suppose that diploperennial teosinte constitutes a genetic resource of great value, which can be used in projects whose objective is the increasing of yield of those cultivated species.

The heterotic expression of those traits of economical importance such as yield can be detected early in F1 individuals. Although these plants do not join appropriate agronomical characteristics, to be directly cultivated, the additional generated heterosis can be availed in projects of controlled introgression, as communicated in this issue.

I.G. Palacios and J.L. Magoja

Variation within *Zea*: numerical analysis of 43 traits

Taxonomy and phylogeny of the taxa of *Zea* is still a matter of discussion, and there are completely opposite interpretations despite the large amount of information produced in the last few years. Especially after the last taxonomic treatment of the genus (Doebley and Iltis, 1980) there was produced a wide and diverse body of information that, through morphological, cytological and biochemical studies, has mainly contributed to a correct characterization of the taxa within *Zea*. Nevertheless, at times, the same available data can lead, depending on the method, to completely different interpretations.

It is because of that, as we have been doing up to now (MNL 59:61, MNL 60:79) and like other authors have also done, we propose an objective handling of the available information through appropriate methodologies, in which each author's preconceived ideas do not have any influence. Grouping methods by numerical techniques based on a great number of equal importance traits seem to be adequate for this purpose. The objective of this article is to show the results obtained for taxa within *Zea*, on the basis of 43 traits.

The greatest part of these traits, especially the quantitative ones, were obtained after the evaluation of small populations of the taxa within the genus, and all of them were cultivated under similar environmental conditions. The data obtained from a large number of plants belonging to each of the taxa were used in partial clustering that we have shown before (MNL 59:61; MNL 60:79).

Now, our purpose is to show as a whole those groups carried out on the basis of all our available information. *Z. perennis* (Zp), *Z. diploperennis* (Zd), *Z. luxurians* (Zl), *Z. mays* ssp. *parviglumis* var. *parviglumis* (Zmpp), *Z. mays* ssp. *parviglumis* var. *huehuetenangensis* (Zmph), *Z. mays* ssp. *mexicana* (Zmmx) and *Z. mays* ssp. *mays*, constitute the 7 operational taxonomic units (OTU's)

Forty-three characters were scored for each OTU as follows: (1) tassel central spike distichous (0), polystichous (1); (2) tassel branch number; (3) tassel branching axis length (cm); (4) tassel central spike length (cm); (5) lateral tassel branch internode length (mm); (6) tassel branching abscission layer absent (0), strongly to weakly developed (1), strongly developed (2); (7) tassel branching rachis flattened (0), rounded (1); (8) distance between the two primary lateral veins of male spikelet outer glume (mm); (9) number of veins between primary lateral veins of male spikelet outer glume; (10) total vein number of male spikelet outer glume; (11) male spikelet outer glume wing absent (0), present (1); (12) male spikelet outer glume primary lateral vein narrow and sparse (0), wide and prominent (1); (13) male spikelet outer glume stiff (1), papery (2); (14) male spikelet outer glume flattened (0), rounded (1); (15) number of fruit cases per spike; (16) female spike distichous (0), polystichous (1); (17) female spikelet arrangement single (1), paired (2); (18) kernel enclosed (1), naked (0); (19) cupule orientation vertical (0), horizontal (1); (20) kernels per cupule one (1), two (2); (21) cupulate fruit case trapezoidal (2), triangular (1), horizontally compressed (0); (22) kernel small (0), intermediate (1), large (2); (23) fruit case weight (mg); (24) fruit case length (mm); (25) fruit case width (mm); (26) fruit case thickness (mm); (27) pericarp thickness (μm); (28) aleurone layer thickness (μm); (29) starch granule size (μm); (30) zein body size of zone 1 (μm); (31) zein body size of zone 2 (μm); (32) zein body size of zone 3 (μm); (33) endosperm protein content (%); (34) Landry-Moureaux saline soluble proteins (%); (35) zein; (36) glutelin-1; (37) glutelin-2; (38) glutelin-3; (39) growth habit perennial (1), annual (0); (40) rhizomes present (1), absent (0); (41) chromosome number 2n = 40 (2), 2n = 20 (1); (42) chromosome knob position terminal (1), terminal and internal (2); (43) number of tillers per plant.

Given a basic data matrix (BDM) (Table 1) of 43 characters by 7 OTU's the data were analyzed by cluster analysis. The BDM was standardized by characters, and phenograms were derived by the Pearson product-moment correlation coefficient applying the unweighted pair group method using arithmetic averages (UPGMA) (Figure 1A) and the "Mean Taxonomic Distance" between pairs of OTU's served as input in the calculation by UPGMA (Figure 1B). The "Manhattan Distance" between pairs of

Table 1. Basic data matrix.

	Characters														
OTU's	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Zp	0	2.8	1.6	9.4	3.7	2	0	2.4	12.0	17.9	1	1	1	0	5.1
Zd	0	6.3	1.8	7.9	4.7	2	0	2.5	10.5	16.6	1	1	1	0	7.5
Zl	0	14.0	3.9	6.3	4.7	2	0	2.4	15.3	25.8	1	1	1	0	6.4
Zmpp	0	57.9	9.1	6.1	4.4	1	1	1.7	4.3	8.8	0	0	2	1	8.8
Zmph	0	29.4	8.6	8.3	5.2	1	1	1.9	6.8	13.7	0	0	2	1	8.6
Zmmx	0	24.5	14.1	12.0	4.4	1	1	2.5	5.3	10.2	0	0	2	1	11.2
Zmm	1	22.6	13.5	21.8	5.9	0	1	2.1	6.7	13.8	0	0	2	1	NC

	Characters													
OTU's	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
Zp	0	1	1	0	1	2	0	54.6	7.0	4.1	3.3	17.0	42.3	17.3	1.7
Zd	0	1	1	0	1	2	0	59.7	7.5	4.4	3.5	21.0	39.7	12.5	1.5
Zl	0	1	1	0	1	2	0	62.3	8.7	4.1	3.6	13.6	31.8	17.6	1.7
Zmpp	0	1	1	0	1	1	0	36.4	6.1	3.6	2.7	12.8	28.3	17.1	1.7
Zmph	0	1	1	0	1	1	0	29.0	6.2	3.6	3.0	13.8	29.5	17.0	1.3
Zmmx	0	1	1	0	1	1	1	104.6	8.4	5.5	4.4	23.9	42.0	14.9	1.4
Zmm	1	2	0	1	2	0	2	NC	NC	NC	NC	95.8	45.9	16.0	1.8

	Characters												
OTU's	31	32	33	34	35	36	37	38	39	40	41	42	43
Zp	1.6	1.4	21.0	1.8	57.1	20.7	3.3	9.2	1	1	2	1	NC
Zd	1.2	1.0	27.0	3.1	67.1	9.2	7.5	10.0	1	1	1	1	NC
Zl	1.5	1.1	23.6	3.3	70.5	7.9	2.3	9.9	0	0	1	1	NC
Zmpp	1.2	0.9	26.5	2.6	71.5	7.1	1.9	9.3	0	0	1	2	11.8
Zmph	1.0	0.9	23.4	1.8	67.9	12.9	2.4	12.2	0	0	1	2	23.9
Zmmx	1.4	1.0	17.7	4.3	58.9	7.4	2.4	14.6	0	0	1	2	4.8
Zmm	1.5	1.4	11.4	5.7	51.9	12.3	9.6	13.0	0	0	1	2	NC

OTU's served as input in the calculation of "Prim Network" and "Wagner trees" (Figure 2).

The cluster of taxa, shown in Fig. 1, is partially congruent with those previously carried out (MNL 59:61, MNL 60:79, 60:81) but based on partial information and a smaller amount of data. As can be seen in Fig. 1, as much in one phenogram as in the other a characteristic cluster is repeated, which comes out to be independent of the method employed. Three groups are established, one consisting of Zp-Zd-Zl, which represents the most primitive teosintes of the *Luxuriantes* section (Doebley and Iltis, 1980). Another group consisting of Zmpp and Zmph is

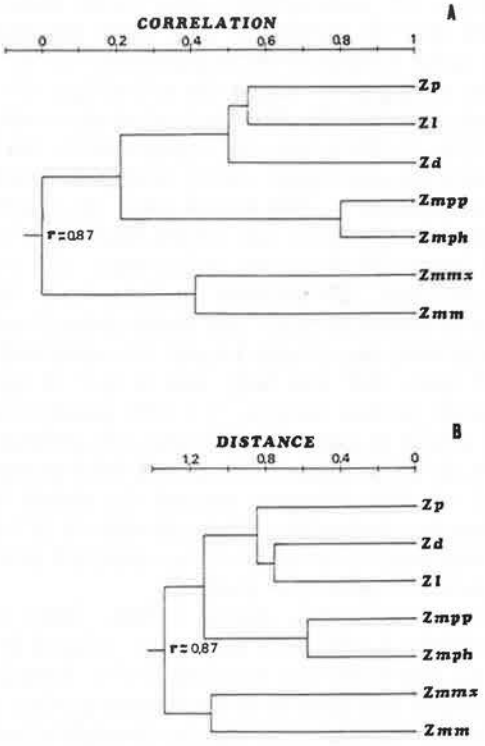

Figure 1. Phenograms of 7 OTU's resulting from the: (A) UPGMA cluster analysis of the OTU x OTU correlation matrix (B) UPGMA cluster analysis of the OTU x OTU distance matrix. r = cophenetic correlation coefficient.

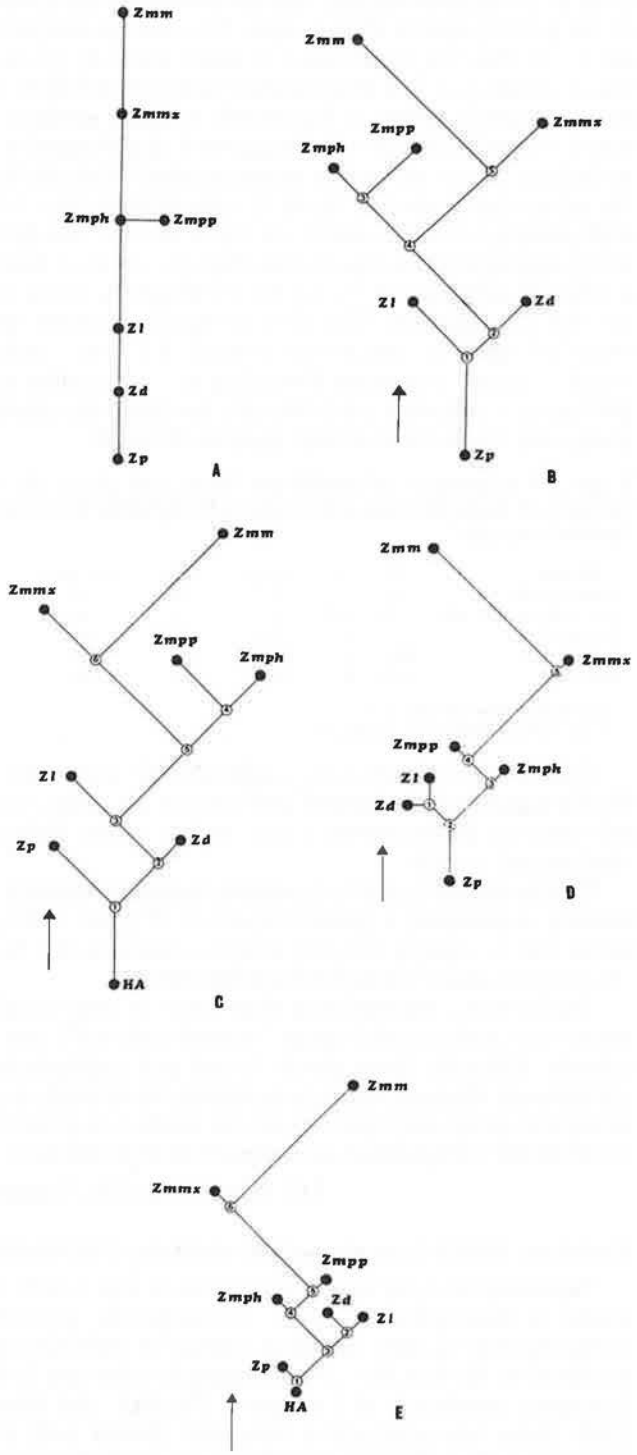

Figure 2. Prim Network and evolutionary trees for the genus *Zea* (A). Prim Network for the 7 OTU's based on 43 characters. (B) Wagner tree for the 7 OTU's based on 43 characters, considering *Z. perennis* as ancestor. (C) The same as B and considering a hypothetical OTU as ancestor (HA). (D) Wagner tree based on 16 relevant characters, considering *Z. perennis* as ancestor. (E) The same as D and considering a hypothetical OTU as ancestor (HA). Number corresponding to HTU's represents the construction sequence of the tree. Arrows indicate the evolutionary direction.

more related to the primitive group of teosintes than to the remaining group, Zmmx-Zmm. It is important to emphasize the fact that different clusters previously carried out (MNL 59:61), and on the basis of different traits, always relate Zp-Zd and Zl in the same group. In contrast, the linkage of the rest of the taxa has been variable, though by now the clusters in Fig. 1 can be considered as much more exact because they are based on a great number of traits.

The taxa of the Zea Section (Doebley and Iltis, 1980) constitute two groups, where Zmpp-Zmph would be much more related to the most primitive teosintes than to the taxa of their own section. This fact states the need of re-examining the taxonomy of section *Zea* on the basis of appropriate studies. If Zmpp, Zmph and Zmm-Zmmx were variants of the same species they must be linked more closely.

Prim Network and Wagner trees represented in Fig. 2 show a high congruence with the ones previously shown (MNL 59:61; MNL 60:81). The taxa of the genus can be related between two extremes: Zp and Zmm. Just in these two species (perennial teosinte and maize) all the necessary basic information is gathered to produce the other variations shown in the remaining taxa.

Our experience on hybrids between perennial teosinte and maize (see previous MNL issues), with the information that we are considering about the variation in *Zea*, suggest to us that Zp and Zmm can be considered basic species with which we can experimentally build all (or the greatest part) of the variation in the genus.

Our main idea about the evolution of *Zea* is like that of other authors, that maize introgression in teosinte (in a primitive teosinte) could have been the main factor in the differentiation of the taxa of this genus. If it were true and could be experimentally demonstrated, the evolutionary trees that we've simulated would be indicating the direction in which introgression increases. The teosintes placed at the bottom of the trees are those which have kept more differences with maize. As we study the top of the trees, the degree of introgression is higher up to a maximum represented by Zmmx, which can be essentially considered a maize which has kept the few necessary genes for wild life.

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Analysis of variability in plants produced with pollen from cultured tassels

In recent Newsletters, we reported that immature tassel meristems of both cv. Se60 and Oh43 can be cultured successfully from anther initiation through normal meiosis and microsporogenesis to production of trinucleate, normal pollen (MNL 55:116, 1981; MNL 59:72, 1985). The pollen so produced germinates on agar (MNL 59:73, 1985) and on silks and also fertilizes ovules to produce mature kernels (MNL 60:89, 1986). We report here further observations from studies undertaken among the progeny derived from in vitro generated pollen in comparison with those derived from in vivo generated pollen to evaluate variability (tissue culture induced somaclonal variation) by: 1) phenotypic analysis, 2) chromosome analysis and 3) gel electrophoresis.

The kernels produced with in vitro pollen from both cultivars (Se60 and Oh43) were grown in our London nursery during the 1985 and 1986 field seasons and the following observations were taken: 1) plant height; 2) number of leaves/plant; 3) leaf width; 4) tassel length; 5) number of tassel branches; 6) pollen fertility; 7) P% (P = number of kernels per ear/number of ovules per ear) and 8) 100 seed dry weight. The kernels from either cultivar had 100% germinability and yielded mature, healthy, normal and fertile plants. No morphological abnormalities were detected among the progeny (N = 290) either in F1 or F2 generations. Statistical analyses of these characters showed that the plants derived from in vitro pollen (R1) and their selfed progeny (R2) were similar in most respects to in vivo plants for both genotypes. Moreover, the analysis of chromosomes in root tips (N = 45) showed no changes in the chromosome number. The analysis of meiotic chromosomes in these plants is in progress.

Patterns of polypeptide synthesis in plumules and radicles of the seedlings derived from in vitro and in vivo pollen were analysed by 1D and 2D SDS PAGE, according to the procedures used routinely in our laboratory (Can. J. Biochem. 60:569, 1982). Striking differences, both quantitative and qualitative, were observed in the polypeptide patterns 1) between Oh43 and Se60, and 2) between plumules and radicles, no qualitative or quantitative differences were observed in the polypeptide patterns of 1D-gels between in vitro and in vivo seedlings. However, minor quantitative differences between in vitro and in vivo seedlings were detected for a few polypeptides by 2D gel electrophoresis. The significance of these minor differences has yet to be explored.

We conclude that the pollen generated from in vitro cultured tassels produces plants which are similar in most respects to plants produced from in vivo grown pollen with no observable morphological and chromosomal alterations.

D.R. Pareddy, R.I. Greyson and D.B. Walden

A comparison of the response of seedlings to heat shock, cadmium chloride and lannate

The response of plumules and radicles of etiolated maize seedlings (5 days old) to heavy metal treatment involves both a general reduction in protein synthesis and the induction of a group of polypeptides with M_r's and pI's similar to those of heat shock proteins (HSPs) (MNL 60:92, 1986). The carbamate insecticide lannate (active ingredient:methomyl, $C_5HN_2OS_2$) has a similar impact on protein synthesis in plumules but not radicles of etiolated maize seedlings.

Fluorographic analysis of PAGE separations of proteins extracted from plumules and radicles treated with varying doses of cadmium chloride or lannate (3 hours) and labelled with ^{35}S-methionine (during the last 2 hours of treatment) revealed that cadmium chloride-induced polypeptides are synthesized following treatment with 0.2 mM but not after treatment with 0.02 mM cadmium chloride. Lannate-induced polypeptides are synthesized in plumules of maize seedlings following treatment with 8.5 ml/100 ml H_2O but not following treatment with 3.4 ml/100 ml H_2O. The recommended field dose for lannate is 1.7 ml/100 ml H_2O.

Protein synthesis (monitored as incorporation of ^{35}S-methionine into TCA precipitable material, cpm/ug protein) is reduced approximately 40% following treatment with 0.2 mM cadmium chloride but is not altered significantly by treatment with 0.02 mM cadmium chloride. Following treatment with lannate (8.5 ml/100) protein synthesis in plumules is reduced approximately 60%. It is interesting to note that exposing intact maize seedlings to a heat shock (42.5 C, 3 hours) does not markedly reduce protein synthesis in plumules and radicles. Raising the concentration of cadmium chloride to 2 mM results in a 90% reduction in protein synthesis of treated plumules and radicles and synthesis of cadmium chloride-induced polypeptides remains evident. Following treatment of plumules with 17 ml/100 ml H_2O lannate, protein synthesis is reduced by at least 90% while synthesis of lannate-induced polypeptides remains evident.

Fluorographic analysis of PAGE separations of immunoprecipitates obtained using polyclonal antibodies raised against low M_r (18 Kd) HSPs has revealed the presence of a single polypeptide band (M_r, 18 Kd) immunoprecipitated from proteins synthesized in cadmium chloride treated (5 mM; 3 hours) plumules and radicles and in lannate treated (8.5 ml/100 ml H_2O; 3 hours) plumules. Cadmium chloride treatment administered to plumules and radicles of maize seedlings therefore induces the synthesis of a group of polypeptides with the same M_rs and pIs as 18 Kd HSPs and which are recognized by antibodies raised against these HSPs. Lannate treatment, however, only induces synthesis of these polypeptides in treated plumules.

Following treatment with lannate (8.5 ml/100 ml H_2O; 3 hours) protein synthesis in radicles of intact maize seedlings is reduced by at least 90%. HSP-like polypeptide synthesis in radicles is not apparent following treatment with any concentration of lannate so far tested. Antibodies raised against 18 Kd HSPs do not precipitate a polypeptide from proteins extracted from lannate treated radicles. Lannate is a solution (1.48 M) of methomyl in methanol (68%) (Dupont). Methanol treatment alone does not induce the synthesis of HSP-like polypeptides in plumules or radicles at any of the concentrations tested (1.2%, 2.4%, 5.0% or 12.0%; these concentrations represent the amount of methanol in 1.7, 3.4, 8.5, 17.0 ml/100 ml H_2O lannate respectively). General protein synthesis decreases as the concentration of methanol increases but the decrease is not as rapid as following treatment with increasing concentrations of lannate. It remains to be determined whether methomyl alone will induce the synthesis of HSP-like polypeptides or whether this effect is synergistic, requiring the presence of methanol.

Methomyl is selectively toxic to maize lines carrying the male sterile (T) cytoplasm. The effect of methomyl on plants with the T cytoplasm is similar to that of T-toxin isolated from *Helminthosporium maydis* race T (HMT) (Humaydan and Scott, 1977; Hortscience 12:312-313). Both methomyl and T-toxin block oxidative phosophorylation in mitochondria isolated from plants with the T-cytoplasm (Berville et al. 1984, Plant Physiol. 76:508-519). A preliminary comparison of the response of plants (Oh51A) with a normal male fertile cytoplasm (N or carrying the male sterile cytoplasms T, S or C) to heat shock and lannate does not reveal any major differences in the polypeptide synthesis patterns (after ID-PAGE) in plumules of the four maize lines. Currently the response of isolated mitochondria from these four lines to heat shock and lannate are being compared.

Carol A.B. Rees, Annette M. Gullons and D.B. Walden

Multivariate analyses of data from two-dimensional electrophoresis of polypeptides

We have reported (MNL 59:77) on the variation observed among the arrays of polypeptides synthesized by developing maize embryos. Qualitative differences in the species of polypeptides synthesized and quantitative differences in the levels of synthesis were detected among the fluorograms obtained from embryos of different ages. Recent work has led to the adaptation of multivariate techniques for use in the analysis of patterns resulting from the two dimensional electrophoretic separation of polypeptides (Fewster and Walden, Computers in Biology and Medicine, in press).

The method involves the division of a stained gel or fluorogram into a number of discrete grid units each of which represents an axis along which variation may occur. By established criteria, we have selected 16 grid units for the study summarized below. The positions of the lines forming the grid are determined with respect to those of a number of widely separated, readily identified polypeptides such that the entire grid structure may be reproduced accurately on all gels included in the analysis. Each grid unit is assigned a numerical value which reflects the number and intensity of spots residing therein. A variety of different analytical procedures may be employed subsequently to examine the data.

Examples of the application of these methods to a study of variation in polypeptide synthesis during maize embryogenesis are presented in Tables 1 and 2. The data were obtained from the two dimensional arrays of polypeptides synthesized by embryos of Oh43 at 15, 20, 25, 30, 35, 40, 45 and 52 days after pollination. Table 1 shows the results of a Principal Components Analysis (PCA) where the first two components (PC1 and PC2) comprised 66.6% and 15.5% of the total variation respectively. The greatest contributions to the variation expressed by these components was evident in eigenvectors 3, 5, 7, 10 and 11 which represent the corresponding grid units. PC1 appears to contrast variation in grid unit 5 with that in grid units 3,

Table 1. Principal components analysis (PCA) of fluorograms obtained from developing Oh43 embryos.

PC1				PC2		
EV	VC	ICS	IA	ICS	VC	EV
3	-0.46	-0.93	15	-0.37	0.79	5
5	0.47	-0.90	20	-0.32	0.26	10
7	-0.43	-0.33	25	0.27		
10	-0.27	-0.48	31	0.32		
11	-0.33	0.15	35	0.46		
		0.65	40	0.15		
		1.05	45	-0.56		
		0.79	52	0.04		

EV - eigenvector
VC - vector coefficient
ICS - individual component score
IA - individual age (days after pollination)

Table 2. Deviations from random expectation for the first lattice of a concentration analysis of fluorograms obtained from developing Oh43 embryos.

IA \ GU	5	3	7	10	11
15	-1.90	0.96	0.49	0.30	0.73
20	-1.49	0.76	0.39	0.24	0.58
25	-0.46	0.23	0.23	0.07	0.18
31	-0.63	0.32	0.16	0.10	0.24
35	0.47	-0.24	-0.12	-0.07	-0.18
40	1.60	-0.81	-0.42	-0.25	-0.62
45	1.49	-0.76	-0.40	-0.24	-0.58
52	0.89	-0.46	-0.23	-0.14	-0.35

IA - individual age (d.a.p.)

GU - grid unit

7, 10 and 11. Upon examination of the distribution of individual component scores along this component, a developmental trend emerges in which the density of grid unit 5 is observed to increase and the density of grid units 3, 7, 10 and 11 decrease as embryogenesis progresses between 15 and 55 days. PC2 expresses variation attributable to the combined effects of grid units 5 and 10. In this case a discrimination is made between embryos mid way in development from those which are older and younger, in that the combined relative densities of these two grid units are higher in the former.

Table 2 shows results obtained from a concentration analysis of these data where deviations from random expectation are measured for all grid units of an individual. This analysis partitions the variation into a number of lattices, the first of which is presented here, comprising 57.5% of the total.

The results obtained suggest a developmental trend similar to that which emerged through PCA. As embryogenesis proceeds from early through late development, the deviations associated with grid unit 5 change from negative to positive while the reverse is observed from grid units 3, 7, 10 and 11, indicating shifts from lower to higher and higher to lower densities respectively. Values obtained for these deviations in embryo mid-development are intermediate to those of early and late development.

Although some variation was lost in the process of grouping polypeptides into grid units, good correlation was observed between results obtained with this method and those of conventional forms of analysis. The multivariate techniques described should prove effective in the detection and representation of trends in any investigation in which the analysis of two dimensional separation of molecules is undertaken.

J.G. Boothe and D.B. Walden

Anther culture from stamens of the ears of *an1*

Anther culture is becoming an accepted procedure for the production of isogenic diploid lines from the embryogenic haploid tissues. Although the technique has proven to be difficult with corn, success is possible. In all previously reported work, however, stamens were derived from tassels.

In this study, stamens were obtained from the ears of the anther-ear mutant (*an1*). Ears were harvested when the anthers contained meicoytes at the 'uninucleate' stage and were wrapped in 'Saran Wrap' and stored in the dark for 8 days at 10 C. They were then surface sterilized with 10% 'Javex' and rinsed with sterile water. The anthers were removed and cultured in plastic petri dishes and sealed with 'Para Film'. Two media were tested: 1) YP medium (Genovesi and Collins, Crop Sci. 22:1137-1144, 1982) with TIBA (0.1 mg/l), casein hydrolysate (500- mg/l), myo-inositol (100 mg/l) and sucrose (120 g/l) and 2) MS medium with proline (1500 mg/l), kinetin (5×10^{-7}M), myo-inositol (100 mg/l) and sucrose (60 g/l). Each 60 mm plastic petri dish contained 4 ml of autoclaved liquid or agar (0.6%) medium.

After approximately 6 weeks in the dark at 25 C white calli were produced from some cultured anthers and the results are summarized as follows:

Medium	No. of anthers Cultured	No. Calli embryoids	Percent
YP Liquid	400	0	0
YP Agar	434	10	2.9%
MS Liquid	428	14	3.3%
MS Agar	485	3	0.6%

The bulk of the calli grew but remained non-organogenic. A few plantlets have been recovered and are presently being reared in the light on N6 medium. While no chromosome counts are yet available, there seems little doubt that the calli were derived from the sporogenous tissue.

While this represents a novel use of "ear-derived" anthers, the significance and utility of this observation remains obscure.

V.R. Bommineni and R.I. Greyson

Polypeptide differentiation associated with maturation of organs on tassels and ears

In maize, while both inflorescences are initially bisexual, female spikelets mature into ears and male spikelets mature into tassels. The molecular basis of this differential development has not been studied although plant growth regulators are known to be involved.

We report here preliminary observations on polypeptide variability associated with the maturation of male and female spikelets. Tassels and ears of different developmental stages (lengths) were labelled with diluted ^{35}S-methionine (5 ul in 200 ul H_2O) for 2 h at 27 C. One hundred spikelets were dissected from each labelled inflorescence. In the case of older (> 12.0 mm) inflorescences, their developing organs (ovary, silk or stamens etc.) were isolated. The tissues were homogenized separately in buffer containing 200 mM tris-HCl, 5% SDS, 7.5% β-mercaptoethanol, 1 mM PMSF, 10% glycerol at pH 7.5. Radioactive incorporation was determined by scintillation counting of TCA precipitable material. Protein samples were separated by 1D SDS PAGE and 2D IEF-SDS PAGE and fluorography was performed.

No major qualitative differences between tassel and ear or between young and older inflorescences were detected on the 1D fluorograms. While many polypeptides (>150) were resolved by 2D PAGE and fluorography, most were common to all fluorograms. A few unique polypeptides were consistently related to specific samples. At least two polypeptides were associated with maturation. One polypeptide (18 kd, approx. pH 7.0) was associated with developing ovaries and silks but not with male

flowers. A second polypeptide (25 kd, approx. pH 5.5) was associated with maturing stamens but not with female flowers. In addition, a few polypeptides, unique to either male or female flowers, appeared and then disappeared as the flowers matured. Whether the few polypeptide differences which were detected are related to the regulation of, or are the result of, differentiation remains for further studies.

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More oncogene-related sequences

We reported (MNL 60:91, 1986) on the presence of oncogene-related sequences in maize. In that report, labelled probes (v-Ki-ras and v-src), from Oncor Inc., were used to detect homologous sequences in the maize genome via nick translation and southern hybridization. In this report, we extend the number of oncogenes with related sequences in the maize genome to include v-myc, v-myb, and v-Ha-ras.

Maize DNA was isolated from 5 day old, Oh43 plumules as described by Dellaporta et al. (Molec. Biol. for Plants, pp. 36-37, 1984) and then treated as follows. After resuspension in TE, the DNA was incubated with 250 ug/ml of RNase A at 37 C for one hour, followed by an incubation in proteinase K at 50 C for one hour. The sample was extracted with first phenol/chloroform and then ether followed by dialysis against TE (pH 8.0) for three hours. The DNA was precipitated in 2 M ammonium acetate plus 2 volumes of 95% ethanol and resuspended in TE (pH 8.0) at a concentration of at least 0.4 ug/ul. Digestion of the DNA was carried out overnight with three to four times excess of *Bam*HI or *Eco*RI using the buffer system recommended by the manufacturer (Boehringer-Mannheim).

E. coli and human *Eco*RI digested DNA served as controls; all DNA samples were electrophoretically separated on 0.8% agarose gels. Transfer of the DNA to Zeta-Probe (BioRad) charged nylon membrane using 0.4M NaOH as the transfer buffer was undertaken and the blots were rinsed in 2X SSC for 5 minutes and vacuum baked at 80 C for 30 minutes.

Plasmids containing viral oncogene inserts were purchased from American Type Culture Collection. V-myc, v-fos, v-myb and v-Ha-ras inserts were isolated from plasmid pBr322 sequences by cutting with the appropriate restriction endonuclease, electrophoretically separating the fragments on low temperature agarose gels, slicing the gel segment containing the insert out of the rest of the gel, melting it and then purifying the insert through a BioRad RDP column.

Although contamination of inserts with pBR322 was minimal, unlabelled, denatured plasmid was included in each oncogene hybridization reaction to remove any contaminating labelled pBr322. Oncogene inserts were labelled using the Pharmacia oligolabeling kit to a specific activity of approximately 1 $\times$ 10 exp 9 cpm/ug.

The prehybridization buffer consisted of 1.5X SSPE, 1% SDS, 0.5% "Blotto" (Carnation low fat milk powder), and 500 ug/ml of sheared and denatured salmon sperm DNA; the incubation temperature was 65 C. Hybridization buffer had 10% dextran sulphate included. Wash buffer was 0.1X SSP, 1% SDS at 62 C. Hoeffer's "Hybrid-Ease" chambers were used for prehybridizations, hybridizations and washes.

The probes did not detect any homologous sequences in *E. coli* DNA, confirming an earlier report by Bishop (The Harvey Lectures, Series 78, pp. 137-172, 1982-83).

The v-myc probe detected 2 bands of near identical size (approximately 4.4 kb) in the BamHI digested maize DNA lanes. All previously reported myc-related sequences were detected in the human DNA (c-myc, L-myc, and N-myc). It has been reported (Alitalo et al., PNAS 80:100, 1983) that V-myc contains a 250 bp region with high G-C content (84%) which can lead to spurious hybridization signals. To test for spurious signals, we are removing this region from the insert.

V-myb detected a single sequence in maize of approximately 8 kb. The three myb-related sequences in human *Eco*RI digested DNA were also detected (Franchini et al., PNAS 80:7385, 1983).

Under the hybridization conditions used, no homologous sequences to v-fos were found in maize.

The v-Ha-ras probe produced several hybridization signals in maize *Eco*RI or *Bam*HI digested DNA. V-ki-ras had been previously shown (MNL 60:91, 1986) to detect multiple bands in maize DNA. Cross homology was detected between these 2 probes using the hybridization conditions listed above. Although a few maize hybridization signals were unique to each of the two ras probes, most bands were detected by both probes. This complex hybridization pattern between the 2 probes is currently being investigated using high stringency conditions to determine if any of the common sequences are unique to one probe or the other.

These results and those reported last year emphasize the evolutionary conservation of these sequences, thus implying their importance to cell function. Work is underway to determine if the maize sequences are transcribed and if so, in what tissues and under what conditions.

R. Zabulionis, D.B. Walden and J.D. Procunier

Use of leaf discs to monitor protein synthesis under field conditions

As reported last year (MNL 60:93, 1986), a study to monitor variation in leaf polypeptide synthesis patterns was undertaken for several field management practices and various cultivars of maize. This study involved attempts to report on the phenotypic variation found among the maize cultivars in the field as revealed through electrophoretic and fluorographic procedures in the laboratory. The laboratory work from the 1985 growing season was repeated for the 1986 growing season. A favorable growing season in 1986 produced expected differences among field plots, planting dates and cultivars. The "base-line" of growth measurements (height, number of leaves etc.) for the 1986 season were highly correlated with those reported for the 1985 growing season.

To analyze the detailed information collected from the fluorograms obtained during the two growing seasons, a newly developed technique employing multivariate analyses was performed (P. Fewster and D.B. Walden, in press). These methods involve a principal components analysis and an analysis of concentration, which define and outline any trends in variation (of polypeptide synthesis among cultivars, field plots etc.).

Our system (field grown material, ^{35}S-methionine exposure, electrophoresis, fluorography and statistical analyses) appears sensitive enough to enable preliminary observations on the objectives: 1) to identify proteins that may be unique to cultivars; 2) those that may have a role in heterosis; and 3) those that are of developmental significance.

The analyses revealed that some of the polypeptides synthesized were unique to specific cultivars and specific field plots. Few (less than 1%) differences were found between planting dates of the same cultivar/plot when comparisons were made with leaves of the same age. The variation among cultivars in the control plot was small in general and less than among other comparisons. The stress and optimal field management plots reveal a much greater directed variation in the polypeptide synthesis patterns trends as compared to that of the control or each other. These observations suggest that as the control conditions or those to which the cultivars are most 'adapted' are altered the cultivar responds to this new set of management conditions in ways which result in increased variation in polypeptide synthesis. This variation in polypeptide synthesis may allow the cultivar to adapt to a greater range of growth conditions. Also, polypeptide synthesis patterns throughout development show both similarities and differences between inbreds and hybrids. Our data and analyses confirm the view that hybrids are better able to respond favorably to more extreme management conditions than are the inbred cultivars.

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Effect of erythromycin on seedling growth, thermotolerance and synthesis of 52kD mitochondrial heat shock protein

Corn seedling mitochondria respond to temperature elevation and 50 uM arsenite treatment by the enhanced synthesis *in organello* of a 52 kD protein. We have defined this protein as a mitochondrial heat shock protein (HSP) (Nebiolo and White, Plant Phys. 79:1129, 1985). We are continuing to investigate the potential role of this protein in the mitochondria, as well as in the cellular heat shock response in light of various lines of evidence implicating the mitochondria as the primary target to heat and chemical stress (Nebiolo and Walden, J. Cell Biol. 79:258a, 1986).

Erythromycin (100 -150 uM) inhibits plastid *in organello* protein synthesis, while having no significant inhibitory effect on the mitochondrial translation apparatus (Tassi et al., Plant Sci. Lett. 29:215, 1983; Newton and Walbot, PNAS 82:68, 1985). To ensure that our purified etiolated corn seedling mitochondria prepared according to Forde and Leaver (PNAS 77:418, 1980) were not significantly contaminated by plastids, we treated an aliquot of each sample with 100 uM erythromycin. *In organello* translation products were labelled as previously described (Nebiolo, op. cit.) and subjected to SDS-PAGE/fluorography. Fluorographic profiles of erythromycin-treated and control samples were identical for both control incubation temperature (27C) and heat shock temperature (37C), as well as for mitochondria chemically stressed by 50 uM arsenite treatment. We have concluded that our preparations are not significantly contaminated by plastids and that the 52 kD mitochondrial HSP is not affected by erythromycin treatment. Other controls were run in previous experiments (e.g., measuring amount of bacterial contamination in purified mitochondrial preparations).

We are interested in the potential role of mitochondrial protein synthesis, specifically synthesis of the 52 kD HSP in response to temperature and chemical stress, in seedling growth and seedling thermotolerance. We have grown 3d (27C) corn seedlings (Oh43) for varying lengths of time (24, 48, and 72 hr) in sterile solutions of 100 uM erythromycin, 200 uM erythromycin, 200 uM chloramphenicol (inhibitor of organelle protein synthesis), 10 ug/ml cycloheximide (inhibitor of eukaryotic protein synthesis), and sterile distilled water as a control. Rates of growth, measured as fresh weight/hour of 3d seedlings in the various solutions were all decreased relative to the control. The lowest rate was manifested by seedlings incubated in cycloheximide, the next lowest by 200 and 100 uM erythromycin and the next by chloramphenicol. Similar results were obtained when using 4 and 5d seedlings. Therefore, erythromycin inhibits growth of corn seedlings to an extent greater than chloramphenicol but not as severely as cycloheximide.

To test the effect of erythromycin on the acquisition of thermotolerance (ability to survive an otherwise lethal temperature) we incubated 5d seedlings in the various sterile solutions of erythromycin, chloramphenicol and cycloheximide and at various temperature regimes (27C, control; 45C for 2 hr, lethal treatment; 37C for 2 hr followed by 2 hr at 45C, heat shock and lethal treatment). We found that seedlings in all drugs acquired thermotolerance and growth curves after heat shock were similar to those obtained for controls.

We are investigating the expression of this protein by the mitochondrial genome and its role in the mitochondria during temperature/chemical stress. We have separated membrane and non-membrane fractions of control and heat shocked mitochondria by the method of Boutry et al. (J. Biol. Chem. 258:8524, 1984). By subjecting mitochondrial proteins to SDS-PAGE/fluorography we have localized the 52 kD protein exclusively to the membrane in heat shocked mitochondria. Its role may be to protect membrane components of the respiratory complex from stress. We are currently designing experiments to isolate from the mitochondrial genome the gene coding for the 52 kD protein and to localize the protein in tissue sections using polyclonal antibodies raised against purified protein.

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A cytological approach to the characterization of *dek1*

The embryo-lethal recessive 561 mutant, isolated in our lab following EMS mutagenesis and shown to be

allelic to *dek1* (W. F. Sheridan, M. T. Chang and M. G. Neuffer, MNL 58:98-99, 1984), is characterized by defects both in embryo and in endosperm development, absence of a correctly differentiated aleurone layer and suppression of both carotenoids and anthocyanins. Here a description is given of the cytology of endosperm and embryo tissues of the mutant *dek1*.

Embryos and endosperms of four mutant and four normal sib kernels collected 16 days after pollination (DAP), when the mutant is first recognizable, were separately fixed. The procedure followed to obtain cytological preparations was that described by B.-Y. Lin (Stain Technology 52:197-201, 1977) with slight modifications. Dissociation into cells was achieved mechanically by needles and preparations were obtained by pipetting a few drops of the cell suspension onto clean slides warmed on a hot plate. Silver staining (50%$AgNO_3$ at 60 C) was applied in order to visualize nucleolar structures.

The number of nucleoli per nucleus in normal and mutant kernels is shown in Table 1. The results are strikingly different in the two tissues. In the embryo, in fact, the frequency of cells with two nucleoli in the mutant is significantly lower than that found in non-mutant embryos, the majority of cells being with one nucleolus. In the endosperm, on the other hand, the distribution of nucleoli among the three frequency classes is apparently equal in both mutant and normal. Keeping in mind that endosperm cells at the time of observation (16 DAP) are no longer dividing, these results suggest a total or partial impairment of mutant embryo tissues to go through active divisions.

Table 1. Number of nucleoli/nucleus in embryo and endosperm cells of wild type and mutant at 16 DAP.

Genotype	Frequency (%) of cells with the indicated number of nucleoli in						
	Embryo			Endosperm			
	No. cells scored	1	2	No. cells scored	1	2	3
Dek 1	561	54.7	45.3	364	89.0	10.2	0.8
dek 1	341	85.6	14.4	340	92.9	5.6	1.5

Table 2. Nucleolus and nucleus diameters in embryo cells (possessing one nucleolus due to nucleolar fusion) of wild type and mutant at 16 DAP.

Genotype	No. cells scored	Mean nucleolar diameter (μm ± S.E.)	Mean nuclear diameter (μm ± S.E.)	Correlation between nucleolar and nuclear mean diameters (r)
Dek 1	307	4.29 ± 0.05	16.23 ± 0.14	0.7504**
dek 1	292	6.13 ± 0.09	20.91 ± 0.18	0.6798**
		1.84**	4.68**	

Nucleolar and nuclear diameters for both embryo and endosperm were measured by an eye piece micrometer in cells displaying one nucleolus. The data pertaining to embryo measurements (Table 2) indicate a significant increase in size of the mutant vs. normal embryos. Similar conclusions are obtained in endosperm cells (Fig. 1), where a shift of the mutant frequency distribution towards higher values, if compared to the non-mutant values, is observed.

These preliminary data are consistent with the hypothesis of a difference in kinetics of the two cell populations and further studies will be aimed at the elucidation of these aspects.

Silvana Faccio Dolfini

The effect of PEG on *pro1* mutant

The role of proline in plant cells and tissues has been the subject of intensive research (Dashek and Erickson, Bot. Rev. 47:349-385, 1981). It has been repeatedly observed that plants accumulate a considerable amount of free proline in response to different biological and environmental stresses. Though the physiological significance of this accumulation is not clear, it has been suggested that it is either a consequence of the stress or alternatively an adaptive response of the plant in terms of survival. It seemed therefore interesting to us to analyze the effect of water stress on *pro1* mutants whose lethality is overcome by growth on media supplied with proline.

PEG (MW 6000) solutions at different concentrations (20%, 30% and 40%) were used to simulate water stress conditions. Intact seedlings (1-2 leaf stage) and leaf discs (5mm) were treated for 24 hours with PEG solutions and then analyzed for their proline and soluble proteins content. For whole seedlings, shoots and roots were analyzed separately. As far as mutants are concerned, care was

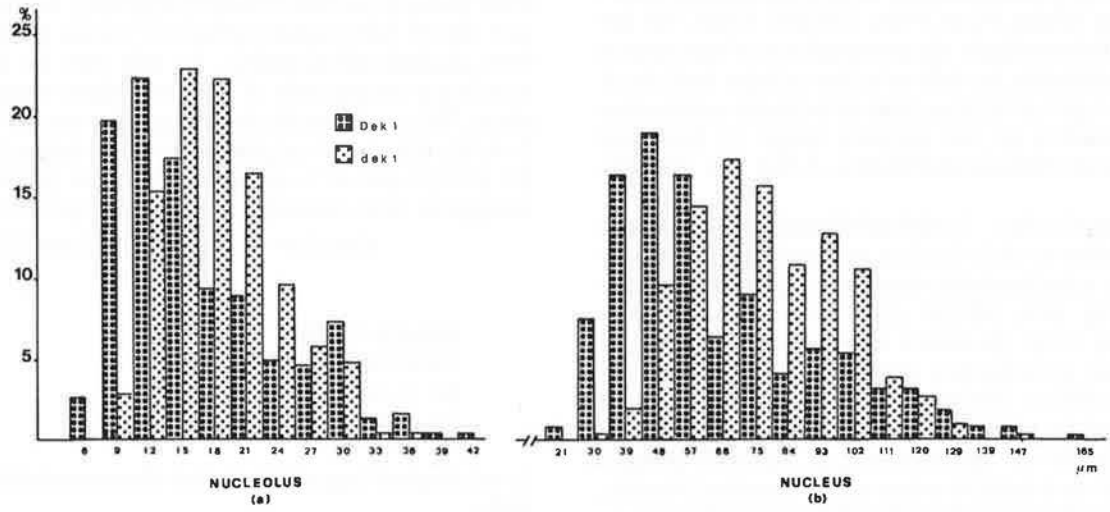

Figure 1. Frequency distribution (%) for nucleolar(a) and nuclear(b) diameters in normal and mutant endosperm at 16 DAP. The values indicated represent the class midpoint.

taken to initiate PEG treatment before any sign of lethality due to the proline requirement. The free proline content, in response to PEG treatments, shows a significant increase in shoots and leaf discs but not in roots where the content is unaffected. The threshold of PEG induction (concentration of PEG at which an accumulation of proline begins to take place) is lower in mutants than in normals.

The polypeptide pattern of soluble proteins present in roots seems in agreement with the hypothesis that the mutant is more sensitive to PEG than the control. Both mutant and normal show changes in some polypeptide classes as a consequence of PEG treatments; however, they differ in the PEG concentration required to induce such changes. So mutant roots begin to show modifications in the polypeptide pattern at 20% PEG while normal roots require 30% or 40%.

Chiara Tonelli and Alcide Bertani

The role of *Sn* in the light-regulated activity of enzymes of flavonoid biosynthesis

Sn is a light-regulated factor, closely linked to *R*, responsible for specific pigmentation of the mesocotyl of the maize seedling. One of the *Sn* accessions, *Sn-bol3*, appears to be present in two variants, one producing intense (*Sn-s*) and the other weak (*Sn-w*) pigmentation of the seedling tissues. We had previously shown (Gavazzi et al. in Plant Genetics, 1985) that the phenylalanine ammonia-lyase (PAL) and UDP glucose 3-0-glucosyl transferase (UFGT) are related to the *Sn* genetic constitution. To further analyze the role of *Sn* on flavonoid biosynthesis, we tested the activity of chalcone synthase (CHS) and chalcone isomerase (CHI), two enzymes involved respectively in the production of naringenin chalcone and in its isomerization to naringenin. The activity was determined on extracts of homozygous *Sn-s* mesocotyls following different time of irradiation (14000 Lux provided by Power stars -HQ1-T400 W/DV OSRAM lamps).

Increases of CHS and CHI were observed in the *Sn-s* tissues with a similar time course (Figure 1); the maximum was observed 48 hours after the onset of irradiation,

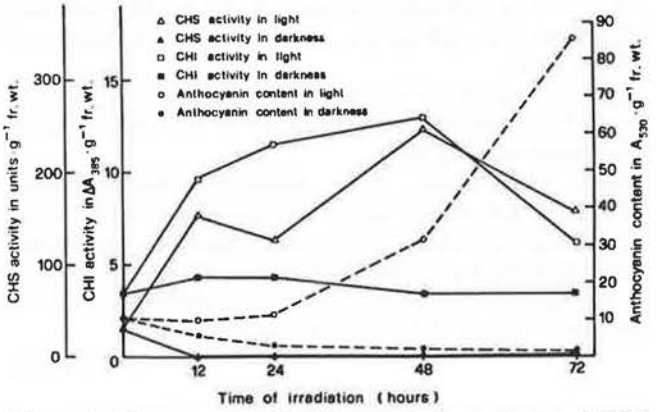

Figure 1. Time course of anthocyanin formation and CHS and CHI activity as determined in homozygous *Sn-s* mesocotyls. Time$_0$ refers to five-day-old seedlings grown in darkness.

which was not coincident with anthocyanin accumulation, still increasing after 72 hours. In the dark both enzymes and pigment do not show any increment. Data in Table 1 show the pigment content and the two enzyme activities

Table 1. Effect of *C2* and *Sn* on anthocyanin content and activity of CHS and CHI in seedlings grown in light or in darkness.

Genotype	T_0			48hD			48hL		
	Anth.	CHS	CHI	Anth.	CHS	CHI	Anth.	CHS	CHI
C2 Sn-s	0.1	49.0	5.6	2.3	35.0	5.6	31.4	105.0	20.8
C2 sn	0.0	1.2	5.6	0.0	0.0	4.0	0.1	11.9	4.0
c2 sn	0.0	0.0	3.2	0.0	0.0	4.0	0.0	0.0	4.0

T_0: onset of irradiation, following five days of growth in darkness. 48hD and 48hL: 48 hours of continuous dark or light after T respectively Anthocyanin content is in $A_{530}.g^{-1}$fr.wt., CHS is in Units $.g^{-1}$fr.wt., CHI is in$\Delta A_{385}.min^{-1}.g^{-1}$.fr.wt.
1 Unit: 1 pmol malonyl CoA converted into p-coumaroyl CoA-dependent, ethyl acetate extractable products per minute.

determined at the onset of irradiation and after 48 hours of growth in continuous light or darkness. The results indicate that both pigment content and enzyme activities are regulated by light as well as by the *Sn* genetic constitution. In homozygous *c2 sn* seedlings (*c2* is the gene responsible for CHS activity) there is a complete suppression of pigments and CHS activity while a low but significant level of CHI is still present. This low level of enzyme activity could be accounted for by a residual non-induced activity of CHI or by the existence of two enzymatic forms of CHI, only one of the two being *Sn* and light dependent.

These observations indicate that the presence of *Sn* promotes the enzymes tested in a coordinate, light-dependent manner suggesting a transactive regulatory role of this gene on the structural genes (or their products) involved in flavonoid biosynthesis in maize.

G. Consonni, M.L. Racchi, S. Shammah, G.A. Gavazzi

First results on the progeny of regenerated plants

The success obtained in plant regeneration from maize tissue culture by means of organogenesis or somatic embryogenesis and the possibility of obtaining somaclonal variants from regenerated plants stimulated us to test some genetic stocks for regeneration capacity and the obtainment of mutants of somaclonal origin. The genetic constitution of the two stocks used in this study is reported in the Table. The background of the first was originally W23 × K55 and it was reproduced by selfing while the second is an inbred line (A188). Calli induced on Murashige and Skoog medium (1962) were then transferred to regeneration medium, according to a procedure described by Armstrong and Green (1985). The yield of embryogenic calli differed in the two stocks as shown.

Genotype	Origin	Number of embryos explanted	Embryogenic calli (%)	Number of R1 plants
A1 A2 C1 C2 R-g	*W23+K55*	*120*	*13.5%*	*180*
A1 A2 c1 C2 r-r	*A188*	*140*	*2.5%*	*56*

Different morphological variants were observed among regenerated (R1) plants, including abnormal leaf and auricle development, white stripes on leaves, and tassel seed. Regenerated plants were often asynchronous in their male and female inflorescence maturation; accordingly they were outcrossed and the progeny grown and selfed.

The resulting ears were then scored for appearance of non-parental phenotypes and the results obtained are the following:

Number of R1 plants tested	Number of selfed ears	Ears with nonparental phenotypes			
		*dek**	*et**	*vp**	*ss**
11	87	7	1	1	3

**dek:* defective kernel; *et:* etched; *vp:* viviparous; *ss:* semisterility.

The frequency of these presumed mutants was in most cases lower than the expected one-quarter except for viviparous, which segregates as a monogenic recessive mutant; one of the seven *dek* and the three cases of semisterility exhibited a ratio of about 1:1. We are planning to ascertain the genetic basis of these presumed mutants through progeny tests, and to characterize them further in relation to their somaclonal origin.

M.L. Racchi and M. Pontoglio

Physiological components of yield

Rate of photosynthesis is expected to be directly related to yield potential and to be a major physiological component of yield in maize. On the contrary, available information generally shows an absence of association between photosynthetic variability and yield components. In fact the rate of photosynthesis may not be a limiting factor in a non-restrictive environment; moreover the photosynthetic activity must be estimated in a large number of samples with good precision and in the correlation analysis linkage effects must be distinguished from pleiotropic ones.

For these reasons we have analyzed physiological components of maize yield and their relevance by means of an appropriate population structure and by growing plants in normal and restrictive conditions. As regards the population structure, a set of random pure lines was extracted from the BSLE population and tested in 1984. Hybrid combinations were obtained from them according to an incomplete diallel crossing design (circulant diallel) and tested in 1985. In both cases two field conditions were used: normal (spring sowing) and restrictive condition (summer sowing) with light and temperature as a limiting factor in the last part of the vegetative cycle. The rate of photosynthesis was estimated as CER by means of an original apparatus based on $^{14}CO_2$ photosynthetic fixation, that ensures high repeatability of the data in a large number of samples.

The correlation coefficients between CER and yield components were (significant coefficients are underlined):

	Pure Lines		Hybrids	
	Normal	Stressed	Normal	Stressed
Yield/plant	0.39	0.41	0.33	0.22
50-kernel weight	0.05	0.43	0.29	0.18
Row number	-0.04	0.36	0.07	0.11
Kernels/row	0.31	0.33	-0.08	0.16
Stalk diameter	0.18	0.38	0.13	-0.16
Flowering time	-0.12	0.07	-0.53	-0.58

The results, interpreted also by means of multivariate statistical techniques, show that, as regards pure lines, CER can be a relevant component of the yield level especially in restrictive conditions. Hybrids are less sensitive to the environmental conditions. For these reasons the relevance of physiological parameters seems to be not independent from the genetic constitution of the material and this fact should be considered in breeding programs based on components of yield.

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Gene expression during male gametophyte development

A substantial part of the genome is expressed in the male haploid phase and the gametophytic-sporophytic genetic overlap has been estimated to exceed 60% (M. Sari-Gorla et al., TAG 72:42-47, 1986; P. Willing & J.P. Mascarenhas, Plant Phys. 75:865-868, 1984). However, all the studies have considered only mature pollen as male gametophyte and there is very little data concerning gene expression during microspore development.

In this report seven genes coding for enzymes were analyzed throughout pollen development, from 5 days after meiosis (DAM) to full maturity (anthesis). Six of these enzymes are present in mature pollen. Four (ADH-1, GOT-1, GOT-2, CAT-1) are multimeric and therefore suitable for the detection of the type of expression, haploid or diploid, of their encoding genes (Sari-Gorla et al., 1986). Two (GOT-3 and β-GLU-2) are invariant and possibly monomeric. For these only the onset of enzymatic activity during development was determined. One (β-GLU-1), absent in mature pollen, was included to verify its sporophyte-specificity.

The analysis indicates that: ADH-1 and GOT-2 show haplo-diploid expression from the earliest stages examined. CAT-1 shows haplo-diploid expression only starting 7 DAM. No activity at all could be detected earlier. An additional anodal band showing catalase activity appears in immature microspores only. The electrophoretic mobility and the absence of heterodimers with CAT-1 suggest this isozyme may be CAT-4. For GOT-1, 3 bands typical of sporophytic genetic control appear in the early stages (up to 9 DAM), while the 2-band pattern expected for haploid expression was found from 9 DAM on. GOT-2 and β-GLU-2 were clearly detectable from 5 DAM. β-GLU-1 was absent throughout pollen development, thus confirming its sporophyte-specificity.

On the whole the data indicate that:

– the male gametophyte is genetically largely independent from the sporophyte even in the first stages of development.

– in some cases (GOT-1) a sporophytic control over gametophytic functions may persist at least for the early steps of microspore development.

– some genes appear to be developmentally regulated (CAT-1, CAT-4) in the "pollen formation" phase. This last observation is confirmed by data regarding heat-shock response during microspore development.

Further analyses for a better characterization of the very early stages (0-5 DAM) are in progress.

Carla Frova

HSPs: temporal onset in developing pollen and genetic variability in the sporophyte

In last year's issue we reported evidence of HSP synthesis in immature pollen, 12 days after meiosis. In this note we present data on HSPs induced at different stages of microspore maturation.

Pollen development within the anther was divided into three stages: (A) From meiosis to first mitotic division, (B)

First and second mitotic division, and (C) Trinucleate pollen maturation. Each stage was analyzed for HSP synthesis by SDS-PAGE in a gradient 5-20% gel system.

High molecular weight HSPs approximately 84 and 72 Kd are induced in all stages following heat-shock (37 C). Some genotypes show an additional 94 Kd band starting from stage B.

In the 40-69 Kd range two stage-specific HSPs were found: a 46 Kd band appeared only in stage A, while a 66 Kd band is synthesized through stages B and C. These two HSPs are not inducible in sporophytic tissues.

In the low molecular weight range only an 18 Kd HSP was detectable, but the pattern through development is not very clear because overall incorporation of ^{35}S-methionine in immature microspores is low and therefore this non-prominent HSP appears very faint. Further analysis is under way to clarify this point.

The analysis of sporophytic HSPs was continued on additional genotypes. Genetic variability for several HSPs was detected. In particular, a low molecular weight 17 Kd band, additional to the normal HSP pattern, was found in several inbred lines. A preliminary genetic analysis shows that the F1 hs17$^+$/hs17$^-$ also synthesizes this band.

C. Frova, G. Binelli and E. Ottaviano

Pollen irradiation and gene transfer

The possibility of achieving transfer of one or a few traits from one genotype to another by the use of irradiated pollen was first described by Pandey in *Nicotiana* (Nature 256:310-313, 1975); however, the subsequent attempts to reproduce the phenomenon in other plants have not been generally successful. In maize, only sublethal doses of radiation can be used for pollen treatment, since no viable seeds are produced at higher doses; in order to study gene transfer in this crop, we utilized a genetic system suitable to distinguish between gene transfer and genetic loss or mutation.

A multiple dominant marker stock was used as pollen source: *C Sh Wx Gl15,* on chromosome 9; pollen was irradiated with 10, 20, 30, 50 Krad of X-rays and applied to multiple recessive females. The F1 seeds were harvested, scored for endosperm markers, and a sample of each treatment (both normal kernels and with unexpected phenotype) was selfed to observe F2 traits.

The results of pollinations using treated pollen are summarized in Table 1, where, according to each radiation dose, the mean number of kernels per ear, the percentage of sterile or male-sterile plants and the F1 endosperm characters are reported. The F1 generation from irradiated pollen revealed a high proportion of defective endosperm seeds, uncoloured seeds, shrunken seeds and uncoloured shrunken seeds.

Table 1.

RD	KNE	S%	KN	F1 seed characters coloured Sh	coloured sh	coloured de	colorless Sh	colorless sh	colorless de
0	89	0	446	446	0	0	0	0	0
10	62	23.3	1233	394	35	451	59	63	231
20	114	8.8	1255	1129	57	0	39	20	10
30	1	-	-						
50	0	-	-						

RD : Radiation dose
KNE : Kernel number per ear
S% : % of sterile or male-sterile plants
KN : Number of kernels assayed
de: defective endosperm

The F1 flowering plants were 954, from 1358 seeds sowed; the segregation for endosperm and seedling traits was analyzed in detail on a sample of twenty-one ears showing loss of endosperm markers or large segregation distortions. Single plant progenies showed complete loss of dominant allele *C* in 21 cases out of 843, of *Gl* allele in one case and of both *Wx* and *C* in one case.

When considering single gene segregation in the whole F2 progeny, plants from irradiated pollen revealed phenotypic ratios different from those of the control plants: a highly significant excess of recessive phenotypes was observed for *wx, gl* and *c* alleles.

M. Sari Gorla, M. Villa and E. Ottaviano

Are there *Mu1* sequences in B chromosomes?

We have carried out an experiment of in situ hybridization with a *Mu* sequence (generously provided by V. Walbot). The entire plasmid pAB5 (Taylor et al., Maydica 31:31-45, 1986) containing the internal 650 bp fragment of the *Mu1* element has been cloned in the single *Eco*RI site of the insertional lambda vector 1149. The entire recombinant phage was labelled by nick-translation with tritiated thymidine to a specific activity of about 20 million dpm per microgram of DNA. In situ hybridization in the presence of dextran sulphate was performed on microsporocytes from several Black Mexican lines containing 4 to 12 B chromosomes. Silver grains on B chromosomes have been observed in the two heterochromatic blocks adjacent to the proximal euchromatic region. Interestingly, the number of silver grains present in B chromosomes of the same microsporocytes varied from zero to ten. This suggests that sequences related to the *Mu* element preferentially accumulate in the same chromosomal region. Few silver grains were also observed in the A chromosome region, but there was no evidence of induction of mutable alleles in the self progeny of these *A C R Su* Black Mexican lines. However, when they were crossed as male parents with stocks recessive for endosperm marker genes, kernels with purple spots or coloured-purple kernels with *su* sectors were observed.

A. Viotti, L. Bernard and N.E. Pogna

A role for DNA methylation in the tissue-specific expression of maize genes?

Using Southern analysis of genomic DNA, digested with methylation-sensitive restriction enzymes, we investigated the methylation state of three families of storage protein genes in the following tissues and organs of inbred line W64A: endosperm at 8 days after pollination (dap), endosperm at 22 dap (w.t. and homozygous for opaque-2), three day etiolated shoot, three day root, immature ear (4 centimeters long) and mature pollen. We used as probes fragments of a cluster of zein genes coding for 22 kd polypeptides, an opaque-2 dependent cDNA clone coding for a 27 kd zein, and a novel cDNA clone related in sequence to a RSP gene. The transcripts of these genes were detected by Slot Dot analysis of total or polyadenylated RNAs (detection threshold: approx. 0.0004% of total RNA) only in the endosperm cells, but not yet at 8 dap, an early and for most aspects undifferentiated stage of development. The DNA of zein and RSP-related genes (coding regions and surrounding sequences) was found to be extensively

undermethylated in the endosperm, while we observed a single, more methylated pattern in the other tissues. Data on pollen DNA obtained with the 27 kd zein clone revealed a similarly methylated pattern. At 8 dap, the demethylated pattern is already and completely established, indicating that DNA demethylation precedes and is not necessarily coupled with the active transcription of these genes. In agreement with these data, we found the demethylated pattern of the 27 kd zein sequences in the 22 dap opaque-2 endosperm as well, where the level of the corresponding transcripts is significantly reduced. The 5-methyl cytosine content of endosperm DNA is not significantly different from that of DNA from the immature ear, as assessed by HPLC analysis, or from that of DNA from the other tissues, as assessed by comparison between ethidium bromide stained or end-labelled digests. Moreover, hybridization experiments with a sequence abundantly represented in root and endosperm RNA, revealed very similar methylation patterns in the two tissues.

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Absence of restoration of *o6 (pro1)* endosperms with proline

Last year (C. Tonelli et al., MNL 60:100) we reported that the *pro1* mutant is functionally allelic to *o6*; accordingly, *o6* seedlings recover when supplemented with l-proline in the growing medium.

This summer we tested the effect of l-proline on young developing *o6* endosperms. On account of the relationship between *o2* and *o6* (both repress zein accumulation and suppress or reduce b32 protein in the endosperm: L. Manzocchi et al., TAG 72:778-781, 1986), we also included *o2* developing endosperms in the test.

A69Y normal, *o2* and +/*o6* plants were selfed, and the endosperms were collected ten days after pollination and cultivated in vitro. Ears segregating + and *o6* seeds were recognized by leaving half of the ear on the plant till maturation and saving the results only for those endosperms deriving from segregating ears. The endosperms were cultivated on agar medium with MS salts, sucrose (3%), asparagine (0.2%) and in the presence or absence of 2 mM l-proline. After 6 days at 26 C, the fresh and dry weight and the content of salt and ethanol soluble proteins were determined. In addition, total proteins were analyzed by SDS-PAGE on pooled normal and *o2* endosperms, and on individual endosperms in +/*o6*.

Presence or absence of proline did not affect the growth of the endosperms, which was about ten-fold the original weight. In the absence of proline, wildtype endosperms accumulated large amounts of zeins, while in *o2* zeins were drastically reduced; endosperms from +/*o6* plants segregated, as expected, high zein/low zein phenotypes, as judged by their electrophoretic patterns. In the presence of proline, essentially the same results were obtained, in particular for the endosperms from +/*o6* plants, where a segregation for high/low zein phenotypes was still observed.

The results suggest that in our culture conditions 2 mM l-proline (a concentration restoring a normal phenotype in *o6* seedlings) does not promote restoration of *o6* endosperms.

Lucia A. Manzocchi and Carlo Soave

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Inheritance of knob heterochromatin

In higher plants, tandemly repeated DNA sequences appear as distinct, heteropycnotic regions located at certain sites on specific chromosomes and look much like beads on a string. They are called knobs by maize cytogeneticists. The genetic effects of knobs in maize include: (1) increased recombination (McClintock, CIW Yearbook 42:148-152, 1943; Rhoades and Dempsey, Genetics 53:989-1020, 1967); (2) neocentromere activity (Rhoades and Vilkomerson, PNAS 28:433-436, 1942); (3) preferential segregation (Longley, Genetics 30:100-113, 1945; Dempsey and Rhoades, MNL 44:56-61, 1970); (4) chromosome breakage and chromatin loss (Rhoades et al., PNAS 57:1626-1632, 1967; Rhoades and Dempsey, MNL 46:48-51, 1972); (5) sex differences in recombination (Rhoades, J. Amer. Soc. Agron. 33:603-615, 1941; Phillips, MNL 45:123-125, 1971).

In wild plants, knobs are typically telomeric; whereas, in cultivated species, they move to internal positions on chromosomes. In maize and annual teosinte, approximately 23 intercalary knob positions have been identified. In diploid perennial teosinte, *Zea diploperennis*, and Guatemalan teosinte, the knobs are telomeric. A cytological study of knobs in F1 progeny of crosses using maize as the pollen parent and *diploperennis* as the female parent demonstrated that the number of terminal knob sites is less than would be expected in Mendelian inheritance, and the number of intercalary knobs is above that expected to be inherited from the male maize parent (Eubanks, MNL 60:103, 1986). The phenomenon of knob transposition and chromosome reorganization as a result of interspecific hybridization is suggested by these data. It contrasts with the basic assumption in maize cytogenetics that knobs, like mutant genes, are stable, heritable characters. Evidence from a recent study of crosses between *diploperennis* and annual teosinte indicates that the number of knobs in the F1 progeny is dependent upon the direction of the cross.

Annual teosinte plant material for the experiment was grown from seed provided by the Southern Regional Plant Introduction Station, Experiment, Georgia (*Zea mexicana*, P.I. # 331779, origin: Miraflores, Mexico), and *diploperennis* plants were grown from seed provided by Professor Hugh Iltis at the University of Wisconsin-Madison (*Zea diploperennis*, Guzman # 777, origin: Jalisco, Mexico). The first phase of the research was carried out at Indiana University in Bloomington in 1985, and the second phase at Vanderbilt University in Nashville in 1986. The cross was made both ways with each species serving as male and female parent. Sporocytes from individual parent plants and their F1 progeny were collected and examined cytologically. The number of knobs observed at pachytene was recorded (Table 1).

The *diploperennis* parent had 6 small terminal knobs. The annual teosinte parent had 4 intercalary knobs. If

Table 1. Chromosome knob number in annual teosinte, *diploperennis* and F1 hybrids.

Plant	Collection	Knob number
Zea diploperennis (2A, 2-5)	Guzman # 777, Jalisco	6
Zea mexicana (22)	P.I. # 331779, Miraflores	4
F1 hybrids		
2A × 22		14
22 × 2-5		3

knobs are inherited in accordance with Mendelian genetics, the number of knobs in the F1 progeny should range from a minimum number of 4 to a maximum number of 10. Interestingly, however, when annual teosinte was the female parent, only 3 knobs were observed on the chromosomes of the F1 progeny. This is 1 knob less than would be expected of the minimum number. These plants had a 25% germination rate, were weak, spindly and male sterile. When annual teosinte was the male parent, 14 knobs were observed, 4 above the predicted maximum number. These plants had a 75% germination rate, were vigorous, highly tillered and male fertile.

These results raise three questions. I. Do the knob data reveal an amplification of repetitive DNA in some crosses and a loss of knob heterochromatin in other crosses when the sexes of the parent plants are reversed? II. Is there transposition of knob heterochromatin in progeny of interspecific crosses? III. Is knob satellite DNA genetically inert or is it transcribed and does it play a role in gene regulation? More work on the inheritance and expression of knob heterochromatin is needed to gain a better understanding of its genetic functions and evolutionary role in the origin of maize.

Mary Eubanks

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A new, improved FPG technique for detecting sister-chromatid exchanges in mitotic chromosomes

We have reported a fluorescence-plus-Giemsa (FPG) technique for visualizing sister-chromatid exchanges (SCE's) in maize mitotic chromosomes utilizing 5-bromodeoxyuridine (MNL 54:88-89, 1980). Recently, certain modifications of the maize FPG technique have been developed. The modifications involved pectinase concentration and treatment time, the concentration of Hoechst 33258 stain, and the temperature during the post-stain treatment.

In the previously described procedures, the pectinase concentration was 0.5% and the treatment time was 1 hr. Under this treatment condition, root tips were still relatively hard. After maceration by a glass rod, many metaphase cells were broken. The broken cells may be the cause of chromosome loss during the later slide preparation procedures. Softening the root tips by pectinase treatment may increase the percentage of intact cells after slide preparation. For this reason, various enzyme concentrations and treatment times were tested. When the pectinase concentration was 1.5% and the treatment time was 2 hrs., the root tips were soft enough to be smashed with a dissecting needle. Then, cells were gently macerated with a glass rod and flattened by placing a cover glass on the suspension and pressing on it. After these treatments, many intact metaphase cells were observed. Intact or slightly damaged cells had a better chance of staying on a slide than chromosomes from ruptured cells during the treatments required to demonstrate sister chromatid differentiation. Goto et al. (Chromosoma 66:351-359, 1978) suggested a practical concentration of 10^{-5} M of Hoechst 33258 to detect SCEs in rat bone marrow cells. This is ten times higher than the concentration we reported previously. To make the 10^{-5} M Hoechst 33258 solution, 1 mg of Hoechst 33258 was dissolved in 1 ml of 100% ethanol, and 1 ml of this solution was added to 200 ml of 0.5 × SSC.

Another factor that affects the success of the sister-chromatid differentiation is the temperature during the UV exposure. In our previous report, the experiments were performed at room temperature. However, the temperature in the laboratory fluctuated during the day and between seasons. In our modified procedure, slides were placed on a slide warmer and this kept the temperature at 45 C during UV exposure.

The rest of the slide preparation procedures were the same as previously described. A far higher percentage (20-40 times as many) of the cells treated with this improved protocol, showed a better sister-chromatid differentiation than with the protocol initially developed.

Tau-San Chou and David Weber

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Dosage analysis of the *D8* allele for dwarfism

The *D8* locus, which is on the long arm of chromosome 1, has an additive effect on the stature of plants. Selfing a known heterozygote produces a ratio of 1 normal (+/+): 2 dwarf (*D8*/+) : 1 mini-dwarf (*D8*/*D8*). The *D8*/+ dwarfs are generally less than ¼ the height of normal (+/+) sibs. Likewise, mini-dwarfs are about ⅔ the size of dwarfs. Unfortunately, mini-dwarfs fail to set seed or produce viable pollen.

In order to study the nature of the *D8* allele, *D8*/+ plants were pollinated by TB-1La hyperploid translocation heterozygotes. Table 1 shows the expected genotypes and chromosome numbers of the progeny of this cross. Half of the progeny are expected to contain the *D8* allele in the presence of 0, 1, or 2 doses of the normal (+) allele.

Table 1. Expected genotypes of progeny from the cross *D8*/+ × 1 1^{B} B^{1} B^{1} (+/+/+). Chromosome number in parentheses.

		Sperm Nuclei			
		1	$1^{B}{}_{B}{}^{1}$	$1^{B}{}_{B}{}^{1}{}_{B}{}^{1}$	1^{B}
Egg Nuclei	+	+/+ (20)	+/+ (21)	+/+/+ (22)	+/- (20)
	D8	D8/+ (20)	D8/+ (21)	D8/+/+ (22)	D8/- (20)

All progeny kernels were from one ear and when planted were separated into two groups. One group consisted of very small kernels and should contain mainly hyperploid embryos (Roman and Ullstrup, Agron. J. 43:450-454, 1951). Approximately half of these should be *D8*/+/+ genotype and will provide the opportunity to study the influence of the *D8* allele in the presence of two normal alleles. The other group consisted of the largest kernels and should contain some hypoploid plants, half of which should be *D8*/- genotype. The +/- hypoploids also are reduced in stature (about half the size of +/+ sibs) and are easily distinguishable from *D8* dwarfs, not only due to their greater height, but also due to their narrow, stiff, pointed leaves.

All kernels were planted in early spring in peat pellets in the greenhouse in order to facilitate root-tip collection. They were subsequently transplanted to the field. Table 2 lists the phenotypes and probable genotypes of all plants for which root-tips were successfully analyzed cytologically.

Table 2. Phenotypic and genotypic classifications of cytologically analyzed progeny from the cross *D8*/+ × 1 1^B B^1 B^1 (+/+/+).

Chromosome #	Phenotype	# of Plants	Genotype
20	narrow-leaf pygmy	5	+/-
20	stunted narrow-leaf pygmy	2	D8/-
20	dwarf	8	D8/+
21	normal	5	+/+
22	normal	14	+/+/+
22	dwarf	9	D8/+/+

It is interesting to note that two expected classes are conspicuously missing. Normal (+/+) plants with 20 normal chromosomes and normal (+/+) plants with 21 chromosomes (1 1^B B^1) do not appear in the progeny. This may be due to the kernel size selection that was performed. An earlier planting of kernels from a different ear of the same cross (*D8*/+ × TB-1La hyperploid) on which the kernels were not as easy to separate into large and small classes produced several normal plants with 20 or 21 chromosomes.

Most importantly, the data show that *D8* is a mutation which causes a gain in function. Nine plants were *D8*/+/+ and had a dwarf phenotype indistinguishable from *D8*/+ dwarfs. Also two plants with 20 chromosomes exhibited a very interesting phenotype. They displayed the hypoploid phenotype (stiff, narrow leaves) yet were only 6 inches tall due to very short internodes. In other words, they were essentially dwarf hypoploids (*D8*/-). Thus, it appears that the *D8* allele has a dwarfing effect in the presence of 0, 1, or 2 normal (+) alleles.

Rick W. Staub and Patricia M. Laurenson

Consistent nondisjunction of B chromosomes in Black Mexican

In reviewing cytological data from crosses in which Black Mexican sweet corn with B chromosomes (BMSC-B) was used as the pollen parent on females lacking B chromosomes, it was observed that the number of B chromosomes in the progeny was almost always even. In fact, of 223 plants from this type of cross, root-tip cytology has revealed that all but four possessed an even number of B chromosomes. Plant RS1044 had 3 B chromosomes and was from a 0B × 9B cross. Plant 86.C10.12 had 9 B's and was from a cross in which the female had no B's and the male had at least 9 B's. Plant 85.103.6 was a 1B/2B chimera (i.e., half 1B and half 2B nuclei in one root-tip) and was from a 0B × 6B cross. Plant 86.C6.11 was from a cross of a female with no B's and a male with at least 7 B's. It was analyzed as having 3 B's, but this was after it had been noticed that most plants from crosses of this type possess an even number of B's. Two more root-tips were analyzed and both were found to have 2 B's.

The cytological data from plants 85.103.6 and 86.C10.12 indicate that B chromosomes may display a low degree of instability in the course of development. That this instability, if it does exist, occurs at a low level is indicated also by comparison of mitotic and meiotic chromosome counts that were performed on 34 plants containing varying numbers of B chromosomes. Of these, only one displayed discordant numbers of B chromosomes in the two analyses. Plant RS650 had 2 B's in root-tip cells and only 1 B in pollen mother cells.

Therefore, it is possible that nondisjunction of all B chromosomes at the second pollen mitosis occurs 100% of the time and that plants containing an odd number of B chromosomes from 0B × B crosses actually represent subsequent instability of B chromosomes in somatic cell divisions. Alternatively, it is possible that, rarely, B chromosomes do undergo normal disjunction in the second pollen mitosis. If this is true, it seems to occur only when the plant possesses a very high number of B chromosomes (at least 9 B's in the two cases presented here).

Thus, it seems quite possible that nondisjunction of B chromosomes at the second pollen mitosis may occur 100% of the time in Black Mexican sweet corn. Utilization of plants with low numbers of B's should virtually guarantee production of male gametes with even numbers of B chromosomes in this inbred line.

In order to study the nature of the influence of genetic background on B chromosome nondisjunction, crosses of BMSC-B to other inbred lines must be made. Preliminary cytological data from A632/BMSC-B hybrids used as pollen sources on 0B plants show 4 progeny with odd B chromosome number out of a total of 21 progeny analyzed. This indicates that the B chromosome nondisjunction rate is a maximum of 81%. Thus, it appears that A632 does not have the proper genetic constitution to support consistent nondisjunction of B chromosomes.

It is interesting to note that early cytological studies of progeny from 0B × B crosses show highly variable amounts of B chromosome nondisjunction (A.E. Longley, J. Agric. Res. 34:769-784, 1927; L.F. Randolph, Genetics 26:608-631, 1941; M. Blackwood, Heredity 10:353-366, 1956). However, none of these researchers used inbred lines, and in fact often intercrossed stocks to utilize hybrid vigor in maintaining B chromosomes. The only case of 100% nondisjunction reported was found by Randolph when he crossed 0B females and 2B males. In the progeny of this cross he obtained 22 0B, 37 2B, and 2 4B progeny. In all other 0B × B crosses, however, he obtained several progeny with odd B chromosome numbers. His sources of B chromosomes were Black Mexican and Golden Bantam varieties of sweet corn. It is possible that in this particular cross, the 2B plant(s) used as the male was from a Black Mexican inbred line.

It seems quite probable that the genetic constitution of plants containing B chromosomes will have a strong influence on the rate of B chromosome nondisjunction. For example, genes that shorten the duration of the division of the generative nucleus or lengthen the duration of replication of centromeric heterochromatin may have a strong positive influence on B chromosome nondisjunction. At any rate, BMSC-B can be used as a pollen source in crosses with 0B plants to assure nearly all progeny will have even numbers of B's. Likewise, if B-A translocations are maintained in a Black Mexican background, the proportions of hypo- and hyperploid progeny could be maximized.

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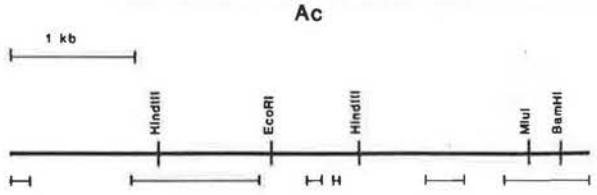
Corrections in the nucleotide sequence of Activator (*Ac*)

In the process of characterizing the *Ac* element present in the *bz-s:2114(Ac)* allele, we have discovered two mistakes in the published *Ac* sequence (R. F. Pohlman, N.V. Fedoroff and J. Messing, Cell 37:635, 1984; M. Mueller-Neumann, J. I. Yoder and P. Starlinger, Mol. Gen. Genet. 198:19, 1984). *bz-s:2114(Ac)* is a derivative of *bz-m2(Ac)*, which has a deletion adjacent to *Ac* (H. Dooner, Plant Genetics, p. 561, 1985). We have cloned both alleles and in restriction mapping we found an *Mlu*I site that was not predicted by the published sequence. The site was also present in *bz-m2(Ac)* and *wx-m7(Ac)* (pJAC, kindly supplied by J. I. Yoder and P. Starlinger). *Bam*HI-*Mlu*I fragments from *bz-s:2114(Ac)*, *bz-m2(Ac)* and *wx-m7(Ac)* were filled in with Klenow, subcloned into M13mp18 in both orientations and sequenced. This sequencing confirmed that the *Mlu*I site in all three *Ac*s resulted from an additional A residue at base pair (bp) 4132. We also found an additional C residue at bp 4225. The additional A residue at 4132 alters ORF 3 by making it 9 bp (three codons) longer, terminating at 4202. Between this and other work (H. Dooner, J. English, E. Ralston, and E. Weck, 1986, Science 234:210), we have sequenced 2300 bp of *Ac* (see figure) and have confirmed all but the two

Figure 1. Restriction map of the transposable element *Ac* with the regions that we have sequenced underlined.



missed nucleotides described above. With the addition of two base pairs, the length of *Ac* is at least 4565 bp.

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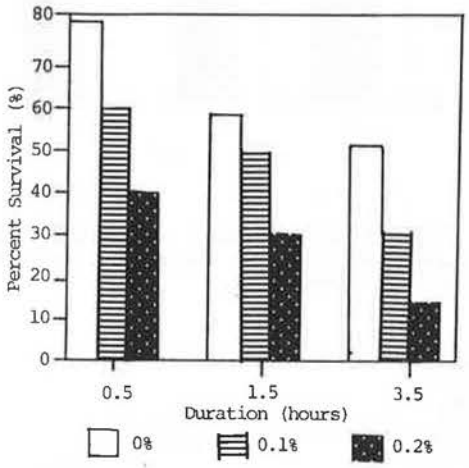
Mutagenesis of tissue cultures

Spontaneous variation found in regenerated plants has been termed "somaclonal variation" and is a common phenomenon in plants. We have been intending to enhance the variation in several elite corn inbred lines through the application of mutagens to maize tissue cultures using EMS, sodium azide, UV-light, X-ray and others. Friable, embryogenic calli of A619, B73 and Mo17 were initiated from immature embryos on MS medium supplemented with either 2,4-D (1.5-4 mg/l) or dicamba (1-8 mg/l), and maintained on the same medium plus proline (10-40 mM) and casein hydrolysate (100-500 mg/l) with a subculture interval of 10 to 14 days. These calli have been maintained for more than ten months.

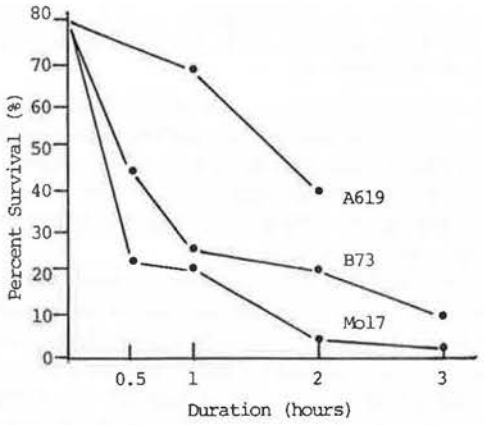
EMS treatment: A619, B73 and Mo17 embryogenic calli were dispersed in liquid MS medium prior to EMS treatment. EMS concentrations of 0.1 to 0.2% (v/v) were applied to the calli for one to four hours. The treated calli were washed three times with fresh liquid medium. Percent survival was assessed under a dissecting microscope. An evaluation of EMS concentration and treatment duration was conducted for B73 only (Fig. 1). It appeared that

Figure 1. Effect of EMS concentration and treatment duration on B73 callus. Each value represents the average of six determinations.



the percent survival of B73 callus decreases with increasing EMS concentration and treatment duration, and that 0.2% EMS was the most appropriate concentration for maize tissue culture. Therefore, 0.2% EMS was applied to A619 and Mo17 calli (Fig. 2). The results also indicated

Figure 2. Response of A619, B73 and Mo17 to 0.2% EMS treatment. Each point represents the mean of three replications.



that an EMS treatment duration of one to two hours is suitable for maize tissue culture. The sensitivity of maize calli to EMS differed among the genotypes and cell lines within a particular genotype. Mo17 callus was more sensitive to EMS treatment than A619 and B73. Regeneration of the treated calli is under way.

Sodium azide treatment: A619, B73 and Mo17 embryogenic calli were dispersed in sterilized 0.1 M phosphate buffer at pH 3.0 with or without added azide. After treatment, calli were washed with liquid MS medium and transferred to maintenance medium. The effect of azide concentration and treatment duration on B73 callus is shown in Figs. 3 and 4. Percent survival decreased with increasing azide concentration and treatment duration. Less than 5% survival was observed in B73 and Mo17 calli

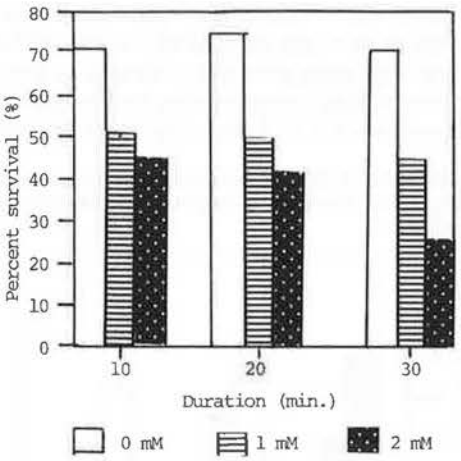


Figure 3. Effect of sodium azide concentration and treatment duration on B73 callus. Each value represents the average of six determinations.

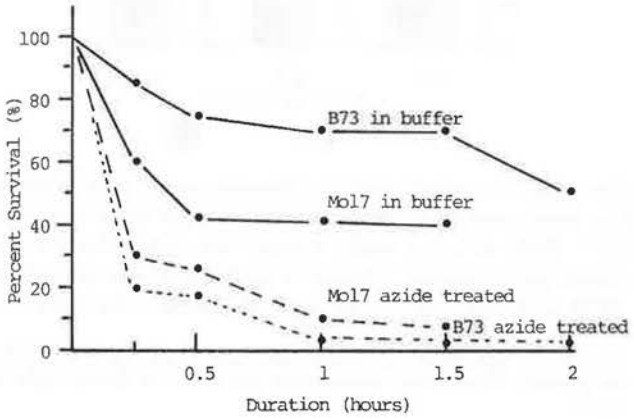


Figure 4. Effect of sodium azide (2 mM) and phosphate buffer on Mo17 and B73 calli. Each value represents the average of two replications.

treated with 2 mM azide for longer than one hour. Phosphate buffer alone seemed to slightly reduce survival of B73 and Mo17 calli. Sensitivity of maize callus to azide was similar to EMS treatment; i.e., it was genotype and cell line dependent, and Mo17 callus was less sensitive to azide than A619 and B73.

A total of 83 R0 plants were regenerated from B73 calli treated either with azide or phosphate buffer alone and grown to maturity in a greenhouse. A hand microscope (Nikken, Tokyo) was used to determine pollen sterility. R0 regenerants with at least 5% pollen sterility were recorded (Table 1). The results indicated that the frequency of R0 regenerants with pollen sterility is higher for those plants from calli treated with either sodium azide or phosphate buffer alone than those plants from control calli. A total of 63 R0 plants produced enough seed (at least 15 seeds) for evaluation of variant segregation in R1 progeny tests. Chlorophyll variants were predominant, although other variants such as a white kernel mutant and some with altered vigor and earliness were also observed among the R1 progenies. R0 regenerants derived from calli treated with phosphate buffer alone produced as many variants as those from azide treated calli, and the frequency of variants from both treatments was higher than the control (Table 1). Therefore, it is not clear if a low pH value (pH 3.0), phosphate buffer, or sodium azide cause pollen sterility and mutation in the R0 regenerants.

Table 1. Effect of phosphate buffer and sodium azide on B73 callus.

Treatment	Treatment time (min)	% R_0 plants with pollen sterility[a]	% variants in R_0 population
1 mM NaN_3	10	41 (9/22)	36 (8/22)
2 mM NaN_3	10	33 (2/6)	20 (1/5)
1 mM NaN_3	20	63 (5/8)	57 (4/7)
2 mM NaN_3	20	30 (9/30)	72 (18/25)
1 mM NaN_3	30	0 (0/1)	100 (1/1)
2 mM NaN_3	30	44 (4/9)	100 (3/3)
PO_4 buffer	20	60 (6/10)	75 (3/4)
PO_4 buffer	30	50 (5/10)	100 (2/2)
Control	0	29 (51/183)	17 (24/138)

a. Number of R_0 plants with at least 5% pollen sterility/total number of R_0 plants.

b. Number of variant R_0 plants / total number of R_0 plants.

UV irradiation: Prior to irradiation, B73 friable, embryogenic callus was dispersed in liquid MS medium and forced through a 600 μm screen. Cell clumps smaller than 600 μm were evenly spread on MS solid medium and irradiated with UV for 0.5 to 8 hours. A 15-W General Electric germicidal lamp emitting at approximately 2600 A with an energy output of 25 ergs/mm^2/sec was used. The results indicated that percent survival of the irradiated callus decreased with increasing UV doses (Fig. 5). Calli irradiated with UV longer than 3 minutes became watery and died one month after irradiation. To date, only a few plants have been regenerated, and one yellow-green variant and one dwarf variant were detected in R1 progeny rows. The progenies segregated 3:1 for the respective characteristics indicating a single recessive mutation. Large-scale plant regeneration is under way.

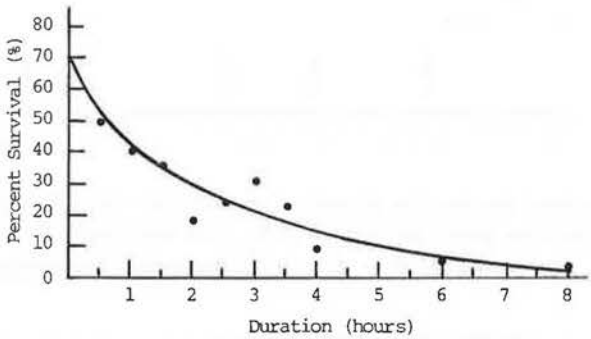


Figure 5. Effect of UV irradiation on B73 friable, embryogenic callus. Each value represents the average of two separate experiments, each experiment consists of at least three replications.

X-ray irradiation (in collaboration with David Cheng and Tracy Yang): Macerated B73 friable embryogenic callus (smaller than 400 μm in diameter) was uniformly spread over a 60 × 20 mm petri dish containing 25 ml of maintenance medium and irradiated with 11 doses (0 to 8.4 kilorads (kR)) of X-ray. Calli irradiated with doses higher than 2.7 kR died one month after irradiation. Three irradiated calli (0.8, 1.3 and 2.7 kR) showed significant increases in growth rate, and one (0.8 kR) showed an increase in embryogenic activity six months after irradiation.

A total of 230 R0 plants were regenerated for evaluation. Pollen fertility and seed set decreased with increasing X-ray dosage (Table 2). Of 230 R0 plants, 83 produced enough seeds (at least 15 seeds) for evaluation of mutant

segregation in R1 progeny tests. The mutation rate increased with higher doses of X-ray.

Table 2. Effect of X-ray irradiation on pollen fertility and mutation rate of B73 regenerants.

X-ray dose (kR)	0	0.2	0.4	0.6	0.8	1.0	1.3	2.7	4.0
% plants w/ above 5% sterile pollen	20	55	25	100	98	73	100	100	100
Mutation rate (%)	9	33	55	--	70	86	100	100	--

Traits such as virescence, defective kernel, striate, white seedling, zebra band, viviparous, wilted, shrunken endosperm and vigorous plants have been observed in R1 progeny rows. Most of the variants segregated 3:1 for the respective traits, indicating a single recessive mutation. The degree of chromosomal alteration increased with X-ray dosage. Several sporocytes were collected and analysed. A deficiency of the proximal portion of the long arm of the chromosome, a trisomic-7, and translocations involving two or three chromosomes have been detected.

A.S. Wang, M.D. Hollingworth and J.B. Milcic

Inheritance of the culture induction response

The induction of somatic embryogenesis and subsequent plant regeneration in maize can be achieved with many inbred lines, although a number of elite inbreds show poor response in vitro. By manipulation of the callus induction medium, it was found that the embryogenesis induction response is largely a physiological phenomenon; a change in the concentration or molecular configuration of the plant growth regulator (PGR) dramatically altered the response of each inbred examined.

Immature embryos from the inbreds B73 and MS71, reciprocal crosses between B73 and MS71 (F1), and self-pollinations of the hybrids derived from these crosses (F2) were isolated when they had attained a length of 1.2-1.8 mm. All culture induction media contained N6 salts, 9% sucrose, and 0.1 to 100 μM of 2-, 3-, or 4-chlorophenoxyacetic acid (CPA). Cultures were scored for embryo formation after three weeks. Treatments which were scored with a "±" (Table 1) produced embryoids at frequencies occasionally approaching those scored with a "+" (optimum induction frequency and growth of embryogenic tissue), but either the growth of the tissue was slow (at concentrations above the optimum) or the concentration of the PGR was high enough only to induce embryoid-like structures in a few isolated cases (at concentrations below the optimum). Treatments scored with a "-" indicate either germination of the immature embryos (concentrations below the optimum) or tissue necrosis with little or no detectable growth (concentrations above the optimum).

Each form of CPA elicited a similar concentration-dependent response. At low concentrations, the scutellum swelled slightly but did not form embryoids. Embryoids formed from the scutellum when the concentration was increased, and a further increase in concentration resulted in toxicity. While each inbred showed a similar overall response pattern, the relative sensitivity of each to different concentrations of the three forms of CPA differed between genotypes. This was particularly evident in the comparison of B73 (a BSSS derivative) and MS71 (an A619 derivative). B73 showed a greater sensitivity than MS71 in response to each concentration of each form of CPA.

Reciprocal crosses between these two inbreds sometimes showed a similar response pattern to that of the maternal parent of the cross, as was especially evident in the case of MS71 × B73. Comparison of the F2 generation embryos showed essentially no difference in sensitivity to

Table 1. Embryogenesis induction by substituted phenoxyacetic acids in B73, MS71 and the F1 and F2 generations of reciprocal crosses.

MS71

Conc., μM	2-CL	3-CL	4-CL
0.1	-	-	-
1.0	-	-	+
10.0	-	+	±
100.0	+	±	-

B73

Conc., μM	2-CL	3-CL	4-CL
0.1	-	-	±
1.0	±	+	+
10.0	+	+	-
100.0	±	-	-

(MS71 x B73)

Conc., μM	2-CL	3-CL	4-CL
0.1	-	-	-
1.0	-	-	+
10.0	-	+	±
100.0	+	-	-

(B73 x MS71)

Conc., μM	2-CL	3-CL	4-CL
0.1	-	-	-
1.0	-	±	+
10.0	±	+	-
100.0	+	-	-

(MS71 x B73)x

Conc., μM	2-CL	3-CL	4-CL
0.1	-	-	-
1.0	-	-	+
10.0	-	+	-
100.0	+	-	-

(B73 x MS71)x

Conc., μM	2-CL	3-CL	4-CL
0.1	-	-	-
1.0	-	-	+
10.0	-	+	-
100.0	+	-	-

\+ OPTIMUM CONCENTRATION FOR CALLUS INDUCTION OF THOSE TESTED.

± NON-OPTIMUM CONCENTRATION; SPORADIC EMBRYOID FORMATION OBSERVED IN SOME REPLICATES (CONCENTRATIONS BELOW THE OPTIMUM) OR POOR TISSUE GROWTH (CONCENTRATIONS ABOVE THE OPTIMUM).

\- NO FORMATION OF EMBRYOGENIC TISSUE IN ANY CASES.

Table 2. Embryogenesis induction by substituted phenoxyacetic acids in the F2 generation of reciprocal crosses between B73 and MS71.

		Phenoxyacetic Acid					
		2-Cl		3-Cl		4-Cl	
Genotype	Conc., μM	N	Freq. (%)	N	Freq. (%)	N	Freq. (%)
(MS71 x B73)x	0.1	180	0	180	0.6	180	1.1
	1.0	180	0.6	180	2.2	180	91.1
	10.0	180	17.8	180	77.2	180	32.8
	100.0	180	81.7	180	0	180	0
(B73 x MS71)x	0.1	162	0	186	0	156	0
	1.0	162	0	166	4.8	164	92.7
	10.0	158	29.1	166	89.2	152	25.7
	100.0	162	89.5	166	0	161	0

the PGRs. In addition, the response tended to be uniform for all embryos in each petri dish; there was no evident Mendelian segregation as would have been expected for nuclear genes (Table 2). The case where partial response was seen (at concentrations just above or below the optimum concentration) appeared to be physiological in nature—swelling of the scutellum with no embryoid formation at lower concentrations and toxicity at higher levels. In these instances, any induction of embryogenesis was sporadic and occurred at a low frequency.

These results indicate that the induction in vitro of somatic embryogenesis in maize is greatly influenced by the concentration and molecular structure of a given PGR and, under the appropriate conditions, can be achieved at relatively high frequencies independent of the genetic background of the explant tissue. Despite the similarity of the response of F1 hybrids to that of the maternal parent of each hybrid in many cases, it is unclear whether the inheritance of culture response is controlled by cytoplasmic genes. Further, the lack of well-defined segregation ratios in the F2 generation indicates a complex mode of inheritance.

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Tp1 is not cell-autonomous

Tp1 is a dominant homeotic mutation whose most conspicuous phenotype is the production of leaves or leaf-like structures in the ear and the tassel. This phenotype could result from a defect in supra-cellular factor(s) that initiate or suppress leaf development, or a defect in the ability to respond to such factor(s). Mutations that affect these two systems can be differentiated by examining their expression in genetic mosaics. Mutations that affect supra-cellular (i.e., diffusible) factors are likely to be non-cell-autonomous, whereas those that affect the ability of cells to respond to such factors are likely to be expressed in a cell-autonomous fashion.

Seeds of the genotype + *Tp1*/*o5-1241* + were irradiated (1 Krad, 250 KV, 15 mA, 2 mm Al) 24 hours after imbibition, grown to maturity, and then screened for sectors expressing the pale yellow phenotype of *o5-1241*. The *o5* locus is located about 10 map units proximal to *Tp1*. Thus, cells expressing *o5-1241* have a high probability of being hemizygous for the wild type allele of *Tp1* because terminal deletions that result in the loss of $o5^+$ will also remove *Tp1*. Although some sectors may result from interstitial deletions that remove $o5^+$ but not *Tp1*, such deletions require 2 chromosome breaks and are therefore much less frequent than terminal deletions, which arise from single breaks.

Seventeen plants with *o5-1241* tassel sectors, 9 plants with ear sectors and 15 plants with tiller sectors were found in a total of 1660 plants. Sectors only encompassed about 1/20 to 1/10 of the circumference of the tassel, but generally occupied a much larger fraction, if not the entire circumference, of an ear or tiller. All but 2 of the tassel sectors extended from the tassel into the vegetative part of the shoot. Tiller and ear sectors generally encompassed all the nodes in these structures.

The feature of primary interest in this experiment was the phenotype of sectors in the tassel and ear, in particular the presence or absence of spathes subtending spikelets. With one exception, all of the sectors in either of these inflorescences had a *Tp1* phenotype indistinguishable from that of surrounding tissue. In the exceptional case, spikelets within the sector were normal in appearance (i.e., lacked spathes). However, because other non-sectored regions of the tassel also had a normal phenotype, it is unclear whether to attribute the phenotype of this *o5-1241* sector to its $Tp1^+$ genotype, or to the poor expression of *Tp1* in surrounding tissue.

Twenty-two of the sectors we observed were located in the L1 layer of the meristem and 15 were located in the L2 layer. The location of the remaining 4 sectors was not determined. Although cells from one of these lineages may replace cells from the other lineage, this usually only occurs during leaf development, not during the growth of the shoot meristem. Thus most of the structures containing *o5-1241* sectors also possessed an epidermis or some sub-epidermal tissue that still carried *Tp1*. In 2 cases, however, spathes arose in regions of the shoot in which *o5-1241* cells in the L1 had displaced L2 cells so that all the tissue in these spathes was genetically *o5-1241*.

These results strongly suggest that *Tp1* is not expressed in a cell-autonomous fashion because wild type cells in proximity to *Tp1* cells can be induced to form mutant structures. What is unclear is the distance over which this induction operates. The fact that *Tp1* cells in the epidermis are capable of recruiting $Tp1^+$ cells in the sub-epidermis, and that *Tp1* cells in one half of a spathe can recruit $Tp1^+$ cells to form the other half demonstrates that this effect extends to at least the boundaries of an organ. The 2 cases involving larger sectors of *o5-1241*

tissue suggest that *Tp1* can operate over even greater distances, but we have not observed enough of these sectors to be confident about this conclusion.

Scott Poethig

Dosage analysis of *Tunicate*

The *Tunicate* mutation causes glumes in the ear and tassel to become abnormally large. Mangelsdorf and Galinat (PNAS 51:147-150) demonstrated that this mutation has 2 components, both of which have a weak *Tu* phenotype, and that these components can recombine to reconstitute a mutation having the severe phenotype of the original locus. This observation raises the possiblity that *Tu* is a duplication, and that its phenotype is a result of the overproduction of the product encoded by this locus. To test this hypothesis, *Tu c2/+ c2* plants were crossed by TB-4L, and plants of the genotypes *Tu/+/+*, *Tu/—*, were identified on the basis of the expression of *c2* in the endosperm. Seeds having colorless endosperms were assumed to have hyperploid embryos, those with pale purple endosperms were assumed to have diploid embryos, and those with dark purple endosperms were assumed to have hypoploid embryos. The hypoploid nature of the latter class of seeds was confirmed by the fact that they produced small, weak plants.

All 3 classes of plants had a *Tu* phenotype, although hypoploids were more severely affected than either diploid or hyperploid plants. The fact that *Tu* is expressed in hyperploids (*Tu/+/+*) indicates that this mutation reflects a gain-of-function rather than a loss-of-function, because a loss-of-function would have been compensated by the 2 wild type alleles in these plants. If this gain-of-function involved the overproduction of a wild type product, as would be expected of a duplication, hypoploid plants (*Tu/—*) should have a less severe *Tu* phenotype than their diploid or hyperploid siblings. This was not the case. The fact that wild type alleles reduce the expression of *Tu* (relative to the hypoploid condition) suggests that *Tu* may encode a product that antagonizes normal gene activity, but other alternatives cannot be excluded.

Scott Poethig

Corngrass: home again

Corngrass was originally mapped to the short arm of chromosome 3 by W. Galinat. Several years ago I obtained a stock of *Cg* which I subsequently used to try to locate the position of this mutation more precisely. These mapping studies demonstrated that the "*Cg*" mutation in this stock was unlinked to genes on 3S (MNL 58:170), creating some doubt about the actual location of *Cg*. I have since discovered that the mutation in this stock is tightly linked to *Tp1* on chromosome 7, and that the stock was originally derived from an outcross of *Cg* to *Tp1;* it seems likely, therefore, that the mutation I had been working with is *Tp1* rather than *Cg*. This conclusion is supported by the observation that the *Cg* mutation in the Coop's stock is linked to *h* on 3S (32% recombination, n = 136). A detailed linkage analysis is currently in progress.

A second dominant Corngrass-like mutation, which was discovered by J. Beckett, also maps to 3S. This mutation has a much less severe phenotype than *Cg*, and often displays poor penetrance. Although its most common effect is to increase the number of tillers, in some backgrounds it also causes an increase in node number, and the production of leaves in the tassel and ear. A detailed linkage analysis of this mutation is also in progress.

Scott Poethig

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Color in corn: natural factors

There may be ancient foundations for the multifaceted color systems that are so prominent in maize. All four teosinte species have white endosperm, no aleurone color and tan pericarp. If there were an ancestral, non-teosintoid wild maize which, after domestication, introgressed with one or more teosinte species (MNL 53:53-54), it could have had many contrasting alleles that controlled color in the endosperm, aleurone and the pericarp-cob systems. Yet other alleles could have been produced by the mutagenic action of transposable elements mobilized by the introgression.

Why might a wild maize have had an array of alleles distinct from those in the teosintes? Perhaps color was important in its reproduction, as an attractant to seed-eating birds! The husks might have been light red, membranous and loose, surrounding small red cobs holding kernels with yellow endosperm. The cob's glumes might have been especially red and partly visible between the loose kernels. Perhaps there was a slightly reddish or purplish tint in the pericarp and/or aleurone. Red and yellow are a common combination that attracts birds to flowers and fruits, but I can think of no other grass in which color acts as such an attractant.

What seem to be the oldest archaeological cobs, those from the Tehuacán area of Mexico, have very narrow rachillas (stems subtending kernels), and their lower glumes are very thin, short and reflexed away from the kernel (MNL 59:43). It would have been easy for birds to detach kernels, eating many, scattering some. But why would maize need to attract birds when presently they come in flocks to fields of much less colored maize? Perhaps small plants of ancestral wild corn were scattered along disturbed river banks and gravel bars, and attraction was needed to assure distribution of seed before the plants fell over with the ear still holding its load of 40-70 kernels. Birds would have helped to spread seed and to thin the potential stand in the following generation. After domestication Mexican farmers would have selected uncolored maize with tight husks to reduce bird damage.

This is theorizing with little evidence and much supposition, but it would be interesting to see if a maize combining these colors were to attract many more birds (grackles?) than would a white-kernelled, white-cobbed form. Preferably this would be tested in Mexico.

Robert McK. Bird

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The sweet butterscotch smell of *Zea*

We report that some progeny of crosses between *Zea luxurians* (Z.l.) and *Z. diploperennis* (Z.d.) smell like butterscotch. One of us (RMB) first noticed the odor on two F1 plants about to flower in the winter greenhouse at

Columbia, MO—the tassels being the source. These plants were transplanted outdoors in the spring where they flowered again with the same odor.

The following winter both plants were shipped to Raleigh, NC. One survived and continues to grow—and to give the strong odor of butterscotch from its tassel at flowering time. This plant (Neuffer 38:704.2) is the product of the cross of Z.l. (Neuffer 36:797-1, accession 759: derivative of USDA seed of *Z. luxurians* PI306615 collected near El Progresso, Jutiapa, Guatemala) by pollen parent Z.d. (Beckett 7931-1: Guzman 777 [original discovery], ex H. H. Iltis, ex Missouri Botanical Garden, collected at La Ventana, Jalisco, Mexico).

The second of us (SAM) determined by olfactoral examination through several flowerings that a butterscotch odor also exuded from the plant Hybrid #6 of the parentage Z.d. (Modena Mo81043/*100-1 : Guzman 1120, ex H. H. Iltis, collected east of Las Joyas, Jalisco, Mexico) by Z.l. (Modena G-36*6 : Iltis G-36, ex H. H. Iltis, collected west of Agua Blanca, Jutiapa, Guatemala). Like the above plant, the tassel odor was strong during flowering. The odor was stronger on the female spikes, leaf blades and especially the leaf sheaths. The plants (clones) were often damp at night or the early morning hours and the perfume was transferred to the hand with a touch. The essence is volatile. The odor was present for several weeks during the fall and the spring flowerings, but not between flowerings nor during the summer.

Several hybrid plants do not have this perfume at all. These are: Hybrid #3 of parentage Z.l. (Modena G-42*2 : Iltis G-42, ex H. H. Iltis, collected north of Ipala, Chiquimula, Guatemala) by Z.d. (Modena Mo81043/*100-3 : Guzman 1120); Hybrid #5 of parentage Z.l. (Modena G-36*3 : Iltis G-36) by Z.d. (Modena Mo81043/*100-3 : Guzman 1120); Hybrid #8 of parentage Z.d. (Modena Mo81043B : Guzman 1120) by Z.l. (Modena G-5*2 : Iltis G-5, ex H. H. Iltis, collected north of El Progresso, Jutiapa, Guatemala); Hybrid #9 of parentage Z.d. (Modena Mo81043A : Guzman 1120) by Z.l. (Modena G-5*1 : Iltis G-5). Neither of us has observed the odor on Z.l. plants, and the clones of the Z.d. parents do not have this odor.

We speculate that the butterscotch odor arises because of a secondary metabolite synthesized in hybrid plants. There may be several steps in the chemical synthesis of this metabolite, but the pathway suffers a synthetic or regulatory genetic block in each species—but at a different point. There may also be variation for several block points since only some combinations of Z.l. and Z.d. complete synthesis of the metabolite.

Robert McK. Bird and Stephen A. Modena

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Acp4 is the most distal marker on chromosome 1L

Table 1 shows data from 200 testcross progeny segregating for two morphological and two isozyme markers on chromosome 1L. We conclude that *Acp4* is clearly distal to *bm2*, thus becoming the most distal marker mapped on 1L. Previous data on recombination between *Dia2* and *Acp4* (J.F. Wendel et al., MNL 60:109-110) also suggest

Table 1. Testcross data.

Cross: gs, Phi1-4, bm2, Acp4-2 x gs, Phi1-4, bm2, Acp4-2
* , Phi1-5, * , Acp4-5

		SCO region *			DCO regions *		
	Parental Types	1	2	3	1,2	1,3	2,3
n = 200	65	11	5	12	1	0	1
	59	14	13	17	1	1	0
TOTALS	124	25	18	29	2	1	1
Recombination % (SE)		14.0 (2.5)	10.5 (2.2)	15.5 (2.6)			

*Regions 1, 2, and 3 correspond to the segments gs,Phi1, Phi1,bm2, and bm2,Acp4 respectively. No triple crossovers were observed.

that *Dia2* may be distal to *bm2*.

Estimated map distances:

gs - 14.0 - *Phi1* - 10.5 - *bm2* - 15.5 - *Acp4*

P.H. Sisco, J.F. Wendel and C.W. Stuber

The *Sod* genes of maize

The four SOD isozymes of maize are coded by the four nonallelic nuclear genes: *Sod1, Sod2, Sod3,* and *Sod4* (Baum and Scandalios, J. Hered. 73:95-100, 1982). The cytosolic isozymes, SOD-2 and SOD-4, and the chloroplast isozyme, SOD-1, are copper and zinc-containing homodimeric enzymes. The mitochondrial isozyme, SOD-3, is a manganese-containing homotetrameric enzyme (Baum and Scandalios, Arch. Biochem. Biophys. 206:249-264, 1981, Baum and Scandalios, Plant Physiol. 73:31-35, 1983).

To investigate these genes we have constructed a cDNA library from W64A scutellar mRNA. The cDNA library was screened with synthetic oligonucleotide probes complementary to the mRNA coding for the N-terminal of SOD-1, SOD-2, SOD-3 and SOD-4 proteins. Positive clones from this work have been characterized by restriction endonuclease analysis, partial DNA sequence analysis, *Sod2* has been completely sequenced (Cannon, White and Scandalios, PNAS in press), and hybrid select translation. Results indicate that *Sod2* and *Sod4* are similar in sequence, especially within the coding region. Most differences in DNA sequence between *Sod2* and *Sod4* occur in the third base of the codon and result in conservative amino acid replacement. The 5′ and 3′ noncoding regions of *Sod2* and *Sod4* cDNAs contain noticeable regions of similarity with shifts and minor changes of the DNA sequence. These results suggest that both *Sod2* and *Sod4* originated from a single progenitor *Sod* gene.

The mitochondrial associated *SOD-3* (Mn) of maize has been restriction mapped and partially sequenced. The restriction profile is different from the SOD-2 and SOD-4 cDNAs. Preliminary DNA sequence results for the 5′ end indicate that the protein contains a highly charged signal peptide of at least 25 amino acids.

Sod1 positive clones have been isolated and are in the preliminary stages of characterization. Additional collaborative work with Dr. T. Helentjaris, NPI, has shown the genomic location of the *Sod2* and *Sod4* genes to be on the short arm of chromosome 1 and the long arm of chromosome 7 respectively.

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A search for cytoplasmic restoration of genetic male sterility among regenerated plants and their progeny

The T cytoplasm undergoes changes during in vitro culture that result in a shift from male sterility to male fertility. The male fertile phenotype exhibits cytoplasmic inheritance and seems to be closely associated with an alteration in the mitochondrial genome (B. G. Gengenbach et al., Theor. Appl. Genet. 59:161-167, 1981). The reversion to male fertility represents a case of cytoplasmic restoration of male sterility because the revertant, male fertile plants should be *Rf1 Rf1 rf2 rf2*. This suggested to us that tissue culture may be a method of inducing cytoplasmic variants in the normal (N) cytoplasm that restore fertility in plants homozygous for one of the nuclear, recessive male sterile genes. If obtained, such variants would be useful in hybrid seed production by facilitating production of all-male sterile progeny containing normal cytoplasm.

Organogenic callus cultures were initiated from immature embryos heterozygous or homozygous for a known male sterile gene (Table 1). From 3 to 20 months after culture initiation, plants were regenerated and grown to maturity in the glasshouse or field. Male fertile, regenerated plants were self-pollinated. Progeny of these plants were grown in the field and scored for male fertility.

Table 1. Summary of cultures and regenerated plants containing male-sterile genes.

ms genotype	Culture age	No. cultures	No. regenerated plants
+/ms1	3-4	2	2
+/ms5	3-4	5	9
+/ms7	3-4	2	2
+/ms7	8-9	2	23
ms7/ms7	8-9	1	17
	12	1	14
	20	1	64
+/ms8	3-4	5	5
	8-9	3	15
ms8/ms8	3-4	2	3
	8-9	3	49
	12	2	33
	20	1	102
+/ms9	3-4	3	4
	8-9	2	16
+/ms10	3-4	4	4
	8-9	2	10
+/ms12	3-4	3	3
ms2/ms12	8-9	3	44
	12	1	10
	20	1	12
+/ms13	8-9	3	10
			459

Eleven tissue cultures were established representing 8 male sterile genes. Three cultures were apparently homozygous for the male sterile gene and only produced completely male sterile plants (348). Eight cultures were heterozygous for a male sterile gene and produced male fertile plants (111). Approximately 15% of these plants had high levels of aborted pollen (50-75%), indicating the presence of a chromosomal aberration presumably induced by the tissue culture process. Progeny of most male fertile regenerated plants segregated as expected (3 fertile:1 male sterile). Progeny of two regenerated plants had an excess of male sterile segregants and actually fit a 9:7 (fertile:sterile) ratio. Progeny of one regenerated plant did not segregate for male sterility. These plants were self-pollinated and male sterile segregants were observed in the subsequent generation. Overall, 459 regenerated plants were evaluated for cases of cytoplasmic restoration of male sterility in the normal cytoplasm. No cases of restoration were detected among these plants.

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Sodium azide as a tissue culture mutagen

Sodium azide is a potent seed mutagen (A. Kleinhofs et al., Mutation Research 55:165, 1978) that has been shown to be mutagenic in maize (R.W. Briggs, personal communication; K.A. Hibberd and C.E. Green, MNL 52:73, 1978). Azide has potential in tissue culture mutagenesis for inducing biochemical mutants because it is a point mutagen and is not known to be highly mutagenic in mammals. Azide mutagenicity in friable, embryogenic (type II) cultures was evaluated using selection for lysine plus threonine resistance described by Hibberd and Green (PNAS 79:559, 1982) as a model selectable marker. Two friable, embryogenic callus lines, designated B and S, were initiated from advanced generations of A188 × B73 crosses. Callus was incubated in citrate buffered azide solution, pH 4.45, for 1 h on a gyratory shaker. After washing, 0.5 g fr. wt. of callus was plated on a 7 cm Whatman #1 filter paper overlying non-selective medium. After 3 d recovery, the filter paper with cells was transferred to selective medium containing 3 mM lysine and threonine (LT). After 10 wk culture on LT medium, surviving colonies were scored as resistant variants (Tables 1 and 2). The LD50 azide for B and S callus lines was 1.1 and 2.5 mM, respectively. Even after adjusting the number of variants selected for azide toxicity, the recovery of LT resistant variants decreased with increasing azide. This may be because the plating procedure preferentially identified pre-existing, spontaneous variants which existed as relatively large cell aggregates at the time of plating. Any

Table 1. Effect of sodium azide treatment of B genotype friable, embryogenic maize callus on the numbers of selected LT resistant variants.

Azide Treatment	Variants a Selected	Predicted b 11 d Growth	Adjusted Variants/Plate
-mM-		-%control-	
0.0	31	101.8%	1.2
0.2	12	90.2%	0.51
1.0	4	55.7%	0.28
2.0	0	30.5%	0
4.0	0	0.6% c	0

a. Totals from two experiments of 6 and 20 plates taken after 10 wk on selective medium.
b. Predicted values from regression of dry wt growth vs. azide concentration.
c. Observed value, not predicted value.

Table 2. Effect of sodium azide treatment of S genotype, friable, embryogenic callus on the numbers of LT resistant variants selected.

Azide Treatment	Variants a Selected	Predicted b 11 d Growth	Adjusted Variants/Plate
-mM-		-% control-	
0.0	68	100.0%	3.2
0.2	17	95.8%	1.1
1.0	27	76.4%	1.8
1.5	8	66.3%	0.6

a. Taken after 10 wk on selective medium from 21, 16, 20 and 20 plates for 0, 0.2, 1.0, and 1.5 mM azide treatments, respectively.
b. Predicted values from regression of dry wt growth vs. azide concentration.

azide-induced mutants, existing as 1-8 cell colonies when selection was applied, may not have been selected due to decreased selection efficiency of the small cell colonies. The decrease in selected variants may have reflected the toxic effects of azide reducing the pre-existing population of LT resistant variants in our cultures and reducing the viable cell plating density. We are conducting further experiments to evaluate this explanation.

A more likely hypothesis is that azide was not highly mutagenic in the maize tissue cultures. Azide is known to be metabolically activated in barley embryos into the presumptive mutagenic metabolite, *o*-azidoalanine. To determine if maize callus synthesized the azide mutagenic metabolite, extracts of azide treated callus were compared with embryo extracts from azide treated B73 × A188 kernels for mutagenic metabolite synthesis using the Ames Mutagenicity Test. Free azide is volatilized in this assay so only levels of mutagenic metabolite were determined. Kernels were pre-soaked 8.5 h and then treated with 1 mM azide. Embryos were dissected and extracted for mutagenic metabolite. A 1 mM azide kernel treatment induced light green, albino, brown-spotted or yellow-gold stripes in 56% of the plants arising from the treated kernels indicating that somatic mutations were induced by azide treatment of embryo tissues. Callus was treated with 1 mM azide as before and sampled over time. Mutagenic metabolite levels 4 h after azide treatment were similar in callus and embryos (Table 3). Mutagenic metabolite levels declined in the callus to slightly above control levels of 20 h after treatment, which was before callus growth had resumed. Embryo metabolite levels remained high up to 40 h after azide treatment, by which time germination had resumed as indicated by the appearance

Table 3. Mutagenic metabolite in tissue extracts sampled at different times after sodium azide treatment detected by scoring reversion of his-*Salmonella typhimurium* strain TA1530. Data are averages of two plates for each of two treatment replications with the average spontaneous frequency of 44 revertants per plate subtracted. Extracts of untreated tissues induced 0 revertants above the spontaneous frequency for embryos and S callus.

		Hours After Azide Treatment		
Tissue	Azide Treatment	4	20	40
	-mM-	-His+ revertants-		
Embryo	1.0	1480	1800	980
S Callus	1.0	1160	42	35

and elongation of the radicle. Friable, embryogenic maize callus appeared to possess an active detoxification pathway, which is hypothesized to degrade the azide mutagenic metabolite before respiratory-arrested cells resumed growth and were mutagenized. The lack of evidence for azide mutagenesis at the Ltr loci in callus and the potent mutagenicity in kernels correlates with the decrease of mutagenic metabolite in callus tissue and higher levels persisting in embryos of treated seed, suggesting that azide mutagenesis is tissue specific in maize.

Using the filter paper selection procedure, 180 LT variant callus lines have been selected. Plants have been regenerated from 20 of the most promising lines and each line will be genetically characterized to determine if the trait can be recovered in the progeny of regenerated plants. Since selections were carried out in two different callus lines, we can be confident that mutants selected will represent at least two different genetic events. Multiple selections within the same line may not represent different mutations. Thus, to maximize selection of different alleles and genes conferring LT resistance in the absence of a mutagenic treatment, we suggest selecting among different callus lines to insure isolation of different spontaneous mutations.

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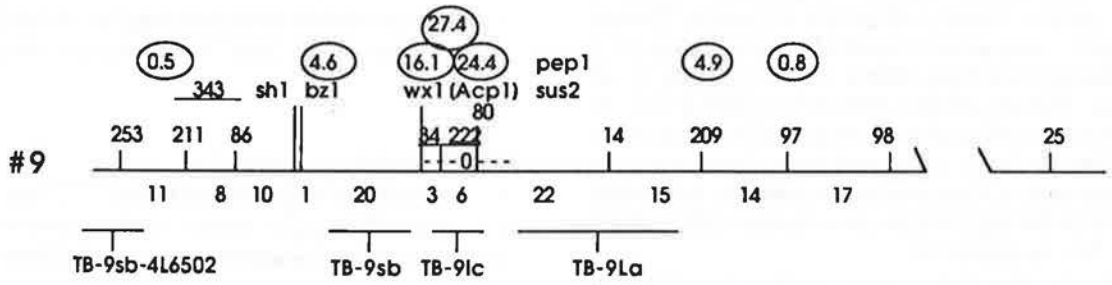
A strategy for pinpointing and cloning major genes involved in quantitative traits

The application of gene transfer technology to plant breeding suggests opportunities for exciting improvements, but the approach in general is limited to the transfer of single genes with obvious phenotypes. Presently, one can consider improving a cultivar through the incorporation of herbicide resistance and possibly some disease resistance genes, but this technology does not address those traits of greatest concern to breeders such as yield, standability, maturity, i.e. any trait exhibiting quantitative expression and inheritance. This limitation stems principally from our lack of understanding as to exactly how single genes influence complex traits as well as to a lack of methods to identify and clone such genes.

D. Robertson previously suggested (MNL 58:10-11, 1984 and 59:8, 1985) that different alleles at a single locus could result in a broad range of phenotypes. In other words, the level of expression or type of gene product produced from an individual locus can determine whether the isolate is recognized as a simply inherited, extreme phenotype mutant or as a variant for a trait with multigenic expression and inheritance. Therefore the same gene with different mutations could be identified as a "qualitative" locus but in other cases as a "quantitative" locus. He then hypothesized that the cloning of QTLs (quantitative trait loci) could be facilitated by recognizing this relationship and targeting those loci with similar but more extreme phenotypes. For example, cloning a gene corresponding to an extreme dwarf or defective kernel phenotype would then permit the isolation of other alleles at the same locus from lines with more moderate and useful alterations in plant height or seed yield characteristics. A difficulty with this approach is that although it does allow one to clone a QTL for a trait of interest, it does not predict whether that gene contributes any measurable effect to the trait in the lines of interest, i.e., is it a major gene?

We have recently explored a new method for identifying major QTLs, as well as testing whether QTLs can be associated with loci corresponding to extreme phenotype mutants in maize. Such a methodology would then facilitate cloning major QTLs through conventional transposon tagging. RFLP analysis (Nienhuis et al., Crop Science, in press) can be used to pinpoint major QTLs when examining quantitative traits, much as isozyme analysis has been previously used. The advantage here is that with our current RFLP map of maize (greater than 250 marker loci located on all ten chromosomes), we can systematically analyze individuals from segregating populations for their expression of the trait of interest as well as check all

Localization of a QTL for Plant Height



chromosomal segments for their genetic contribution to the trait. With these data, one can pinpoint major QTLs with respect to the locations of our RFLP marker loci.

In a preliminary experiment supportive both of Robertson's hypothesis and this strategy, we have examined several quantitative traits by both isozyme and RFLP analyses in collaboration with C. Stuber and M. Edwards at N.C. State. In an examination of a segregating F2 population derived from the cross Tx303 × Co159, several major QTLs for plant height were identified. In the attached figure, we show the results with several markers on chromosome 9 and their genetic contribution to overall plant height (denoted in circles above the marker loci as the percent variance accounted for by them). The advantage of multiple markers is obvious here as one can scan up and down the chromosome to find the marker which accounts for the most variance for plant height and presumably is located closest to the actual QTL, in this case the isozyme, *Acp1* and RFLP #222. We would therefore pinpoint this major QTL, which accounts for at least 27% of the variance in plant height, as being located near the centromere and of interest, also very close to the known GA dwarf locus, *d3*. If one could confirm that this proximity was not just coincidence, then this relationship could be exploited to clone the *d3* allele through transposon tagging and subsequently obtain related alleles from other lines which might function as major genes for plant height in lines with less extreme variance. Interestingly, this type of analysis allows one to deduce that the QTL for shortened plant height near *d3* is recessive in gene action as also is *d3*.

This type of analysis can identify the genomic location of those genes accounting for the most variance for a particular trait, which we feel are most likely to be "rate-limiting" at least in the particular lines examined. Consequently changes in gene expression at that locus are most likely to result in changes in the overall phenotype and we believe these genes would therefore be the best candidate genes to clone, alter, and transfer to effect plant improvement. Hence one might be able to significantly affect quantitative traits through the introduction of single genes. An attractive precedent here is the result of Palmiter et al. (Science 222:809-814, 1983), who were able to obtain a much greater than naturally observed variation in body size, an obvious quantitative trait, after introduction of a single altered growth hormone gene into mice.

We cannot overemphasize the importance of the conventional maize map with all of its mapped morphological markers in the successful application of this approach. As we improve the correlation of our RFLP linkage map with the conventional map, major QTLs can first be identified as tightly linked to RFLP markers and then subsequently to extreme mutant phenotype loci located on the conventional map. Obviously for the purposes of this approach, one should not discontinue efforts to map morphological markers and we should in the future stress the further correlation of the conventional and RFLP linkage maps.

Tim Helentjaris and Donna Shattuck-Eidens

RFLP mapping of cloned genes

Through the use of restriction fragment length polymorphisms (RFLPs), an extensive genetic linkage map of the maize genome has been constructed (Helentjaris et al., PNAS 83:6035, 1986). Our most current version of the map consists of approximately 300 loci detected through the use of RFLPs. This includes 21 clones of known identity, many of which were previously unmapped as to chromosomal location. The following is a list of these identified clones, their approximate chromosomal location, and the individuals who provided them.

Identity	*Locus*	*Chromosome*	*Provider*
Anthocyaninless	*51-A1*	3L	H. Saedler
Actin[a]	*368-Act1*	8[b]	R. Meagher
Alcohol dehydrogenase-1	*21-Adh1*	1L	M. Freeling
Alcohol dehydrogenase-2	*228-Adh2*	4S	M. Sachs
Aleurain (barley)[a]	*348-Alr1*	2L	J. C. Rogers
	349-Alr2	7L	
Booster (presumptive)	*248-B1*	2S	V. Chandler
Bronze-1	*8-bz1*	9S	D. Furtek
Endogenous Mu	*347-EMu*	2[c]	V. Chandler
Heat shock protein (70Kd)	*119-Hsp1*	8L	D. Ho
NADP malic enzyme[a]	*231-Me1*	3S	P. Collins
	330-Me2	6L[b]	
Pericarp color	*370-P1*	1S	T. Peterson
Phosphoenol Pyruvate Carboxylase	*332-Pep*	9L	B. Taylor
Phytochrome (barley)[a]	*251-Phy1*	1L	P. Quail
	369-Phy2	5S	
Pyruvate, Pi dikinase[a]	*229-Pdk1*	6L	P. Collins
	230-Pdk2	8[c]	
Ribulose Bisphosphate Carboxylase (small subunit)[a]	*227-Ssu1*	4L	B. Taylor
	331-Ssu2	2[c]	
Shrunken	*15-sh1*	9S	B. Burr
Sucrose synthase-2	*121-Css1*	9L	L.C. Hannah
Superoxide dismutase-2	*419-Sod2*	7L	R. Cannon
Superoxide dismutase-4	*412-Sod4*	1S	R. Cannon
Triosephosphate isomerase[a]	*345-Tpi4*	3S	M. Marchionni
	346-Tpi5	5S[b]	
	344-Tpi3	8S	
Waxy	*16-wx1*	9S	N. Fedoroff

"a" = Indicates duplicated loci; that is, individual clones detect more than one fragment, which have been mapped to different locations

"b" = Locus has been assigned to chromosome, however, linkage data not available

"c" = Located near centromere

Once an RFLP linkage database has been established, identifying the genomic location of previously unmapped sequences can be accomplished quickly and easily. The use of unmapped as well as conventionally mapped sequences of known identity is of great utility in developing an RFLP linkage map. They establish a correlation of RFLP linkage groups to the conventional maize map and in many cases, an orientation to the chromosomal arms. Anyone who is currently generating clones of known identity and would be interested in having their genomic location determined, please feel free to contact us*.

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Sequencing of mtDNA related to a recombination event involved with male fertility and/or toxin resistance in T cytoplasm

Restriction enzyme analysis of mitochondrial DNA (mtDNA) from normal (N), cytoplasmic male sterile (cms-T) and fertile revertants (regenerated from callus from immature embryos of cms-T) has identified a 6.6 Kb *Xho*I fragment that is unique to cms-T mitochondrial genome with only one exception (Brettell et al., Theor. Appl. Genet. 58:55, 1980; Gengenbach et al., Theor. Appl. Genet. 59:161, 1981; Umbeck and Gengenbach, Crop Sci. 23:584, 1983).

Comparative studies of the 6.6 Kb *Xho*I fragment from the cms-T mtDNA and the regions homologous to this fragment in the N genome and a fertile revertant, V3 (WF9T/W22 × A188Nrf × W22rf), provided us a clue to the molecular basis of male sterility and/or T-toxin sensitivity in T cytoplasm.

Southern analysis of cosmid clones containing the 6.6 Kb *Xho*I fragment from cms-T mtDNA and homologous regions from N and the fertile revertant V3 mtDNA show that a number of events are necessary to explain the formation of the 6.6 Kb *Xho*I fragment in cms-T and its subsequent loss in the fertile revertant. This includes the formation and the loss of a 4 Kb repeat and at least two independent recombinational events (Fauron et al., Curr. Genet., in press). The region of interest could be narrowed down to a 1.5 Kb *Ava*I fragment internal to the 6.6 Kb *Ava*I fragment. The 1.5 Kb *Ava*I fragment contains part of two reading frames, ORF13 and ORF25, identified by Dewey et al. (Cell 44:439, 1986). This 1.5 Kb *Ava*I fragment hybridizes to a 2.1 Kb *Ava*I fragment in N and V3 (Abbott and Fauron, Curr. Genet. 10:777, 1986). Fine mapping of the 2.1 Kb *Ava*I fragments of N and V3 shows that the two are almost identical (Fauron et al., in press). The data suggest that the two parts of a DNA sequence in N, that have been split and dispersed in T mtDNA, are brought back together through a recombination event in the V3 mtDNA, the fertile revertant.

Sequence analysis of those two 2.1 Kb *Ava*I fragments and the cms-T specific 1.5 Kb *Ava*I fragment has enabled us to localize the recombination site. The break point is

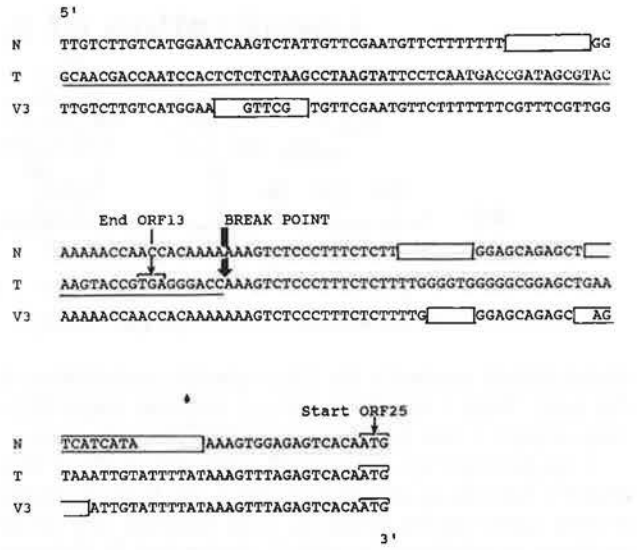

Figure 1. The nucleotide sequence of homologous regions of maize mtDNA from N, cms-T, and a fertile revertant, V3, showing the point of sequence divergence between T and N and V3. The underlined sequence is unique to cms-T. The "boxed" sequence represents the difference between N and V3.

located 6 bp past the ORF13 stop codon (Figure 1). Upstream from this point, the T sequence represents the 3' end of the ORF13 unique to cms-T. It is interesting to see that the recombinational mechanism that gave rise to the 2.1 Kb *Ava*I fragment in V3 has reconstructed an exact N-like sequence at the break point. Between the break point and the beginning of ORF25, the region seems very unstable as seen by the many changes between the 3 genomes.

Another fertile plant, V32, regenerated from a different embryo [WF9T/W22 × A188Nrf × A188Nrf], was sensitive to T toxin and produced four seeds [A,B,C,D] of which A gave a partly sterile and toxin sensitive plant and D a fertile toxin resistant plant. Interestingly V32A, V32B mtDNA contains the 1.5 Kb *Ava*I fragment while the V32C, V32D mtDNA contains the 2.1 Kb *Ava*I fragment.

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Delayed pollen development in maize × *Tripsacum* hybrids

Eight maize lines and hybrids were used as female and crossed by tetraploid *Tripsacum dactyloides* L. "Huey". From these crosses, five maize stocks [Strawberry Pop, Ladyfinger Pop, 311255, Aho (Alexander High Oil), and Mo20W/Aho] showed good seed set (over 50 viable kernels per ear). The other three stocks, Tom Thumb, Mo20W and a synthetic tetraploid, showed normal early kernel development but gave nonviable, collapsed mature kernels. Viable kernels from the above crosses were germinated, root tips were fixed for chromosome counting, and seedlings were transplanted into 10 inch pots. Fresh young and mature anthers from these plants were examined by the acetocarmine-haematoxylin staining technique. The results are summarized as follows:

1. Exposed mature anthers failed to dehisce; no pollen shedding was observed.
2. Microspores from single, fresh young and mature anthers showed clear size variation.
3. In most cases, microspores from single mature anthers were aborted or degenerated, as determined by their non-staining cell contents and transparent appearance.
4. Development after meiosis was delayed and developmental differences among the microspores were observed. These variations occurred at various developmental stages during pollen development.
5. After meiosis, the genomes of maize and *Tripsacum* may not fuse together to form a single nucleus. Microspores that carry one large and one small nucleus are frequently seen. This phenomenon may represent a tendency of maize and *Tripsacum* chromosomes to fail to aggregate after division.
6. After meiosis, microspore development can be classified as (a) uninucleate, (b) first mitotic stage, (c) binucleate, (d) second mitotic stage, (e) trinucleate, or (f) mature pollen. From one young anther, the following counts were obtained:

   1000 aborted or degenerated
   11 tiny aborted or degenerated
   40 uninucleate (a)
   7 binucleate (c)

   In addition to aborted or degenerated spores, the following counts were obtained from a mature anther:

   62 uninucleate (a)
   26 first prophase (b)
   14 first metaphase (b)
   16 first anaphase (b)
   5 first telophase (b)
   5 binucleate (c)

   No microspores reached the second mitotic stage in either anther. Therefore, no mature pollen is present when the anthers are mature.
7. Microspore degeneration and delayed microspore development cause complete male sterility in maize × *Tripsacum* hybrids.
8. No clear starch accumulation was observed in the microspores, but many oil-like droplets of various sizes accumulated in the cytoplasm.

Degeneration and delayed development of the microspores can be attributed in part to unequal distribution of genetic material after meiosis. The hybrids carry 46 chromosomes (10 from maize and 36 from *Tripsacum*), which represent one complete set of maize chromosomes and two complete sets of *Tripsacum* chromosomes. Although they carry two different sets of metabolic assembly lines, F1 plants can survive and grow even though these assembly lines may be working independently. During meiosis, the maize chromosomes are divided randomly between two daughter cells (or lost). The frequency at which a single cell receives a complete set of maize chromosomes should be $(1/2)^{10} = 1/1024$ (or about 0.1%). However, all the cells will receive one complete set of *Tripsacum* chromosomes. Therefore, all the meiotic products should function properly. In our experience, no single microspore reaches maturity, which may imply that unequal distribution of maize genetic material may cause high interference with normal metabolic processes during microspore development.

A significant goal in the study of pollen development is finding genes that control microspore degeneration (West and Albertsen, MNL 59:87) or cause delay in development. Such results can help us understand the genetic control of male sterility.

Ming-Tang Chang and J.B. Beckett

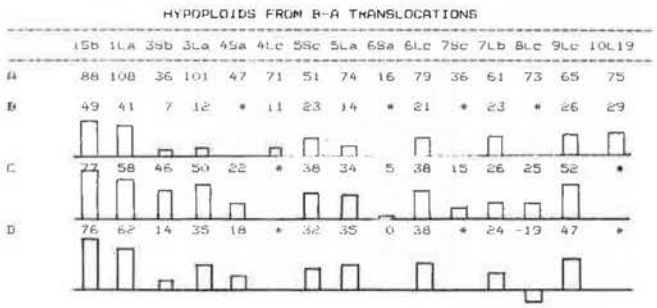
Genetic effects of hypoploidy on kernel weight, plant height and leaf width

Maize hypoploids are derived from non-disjunction of the B-A chromosome from a B-A translocation stock. In a genetic sense, a hypoploid has part of one chromosome arm missing, and is hemizygous for that particular arm segment. Preliminary study showed that a B-A translocation per se will not have any significant effect on kernel weight, plant height, and leaf size. This result suggests that the B chromosome and the break point of the translocation will not have a critical deleterious effect on proper genetic function of the genome. But in hypoploids, when one chromosome arm is missing, different chromosome arms show different effects on kernel weight, plant height, and leaf size. Our studies show that the degree of effects is not highly correlated with the length of chromosome arm fragment, but is highly dependent on the missing arm. In other words, certain chromosome arms are more critical than others for the expression of those quantitative traits. This implies a non-random distribution of genes that are important for the expression of a quantitative trait. It also implies clearly that genetic dosage effects can be immediately observed, if gene doses are altered.

Materials used for this study came from progenies in an ongoing project for converging TB's to inbred lines. TB-carrying pollen parents at the BC3 level with A619 were crossed onto genetic testers for identification of hypoploids. Samples of 100 kernels were weighed and the percent reduction for hypoploid versus sib endosperms was calculated. Hypoploid plants were identified in the field and plant height and leaf width were measured. The physical arm length missing (Table 1, A) was calculated from data on breakpoints (Kindiger et al., MNL 60:50) and arm lengths (Maize for Biological Research, p. 52).

Results showed that the hypoploids of TB-1Sb, TB-1La, TB-5Sc, TB-7Lb, TB-9Lc and TB-10L19 have substantial effects on kernel weight (Table 1, B). A 0.48 correlation coefficient between physical chromosome arm length and

Table 1. Genetic effect of hypoploidy on kernel weight, plant height, and leaf width. A, physical arm length that is missing; B, percent kernel weight reduction; C, percent plant height reduction; D, percent leaf width reduction. Physical arm length is calculated by arm length times percent of arm; for example, hypoploid 1Sb = 92.53 × 0.95 = 88.
*** = Data not available.**



kernel weight was obtained, which implies that kernel weight reduction was not directly proportional to physical arm loss. The plant height of hypoploids is consistently smaller than that of their normal sibs, but TB-1Sb, TB-1La and TB-9Lc have greater effects than others on plant height (Table 1, C) and on leaf width (Table 1, D).

Plant and leaf color of the hypoploids is also variable. Hypoploids for TB-7La and TB-8Lc are dark green, TB-7Sc and TB-9Lc are yellow green, and those for TB-1Sb, TB-3Sb, and TB-5La are pale green. The leaf size and leaf shape of the hypoploids are modified also. TB-1Sb, TB-5Sc, TB-5La, and TB-6Lc have long, narrow leaves, TB-1La has short, narrow leaves, TB-4Sa and TB-8Lc have short, broad leaves, TB-3La and TB-7La have short, erect leaves.

Based on these preliminary results, it is evident that certain chromosome arms show a positive correlation of their genetic effects on kernel weight, kernel size, leaf width, and plant height. The long and short arms of chromosome one are very critical for normal kernel, normal leaf, and normal plant development. Since these preliminary results were based on F1 materials from crosses on different testers, the background and environment may have great effect on the phenotypic expressions. Also, the materials that were screened came only from one inbred line, and the hypoploid phenotypes cannot be used for a generalized description. In any event these results show that certain important quantitative traits are influenced by dosage of certain chromosome arm regions, and these regions are important for crop characters and their improvement.

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Metroglyph analysis of morphological variation in maize grown in Kashmir

The present investigation attempts to classify 15 local maize varieties (Table 1) into morphologically distinct complexes. The results of the metroglyph analysis are presented in Figure 1 while class intervals and index values for 12 characters appear in Table 2.

The varieties could be separated into 3 distinct complexes (Fig. 1). Complex I is comprised of 2 varieties (Table 1) having orange flint grains. One of the varieties (Tchi II) possessed one short ray while Voz. IA was without any ray.

Table 1. Source and grain type of various local maize collections.

Variety	Source	Grain type
1. Tchi II	Shopian	Flint orange
2. Voz.IA	Pahalgam (Ganeshbal)	Flint orange
3. Tchi I	Pahalgam	Flint white
4. Tchi VI	Baramulla	Flint yellowish white
5. Badh IIA	Shopian (Out-skirts)	Flint orange
6. Badh IIB	Shopian (Out-skirts)	Flint white
7. Badh IV	Pulwama	Flint orange
8. Voz.IB	Pahalgam (Lidroo)	Flint orange
9. Niv IVA	Pulwama (Out-skirts)	Flint orange
10. Mish IIC	Shopian (Out-skirts)	Dent white
11. Fero VB	Tangmarg (Ferozpur)	Dent white
12. Tchi III	Mahind	Flint orange
13. Badh V	Tangmarg	Dent white
14. Niv VA	Tangmarg (Outskirts)	Flint orange
15. Kani IVB	Pulwama (Out-skirts)	Flint white

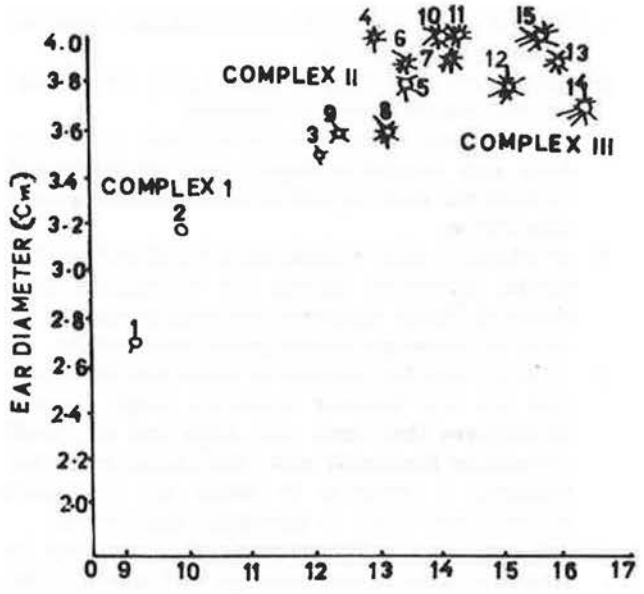

FIG.1. METROGLYPH DIAGRAM IN MAIZE

Table 2. Class intervals and index values for 12 characters of maize in Kashmir province.

Characters	Index Values 1		2		3	
1. Number of leaves/plant	11.8	♂	11.8 - 13.5	♂	13.5	♂
2. Plant height (cm)	150.0	♂	150.1 - 169.9	♂	170.0	♂♀
3. Leaf-index (cm)*	40.0	♂	40.1 - 60.0	♂	60.1	♀
4. Number of tassel branches/plants	16.0	♂	16.0 - 22.0	♂♀	22.1	♀
5. Shelling %age	65.0	♂	65.1 - 73.0	♀	73.1	♀
6. Ear height (cm)	60.0	♂	60.1 - 80.0	♀	80.1	♀
7. Ear length (cm)	12.0		12.1 - 14.0		14.1	
8. Ear diameter (cm)	3.0		3.1 - 3.7		3.7	
9. Number of ears/plant	1.5	♂	1.5 - 1.8	♀	1.8	♀
10. Grain moisture (%)	28.0	♂	28.1 - 34.5	♂	34.5	♂♀
11. Days to 75% silking	81.0	♂	81.0 - 87.0	♂♀	87.1	♀
12. Tassel length from flag-leaf (cm)	38.0	♂	38.1 - 44.0	♂	44.1	♂

The main characteristics of this complex were minimum number of leaves and tassel branches per plant; low grain moisture, tassel length from flag leaf, plant height, leaf index, shelling percentage and ear height. These varieties also required less days to 75 percent silking and had medium number of ears per plant.

Complex II is comprised of 9 varieties out of which Mish IIC, Fero VB had dent grains and the other 7 had flint grains. The grain colour varied from white (55.5%), yellow white (11.2%) to orange (33.3%). The varieties of this complex possessed a minimum of 3 rays each and were characterized by having low plant height and a minimum number of leaves and ears per plant. The rest of these characters were classed as medium.

Complex III contained 4 varieties. Excepting Badh V all had flint grains which were either white or orange in colour (50% each). Each variety was represented by a minimum of 9 rays. Except for higher percentage of grain moisture at harvest, varieties within this complex possessed medium characters.

Comparison of complexes revealed similar behaviour between Complexes II and III for leaf-index, number of tassel branches per plant, shelling percentage, tassel length from flag leaf and number of ears per plant whereas Complexes I and II and Complexes I and III were similar for number of ears per plant. Complex II emerged as an intermediate complex between Complexes I and III. Collections in Complex I had flint grains whereas both flint and dent grain types appeared in Complexes II and III.

The varieties that do not have flowering time overlapping with others are bound to retain their identity as no gene exchange will take place from one population to another. In the present study Tchi II is a pertinent example to quote. This variety was distinctly more early maturing than the rest of the Tchi varieties in the first place and secondly than all other varieties included in the present study (excepting Voz. IA). This variety is represented on the glyph only by 1 ray while all other Tchi varieties have more than 2 rays. Variety Voz. IA was without a ray (Complex I) thereby showing that this variety is closer to Tchi III. Incidentally the latest maturing variety was also from the Tchi group, i.e., Tchi III (Complex III), which was later flowering than 10 of the 15 varieties. The next early flowering variety was Badh IIA (Complex II), which was significantly earlier than 11 of the 15 varieties. Tchi III and Badh IIA (latest and next early) did not show a consistent significant difference either from their own groups or the rest of the varieties in all the characters (excepting number of leaves per plant). It may be due to the fact that some gene exchange did take place through some late flowering plants of early flowering populations and early flowering plants of flowering populations. Also, early maturity varieties (Tchi II and Voz. IA) have minimum ear length (9.1 cm) and ear diameter (2.7 cm) and required less days to silk, while the late maturing varieties (Tchi III, Badh V, Badh IV, Badh VA and Kani IVB) had maximum ear length (16.2 cm), ear diameter (4.0 cm) and required more days to silk.

Early maturing hybrids are generally adapted to semiarid agriculture. From the present study this picture emerged from the maturity of Tchi II and Badh IIA. It is worth mentioning here that maize in Kashmir is generally a rain fed crop, more particularly in areas like Pahalgam, Shopian and Tangmarg. Both of the above mentioned varieties that were early maturing were from one such area, i.e., Shopian. The latest maturity variety, i.e., Tchi III from Mahind, was observed at the time of collection to be growing totally under irrigated conditions.

P.N. Jotshi, B.K. Bhat and M.K. Bhan

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MMS induced aneuploidy in corn roots

Kernels were imbibed in distilled water for 24 hr, then treated with 3×10^{-3} M methylmethanesulphonate (MMS) for 1, 2 or 4 hr separately. Anaphase bridges and/or fragments were found 48-96 hr from the end of treatment (Table 1) and even after 96 hr recovery from 4 hr treatment, 11.8% anaphase configurations were seen to be aberrant. These are chromatid changes, present in meristems many of whose cells have undergone 2-3 cycles since MMS treatment. Evidence that at least 2 cell cycles have occurred since treatment is this: i) metaphases/anaphases with non-congressed chromosomes were present 44 hr after treatment; ii) 24-72 hr later metaphases with 17-22 chromosomes, i.e. numerical aneuploids, were present (Table 2). The aneuploid cells had undergone a replication cycle and were dividing again. The presence of 2n + 1 and 2n-1 metaphases, in approximately equal numbers, con-

Table 1. Frequencies of metaphases and anaphases with bridges/fragments of non-congressed chromosomes 54 and 72 hr after treatment with 3×10^{-3} M MMS for 1, 2 or 4 hr. Data other than 54 and 72 hr after treatment are not included.

hr after MMS	hr in MMS	metaphase number scored	metaphase %with non-congressed Chromosomes	anaphase number scored	anaphase %with fragments/ bridges
54	1	148	11.5	100	6.0
	2	132	9.8	112	7.1
	4	128	14.8	98	9.2
72	1	168	7.7	125	5.6
	2	112	7.1	102	7.8
	4	124	12.9	116	16.4

Table 2. Numbers of aneuploids at metaphases 96 hr after treatment with 3×10^{-3} M MMS with aneuploid complements. A total of 268 metaphases were scored.

Chromosome Number	22	21	20+frag.	19	19+frag.	18	17
No. of aneuploid at metaphases	3	13	1	15	1	3	3

firms that the cells with non-congressed chromosomes divide again, and accordingly they may be capable of initiating stable lineages of aneuploid cells. Many reports emphasize one particular advantage of chemical mutagens; i.e., they induce high frequencies of point mutations and low frequencies of chromosome aberrations. The opposite result occurs with ionizing radiations. The results from the present study show that chromatid aberrations and chromosome non-congression are frequent after MMS treatment. This response may be specific to root meristems and may involve a stage that is particularly sensitive to MMS; i.e., the formation of alkylated bases such as 0^6-methylguanine in DNA of meristematic cells as they are proceeding from G1 into S of the first cell cycle in the root of germinating corn. Continued formation, over 2-3 cell cycles, of cells with aneuploid complements means that the root meristems are, at least for some days, complex chimeras. Some complements will lead to cell death or to failure to proliferate: this will result in diplontic selection and therefore to a reorganization of meristems. Cells capable of initiating lineages will be able to repopulate disrupted meristems and to produce new growth. The chromosome changes seen in meristems 2-3 cell cycles after treatment provide a basis for diplontic selection; for the establishment of a new meristem from, perhaps, a few initial cells.

Ji-Ping Zhao and Douglas Davidson

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Linkage between silk browning and cob color

Previous issues of MNL have reported an association between the browning of silks at the tips (5-10 minutes after they are cut) and red cob color (MNL 59:40) and *P* locus control of silk browning (MNL 60:55). The results of tests we have conducted suggest a very close linkage relationship between factors for cob color and silk browning (<2% crossovers).

We crossed a red-cob silk browning line (T204) with a white-cob green-silked line (NC34) and observed segregation in the F2 and first backcrosses. The backcross to T204 was all red-cobbed with browning silks, but the F2 and backcross to NC34 segregated about 3:1 and 1:1, respectively, for both red cobs and silk browning. The white-cob silk browning, and red-cob green silk classifications represented about 1.7% of the progeny in each of the F2 and BC2. Each generation was represented by at least 350 plants when summed over two years and no classification within generations contained fewer than two individuals.

These data clearly suggest two separate, but closely linked, dominant factors in control of red cob color and silk browning.

N.W. Widstrom

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A new B-A translocation: TB-2Sa

A translocation between a B chromosome and the short arm of chromosome 2 bearing the *B-Peru* allele has been recovered and designated as TB-2Sa. When homozygous for an *r* seed-color allele the hyperploid progeny are easily identified by their deeply colored scutella and colorless aleurones. Because no *wt* seedlings were observed (0/29) among the hypoploid progeny of a cross of *gl2 wt, r-g* × hyperploid TB-2Sa *r-g*, the breakpoint of the translocation probably lies between the *B* and *wt* loci. This translocation should prove useful in some studies of the distal half of 2S because the only other B-2S translocation available to date is an unmarked compound translocation involving 3L.

Thirty-seven *B-Peru r-g* multi-B-chromosome plants, whose tassels were just beginning to emerge from the whorls, were treated with 500 to 594 r of gamma irradiation given at a rate of 0.95 to 1.13 r/sec. Irradiation was accomplished by hanging the potted plants upside-down next to a retractable in-air ^{60}Co source at the University of Missouri Research Reactor. Pollinations were made from the irradiated plants onto various *r-g* testers 5 to 10 days after irradiation. The plant that gave rise to TB-2Sa received 500 r at a rate of 0.95 r/sec and was used as a pollen source six days after irradiation.

Among the 53,770 treated F1 progeny 234 TB-2S candidates were selected on the basis of their colorless aleurones and colored scutella. The selected F1's were planted in flats and screened for germination (205 germinated) and viability. Of the 143 individuals transplanted into the field, 64 were tested by crossing onto a *gl2 wt, r-g* tester. Only eight of those tested segregated for more than one non-corresponding (colorless aleurone/colored scutellum) kernel each and are considered to be likely candidates for TB-2S lines marked with *B-Peru*.

Further testing has only been performed on one of the candidates, TB-2Sa, which gave 2 kernels with colored scutella out of 45 with colorless aleurones from the F1 × tester cross. Both of these kernels were planted and crossed again onto *gl2 wt, r-g*. Only one of these crosses resulted in segregation of non-corresponding kernels. Out of the 304 kernels from that one cross (179 colorless and 125 colored aleurones) there were 37 hyperploid and 29 hypoploid (colored aleurone/colorless scutellum) kernels. The 37 hyperploids are being reserved for stock increases and further mapping of the breakpoint. Sandbench testing of the 29 presumptive hypoploids resulted in 13 *gl2* and no *wt* seedlings. Thirty of the colored/colored euploid kernels were scored in the sandbench and 5 were *gl2* and none were *wt*. The presence of a few *gl2* seedlings in the fully colored euploid class is thought to be due to crossing-over between *B-Peru* on the translocated chromosome and *gl2* on the normal homolog. The distance between *B* and *gl2* is 19 map units.

The initial idea for this experiment was provided by Dave Hoisington, the stocks were provided by Jack Beckett and Ed Coe, and the gamma-rays were provided by Vicki Spate and all of the other fine folks at MURR.

Craig Echt

Factors affecting expression of *Les1* in leaves

Last year it was reported that light is a required factor for leaf lesion induction in *Les1/Les1* plants (C. Echt, MNL 60:49). Through a series of light and temperature shift experiments the time-course, and the physiologically separable stages, of light-induced necrosis of *Les1/Les1* leaf tissue now have been determined. The term 'light-induction' is defined as the process of exposing dark-adapted leaf tissue to light for a length of time sufficient to induce necrosis of that tissue in *Les1/Les1* plants.

The time course of light-induced necrosis proceeds as follows (at 25 C): the first 2 hours of exposure to light represent a lag phase during which time induction can be reversed by returning the leaf section to the dark; the time from 2 to 6 hours after exposure is when an increasing proportion of the cells become irreversibly set to die; necrosis occurs over a period of time from 8 to 20 hours after exposure. The observed family-to-family variability in the timing of the various stages is presumably due to segregation of unidentified loci that modify *Les1* expression.

The three stages of light-induced necrosis that have been determined so far are (1) development of lesion-forming potential in the maturing leaf, (2) induction, and (3) necrosis. Each of these stages has a different light and temperature requirement. Lesion-forming potential is independent of light and temperature and appears to be determined solely by the age of the leaf. Induction generally requires light and has a temperature optimum of around 25 C. Temperatures between 30 C and 35 C during the induction period decrease the necrosis response by 90-95%. Necrosis is independent of light and is inhibited by temperatures over 30 C.

An "action spectrum" of light-induction was determined with the use of monochromatic light filters obtained from Carolina Biological Supply Co. White light is most efficient for induction. Induction did not occur with blue light (350-550 nm, peak at 450) and was reduced by about 90% with red light (550-750 nm, peak at 650). Far-red light (650-750+ nm, max. at >750 nm) does not inhibit the induction response elicited by white or red light. It thus appears that light induces lesion formation by a general stimulation of photosynthetic activity. The active photosynthetic product(s) must be translocatable, however, because lesions can form within sectors of white leaf tissue bordered by green tissue (Hoisington, MNL 60:57) and, on occasion, within dark-adapted leaf sections prior to exposure to light (C. Echt, MNL 60:49).

Several experiments suggest that translational and transcriptional events may be involved in lesion formation. It was found that cycloheximide (50-200 μM) is effective at inhibiting light-induced necrosis when administered through cut leaf tips for 2 hours prior to, and during, a 7 hour light-induction period. Light-induced necrosis is also prevented when a 6 hour light exposure at 25 C is preceded by a 4 hour light exposure under heat shock conditions (42 C, 100% R.H.). In contrast, necrosis proceeds normally when a 6 hour exposure at 25 C is preceded by a 4 hour exposure at 30 C.

These observations are consistent with the hypothesis that a translocatable photosynthetic metabolite is involved in the induction of the *Les1* phenotype and that the expression of *Les1* may require the activation of a specific gene or genes.

Craig Echt

Root lesions in *Les1/Les1* seedlings

Production of localized tissue necrosis by the *Les1* allele occurs not only in the leaves and leaf sheaths of *Les1/Les1* plants but apparently also in the roots. Sibling comparisons were made between 27 *Les1/Les1*, 12 +/*Les1* and 38 +/+ 10- to 12-day-old seedlings from several different F2 families. *Les1/Les1* seedlings exhibited small areas of tan discoloration and cellular collapse along the primary roots and in the central portions, or at the points of attachment, of some of the adventitious branch roots of the primary and lateral roots. The roots of all +/+ and most +/*Les1* plants were unblemished.

The lesions on the primary roots were 2-5 mm long and about 1 mm wide. Lesions on the branch roots covered the circumference of the root and were 2-4 mm long. When lesions were present at the base of the branch roots they occasionally extended into a patch on the primary or lateral root.

The extent of lesion expression was variable. Some *Les1/Les1* root systems had a decidedly brown appearance, while on others only a few lesions could be found. Whether this variation is genetic, physiological, or environmental is not known. Lesion expression on +/*Les1* roots, when it was found, was always low. The severity of root-lesion expression did not appear to be correlated with the severity of leaf-lesion expression. This lack of correlation between expression in leaves and roots suggests that root lesions are not simply a physiological result of leaf-lesion formation. It also indicates that *Les1* expression may be controlled by different environmental or physiological factors in different tissues.

The plants were grown in 8″ × 1.5″ (top dia.) conical plastic containers (Super-cell Cone-Tainers) filled with Pro-mix soil mixture. Following germination at 30 C, the seedlings were placed in growth chambers set for 14h 25 C days and 10h 20 C nights with an irradiance of 350 uE/m^2/sec. *Les1* genotypes were scored on the basis of *wt1* expression (*wt1* is a closely linked marker) or on the basis of relative severity of leaf-lesion expression. The roots were exposed intact by removing the soil plugs from the containers and separating the roots from the soil in a basin of water.

Craig Echt

Low frequency transmission of *Les1 wt1* gametes

Lesion expression in *Les1* homozygotes is earlier, more uniform, and more severe than in *Les1* heterozygotes. Consequently, it is preferable to use homozygous material for biochemical and physiological investigations of *Les1* expression. One can actually score *Les1* homozygous seedlings before lesion expression is initiated with the aid of the closely linked recessive marker *wt1* (white-tipped first and second leaves) (Hoisington, MNL 60:51). Such marked stocks are generated simply by selfing or intercrossing + +/*Les1 wt1* lines. (*Les1/Les1* plants die as seedlings in the field.) The expected ratio of 1 *wt1*:3 *Wt1* seedlings was not found, however, in most of the stocks generated in this manner.

Table 1 lists the percentages of *wt1* seedlings observed for several different types of + +/*Les1 wt1* × + +/*Les1 wt1* crosses. The +/*wt1* × +/*wt1* crosses used as controls were made with segregating +/*wt1* (non-*Les1*) siblings. The two F1 families used in these crosses had as their male parent the same + *wt1*/*Les1 wt1* individual. In all crosses 25% of the progeny are expected to be *wt1*.

Table 1. Effect of *Les1* on transmission of *wt1*.

cross	# wt/# total	% wt	# ears tested
non-Les1			
(Mo20W/wt1) ⊗	54/225	23.9	5
(W23/wt1) ⊗	24/82	29.3	3
(Mo20W/wt1) X (W23/wt1)	50/200	25.0	2
(W23/wt1) X (Mo20W/wt1)	44/215	20.5	7
Les1			
(Mo20W/Les1 wt1) ⊗	179/770	23.2	7
(W23/Les1 wt1) ⊗	128/990	12.9*	5
(Mo20W/Les1 wt1) X (W23/Les1 wt1)	301/1893	15.9*	6
(W23/Les1 wt1) X (Mo20W/Les1 wt1)	96/751	12.8*	5

* Values are significantly different from the expected 1:3 ratio ($p > .10$).

The data indicate that all crosses involving W23 and the *Les1 wt1* chromosome have a striking reduction in the number of *Les1 wt1* progeny. Germination frequencies for all families were between 97% and 99%. Semi-sterility was not evident on any of the ears from + +/*Les1 wt1* plants. Pollen abortion in the + +/*Les1 wt1* plants was not examined. Sufficient data on the reciprocal crosses between + +/*Les1 wt1* and +/*wt1* plants are not yet available.

Under-representation of *Les1 wt1* seedlings cannot simply be due to poor expression of the *wt1* trait for two reasons. First, very few of the non-*wt* seedlings gave lesion expression characteristic of *Les1* homozygotes. This type of lesion expression would be expected among the *Wt1*/- class if the *Les1 wt1* chromosome was transmitted at a normal frequency but *wt1* was poorly expressed. Second, outcrosses of the original recombinant + *wt1*/*Les1 wt1* individual (recovered by D. Hoisington), and of the + +/*Les1*

wtl F1 individuals, onto various inbred lines resulted in a lower than expected frequency of +/*Les1* plants in the progeny. In these crosses *Les1* gametes (microspores) were transmitted at a frequency of 31% (32/103) from the original + *wtl*/*Les1 wtl* individual, 38% (29/77) from several Mo20W/*Les1 wtl* F1 individuals and 18% (40/223) from several W23/*Les1 wtl* F1 individuals. Similar crosses with +/*Les1* plants give the expected transmission frequency of 50% for *Les1* gametes (Neuffer & Calvert, J. Heredity 66:265).

Further tests are needed to explain why the lower transmission of *Les1* gametes that was observed following outcrossing of the Mo20W/*Les1 wtl* family was not observed following its selfing. If *Les1* is directly involved in the reduction of *Les1 wtl* gametes, however, then another interesting effect of the *Les1* mutation has come to light.

Craig Echt

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Glossy seedlings - update

Testing of the unknown glossies in the Coop collection is nearing completion. Tests are still incomplete on a half-dozen items but otherwise no new glossies have been found.

Two anomalies are still under investigation. The first involves *gl1* and *gl7*. These are on chromosome 7 and 4 respectively yet they give glossy seedlings in F1. In all tests conducted thus far they behave as functional alleles. A somewhat similar case involves *gl3* and *gl15*. In the routine tests for allelism, involving crosses of known heterozygotes, a few cases were found in which the unknown glossy indicated allelism with both *gl3* and *gl15*. This behavior was confirmed using homozygous glossies as parents. Additional tests of such exceptional behavior are in progress.

The stock designated *gl5* represents a duplicate gene situation, both recessive alleles being required for glossy expression. I have used the symbols *gl5-1* and *gl5-2*. As this usage does not conform to accepted nomenclature, I have designated *gl5-2* as *gl20*. *gl5* is linked with *su* with 12% crossing over. *gl20* remains unlinked.

gl14-wt linkage: Preliminary data indicated linkage of *gl14* and *wt*. The backcross data available are as follows: *Gl14 Wt* 113 : *Gl14 wt* 18 : *gl14 Wt* 45 : *gl14 wt* 76, indicating approximately 25% crossing over.

G.F. Sprague

Dt6-Su-Gl3

Dt6 was derived from virus-exposed cultures. The pattern is more extreme than *Dt1* in dosage response. In homozygous form the lower third to half of the kernel appears almost self-colored. In heterozygotes dotting is more frequent on the sides than crowns of the seeds. *Dt4* in the Coop stocks is supposed to be on chromosome 4 but in repeated tests I have been unable to establish any linkage with *su*. On that basis I have assumed *Dt6* to be new. Limited backcross data indicate the following linear order: *Dt6* 13 *Su* 41 *Gl3*.

G.F. Sprague

y11* and *y12

Two new lemon yellow types have been identified. Allelism tests have been completed with *y1*, *y3*, *y8*, *y9* and *y10*. Both give normal yellow:lemon segregations in F2 and the lemon-yellow seeds give rise to normal green seedlings and are fully viable.

G.F. Sprague

sh5

This trait was found in one of the virus-derived-mottled cultures. The crowns of the affected seeds rarely collapse. Most of the reduction affects the sides of the seeds. Classification is reasonably accurate. The gene is located on chromosome 5. The backcross data involving *Pr* are as follows: *Pr Sh* 977, *Pr sh* 255, *pr Sh* 301, *pr sh* 1021. *sh4* has been reported on chromosome 5 but tests for allelism were negative. The symbol *sh5* is suggested.

G.F. Sprague

A new allele at the *Y1* locus with pleiotropic effect

A pale golden plant type was observed unlike *g1*, *g2* or *g4*, more closely resembling *pg11*, *pg12*. All tests for allelism were negative. This type was later shown to be either closely linked or allelic to *y1*. The endosperm color is a straw yellow rather than white. In the F2 populations available all *Y* seeds gave normal green plants and all straw or lemon colored seeds gave yellow green or golden plants with moderate to severe blotching of the leaves. Leaf tissue in these affected areas eventually dies. In the plants that survive, the blotching becomes less extreme as the plant develops. This should be a useful trait as individuals may be classified in the seed, seedling or mature plant stage. Dr. Robertson informs me this may be similar to a type called pastel described by Anderson many years ago.

G.F. Sprague

Miniature germ - *mg*

Miniature germ was reported by J. B. Wentz (J. Hered. 15:269-272). This stock was never included in the Coop. A similar phenotype was found as a mutant in one of the breeding cultures and segregation indicated a single factor. Seed size is essentially normal but germ size is reduced to one-fourth or one-third of normal. In the sand bench *mg* seedlings are somewhat delayed in emergence. Under field conditions *mg* plants attain normal size and are fully viable. No linkage data are available but this should prove to be a useful seed trait.

R.J. Lambert and G.F. Sprague

Knob composition of commercial inbred lines

Over the past few years, we have made considerable progress in understanding the genetics of flowering time in maize. As a part of our studies, we have determined the knob composition of a number of inbred lines. These include some of the currently used inbreds, or those which have been used extensively in the present day maize germplasm in the U.S.A. The cytological analyses regarding the number, size and position of knobs were performed with acetocarmine squashes of microsporocytes mostly at pachytene stage during meiosis. With regard to size, the knobs were divided into six arbitrary classes. This classification was subjective since no attempt was made to measure the size of the knob.

Table 1. Number, size and position of heterochromatic knobs in maize (*Zea mays* L.) inbreds and varieties.

Inbred/Race/ Variety	Derivation†	Origin	Source	Knobs on Chromosomes*†																	Total Number of Knobs
				1		2		3		4		5		6			7		8		9	
				L	S	L	S	L	S	L	S	L	S	L_1	L_2	L_3	L	S	L_1	L_2	S	
Reid's yellow dent	(Dent x Flint)	Iowa	USDA PI 408705							L-VL							L-VL					2
Lancaster Sure Crop	(Dent x Flint)	Iowa	USDA PI 213697														L-VL					1
B14	BSSS	Iowa	U. of Ill., J.R. Laughnan							L							L				ML	3
A632	(Mt42xB14)$B14_3$	Minnesota	U. of Ill., J.R. Laughnan			S				VL							L				M	4
A634	(Mt42xB14)$B14_3$	Minnesota	U. of Ill., J.R. Laughnan			L				L						M	L				S	5
A635	(ND203xB14)$B14_2$	Minnesota	U. of Minn., R.H. Peterson							L							L					2
B37	BSSS	Iowa	U. of Ill., R.J. Lambert							L							L					2
B73	BSSS, cycle 5 sel.	Iowa	U. of Ill., R.J. Lambert				S			L							L				ML	4
A640	ND203xB14	Minnesota	U. of Minn., R.H. Peterson					ML		L							L					3
A631	(A509 X WF9)$WF9_3$	Minnesota	U. of Minn., S(?) R.H. Peterson							L							L					3
A659	Minn. Syn. 3	Minnesota	U. of Minn., L(?) R.H. Peterson							L							L-VL					3
A685	(ND203 X A635)$A635_3$	Minnesota	U. of Minn., R.H. Peterson							L							VL				VS	3
A641	ND203xB14	Minnesota	U. of Minn., R.H. Peterson					ML		L							L					3
CM105	V_3xB14_2	Canada, Morden	Funk Seeds Intl.					S		L							L				ML	4
CM109	V_3xB14_2	Canada, Morden	Funk Seeds Intl.				S										L				ML	3
WF9	Wilson Farm Reid sel.	Indiana	U. of Ill., C. Wilson			L(?)				L						S-M	VL		L			5
CI187-2 (Krug)	Reid's yellow dent sel.	USDA, Illinois	Missouri, M.S. Zuber							VL						M	VL					3
Oh43	Oh408xW8	Ohio	U. of Ill., J.R. Laughnan							L		S(?)			S		L				M	5
Oh45	Oh408xW8	Ohio	U. of Ill., J.R. Laughnan			M				L					S		L				S-M	5
Va26	Oh43xK155	Virginia	U. of Ill., E.B. Patterson							L					S	S	L					4
Oh545	[(M14x187-2)Oh45] x [(45Ax45T)Oh45]	Ohio	U. of Ill., R.J. Lambert							L							L				L	3
A619	(A171xOh43)Oh43	Minnesota	U. of Ill., J.R. Laughnan			L				VL					S		VL				ML	5
LH38	A619xL120	Holden's	Holden's, Iowa			L		L		L							L					4
C103	Lancaster Sure Crop sel.	Connecticut	U. of Ill., E.B. Patterson																		L	1
Mo17	CI187-2xC103	Missouri	Missouri, M.S. Zuber														L					1
W23	Gold Glow sel.	Wisconsin	U. of Ill., E.B. Patterson			L									S	S(?)	L				S	5
F2	O.P. Lacaune	France	Funk Seeds Intl.																		M	1
RSSSC	Stiff Stalk Synthetic, Illinois version	Illinois	U. of Ill., R.J. Lambert from S. Eberhart, Funk					L		ML		L(?)				S	L				M	6
Minnesota 13	Reid's yellow dent sel.	Minnesota	U. of Minn., R.H. Peterson					L		ML					S		VL					4
ND203	Haney Minn. 13	N. Dakota	N.D. State Univ.			L(?)						L					L				VL	4
Early Early Synthetic	d	Illinois	U. of Ill., D.E. Alexander																		VS	1

Table 1, continued

Inbred/Race/ Variety	Derivation	Origin	Source	Knobs on Chromosomes*+ 1 L	1 S	2 L	2 S	3 L	3 S	4 L	4 S	5 L	5 S	6 L_1	6 L_2	6 L_3	7 L	7 S	8 L_1	8 L_2	9 S	Total Number of Knobs
Minnesota Early Synth.		Illinois	U. of Ill., D.E. Alexander	S(?)						L							L				S	4
Argentine Flint**	Colorado majorado sel.	Nebraska	Hoeg Hybrids, L.C. Hoegemyer			ML				ML							L		M	S		5
Apachito**	Mexican race	Mexico (CIMMYT E-7)	N. Carolina, M.M. Goodman		S(?)		S			S					S							4
Azul**	Mexican race	Mexico (CIMMYTE-10)	N. Carolina, M.M. Goodman						ML(?)	L(?)							L(?)					3

†Henderson (1976, 1980) and Baker (1984).
+No knob was found on chromosome 10.
*L and S denote the long and short arm of the chromosome, respectively. Knob positions according to McClintock et al. (1981).

Size classes (in the body of the table):

VS = very small
S = small
M = medium
ML = medium-large
L = large
VL = very large

(?) = the identification of the knob is not positive.
**Argentine Flint and the Mexican flints (Apachito and Azul) have one and two B chromosomes, respectively.
d = derived by D. E. Alexander from these inbreds: ND276, W103, Co131, WD, CMV3, Tom Thumb, Gaspe Flint, ND1, and some others.

The data presented in Table 1 provide information on 35 inbred lines regarding the number, size and position of knobs together with some background information. The knob number ranged from 1 to 6 with most lines having 3 to 4 knobs. Of 23 possible locations, the knobs were found at 13 in the lines analyzed. The knob on the long arm of chromosome 7 (7L knob) was the most frequent, being present in all the lines (or races) except C103, F2, Apachito and Early Early Synthetic. Two other knobs were also found with high frequency; one on the long arm of chromosome 4 (4L) and the other on the short arm of chromosome 9 (9S). The knobs on chromosome 1 were the least frequent while no knob was observed on chromosome 10. Two Mexican flints (Apachito and Azul) and the Argentine flint (Colorado Halidaisi majorado) have supernumerary B chromosomes in their genomes. Three flints, Wilbur's, Parker's and Tama (not shown in the table), were also analyzed, and as expected none had a cytologically observable knob.

The Lancaster Sure Crop and its derivatives such as C103 and Mo17, the cold tolerant French inbred F2, and Early Early Synthetic (probably the earliest maturing line of maize) had only one knob. The inbreds related to Oh43 family have the highest average knob number while those related to Iowa Stiff Stalk Synthetic have an average knob number intermediate between those of the Lancaster derivatives and the Oh43 family.

Sajjad R. Chughtai and Dale M. Steffensen

Some insights into the genetics of flowering time

Heterochromatic knobs in maize exhibit a highly nonrandom geographical and racial distribution in spite of the apparent polymorphism for their number and size. The frequency of knobs, and even the size of a given knob in a particular race, is inversely related to the changes in latitude and altitude. Furthermore, the knob number is positively correlated with the time to reach maturity. Generally, low knob lines flower before high knob lines.

The maturity and flowering time in maize have long been considered quantitatively inherited characters. However, a large number of studies have failed to resolve the major controversy regarding the number of genes that differentiate parental lines with respect to flowering time; the estimates range from as few as two genes to as many as twenty-nine (see references in Bonaparte, Can. J. Genet. Cytol. 19:251-258, 1977).

For about 4 years, we have gained insights into the genetics of flowering time in maize. The focal point has been to elucidate the role of knob heterochromatin in determining the rate of floral development. Undoubtedly, the genetics of flowering time is not simple. We have been able to partition variously involved factors. Certain factors have proved critical in the analysis, especially inbreds and varieties with various genetic backgrounds that are essentially day-length neutral. The knobless genotypes with their F1's and F2's have been most important since they provided a simple genetic system.

Since the rate of development is related to the number of knobs, our first assumption was to consider the degree of delay in pollen shedding in relation to knob number. One might expect that the higher the knob DNA content, the longer the cell cycle would be and thus the slower the development. This simplest model proved to be incorrect in crosses using Zapalote chico, a high-knob Mexican race; the F1 hybrids flowered at more or less the same time as the knobless or low-knob parent. Similarly, the F1 hybrid between Zapalote grande (another high-knob and late maturing Mexican race) and the knobless Tama flint flowered at precisely the flowering time of its knobless parent during the 1st week of January, while Zapalote grande did not flower until the last week of February in our winter 1984-85 nursery at Molokai, Hawaii.

In our summer 1985 planting at Urbana, the knobless flints (Tama, Parker's and Wilbur's) were extensively crossed to a large number of inbreds of diverse genetic backgrounds. The parents and the F1 progeny from a multitude of crosses were grown out in our New Zealand and Molokai winter 1985-86 nurseries. With very minor exceptions, the F1 hybrid corresponded in flowering, within a day or two, with the knobless parent regardless of the knob composition, maturity group or the genetic background of the other inbred parent. Simply stated, the knobless genotypes are completely dominant over the

genotypes with knobs and the knobless parent determines the rate of development in these hybrids.

Further analyses regarding the segregation of knobs in the F2 generation enabled us to understand the genetics. Initially, the segregation of knobs was followed in F2 progeny of crosses of Wilbur's knobless flint to four inbreds, KYS, A634, Oh43 and A619, each with 5 knobs. Not unexpectedly, "polygenic" inheritance-type data were obtained. The data from crosses of the knobless flints to low-knob inbreds made the picture clearer. The F2 progeny from such crosses did segregate in a discontinuous fashion, rather than continuous. The F2 progeny gave a bimodal distribution pattern when segregating for a single knob. The large F2 populations from selfed F1's involving the genetically related inbreds with known knob compositions (i.e., B14 × B37, B14 × A635, B14 × B73, and Mo17 × C103) were analyzed in 1986 plantings at Urbana. These are segregating for only one or two knobs and thus served as the model system. These and various other crosses exhibited a discontinuous and simple Mendelian inheritance pattern. From these data we conclude that each homozygous knob combination (e.g. 9S/9S) delays plant development by three to four days. A heterozygous knob has no delay effect. Thus, the delay in development and the later time of flowering is positively correlated with the number of homozygous knobs. In simple terms, it is not just the number of knobs nor the amount of knob DNA, but the number of homozygous knobs causing delays. The observation that homozygous knobs delay development is consistent with the association between knob number and the time to reach maturity for the races of maize. Our best data are for 4L, 7L and 9S knobs. Interestingly, these are the most frequent knobs in U.S. corn belt inbreds. Other knobs are being analyzed.

Since the amount of knob DNA is not related to the rate of development and thus its effect in delaying development is not via lengthening the cell cycle, our alternate hypothesis is that knob DNA affects development via controlling the expression of the bracketing genes by a cis-acting position effect. The examples of cis-acting position effect variegation showing spreading effect that might involve DNA binding proteins are well known in *Drosophila* and mammalian X-chromosome inactivation.

We propose the presence of a protein that specifically binds to 180-bp repeat of knob DNA, brings about a conformational change and is probably involved in condensation of the knob DNA which could shut the neighboring genes off by a spreading effect.

The proposed hypothesis could explain the complete dominance of the knobless genotype, the knob-mediated developmental delay in homozygous and its absence in heterozygous knob condition. Thus, the F1 hybrids between knobless and knobbed inbreds involve the co-existence of "two genomes" which differ temporally in their RNA and DNA synthetic activity. Since replication and transcription are temporally correlated, the knobless genotype should replicate early in the S phase, and so most of these newly replicated genes will be expressed. On the other hand, those genes closely linked to knobs will not replicate until the end of S when the knob DNA replicates (Pryor et al., Proc. Natl. Acad. Sci. 77:6705-6709, 1980).

If our working hypothesis on the role of knob DNA in gene expression is correct, then it might provide clues to understanding the dogma of hybrid vigor and combining ability. For example, in crosses between knobless (or low knob) and high-knobbed inbreds (e.g., Mo17 × B73 with good combining ability), the early replication and expression of knobless genome coupled with the late expression of knob-controlled genes will provide overlapping and extended periods of gene activity.

Studies are being actively pursued to test our various predictions, especially the role of knob heterochromatin in maize development.

Sajjad R. Chughtai and Dale M. Steffensen

A correlation between knob number and leaf number—a counting mechanism

Most of the very early races or lines of maize have only 9 or 10 leaves at maturity. Since their mature seeds already have 5 or 6 preformed leaves, the remaining 3 or 4 must have cell initial areas that are already present in the apical dome. Since ears are initiated at the base of leaves 3 or 4 in mature seed, the anlagen of ear buds should be in place. Also the cells at the top of the dome, which form the tassel, should have more preformed cells than lines that grow a lot more.

One of the earliest selections that we know about is Early Early Synthetic (EES) from D. E. Alexander. EES is knobless except for a very small knob at 9S. Its background is mostly Gaspé. It is well known that Northern races and knobless flints are the earliest to come to maturity. If one plots the number of homozygous knob sites on one axis and the number of leaves on the other, a rather good correlation results. Knobless or low knob lines (1-2) have the fewest leaves. Intermediates with 3 to 5 knobs have more leaves and of course Southern and Mexican races (6 to 16) have the most leaves. Our morphological and cytological observations would suggest that knobs have a function to delay flower formation by controlling the initiation of more leaves. Since this significance on flower regulation was only realized during the summer of 1986, considerably more information must be obtained. However enough is known now about the maize of North America (Chiapas to Canada) to be convinced of the proposed correlation. Finally, it appears that the placement of the topmost ears may be the best predictor of maturity. The pre-set placement of the ear on the shoot is under genetic control and regulated by knob composition. At tasseling the ear also takes over apical dominance. This switch of dominance is of theoretical and practical interest.

Dale M. Steffensen and Sajjad R. Chughtai

Mapping genes for ear development

During a study designed to locate zein polypeptides on chromosome 4, an unrelated phenomenon was discovered involving ear development. The progeny exhibited segregation from a cross of the female, W153R/T4-9g (4 S.27;9L.27), to pollen of Mo17, for the size of the ears. The large ears with large shanks having 8 or 9 nodes were the semi-sterile plants. Plants with normal pollen had smaller ears and shorter shanks. This "hybrid vigor" persisted in the second and third backcross generations using the semi-sterile female plants crossed to Mo17. All types of crosses have been made to Mo17 using other translocations with

chromosome 9 (not with 4) and several different chromosome 4 translocations not involving 9. Other inbreds were used in the same test. Thus far the ear factor(s) follows chromosome 4 of Mo17.

Another genetic feature surfaced in the third backcross generation of T4-9g/Mo17 × Mo17. Semi-sterile plants flowered three days earlier than sibs with normal pollen. There was a bimodal distribution with little overlap. In our knob studies with Mo17 we found a gene, which we call "delay factor." This delay factor slows down tassel and ear development toward the end of maturity. It could be related to the increase of the number of nodes in the shank. Conventional genes on chromosome 4 are being used to map this delay factor. Zein genes are included. C103, one of the parents of Mo17, seems to possess this delay factor. Lancaster and other derivatives would be expected to transmit it too.

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An unstable factor for orange pigment

In the 1982 News Letter (p. 160) we reported on an unstable pigmenting factor sent to us by Dr. Charles Burnham. The pigment determined by this factor is similar if not identical to the *P*-controlled (phlobaphene) pigment normally produced in the cob and pericarp, but the factor is different from *P* in that the pigment may form in any or all tissues, and concentrations can be excessive. Sectoring is common, but unpredictable.

Although we have not yet been able to obtain stable lines of this 'Burnham factor' with predictable distribution and concentration patterns, we have been able to obtain some information on its inheritance, expression and function.

Inheritance: The factor, tentatively designated *Ufo* (*U*nstable *f*actor for *o*range), is dominant, and, from reciprocal cross data, is fully and equally male and female transmissible. It segregates independently from *P* and *sm*.

Interaction with other genes:

a) *Ufo* expression requires *P-WR* or *P-RR*. It is not expressed in *P-WW* or *P-MO* plants, and it has little or no expression with an intermediate 'grainy' pericarp *P* allele (obtained from the collection of *P* alleles backcrossed into 4 County 63 by R.A. Brink (see MNL 40:149-160,1966).

b) *sm* (salmon silk) normally behaves as a recessive in the presence of *P-RR* or *P-WR*, but behaves as a dominant in the presence of *Ufo*, e.g., *P-WR Ufo*/+ *Sm*/*sm* plants have salmon silks.

c) *C2-Idf* inhibits the expression of *Ufo*. *P-WR C2-Idf*/*C2 Ufo*/+ plants are green, but these plants when self-fertilized yield *C2 Ufo*/+ progeny that are orange.

d) *a1 P-WR Ufo*/+ plants are brown in tissues that would be orange in otherwise similar *A1* plants.

Function: The *P* locus controls the formation of flavones, the 3-deoxyanthocyanins (the salmon silk pigments), and the (leuco) flavan-4-ols that polymerize to form the visible phlobaphenes. One possibility is that *Ufo* may block flavone synthesis and thereby cause an excessive build-up of phlobaphenes and 3-deoxyanthocyanins. This appears unlikely, as *Ufo* tissues seem to have normal amounts of flavones, at least at developmental stages just prior to the formation of the orange pigment.

Effect on growth: A notable effect of *Ufo* is that plant and tissue growth is retarded in direct proportion to the concentration of the pigment. Testcrosses of *P-WR* plants heterozygous for *Ufo* produce *Ufo*/+ progeny that are on the average 20% shorter than their +/+ sibs. Homozygous *Ufo* plants are usually very short, and silk growth is minimal, making it difficult to maintain true breeding lines of *Ufo* in a form or a background that allows strong expression.

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Phase change involves a two-gene switching system

There is morphological evidence that the switching off of one phase and turning on of the next phase are independent genetic events. In the case of the corngrass (*Cg*) and teopod (*Tp*) mutants, the floral phase may be turned on without the vegetative phase being switched off. The overlapping and sometimes simultaneous expression of both phases accounts for the phenotypes of these mutant genes. When the vegetative phase is finally shut down, the demarcation is not always at right angles to the axis but the switching may follow along wedge-shaped sectors flowing up into the pure floral region. This is suggestive of an Activator-Dissociator type of action such as the somatic cell heredity known for aleurone color.

If two independently acting genes are responsible for this type of phase change, then a state should also be found in which the vegetative phase is turned off but the floral phase is not yet turned on. Since both the vegetative and floral phases have nodes, this hypothetical state with a barren axis might also have nodes or at least rudimentary nodes. Since the pre-phase non-specialized condition is one of short internodes, the barren nodes would be close together. Since an Activator-Dissociator type of switching is envisioned, the interface between the pre-phase barren axis and any eventual switch to the floral phase should sometimes follow wedge-shaped sectors. The delayed switch to the floral phase should affect both tassel and ear. It is expected that by a year from now, I shall be able to report on the inheritance of such an interrupted phase change condition.

One problem is how a given complex of genes becomes identified with one switching element, like an antibody with an antigen, and another complex of genes with a second switching element. Another problem is how the two switching elements normally synchronize their on-off activities during phase change. Perhaps the mutant genes that cause defective switching may be utilized to reveal the genetic process of switching and the control of morphogenesis.

Walton C. Galinat

New evidence supporting multiple domestications

The different patterns of chromosome knobs and other diverse traits of maize have led McClintock, Randolph and Kato to independently suggest that modern maize stems from multiple domestications of its wild ancestor.

New evidence supporting at least two independent domestications from two different teosintes comes from

the comparative morphology of the cobs and kernels of two ancient indigenous races of maize from Mexico. It appears that the emergence of the maize kernel from the cupulate fruit case of teosinte about 8,000 years ago was accomplished by two different systems during two or more independent domestications. Understanding these systems has a bearing on corn improvement. In the Nal Tel-Chapalote system, the kernel emerged by an elongation of the rachilla within the female spikelet. In the Palomero Toluqueno system, emergence was by way of an elongation of the kernel while its rachilla remained short. The present descendants with cobs based on rachilla elongation are most common in U.S. corn, especially in the Northern Flints. The second system, by kernel elongation, has among its descendants the Gourd Seed - Shoe Peg types and, to a lesser extent, the Southern Dents in general.

The Corn Belt Dent is known from historical records to be a hybrid between the Southern Dents and Northern Flints. Much of its heterosis appears to stem from combining these diverse parents. Further back, the source of the heterosis may stem in part from either early isolates of domesticated teosinte or from independent domestications of two different teosintes.

It is suggested that the Nal Tel-Chapalote system came from domesticated Guerrero teosinte while the Palomero Toluqueno system came from Chalco teosinte.

Walton C. Galinat

The use of Palomero Toluqueno (white rice popcorn) in sweet corn improvement

Historically, U.S. sweet corn has had its evolutionary roots in the Northern Flint-long rachilla type of cob. Yet in recent years the consumer demand has been for deep kernels and a high number of kernel rows. These are characteristics of the Palomero Toluqueno - white rice popcorn type of cob and kernel. We have made progress in sweet corn improvement by going directly to the source of these traits. The thick pericarp of this popcorn is easily removed by breeding. The vestigial glume gene may have to be used with this material in order to allow full eating access to its deep kernels. With little or no rachilla to elevate the kernel above the chaff, there would be serious interference with attempting to eat intact kernels off from the cob.

Walton C. Galinat

The origin of thick-cob, eight-row maize

Previously I have reported that the string cob trait is controlled by two incompletely dominant genes (Galinat, MAES Bul. 577, 1969). It appears that all of the most ancient cobs from dry caves in New Mexico, Tehuacan-Mexico, Panama, Venezuela, Ecuador and Peru carry the string cob trait and are eight-rowed. In each case where a sequence was available leading to more productive ears, the first steps involved an increase in both the number of kernel rows and the thickness of the rachis. Much later, sometimes thousands of years later, thick-cob, eight-row types of ears bearing large kernels appeared, all of independent origin. Apparently the thick-cob, eight-row types of ears are a result of recombination between the thick cob, high-row types and the slender cob, eight-row types. This conclusion is based both on archaeological remains and on experimental results.

In the American Southwest, the source of the two recessive genes for thick cob was the 12 to 14 rowed race, Chapalote, while the more primitive eight-rowed condition was probably derived from persistent remnants of the original maize to reach the area, or less probably due to row-number reductions from teosinte introgression. That the reduction in row number may leave the previous vascular supply system of the higher ranking intact was observed first by Laubengayer (Ann. Mo. Bot. Gard. 35:337-342, 1948). Increased vascularization in eight-rowed maize may lead either to induration of the rachis if the kernels remain small, or to increased kernel size and rachis size. Maiz de Ocho took the large-kernel, thick-rachis pathway and with the day-neutral, early flowering traits, it was the frontier maize in the spread of maize to the North and Northeast.

Walton C. Galinat

On the return of a hopeful monster: cauliflower ear maize

The first cauliflower ear of maize was reported by Collins (Smithsonian Inst. Report Plate 25, 1937). It was a result of combining homozygous ramosa (*ra ra*) with heterozygous full tunicate (*Tu tu*) in a family segregating these two genes. The combined phenotype of these two genes is a cauliflower ear monster incapable of propagation except as a recombinant in a segregation. But now, 50 years after its first appearance, this monster has returned with renewed hope for a successful role in macroevolution.

Since the early observations of Collins, mutations to intermediate tunicate alleles (*tu-l* and *tu-d*) have been discovered by Mangelsdorf (Mangelsdorf and Galinat, P.N.A.S. 51:147-150, 1964), and in contrast with full tunicate, these weaker alleles may be propagated as homozygotes. We have produced the double homozygote (*tu-l tu-l, ra ra*) and have found that its phenotype is identical to that observed by Collins (1937) for homozygous ramosa with heterozygous full tunicate. That is, both are cauliflower sterile as a lateral ear in a background of modern dent corn. Apparently, an incongruous combination of genetic instructions causes the female flowers to abort in a parallelism to a head of cauliflower. Under internal conditions at a lowered level on the plant that cause the pistil to become extremely precocious, the combination of the tunicate (long glume) gene that further increases female earliness with the ramosa gene that puts developmental priority on a branching of the rachis results in an inadequacy by the supply system to function soon enough to nurture its spikelets. But in a different position on the same plant where pistil development is less precocious, such as the inflorescences of long tillers, the relational balance may be improved, female fertility normal and the way opened to successful macroevolution. The tassels of tunicate ramosa have normal male fertility.

Walton C. Galinat

Duplication of the nucleolus organizer in the genome of maize

Cytogenetic analysis was undertaken in an alien addition stock of maize derived from an intergeneric cross between recessive marker gene stock for chromosome 2 in the maize parent and *Tripsacum dactyloides* L. In this strain a duplication of the nucleolus organizing region (NOR) was observed. The two chromosomes, each carrying

a segment of the NOR, form two distinct nucleoli each with a bivalent attached to it. Microsporogenesis is normal and at pachytene the two chromosomes involved in this cytologically visible abnormality do not show any pairing relationships. This accounts for the absence of higher associations like quadrivalents at diakinesis and metaphase stages of meiosis. The break must have occurred in the NOR itself, resulting in a breakage-transposition-duplication of the nucleolus organizer. At the tetrad stage of meiosis we can see two nucleoli in each of the tetrad cells. Occasionally one of the tetrad cells shows a single large nucleolus as a result of fusion. Since there is no deficiency they are all functional. This homozygous translocation is transmitted normally and the progeny breed true.

Chandra V. Pasupuleti

Cytological techniques for the study of maize chromosomes

A paper on the cytological techniques for the study of maize chromosomes is in the process of being published at the suggestion of Dr. Charles R. Burnham. The information and the technique are nothing new. But it is a specific, straight-forward, step by step procedure from the time of collection to the finish, including photomicrography, where the data are well documented with self explanatory photographs. It is made easy for a beginner, working on meiotic studies with special emphasis on the pachytene stage of meiosis in maize, where the morphology of the individual chromosomes is essential for cytogenetic analysis.

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Cloning of a *Ds1*-homologous element at the *A1* locus

The *a-m-4* mutable allele of the anthocyanin locus, *A1*, originated from a wildtype *A1* allele in a plant carrying the controlling elements Dissociation (*Ds*) and Activator (*Ac*) (B. McClintock, Genetics 38:579-599, 1953). *Ac* controls transposition both of itself and of *Ds* causing both insertional inactivation of genes and subsequent reversion (i.e., a mutable phenotype). Since *a-m-4* mutability is controlled by, but unlinked to *Ac*, it is thought that the *a-m-4* mutation resulted from a *Ds* insertion. *Ds* elements isolated so far fall into two classes based on their degree of homology with the *Ac* element. We were interested in identifying the *Ds* element thought to be present in *a-m-4* and in determining its position relative to the *A1* gene.

Molecular analysis of *a-m-4* was facilitated by the availability of a cloned *A1* gene and two cloned *Ds* elements for use as hybridization probes. To isolate *a-m-4* clones we constructed a genomic library in lambda EMBL4 from an *a-m-4* homozygous plant (no *Ac* elements present in the line, seed provided by M.G. Neuffer) and screened the library with an *A1* probe (C. O'Reilly et al., EMBO J. 4:877-882, 1985). We then screened *A1*-homologous clones with the *Ds* probes derived from two different *Ds* elements; *Ds1*, which is nonhomologous to *Ac* (isolated from the *wx-m1* allele, and provided by S. Dellaporta; S.R. Wessler et al., EMBO J. 5:2427-2432, 1986), and *Ds-5933*, a deletion derivative of *Ac* (isolated from the *sh-m5933* allele, and provided by H.P. Döring; H.P. Döring et al., Nature 307:127-131, 1984). Three *A1*-homologous clones hybridized to the *Ds1* probe but none hybridized to the *Ds-5933* probe. Our preliminary results suggest that the mutability of *A1* gene expression in the *a-m-4* allele is due to a *Ds1*-homologous element.

We constructed a restriction enzyme map of a representative *a-m-4* clone and determined the region of *Ds1* homology by Southern analysis and hybridization to the *Ds1* probe (Fig. 1). A comparison of the preliminary *a-m-4*

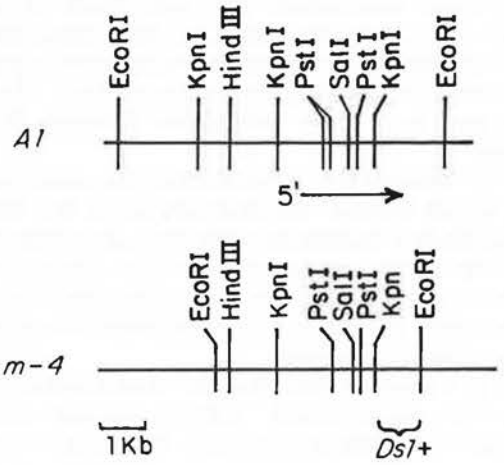

Figure 1. The *A1* and *a-m-4* restriction enzyme maps shown are aligned at sites conserved between both alleles within the transcribed region. The arrow indicates the direction and extent of transcription of *A1* (Schwarz-Sommer et al., EMBO J., in press). The bracket indicates the *Ds1* -homologous region of *a-m-4*.

data with a wildtype *A1* restriction enzyme map showing the region of *A1* transcription indicates that the *Ds1* homology is located toward the 3′ end of the gene. The *Ds1*-homologous region is 3′ of other transposons at *A1:* those reported in Zs. Schwarz-Sommer et al. (EMBO J., in press), the *Mu1* element in *a1-Mum2* (5′ of the *A1* transcription unit), the two *rDt* insertions in *a-m-1:Cache* and *a1* (in exons 3 and 4), and the *rMrh* insertion in *a-mrh* (also in exon 4).

Differences in *Eco*RI sites between *a-m-4* and *A1* (shown in Fig. 1) may be restriction fragment length polymorphisms resulting from minor sequence alterations or from more extensive differences. Such *Eco*RI polymorphisms are common between *A1* alleles (for example, *A1* alleles reported in Zs. Schwarz-Sommer et al. EMBO J., in press; and *a1, a-m-1:Cache* alleles which were cloned on 10kb *Eco*RI fragments).

A genomic Southern blot of DNA from *a-m-4* or *A1* homozygous plants probed with a fragment of the wildtype *A1* gene is shown in Figure 2. The sizes of the major hybridizing bands agree with the restriction maps constructed from *a-m-4* and *A1* clones. Maize genomic Southern blots probed with wildtype *A1* sequences often result in one major hybridizing band and a second minor band, which may be either smaller or larger than the major band. As shown in Figure 2, this is the case with DNA prepared not only from plants carrying mutable alleles of *A1*, such as *a-m-4*, but also from those carrying a wildtype *A1* allele. This might result from the presence of a second

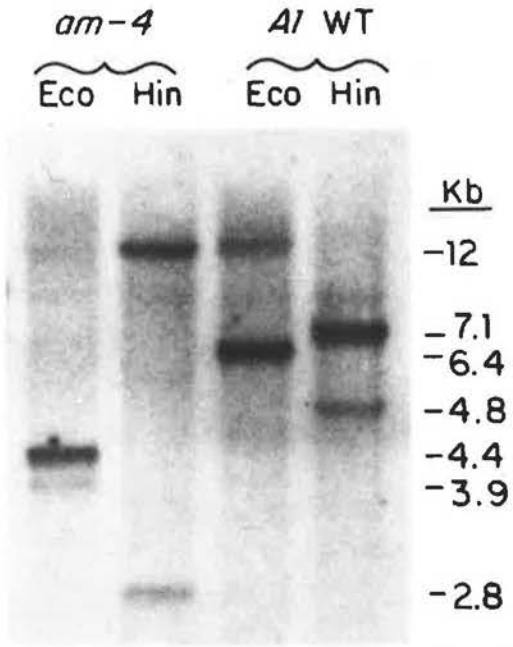

Figure 2. Southern blot of 10μg per lane of *Eco*RI or *Hin*dIII digested genomic DNA isolated from either *a-m-4/a-m-4* (lacking *Ac*) or *A1/A1* plants and probed with a nick-translated 0.6kb *Pst*I restriction fragment of the wildtype *A1* gene.

locus that shares incomplete sequence homology with *A1*. Alternatively, the minor bands may represent somatic rearrangements of *A1* sequence in certain cells even when known transposons are not active in the genome. One *A1*-homologous clone isolated from the *a-m-4* library seems to have the same structure as the minor bands seen on the genomic Southern of *a-m-4/a-m-4* DNA (Fig. 2). We will analyze both the *Ds1*-hybridizing genomic *a-m-4* clone and this second type of genomic clone in order to determine their relationship.

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Sequence characteristics of the *rDt* controlling element

Dotted is a two-element transposon system consisting of the dotted element *Dt*, plus a second receptor element for *Dt*, *rDt* (M. Rhoades, J. Genetics 33:347-354, 1936). *Dt* not only controls its own transposition, but also controls the expression of several alleles of the anthocyanin locus, *A1*. The presence of the *rDt* element in these *A1* alleles is inferred from their mutability in response to *Dt* (B. McClintock, Cold Spring Harbor Symp. Quant. Biol. 21:197-216, 1956). To address the mechanism of *Dt*-controlled *A1* mutability, we cloned, sequenced, and determined the insertional position of *rDt* elements found in two mutable *A1* alleles.

The alleles studied were *a1*, the standard recessive allele which probably originated from *A-r* (present in N. American races), and *a-m-1:Cache*, which originated from *A:Cache* (M.G. Neuffer, Genetics 46:625-640, 1961). Both alleles are recessive in the absence of *Dt*, presumably due to insertional inactivation of *A1* by the *rDt* element. These two alleles also differ in their response to *Dt*, *a-m-1:Cache* being more mutable.

To clone *a1* and *a-m-1:Cache*, we constructed genomic libraries from plants carrying these alleles, and then screened these with an *A1* probe (C. O'Reilly et al., EMBO J. 4:877-882, 1985). We characterized positive clones by restriction enzyme mapping and by nucleotide sequencing. These data, plus our previous data on the *A1* gene itself (Zs. Schwarz-Sommer et al., EMBO J., in press), have allowed us to characterize the *rDt* element at the molecular level.

Comparisons of the restriction enzyme maps of *A1*, *a1*, and *a-m-1:Cache* identify similar 0.7 kb insertions in the latter two alleles. The sequences of the two insertions are very similar (>80% homology), but they are inserted at *A1* in opposite orientations. The ends of the insertion contain 11 bp inverted-repeats, and the insertion is flanked by a 9 bp direct-repeat of *A1* sequence in each case. The insertions in *a-m-1:Cache* and *a1* interrupt the *A1* coding sequence in exons 3 and 4 respectively, and each generates a translational stop signal.

Since we found insertions of similar nucleotide sequence in two *Dt*-responding *A1* alleles of independent origin, we believe the insertions are the *rDt* controlling element. The *rDt* element is 0.7 kb long and has molecular characteristics typical of other maize controlling elements; *Ac*, *Ds*, and *Spm* (terminal inverted repeats flanked by a target site duplication). As expected, the recessive null phenotype of both alleles can be explained by the *rDt* insertion within the coding sequences of *a1* and *a-m-1:Cache*. In this respect *rDt* is similar to *Ds* and other transposons, which can cause insertional inactivation of the gene.

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A flint type endosperm gene

An ear with small flint type endosperms was discovered in selfed progeny of Oh43, *zpg-3* mutants. This phenotype is controlled by a recessive gene named *sft* (small flint type). The cross *sft/sft* × +/+ (normal Oh43) produces all flint kernels and the reciprocal (+/+ × *sft/sft*) all normal dent kernels. Selfs of F1 (*sft*/+, and reciprocal) plants produce ears with all normal dent endosperms. Flint type endosperms are not produced. Ears are well filled and appear normal. Endosperms of the backcross F1 × *sft/sft* seeds are all normal and of the reciprocal (*sft/sft* × F1) all flint. Selfed plants from the above backcrosses produce ears which have either all small flint type endosperms or all normal dent endosperms. Thus, *sft/sft* plants produce only flint endosperms and *sft*/+ plants only normal endosperms regardless of the pollen parent genotype. The type of endosperm produced is dependent upon a contribution from maternal tissue. Gene action appears similar to that of the *de-p1* gene reported by Mangelsdorf (1926).

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Chemically induced alteration of floral sexuality

Sex determination in monoecious plants like maize has been shown to be influenced by genotype, daylength, and plant hormones. In a recent series of experiments to determine the physiological effects of an anti-dehiscence chemical, 3-(p-chlorophenyl)-6-methoxy-s-triazine-2,4(1H, 3H) dione triethanolamine, DPX-3778 (E.I. du Pont de Nemours), the sex determination of the tassels was found to be altered depending on the concentration and timing of DPX-3778 application. Chemical treatments have been conducted on two hybrids (Seneca 60 and Golden Beauty) and an inbred (Oh43). Multiple applications of 0.005% DPX-3778 to the leaf sheath of maize plants or the addition of 1 ppm of DPX-3778 to hydroponic growth medium approximately 4 weeks before anthesis caused almost complete reversion of staminate inflorescences to pistillate inflorescences (Fig. 1 and 2). In addition to the normally developed gynoecium of the upper floret, DPX-3778 treatment resulted in the full development of the gynoecium of the lower floret of the ear spikelets. Both the spikelets in the tassel and ear inflorescences could produce viable seeds if pollination occurred (Fig. 3). The net result of DPX-3778 treatment is "male sterile" inflorescences. These results could be duplicated in northern teosinte (*Zea mays* subsp. *mexicana*, race Nobogame) (Fig. 4). Our results indicate that DPX-3778 could be useful for elucidation of sex determination in grasses.

P.C. Cheng and D.C. Wright

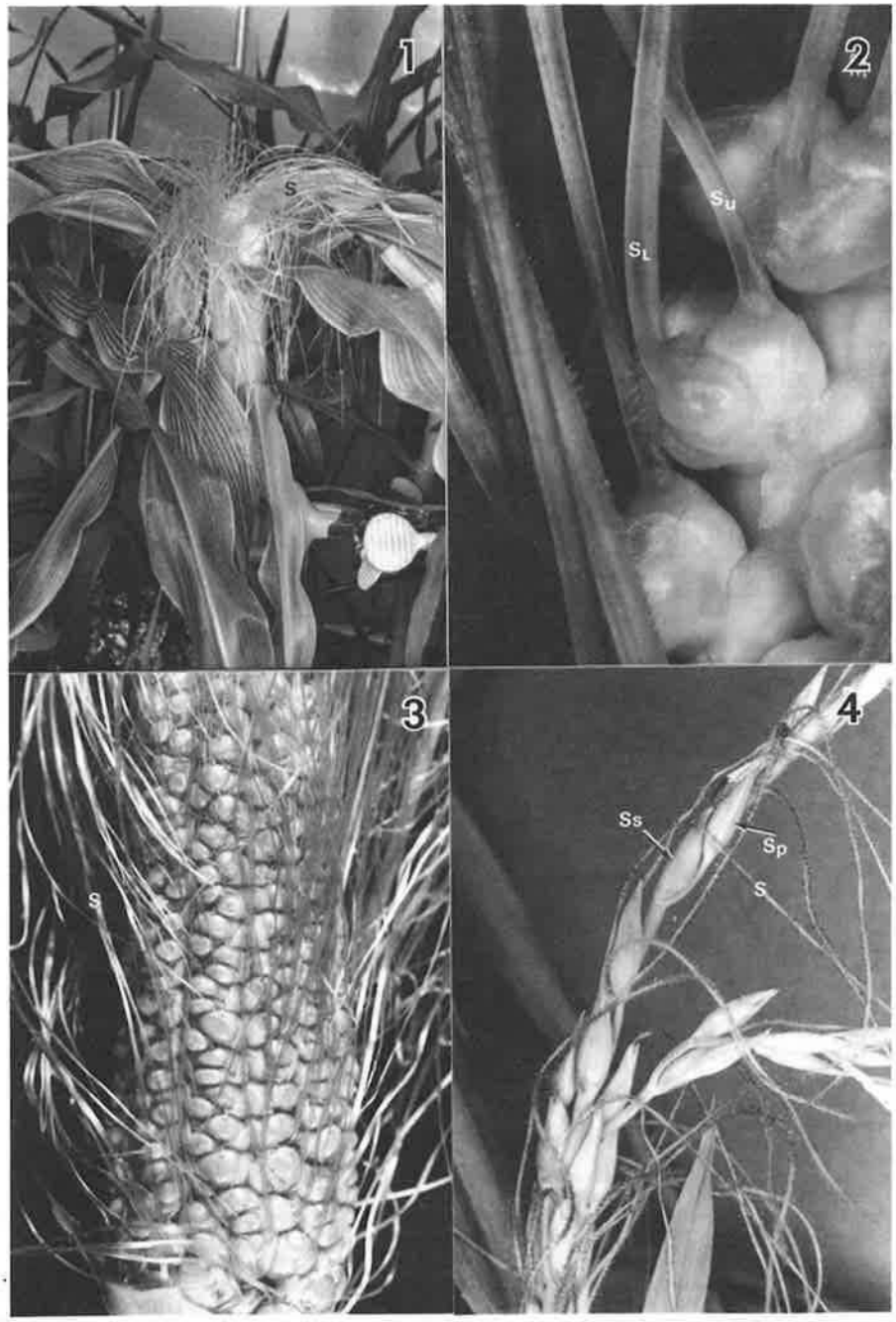


Figure 1. Tassel inflorescence of treated plant (var. Golden Beauty). Note the development of "silks" (S). The plant was treated with DPX-3778 at a concentration of 1ppm in hydroponic medium.

Figure 2. Female tassel spikelets from DPX-3778 treated plants. The treatment was conducted approximately at the spikelet-pair primordium stage. Note the two silks (SU and SL) developed from the upper and lower florets. The spikelet has its outer glume removed. Note the short pedicel of both spikelets.

Figure 3. Kernel arrangement on the ear of a DPX-3778 treated plant (var. Golden Beauty). Due to the development of lower florets which compete for space, the placement of kernels is no longer in regularly arranged rows.

Figure 4. Terminal inflorescence of DPX-3778 treated northern teosinte (*Zea mays* subsp. *mexicana*, race Nobogame). The chemical was applied to the plant at the 5th leaf stage (3rd "true" leaf stage), via hydroponic medium at a concentration of 1ppm. The spikelets are bisexual, however, the anthers usually do not reach maturation.

Key to labeling: S: silk, SU: silk of upper floret, SL: silk of lower floret, Sp: pedicelled spikelet, Ss: sessile spikelet.

* in symbol identifies loci needing documentation, symbol standardization and/or allelism tests
BS = base sequence; BSH = broad-sense heritability; gca, sca = general and specific combining ability; QTL = quantitative trait loci;
RM = restriction map; R/S = resistance/susceptibility or tolerance
r refers to numbers of references in the list of Recent Maize Publications

CHROMOSOME 1
Adh1-Usv somaclonal mutant from Adh1-S, BS --Brettell &, r60
bz2-mu1 Mu1-elicited; modification of Mu elements correlated with loss of somatic reversion --Chandler &, r73; Walbot &, r657
dek22 on 1L --Clark &, r83
Acp4, Adh1 allozymes in Corn Belt dents, flints and South American dent populations; QTL associations in F2 from Wf9/Pa405 --Kahler &, r260, r261
Adh1 BS, methylated sites --Nick &, r420
Adh1-1S, Adh1-1F, 5' and 3' BS --Sachs &, r526
Adh1: Mu1 insert in intron --Vayda &, r645
Mpl1, possible allele D8, approx. 4 mu distal to lw1 --Harberd &, 61:23
P1-WR, f1, Flt1, Krn1, Zer1, Pd1, Tr1, bm2 linkages --Miranda &, 61:32,34
zb4 (5) P1 (9) nec2; dek1 (7) nec2 --Hoisington, 61:47
l16, pg15, v25, Les2, Les5 on 1S; hcf13, ij2, l17, pg16, py2, spc2, zb7, Les7, Msc1, T1r1 on 1L --Neuffer &, 61:50
gs1 (14) Phi1 (10) bm2 (16) Acp4 --Sisco &, 61:86
NPI370-P1, 412-Sod4 on 1S; 21-Adh1, 251-Phy1 on 1L --Wright &, 61:89
Hypoploids for TB-1Sb and TB-1La: endosperms, reduced kernel weight, plants, reduced height, leaf width; TB-1La leaves shortened --Chang &, 61:91

CHROMOSOME 2
rRNA5S, BS --Barciszewska &, r22
dek23 on 2L --Clark &, r83
Px1 allozymes in Corn Belt dents, flints and South American dent populations; location on 2L, QTL in F2 from Wf9/Pa405 --Kahler &, r260, r261
bl-peru-mu1 Mu1-elicited --Walbot &, r657
Pd2, Tr2, lg1, g12, Flt2, Krn2, Lsc1, Zer3, Bl, Fl1 linkages --Miranda &, 61:27,32
gl2 (18) wt1 (15) Les10; Bl (18) ts1 (8) Les10 Hoisington, 61:48
nec4, v26, Les1 on 2S; l18, spt1, v24, Les4 on 2L --Neuffer &, 61:50
NPI248-Bl on 2S; 348-Alr1 on 2L; 347-EMu, 331-Ssu2 on 2 near centromere --Wright &, 61:89
TB-2Sa uncovers gl2, Bl-Peru, not wt1 --Echt, 61:94
gl14 (25) wt1 --Sprague, 61:96

CHROMOSOME 3
E4, Got1, Pgd2 allozymes in Corn Belt dents, flints and South American dent populations; QTL associations in F2 from Wf9/Pa405 --Kahler &, r260, r261
a1-m13, a1-m16, Uq-regulated --Pereira &, r471
Hex1 located by TB-3Sb; alleles -2, -3, -4, -5, -6, -n; E8 (24) Hex1 (17) Tpi4 (10) Pgd2 (24) Got1 (5) Me1 (21) Mdh3 --Wendel &, r670
brn1 (19) d1 (21) Lg3; brn1 (36) c11 Stinard, 61:6
lg2, Pd3, Flt3, Krn3, Tr3, a1, Mer2 linkages --Miranda &, 61:32
gl19, Wrk1 on 3S; spc3, wlu1, Spc1 on 3L --Neuffer &, 61:50
hl (32) Cg1 --Poethig, 61:85
NPI231-Me1, 345-Tpi4 on 3S; 51-A1 on 3L --Wright &, 61:89
Hypoploids for TB-3Sb and TB-3La have reduced plant height; TB-3Sb leaves pale green, TB-3La leaves short, erect --Chang &, 61:91
a1-m-4, Ds1 insert: RM; a1, a1-m-1:Cache, rDt inserts 0.7kb --Sorrentino &, 61:102,103

CHROMOSOME 4
Fatty acid composition affected by 4L dosage, TB-4Lb --Shadley &, r564
ptd*-1130, pitted endosperm, on 4L --Sheridan &, r573
c2-mu2 Mu1-elicited --Walbot &, r657
c2-m1 Spm/En-tagged, C2 clone; c2-m3 and revertant c2-m3R; RM --Wienand &, r685
Krn4, Fas4 linkages --Miranda &, 61:29
Ga1 (40) o1; Pd4, Tr4, Ts5 linkages --Miranda &, 61:32
orp1 uncovered by TB-4Sa; linkage with TB-4Lf --Neuffer &, 61:44
spt2, wt2, Ysk1 on 4S; nec5 on 4L; Ms41 near T4-9b (4L.90) --Neuffer &, 61:50,51
NPI228-Adh2 on 4S; 227-Ssu1 on 4L --Wright &, 61:89
Hypoploids for TB-4Sa have short, broad leaves --Chang &, 61:91
gl5 (12) su1; Dt6 (13) su1 (41) gl3 --Sprague, 61:96

CHROMOSOME 5
Fatty acid composition affected by 5L dosage, TB-5La --Shadley &, r564
a2-mu1, a2-mu2, a2-mu3 Mu1-elicited --Walbot &, r657
Flt5, Krn5, Fas5, pr1, Pd5, Tr5, Ger1 linkages --Miranda &, 61:29,32
anl1 distal to a2 --Coe, 61:47
nec6, Msc2, Rgd2 on 5S; grt1, nec7, ppg1, wga1, Hsf1 on 5L --Neuffer &, 61:50
NPI369-Phy2, 345-Tpi5 on 5S --Wright &, 61:89
Hypoploids for TB-5Sc and TB-5La: endosperms, reduced kernel weight; plants, reduced height and leaf width; TB-5La leaves pale green --Chang &, 61:91
sh5 not allelic to sh4; sh5 (22) pr1 --Sprague, 61:96

CHROMOSOME 6
Enp1, Idh2, Mdh2, Pgd1 allozymes in Corn Belt dents, flints and South American dent populations; QTL in F2 from Wf9/Pa405 --Kahler &, r260, r261
rDNA spacer, RM; new site in tripsacoid maize --Lin &, r326
rDNA spacer, BS --McMullen &, r372; Tolczyki &, r622
Hex2 located by TB-6Lc; alleles -0.5, -1, -1.7, -2, -2s, -3, -4, -6, -n; Pgd1 (3) Enp1 (29) Pl1 (13) Hex2 (43) [Idh2 (2) Mdh2] --Wendel &, r670
Adk1 located by TB-6Sa; Adk1 (8) rgd1 (8) Pgd1 (4) Enp1 --Wendel &, 61:19
y1, py1, Tr6, Pd6, Krn6, Flt6, Zer2, Plp linkages --Miranda &, 61:32
gs3 on 6L --Neuffer &, 61:50
NPI330-Me2, 229-Pdk1 on 6L --Wright &, 61:89
Hypoploids for TB-6Lc in endosperm have reduced kernel weight, in plants reduced height and leaf width --Chang &, 61:91

CHROMOSOME 7
E1 allozymes in Corn Belt dents, flints and South American dent populations --Kahler &, r260
Flt7, Krn7, Fas7, Bn1, Ger2 linkages --Miranda &, 61:29
v27, wlu2 on 7L; Les9 on 7 --Neuffer &, 61:50
NPI349-Alr2, 419-Sod2 on 7L --Wright &, 61:89
Hypoploids for TB-7Lb in endosperm have reduced kernel weight, in plants short, erect, dark green leaves --Chang &, 61:91

CHROMOSOME 8
ms43 uncovered by TB-8La --Golubovskaya &, r177
Krn8, Fas8, Flt8, Ger3 linkages --Miranda &, 61:29,34
hcf*-1113-3, Mu-elicited; uncovered by TB-8Lc --Cook &, 61:44
v21, wlu3 on 8L; Clt1, Bif1, Sdw1 on 8; pro1 (33) ms8 (12) j1 (9) de*-1386A; Bif1 (27) pro1 (45) v16 (23) de*-1386A --Neuffer &, 61:50,51
NPI344-Tpi3 on 8S; 119-Hsp1 on 8L; 230-Pdk2 on 8 near centromere; 368-Act1 on 8 --Wright &, 61:89
Hypoploids for TB-8Lc have dark green, short, broad leaves --Chang &, 61:91

CHROMOSOME 9
bz1-E1, -E2, -E3, -E4, -E5, -E6, -E7, -E8, -E9, -E10, EMS-induced; -m2(DI), -m2(DII) from bz1-m2 --Dooner, r116; Dooner &, r118
Acp1 allozymes in Corn Belt dents, flints and South American dent populations; QTL associations in F2 from Wf9/Pa405 --Kahler &, r260, r261
wx1 RM, BS of genomic and cDNA --Klosgen &, r283
Css1 near centromere; RM --McCarty &, r369; Behrendsen &, 61:60
C1 RM; c1-m668655, -m668613 En insert, c1-m2 Ds insert --Paz-Ares &, r462
bz1-mu1, bz1-mu2 Mu1-elicited --Walbot &, r657
wx1, Flt9, Krn9, Ger4 linkages --Miranda &, 61:35
Sh1 5' region, DNaseI hypersensitive sites --Frommer &, 61:44
baf1, barren-stalk-fastigiate, uncovered by TB-9Sb, close to wl1; bz1 (<10) 17; bk2 (5) v30 (19) Wc --Coe &, 61:46
wlu4 on 9L; Zb8, Les8 on 9; G6 distal to TB-9Sb, G6 (18) wx1 --Neuffer &, 61:50,51
Ss2 close to bz1 --Gupta &, 61:57
QTL for plant height very close to d3; NPI-probe RFLP map for chromosome 9 --Helentjaris &, 61:88
NPI1-Bz1, 15Sh1, 16Wx1 on 9S; 332-Pep, 121-Css1 on 9L --Wright &, 61:89
Hypoploids for TB-9Lc in endosperm have reduced kernel weight, in plant reduced height, yellow green leaves --Chang &, 61:91

CHROMOSOME 10
r1-r:n46, n142, n35, n101 (ex R1-r:standard); r1-g:1557-2 (ex r1-r:n35); R1-sc:124 (ex R1-st); r1-m1, -m3, -m9 (Ds-elicited from R1-sc:124); r1-g:nc3-5 (derivative of R1-st); R1-g:8pale (variant of R1-g:8, ex R1-r:standard) alleles --Dooner &, r118
Sn1, derived from r1-ch:bol1, -bol2, -Co-op; 1.5 mu distal to R1 --Gavazzi &, r171; Consonni &, 61:75
Glu1 allozymes in Corn Belt dents, flints and South American dent populations; QTL associations in F2 from Wf9/Pa405 --Kahler &, r260, r261
Fatty acid composition affected by 10L dosage, TB-10L19 --Shadley &, r564
wx1-m1, Ds excisions Wx1-S5, -S9, RM, BS --Wessler, r674
Flt10, Krn10, Ger5 linkages --Miranda &, 61:34
orp2 (19) r1 --Neuffer &, 61:44
g121, 119, Les6 on 10S; 113, v29, Vsr1 on 10L --Neuffer &, 61:50
Hypoploids for TB-10L19 in endosperm have reduced kernel weight --Chang &, 61:91

UNPLACED
hcf*-Mu106, hcf*-Mu122 --Barkan &, r23
bno*-747B, brown opaque endosperm; cp*-1418, collapsed endosperm --Sheridan &, r573
y11, y12, mg1 --Sprague, 61:96
Ufo1, unstable factor for orange --Styles &, 61:100
sft1, small flint type --Dollinger, 61:103

CHLOROPLAST
atpB, BS; promoter deletion mutants map its structure --Bradley &, r52
rbcL 3' non-coding region hybridizes to junctions of inverted repeat --Brears &, r59
mtDNAS1 sequence homologies to ctDNA-psbA --Sederoff &, r560
psbG RM, BS --Steinmetz &, r592
rDNA5S, tRNA-Arg(ACG), tRNA-Asn(GUU) and intergenic regions, BS --Dormann-Przybyl &, r120

MITOCHONDRION
cms-T, RM, altered transcribed sequences --Abbott &, r1
cms-S, S2 plasmid, RM --Bedinger &, r30
BS of inverted repeats in cms-Vg S1 and S2 vs. main mtDNA and in cms-Vg369 (revertant) and normal --Braun &, r58
Map locations of coxI, coxII, cob, atpA, rRNA5S, rRNA18S, rRNA26S --Dawson &, r101
cms-T sequence TURF2H3, BS and hybridizations to atp6, rRNA26S, tRNAarg --Dewey &, r108
cms-RD81-47-13, -15, -16, 82-1179-16, 78-419-13; cms-ML81H-51-1, 78-409-7 revertants; S1, S2 plasmid content is nucleus-dependent --Escote &, r139
cms-S cytoplasms carry autonomously replicating RNA plasmids --Finnegan &, r152
Direct repeat 5.27kb, intragenomic recombination sites adjacent to R1(S1) and R2(S2): BS --Houchins &, r234
atpA, BS; 2 copies in B37N, 1 in T, C and S --Isaac &, r243
coxI, BS; Wf9N vs. S in region of R1 adjacent --Leaver &, r319
cms-1j1 isolated from progeny of ij plants --Lemke &, r321
1.9kb plasmid, BS --Ludwig &, r338
NCS2, NCS3 restriction fragment modifications --Newton &, r418; Feiler &, 61:45
S1 sequence homologies to ctDNA-psbA --Sederoff &, r560
URF13-T, ORF25, atp6 regions in N, cms-T, -C, -S --Kennell &, 61:57
ORF13T, ORF25 region BS in N, cms-T, and fertile revertant V3 --Fauron &, 61:90

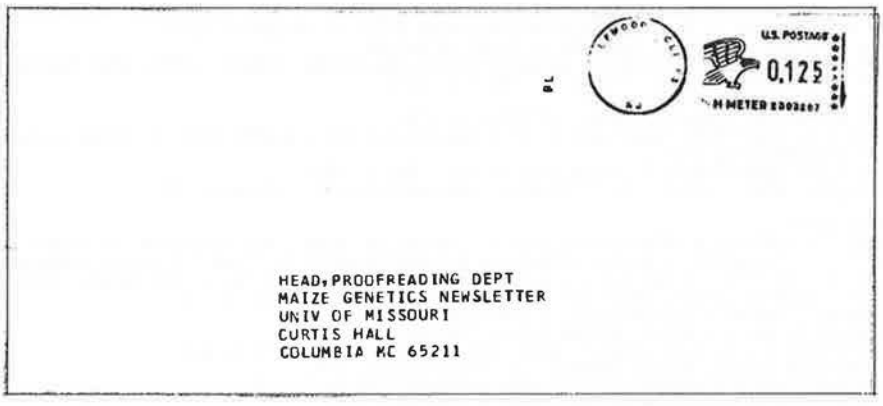
TRANSPOSABLE ELEMENTS (see also specific loci affected)
Ac, Ds9, Ds6, Ds2d2 maps; Spm-s, Spm-w, dSpm-8, dSpm-13, dSpm-7995, dSpm-7997, dSpm-8004 maps --Banks &, r20
Mu1.4B37 element, RM; modified relative to Mu1 --Chandler &, r72
Ac: defects in wx-m9(Ds), in ORF1, and bz-m2(DI), in ORF2, do not complement for transposition -Dooner &, r117
Ac, errata for BS --Muller-Neumann &, r 408; English &, 61:81
En-1 BS --Pereira &, r470
BS1, TZ86 insertions, Uq --Peterson, r473
Uq, Mrh in BSSS --Peterson &, r477

cDNA/GENOMIC CLONES/PROBES
PEP carboxylase clone pPC2 hybridizes to 1-2 two major bands; RM --Harpster &, r218
P-ePrv carboxylase clone pH1 hybridizes to 3+ bands; Prv,Pi dikinase clone pH2 to 2+ --Hudspeth &, r237
Triosephosphate isomerase clone hybridizes to 9+ bands; BS --Marchionni &, r352
Zein clones for Mr 22,000, 19,000 and 15,000, homologies among and between, RMs and BSs --Marks &, r355, r366; Pedersen &, r465; Wang &, r660, r661
Glutathione-S-transferase III clone hybridizes to 1 band; BS --Moore &, r393
Poly (dT-dG).poly(dC-dA), 6,500 copies --Morris &, r401
Histone H4, 2 clones, RM, BS; multiple dispersed repeats --Philipps &, r482
Repetitive sequences: knob probe pZmK6 185bp, tandem arrays; ARS probes pZmA1111 525bp, pZmA311 600bp, dispersed and tandem --Rivin &, r512
Heat-shock protein 70, BS --Rochester &, r516
Glutathione-S-transferase I clone hybridizes to 1 band in Mo17; RM, BS --Shah &, r565
LHCP-II: 6 clones isolated, hybridizing to 6 of a family of about 12 genomic sequences --Sheen &, r570

RESISTANCE/TOLERANCE/HERITABILITY
Selection for stalk strength in MoSQA and MoSQB associated with plant height, ear height, maturity, yield --Berzonsky &, r37, r38
Inbreds show 56% variation over 11 generations, more in selfed than in sibbed progenies (10 lines, 10 traits) --Bogenschutz &, r48
Bipolaris maydis race O R/S: up to 97% of variation additive, 2 to 47% dominance; BSH 30 to 69%; 2 to 15 factors --Burnette &, r66
R/S to bird damage correlated with husk weight, length and extension in sweet corns --Dolbeer &, r115
Allozyme selection; performance prediction by heterozygosity --Frei &, r156, r157
Kanamycin R transferred stably by electroporation into BMSI callus --Fromm &, r159
Gibberella zeae isolate U5373 R/S in 2 R vs. 2S inbreds: additivity predominates --Gendloff &, r172
Delayed senescence and high stalk sucrose, 1 major dominant gene --Gentinetta &, r174
Opine synthesis following Agrobacterium introduction into wounded seedlings --Graves &, r187
Maize streak virus DNA transferred via Agrobacterium inoculation --Grimsley &, r191
Recurrent selection in BS13 and BSSS2 for cold tolerance --Hoard &, r229, r230
Regenerability of hybrids dominant/semidominant --Hodges &, r231
Gibberella zeae R/S, % infected plants (IP), and spread (SI), BSH 0.73, 0.84, and 0.54; IP and SI separable --Hunter &, r238
Recurrent selection in Tuxpeno for reduced plant height --Johnson &, r254
Frequencies, genetic distances for allozymes in Corn Belt dents, flints and South American dent populations --Kahler &, r260

QTL associations with allozymes in F2 of Wf9/Pa405; segmental heterosis, overdominance --Kahler &, r261
Yield with grain-filling period, rate of fill and shorter lag period in diallel; gca > sca --Katsantonis &, r269
Ostrinia nubilalis R/S, recurrent selection in BS9 rapid for first-brood; less rapid for second-brood; yield reductions --Klenke &, r280, r282
High per se yield of random inbreds from BSSS partially dominant --Lamkey &, r304
Atrazine R/S BSH 0.66 in diallel --Le Court de Billot &, r315
Recurrent selection in BSSS for yield, *Ostrinia nubilalis* R, *Diabrotica virgifera* R --Oyervides-Garcia &, r439
Full-sib selection in 8 tropical populations, 6 traits, international testing --Pandey &, r448
Allozyme correlations in Hays Golden mass selected for yield or prolificacy --Pollak &, r491
Cold tolerance in diallel, gca > sca --Pozzi &, r497
Allozyme heterozygosity for prediction --Price &, r498
Selection for stalk strength, *Diplodia maydis* R, *Ostrinia nubilalis* R, DIMBOA --Rehn &, r508
Corynebacterium nebraskense R/S, 9:7 ratio from A619/A632 --Schuster &, r557
Full-sib selection in Partap for prolificacy --Singh &, r577
Selection for early flowering --Troyer, r632
Methotrexate R selection in tissue culture --Tuberosa &, r636

--Assembled unrestricted by Prof. Ligate



IV. MAIZE GENETICS COOPERATION STOCK CENTER

In the immediate future high priority will be given to the development of the most useful stocks in immediately usable condition. In part this will involve an effort to derive stocks in more vigorous backgrounds, particularly through the intercross of stocks of similar genetic marking followed by re-extraction of parental or new combinations. As an adjunct to this effort, it will be important to develop improved gene combinations incorporating both old and new marker genes. Greater emphasis will be placed on deriving and perpetuating alleles and chromosomal variants in homozygous condition or by testcrosses.

It is important that you be reminded that many of you have valuable genetic stocks that have not yet been submitted to the stock center. Usually, the most appropriate and convenient time to send in stocks occurs as soon as you are willing to share them or at such time as you cease active work with them. It is particularly important that seed stocks be submitted while pedigree information is newly verified and seed viability is at its maximum.

Several other guidelines for submission of seed stocks are appropriate. If feasible, stocks should be provided in homozygous condition if classification of traits in segregating progenies requires cytological or biochemical observations, or if other extended capabilities are necessary. For example, the most useful, and immediately usable, stocks of defined chromosome knob constitution would consist of inbred lines homozygous for knobs at designated locations. Similarly, stocks of isozyme alleles might best be provided in homozygous condition. A moment's reflection may suggest to you the most suitable genotype for perpetuation; it would usually be that genotype that you would prefer if you were requesting the stock. Any additional information or documentation you can provide would be welcome. If known, indicate which genes can be recommended for general linkage studies, and which traits have exhibited a disquieting range of penetrance or expressivity. Finally, if certain stocks are partially converted to inbred line background, that fact should be noted in the pedigree in order that the background might be preserved and the conversion continued.

We continue to welcome constructive suggestions for improving the stock center program. Over time, as the directions and emphasis of research activities are modified, we will need the help of each of you in ensuring that our activities and priorities remain responsive to your needs.

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Chromosome 1

101A sr zb4 P-WW **
101B sr P-WR **
101C sr P-WW
101D sr P-RR
101E sr zb4 P-WR
101F sr P-RR ts2
101K sr an
102A sr P-WR an gs bm2 **
102B sr P-WR an bm2 **
102C sr P-RW ad bm2 **
103A sr P-RR an bm2
103B sr P-RR gs bm2
103C sr P-WR bm2
103D vp5
103E zb4 ms17 P-WW
103F zb4 ms17 P-WW rs2
103G sr P-RR bm2 **
104A zb4 ts2 P-WW br f bm2
104B zb4 ts2 P-WW bm2
104C zb4 ts2 P-WW br f
104E zb4 ts2 P-WR br f
105A zb4 P-WW **
105C zb4 P-WW br **
105D zb4 P-WW br f bm2
105E ms17
106A zb4 P-WW bm2
106B ts2 P-RR
106C ts2 P-WW bm2
106D ts2 P-WW br bm2
106E ts2 P-WW br f bm2
106G ts2 br f bz2 bm2 ACR
107A P-CR **
107B P-RR **
107C P-RW **
107D P-CW **
107E P-MO **
107F P-VV **
107G P-OR
108A P-RR as br f an gs bm2
108B P-RR br f an gs bm2 rd
108C P-RR br f an gs bm2
108D P-RR br f an gs bm2 id
108E P-RR br f an gs bm2 v8983
108F P-RR br f an gs bm2 v8943
109A P-RR an ad bm2
109B P-RR an gs bm2 **
109C P-RR br f an ad gs bm2
109D P-RR ad bm2 **
109E P-WR br f
109F P-RR br
110A P-WR an Kn bm2
110B P-WR an Kn
110C P-WR an ad1 bm2
110D P-WR an bm2
110E P-WR ad bm2
110F P-WR br Vg
110G P-WR br f gs bm2
110H P-WR br f bm2
110J P-WR an br2 bm2
110K P-WR br
110L P-WR br f Kn Ts6 bm2
110M P-WR br an Kn bm2
110N P-WR br2 bm2
111A P-WW rs2
111B P-WW rs2 br
111C P-WW as br f bm2
111D P-WW hm br f
112A P-WW br f ad bm2
112B P-WW br f bm2 **
112C P-WW br f an gs bm2
112D P-WW br Vg
112E as
112H P-WW br **
113A as br2
113B rd
113C br f **
113D br f bm2 v5588
113E br f Kn
114A br f Kn Ts6
114B br f Kn bm2
114C br bm2
114D Vg
115A Vg an bm2
115B Vg br2 bm2
115C v22
115D bz2-m: A A2 C Pr
115I Vg id bm2
116A bz2-m: A A2 C R Pr
116B bz2 ad bm2: A C R
116C an bm2
116D an-bz2 6923 (Df)
116E an br
117A br2 **
117B br2 bm2
117C br2 an bm2
117D tb-8963
117E Kn

(continued)

117G br2 ts2
118A Kn Ts6
118B Kn bm2
118C lw
118D Kn Ts6 bm2
118H rs2 Kn Ts6
119A Adh1-S
119B vp8
119C gs **
119D gs bm2 **
119E Ts6
119F bm2 **
120A id
120B nec2
120C ms9
120D ms12
121A ms14
121B mi
121C D8
121D Lls
121E tiny
122A TB-1La (1L.20)
122B TB-1Sb (1S.05)
125A Les2

Chromosome 2

201A ws3 lg gl2 B
201B ws3 lg gl2 B sk
201C ws3 lg gl2 B sk v4
201D ws3 lg gl2 B sk fl v4
201E ws3 lg gl2 B ts
201F ws3 lg gl2 b
202A ws3 lg gl2 b sk v4
202B ws3 lg gl2 b gs2 v4
202C ws3 lg gl2 fl v4
202D ws3 lg gl2 b sk fl v4
202E ws3 lg gl2 b v4
203A ws3 lg gl2 B gs2 v4
203B al
203C ws3 lg gl2 b sk
203D al lg
203E al lg gl2 B sk v4
204A al lg gl2 b sk v4
204C al lg gl2 B ba2
204F al lg gl2 B sk
205B lg
205C lg gl2
206A lg gl2 B
206B lg gl2 B gs2
206C lg gl2 B gs2 v4
206D lg gl2 B gs2 Ch
206E lg gl2 B gs2 sk Ch
206F lg gl2 B Pl
207A lg gl2 B gl11
208A lg gl2 B gs2 sk v4
208B lg gl2 B sk
208C lg gl2 B sk v4
208D lg gl2 B v4
208E lg gl2 b **
208F lg gl2 b gs2 **
208G lg gl2 b gs2 Ch **
208H gl2 **
209A lg gl2 b gs2 sk Ch
209B lg gl2 b gs2 v4
209C lg gl2 b gs2 v4 Ch
209E lg gl2 b sk
209F lg gl2 b sk fl
209G lg gl2 b sk fl v4
210A lg gl2 b sk v4
211A lg gl2 b fl
211B lg gl2 b gs2 wt **
212A lg gl2 b wt v4 **
212B lg gl2 b fl v4 **
212C lg gl2 b fl v4 Ch **
212D lg gl2 b v4
212E lg gl2 b v4 Ch **
213A lg gl2 mn v4
213B lg gl2 wt **
213C lg gl2 w3
213D lg gl2 w3 Ch
213E lg gl2 b Ch **
214A lg b gs2 v4
214B lg gl2 Ch
214C d5
214D B gl11
214E B ts
215A gl14
215B gl11 **
215C wt
215D mn
215E fl **
215F fl alleles from PI
216A fl v4 Ch
216B fl Ht v4
216C fl Ht v4 Ch

(continued)

216D fl w3
216E fl v4 w3
216F fl w3 Ch
216G fl v4 w3 Ch
217A ts
217B v4 **
217C v4 w3 Ht Ch
217D v4 Ht Ch
217E w3 Ht Ch
218A w3
218C w3 Ch
218D Ht (source A and B)
218E ba2
219A R2: r A A2 C
219B r2: r-g A A2 C
219C Ch
219D Ht Ch
219E w3 ba2
220A Les
220B 2 2T T2/ ws3 lg gl2 (T=Tripsacum)
221A gs2 **
222A TB-1Sb-2L 4464
222B TB-3La-2S 6270
223A Primary Trisomic 2
224A w4670
224B v5537
224F w 062-3
224G yel 8630
224H white pollen (Coe)

Chromosome 3

301A cr **
301B cr d **
301C cr d Lg3
301E cr ts4 na lg2
302A d=d-6016 (rosette)
302B d rt
302E d (tall)
303A d rt Lg3
303B d Rf lg2 **
303F g2=v19=pg14 **
304A d ys3
304B d ys3 Rg
305A d Lg3
305D d Rg
306A d Rg ts4 lg2
306D d Rg ts4
307A d pm
307C pm **
308A d ts4 lg2 a-m: A2 CR Dt
308B d ts4 **
308C d lg2 a-m A2 C R Dt
308D d a-m A2 C R Dt **
308E ra2 **
308G d ts4 a-m: A2 C R Dt
309A ra2 Rg ts4 lg2
309B ra2 ys3 Lg3 Rg
309D ra2 Rg lg2
309E ra2 pm lg2
309F ra2 Lg3 ys3
310A ra2 ts4 **
310C ra2 lg2 **
310D Cg
311A cl
311B cl : Clm2
311C cl: Clm3
311D cl-p: Clm4
311E rt
311F ys3 **
311G ys3 Lg3
312C ys3 ts4 lg2
312D Lg3
316A ts4 **
316B ts4 na
317A ts4 na pm
317B ts4 ba na
317C ts4 lg2 a-m A2 C R Dt
317D ts4 na a-m et: A2 C R Dt
318A ig **
318B ba
318C w7748=y10
319A lg2 A-b et: A2 C R Dt
319B lg2 a-m sh2 et: A2 C R Dt
319C lg2 a-m et: A2 C R dt **
319D lg2 a-m et: A2 C R Dt **
319E lg2 a-st sh2 et: A2 C R Dt **
319F lg2 a-st et: A2 C R Dt **
320A lg2 **
320C na lg2
320D A sh2: A2 C R B Pl dt
320F A sh2: A2 C R b pl
321A A-d31: A2 C R
322A A-d31 sh2: A2 C R B Pl dt
322B A-d31 sh2: A2 C R Dt
322E a-m: A2 C R B Pl dt **
322F a-m: A2 C R b pl dt **

** Some of these stocks are available in a homozygous state.

(continued)

323A a-m: A2 C R Dt **
323B a-m: A2 C R B Pl Dt **
323C a-m sh2: A2 C R B Pl dt
323D a-m sh2: A2 C R B Pl Dt
323E a-m et: A2 CR Dt **
324A a-st: A2 C R Dt **
324B a-st sh2: A2 C R Dt **
324D a-st sh2 et: A2 C R Dt
324E a-st et: A2 C R Dt **
324F a-p sh2 et: A2 C R B Pl Dt
324G a-st: A2 C R dt **
325A a-p et: A2 C R dt **
325B a-p et: A2 C R B Pl Dt **
325C a-x1
325D a-x3
325E a Ga7: A2 C R
325G a3 **
326A sh2
326B vp
326C Rp on 3 **
326D te **
326E yel 5787
327A TB-3La (31.10)
327B TB-3Sb (3S.50)
327C TB-3Lc
327D TB-3Ld
328A Primary Trisomic 3
330A h **
331A TB-1La-3L5267
331B TB-1La-3L4759-3
331C TB-1La-3L5242
331E TB-3Lf
331F TB-3Lg
331G TB-3Lh
331H TB-3Li
331I TB-3Lj
331J TB-3Lk
331K TB-3Ll
331L TB-3Lm

Chromosome 4

401A Rp4
401B Ga **
401C Ga su **
401D Ga-S **
401E Ga-S: y
401F Ga-S: A A2 C F
402A st **
402B st Ts5
402C st f12 **
402D Ts5
403A Ts5 f12
403B Ts5 su
403C Ts5 la su gl3
403D Ts5 la su gl3 ol
404A Ts5 su zb6
404B Ts5 su zb6 o
404C Ts5 su gl3 o
405A Ts5 Tu
405B la
405D la su gl3
406A la su gl3 o
406B la su bt2 gl3
406C f12 **
406D f12 su **
407B f12 su bm3 **
407C f12 su Tu gl4
407D su **
407E su-am **
407F su-am du
408A su bt gl4 **
408B su bm3 **
408C su zb6 **
409A su zb6 Tu **
410A su zb6 gl3 dp **
410D su zb6 gl3 **
411A su gl4 j2
411B su gl4 o **
412E su j2 gl3 **
414B gl4 **
414C gl4 o **
412A su gl4 o Tu
412B su j2 **
412C su gl3 **
412D su gl3 o **
413A su o **
413B su gl4 **
414A bt2 **
408E bm3 **
415A j2
415B j2 c2: A A2 C R
415C j2 C2: A A2 C R
416A Tu
416B Tu-1 1st
416C Tu-1 2nd
416D Tu-d
416E Tu-md
416F Tu gl3
417A j2 gl3

(continued)

417B v8 **
417C gl3 **
417D gl3 o
418A gl3 dp
418B c2: A A2 C R **
418C C2: A A2 C R **
418D C2-Idf (Active-1) : A A2 C R **
418E dp **
418F o **
418G v17 **
419A v23
419B su gl3 ra3
419F a1 Dt6 gl3
420A Dt4 su: a-m A2 C R
420B TB-4L-9S 6504
420H Dt4: a-m A2 C R
420I TB-4L-9S 6222
421A TB-4Sa (4S.20)
421B TB-1La-4L4692
421C TB-7Lb-4L4698
422A Primary Trisomic 4
423A TB-4Lb
423B TB-4Lc
423C TB-4Ld
423D TB-4Le
423E TB-4Lf

Chromosome 5

501A am a2: A A2 C F
501B lu **
501C lu sh4
501D ms13
501E gl17
501F gl17 A2 pr: A C R
501H gl17 a2 bt: A C R
501K gl17 v3
501L lu a2 bm pr A C R
502A gl17 a2 bt v2: A C R
502B A2 vp7 pr: A C R
502C A2 bm bt pr ys: A C R
502D A2 bm pr: A C R
503A A2 bm pr ys: A C R **
503D A2 bt v3 pr: A C R
504A A2 bt pr:A C R
505A A2 bt pr ys in: A C R
506A A2 v3 pr: A C R **
506B A2 pr: A C R **
506C A2 pr v2: A C R **
506D A2 pr na2: A C R
505B A2 pr ys: A C R **
506E A2 pr zb3: A C R **
506F A2 pr v12: A C R **
506L A2 pr br3: A C R
507A a2: A C R **
508A a2 bm bt pr: A C R **
508B a2 bm bt pr ys: A C R
509C a2 bm pr v2 sh4 : A C R
510A a2 bm pr v2: A C R **
510B A2 bm pr eg: A C R **
511A a2 bt v3 pr: A C R **
511B a2 bt v3 Pr: A C R
511C a2 bt pr: A C R **
512A a2 bt v2: A C R
512B a2 v3 pr: A C R
513A a2 pr: A C R **
513B a2 pr: A C R B Pl
513C a2 pr v2: A C R
515A vp2
515C vp7
515D bm **
516A bm yg Ch
516B bt **
516C ms5
516G bm pr yg: A C R
517A v3 **
517B ae **
516D td ae
518A sh4
518B gl8 **
518C na2
518D lw2
518F sh4 v2 **
519A ys **
519B eg **
519C v2 **
519D yg **
519E pr yg: A C R **
520B v12 **
520C br3 **
521A nec3
522A TB-5La
522B TB-5Lb
522C TB-5Sc
523A Primary Trisomic 5

Chromosome 6

601A rgd po y
601B rgd po Y
601C rgd y
601D rgd Y
601E po = ms6
601F po y pl
601G po y Pl
602A po y wi
602B po y pl
602C y = pb = w-n
602D y rhm
602E po y wi pl
602F po y wi pb4 pl
602G po y wi pb4 Pl
603A y 110
603B y 111
603C y 112
603D y w15
603E y pb4
604A y pb4 pl **
604B y pb4 Pl
604F y ms-si
604G y wi pb4 Pl
604H y ms
605A y wi Pl
605C y pg11 : Wx pg12
605D y pg11 wi : wx pg12
605E Y wi Pl
605F Y wi pl
606A Y pg11: Wx pg12
606B y pg11 : wx pg12 **
606C Y pg11 : wx pg12
606D y pg11 su2 : wx pg12
606E y pl
606F y Pl
607A y Pl Bh : c sh wx A A2 R **
607B y pl Bh : c sh wx A A2 R **
607C y su2 **
608A y 110
608B Y 112
609A Y pb4 **
609B Y wi pl **
609C Y wi Pl **
609D Y su2 **
610A wi
610B Pl Dt2 : a-m A2 C R **
610C pl sm : P-RR **
611A Pl sm : P-RR **
611B Pl sm py : P-RR
611C Pl sm Pt py : P-RR
611D Pt
611E w
611F Pl sm Pt:P-RR
612A w14
612B ms6
612C 1*-4923
612D oro
613A 2NOR : a2 bm pr v2
614A TB-6Lb
614B TB-6Sa
614C TB-6Lc
615A Primary Trisomic 6

Chromosome 7

701A Hs o2 v5 ra gl
701B In-D
701C In-D gl
701D o2 **
702A o2 v5 **
702B o2 v5 ra gl
702C o2 v5 ra gl sl
702D o2 v5 ra gl Tp
702E o2 v5 ra gl ij
703A o2 v5 gl **
703B o2 v5 ra gl Pn
703C o2 v5 gl ms7
703D o2 ra gl
704A o2 ra gl ij
704B o2 ra gl sl
704C o2 v5 gl sl
705A o2 gl
705B o2 gl sl **
705C o2 ij
705D o2 bd
706A o2 sl
706B o2 ij bd
707A y8 v5 gl
707B in : A2 pr A C R
707C in gl : A2 pr A C R
707D v5 **
707E vp9
707F y8 gl **
708A ra
708B ra gl ij bd
709A gl **
712B ms7 gl Tp
710A gl Tp
710B gl mn2

** Some of these stocks are avialable in a homozygous state.

(continued)

710E gl o5 = pg
711A Tp
711B ij **
711C gl sl va
712A ms7
713A Bn
713B bd
713C ms7 ra gl ij
714A Pn
714B o5 **
714C o5 mn2 gl
714D va **
715A Dt3: a-m A2 C R
715B o2 ra gl Dt3: a-m A C R
715C gl Dt3: a-m A C R
716A v*-8647
716B yel*-7748
717A TB-7Lb (7L.30)
718A Primary Trisomic 7
719A TB-7Sa

Chromosome 8

801A gl18 **
801B v16 **
801D v16 ms8 j
801E v16 ms8 j nec
801F v16 j gl18 **
801G v16 gl18
802A v16 ms8 j gl18
802B v16 ms8 j yel0245
803A ms8
803B nec
803C ms8 j gl18
803D ms8 gl18
804A v21 **
805A fl3 **
805B nec v21
805C gl18 v21
805D fl3 j ms8
806A TB-8La
806B TB-8Lb
807A Primary Trisomic 8
809A TB-8Lc

Chromosome 9

901B yg2 C sh bz: A A2 R
901C yg2 C sh bz wx: A A2 R **
901D yg2 C-1 sh bz wx: A A2 R
901E yg2 C bz wx: A A2 R **
902A yg2 c sh bx wx: A A2 R **
902B yg2 c sh wx: A A2 R **
902C yg2 c sh wx gl15: A A2 R **
902D yg2 c sh wx gl15 K-S9: A A2 R
902E yg2 c bz wx: A A2 R
924A wd-Ring C-1: A A2 R
903A C sh bz: A A2 R **
903B C sh bz wx: A A2 R **
903D C-I sh bz wx: A A2 R **
904A C sh bz wx gl15 bm4: A A2 R
904B C sh: A A2 R **
904C C sh wx: A A2 R
904D C wx ar: A A2 R **
904E C sh bz wx bm4: A A2 R **
904F C sh bz gl15 bm4: A A2 R **
905A C sh wx K-L9: A A2 R **
905B C sh ms2: A A2 R
905C C bz Wx: A A2 R **
905D C sh wx K-L9: A A2 R K-10
905E C sh wx v: A A2 R **
906A C Ds wx: A A2 R Pr y **
906B C Ds wx: A A2 R pr Y **
906C C-I Ds Wx: A A2 R **
906D C-I: A A2 R **
907A C wx: A A2 R **
907B C wx: A A2 R B Pl **
907C C wx: A A2 R b Pl **
907D C wx: A A2 R B pl **
907E C-I wx: A A2 R y **
907F C-I wx: A A2 R y B pl **
908A C wx ar da: A A2 R **
908B C wx v: A A2 R **
908C C wx v: A A2 R Pl **
908D C wx gl15: A A2 R **
908E C wx gl15: A A2 R pr **
908F C wx da:A R
908G C wx ar: A R
909A C wx Bf: A A2 R **
909B c bz wx: A A2 R **
909C c sh bz wx: A A2 R y **
909D c sh wx: A A2 R **
909E c sh wx v : A A2 R **
909F c sh wx gl15: A A2 R **
909G c sh wx ms2: A A2 R
910A c sh wx gl15 bk2: A A2 R
910B c sh wx gl15 Bf: A A2 R **
910C c sh wx bk2: A A2 R **

(continued)

910D c: A A2 R **
910E c sh wx gl 15 16 **
910F c sh wx gl15 bm4: A A2 R
911A c wx: A A2 R y **
911B c wx v: A A2 R **
911C c wx gl15: A A2 R **
911D c wx Bf: A A2 R **
912A sh **
912B sh wx v **
912E lo2
913A sh wx **
913B sh wx v gl15: A A2 C R **
914A wx d3 **
914D wx d3 gl15
915A wx* **
915B wx-a **
915C w11
915D wx pg12 bm4: y pg11
916A wx v **
916B wx v gl15 bk2 Bf bm4
916C wx bk2 **
916D wx bk2 bm4 **
917A wx Bf **
917C v **
917D ms2
917E gl15 **
917F d3
918A gl15 Bf **
918B gl15 bm4 **
918C Wc bk2
918D Wc **
918E Wx bk2 bm4 **
918F Wx Bf
919A bm4 **
919B bm4 Bf **
919C 16
919D 17
919E wx 17
919F 16 wx
919G 16 11
919H 17 11
920A yel*-034-16
920B w*-4889
920C w*-8889
920E w*-8950
920F w*-9000
920G Tp9 N9 N3 Df3
921A TB-9La (9L.40)
921B TB-9Sb (9S.40)
921C TB-9Lc
921D TB-9Sd
922A Primary Trisomic 9

Chromosome 10

X01A oy
X01B oy R: A A2 C **
X01C oy bf2 **
X01E oy bf2 R: A A2 C **
X01F oy bf2 du
X02A oy ms11
X02D oy du R: A A2 C **
X02E oy du r: A A2 C
X02G oy zn
X02H oy ms10
X02I oy bf2 ms10
X03A sr3
X03B Og **
X03C Og B Pl **
X03D Og: A C R **
X04A Og du R: A C R **
X04B ms11
X04C ms11 bf2
X04D bf2 **
X04E C-I Og B Pl
X05A bf2 zn **
X05B bf2 li g r: A A2 C
X05C bf2 g R sr2: A A2 C **
X05E bf2 sr2
X05F bf2 ms10
X06A bf2 r sr: A A2 C **
X06C nl g R: A A2 C **
X07A nl g r: A A2 C **
X07B nl g R sr2: A A2 C
X07C Y9
X07E nl g r sr2:A C
X07D nl
X09A li zn g r: A A2 C
X09B li g R: A A2 C **
X09F ms10
X09G li g r :A C **
X09H li g r v18:A C
X10A du **
X10C du o7 **
X10D du g r: A C R **
X10F zn **
X10G du v18
X11A zn g **

(continued)

X11B zn g R sr2: A A2 C
X11C zn g r: A A2 C **
X11D Tp2 g r: A A2 C **
X11E g R sr2: A A2 C **
X11F g r: A A2 C **
X12A g r sr2: A A2 C
X12D g R-g sr2 v18: A A2 C
X12E g R:A A2 C
X12F g r-ch K10:A A2 C **
X12G K10 10B B10
X13A g R-g K10: A A2 C
X13B g R-g sr2: A A2 C **
X13D g r-r sr2: A A2 C **
X13F g r-r:A C wx
X13G g r-g:A C
X14A Ej r-r: A A2 C **
X14B g R v18:A C
X15A Ej r-r sr2: A A2 C **
X15C R-g: A A2 C **
X16A r-g sr2: A A2 C
X16B r K10: A A2 C
X16C R-ch :A A2 C B pl **
X17A r-g: A A2 C **
X15D r-ch Pl: A a2 C **
X17B r-r: A A2 C **
X17C R-mb: A A2 C **
X17D R-nj: A A2 C **
X17E R-r: A A2 C **
X17F R-nj purple embryo Chase **
X18A R-lsk: A A2 C **
X18B R-sk-nc 2: A A2 C **
X18C R-st: A A2 C **
X18D R-sk: A A2 C **
X18E R-st Mst **
X18F R-st Mst o7
X18G R-scm2: bz2 A A2 C C2 **
X25A R-scm2: a-st A2 C C2 **
X25B R-scm2: c2 A A2 C **
X25C R-scm122: pr A A2 C C2 **
X25D R-scm2: a2 A C C2 **
X25E R-scm2: c A A2 C2 **
X19a Lc **
X19B w2
X19C w2 l
X19D o7 **
X20A o7: o2 **
X20B l **
X20C v18 **
X20E l yel*-5344
X20F yel*-8721
X20G yel*-8454
X20H yel*-8793
X21A TB-10La (10L.35)
X22A TB-10Sc
X21B TB-10L19
X23A Primary Trisomic 10

** Some of these stocks are available in a homozygous state.

Unplaced Genes

U235A dv
U235B dy
U335A el
U435A l4
U635A Rs
U533A v13
U935A ws ws2
UX35A zb
UX35B zb2
U934B zn2
U734A nec*-8376
U933A o9
U933B o10
U933C o11
U933D o13

Multiple Gene Stocks

M141A A A2 C C2 R-g Pr B Pl
M141B A A2 C C2 R-g Pr B pl
M141C A A2 C C2 R-g b Pl
M241A A A2 C C2 r-g Pr B Pl
MX17A A A2 C C2 r-g Pr b pl
M241B A A2 C C2 r-g Pr B pl
M341A A A2 c C2 R-g Pr B pl
M241C A A2 C C2 R-r Pr B Pl
M341B A A2 C C2 R-r Pr B pl
M341C A A2 C C2 R-r Pr b Pl
M441A A A2 C C2 R-r Pr B Pl wx
M441B A A2 C C2 R-r Pr B pl wx
M441C A A2 C C2 R Pr
M641A A A2 C C2 R Pr wx
MX41A A A2 C C2 R pr y wx gl
M941A A A2 c C2 R Pr y wx
M741A A A2 C C2 r Pr Y wx
M341D A A2 c C2 R-r Pr B Pl
M441D A A2 C C2 r-r Pr B Pl
M441E A A2 c C2 r-r Pr B Pl
MX41B su pr y gl wx : A A2 C C2 R
M841A A su pr : A2 C C2 R
MX41C bz2 a c2 a2 pr Y/y c bz wx r
M841B a su A2 C C2 R
MX40A bm2 lg a su pr y gl j wx g
M841C colored scutellum
MX41D a su pr y gl wx A A2 C C2 R
MX40B ts2 : sk
MX40C lg gl2 wt : a-m A2 C C2 R Dt
M741B A A2 C C2 R-nj : purple embryo
S. Chase
M741C Stock 6 : Hi-haploid R-r $\overline{B}$ Pl

Popcorns

P142A Amber Pearl
P142B Argentine
P142C Black Beauty
P242A Hulless
P242B Ladyfinger
P242C Ohio Yellow
P342A Red
P342B Strawberry
P342C Supergold
P342D South American
P442A Tom Thumb
P442B White Rice

Exotics and Varieties

E542A Black Mexican Sweet Corn
(with B-chromosomes)
E542B Black Mexican Sweet Corn
(without B-chromosomes)
E642A Knobless Tama Flint
E442A Gaspe Flint
E642B Gourdseed
E742A Maiz Chapalote
E742B Papago Flour Corn
E742C Parker's Flint
E842A Tama Flint
E842B Zapalote Chico

Tetraploid Stocks

N103A P-RR
N103B P-VV
N103C a A2 C R Dt
N104A su
N104B pr : A A2 C R
N105A y
N106A gl
N106B Y sh wx
N106C wx
N107A g A A2 C R
N102A A A2 C R Pr B Pl

Cytoplasmic traits

C738A NCS2
C738B NCS3

Cytoplasmic steriles and Restorers

C836A	WF9-(T)	rf rf2
C836B	WF9	rf rf2
C736A	R213	Rf rf2
C736B	Ky21	Rf Rf2

Waxy Reciprocal Translocations

WX01A wx 1-9c (1S.48: 9L.22) * Sx
WX02A wx 1-94995 (1L.19: 9S.20) * Sx
WX03A wx 1-98389 (1L.74: 9L.13) * Sx
WX05A wx 2-9b (2S.18: 9L.22) * Sx
WX08A wx 3-9c (3L.09: 9L.12) * Sx
WX13A wx 4-9b (4L.90: 9L.20) * Sx
WX12A wx 4-95657 (4L.33: 9S.25) * Sx
WX11A wx 4-9g (4S.27: 9L.27) * Sx
WX17A wx 5-9a (5L.69: 9S.17) * Sx
WX14A wx 5-9c (5S.07: 9L.10) * Sx
WX19A wx 6-9a (6S.79: 9L.40) * Sx
WX20A wx y 6-9b (6L.10: 9S.37) * Sx
WX23A wx 7-9a (7L.63: 9S.07) * Sx
WX22A wx 7-94363 (7 cent: 9 cent) * Sx
WX24A wx 8-9d (8L.09: 9L.16) * Sx
WX25A wx 8-96673 (8L.35: 9L.31) * Sx
WX27A wx 9-10b (9S.13: 10S.40) * Sx

Non-waxy Reciprocal Translocations

WX30A Wx 1-9c (1S.48: 9L.24) * Sx
WX30B Wx 1-94995 (1L.19: 9S.20) * Sx
WX30C Wx 1-98389 (1L.74: 9L.13) * Sx
WX31A Wx 2-9c (2L.49: 9S.33) W23 only
WX31B Wx 2-9b (2S.18: 9L.22) * Sx
WX32A Wx 3-98447 (3S.44: 9L.14) *
WX32B Wx 3-98562 (3L.65: 9L.22) * Sx
WX33A Wx 4-9e (4S.53: 9L.26) * Sx
WX33B Wx 4-95657 (4L.33: 9S.25) * Sx
WX34A Wx 5-9c (5S.07: 9L.10) * Sx
WX34B Wx 5-94817 (5L.69: 9S.17) M14 only
WX35A Wx 5-98386 (5L.87: 9S.13) * Sx
WX36A Wx 6-94778 (6S.80: 9L.30) * Sx
WX37A Wx 6-98768 (6L.89: 9S.61) * Sx
WX37B Wx 7-94363 (7 cent: 9 cent) *
WX38A Wx 7-9a (7L.63: 9S.07) * Sx
WX38B Wx 8-9d (8L.09: 9L.16) * Sx
WX38C Wx 8-96673 (8L.35: 9S.31) * Sx
WX39A Wx 9-108630 (9S.28: 10L.27) M14 only
WX39B 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

I143A Inv.1a (1S.30-L.50)
I143B Inv.1c (1S.35-L.01)
I143C Inv.1d (1L.55-L.92)
I143D Inv.1L-5131-10 (1L.46-L.82)
I444A Inv.2a (2S.70-L.80)
I243A Inv.2S-L8865 (2S.06-L.05)
I243B Inv.2L-5392-4 (2L.13-L.51)
I343A Inv.3a (3L.38-L.95)
I343B Inv.3L (3L.19-L.72)
I343C Inv.3L-3716 (3L.09-L.81)
I443A Inv.4b (4L.40-L.96)
I443B Inv.4c (4S.86-L.62)
I543A Inv.4e (4L.16-L.81)
I743A Inv.5-8623 (5S.67-L.69)
I743B Inv.6-8452 (6S.77-L.33)
I843A Inv.6-8604 (6S.85-L.32)
I743C Inv.6-3712 (6S.76-L.63)
I943A Inv.7L-5803 (7L.17-L.61)
I943B Inv.7-8540 (7L.12-L.92)
I943C Inv.7-3717 (7S.32-L.30)
IX43A Inv.8a (8S.38-S.15)
I344A Inv.9a (9S.70-L.90)
IX43B Inv.9b (9S.05-L.87)
IX43C Inv.9c (9S.10-L.67)

CHROMOSOME 1L

I continue to work on mapping new genes that have been localized to 1L, and on mapping known morphological markers to isozyme loci, cytogenetic breakpoints and RFLP clones.

EMS-induced mutants provided by M. G. Neuffer:

wl*-266A has a lethal albino phenotype and is loosely linked to bz2. Data from an F2 progeny in repulsion:

+ +	+ bz2	wl* +	wl* bz2	T
247	127	131	11	516

X^2 =36.1, p <.000; r=.26±.04

No linkage was found, however, with either wx T1-9(4995) (r=.46±.05, 276 F2 progeny) or wx T1-9(8389) (r=.49±.05, 199 F2 progeny).

dek22 (cp*-1113A) has a lethal collapsed-kernel phenotype and is apparently unlinked to bz2 and to wx T1-9(8389). No distortions of an expected 3:1 ratio were found in segregating F2 progeny.

wl*-709B has a variable phenotype, from pale green to white. I have not been able to get Mendelian segregation with this mutant.

white*-495A and white*-571C are albino lethals and have been shown to be allelic by a complementation test. F2 mapping data will be available this spring.

ad*-582 is not allelic to the standard ad1 locus on 1L. Testcross and F2 mapping data will be taken this summer.

zb*-101 has a zebra-striped non-lethal phenotype; mapping data will be available after next summer.

Other unmapped morphological markers:

br2: Scoring of 192 F2 progeny failed to uncover a crossover with hm. It is likely that the two are linked.

rd1: Freeling has reported close linkage to Adh1 (MNL 60:146, 1986). A testcross of bz2 rd1 gs1 in coupling will be scored this summer.

Isozyme-morphological marker mapping:

gs1 bm2 vs. Phi1 Acp4: Data from 200 testcross progeny give the following approximate distances (see MNL 61:86):

Centromere - gs1 (14.0) Phi1 (10.5) bm2 (15.5) Acp4

This makes Acp4 the most distal marker on 1L mapped to date. It also suggests that Dia2 may be slightly distal to bm2. A testcross between Dia2 and Ts6 bm2 is being set up. Testcrosses will be scored next summer for br1 bz2 vs. Amp1 Mdh4 Pgm1 and bz2 gs1 bm2 vs. Adh1 Phi1 Gdh1 Acp4.

Waxy translocation stocks:

Crossing-over, wx-T, from MNL 39:106, 1965, for T1-9(4995)(1L.19) is 2.6% (14/542 plants); for T1-9(8389)(1L.74), 3.3%. My data from testcross, for T1-9(035-10)(1L.89), show 0.7% (1/144 plants).

Mapping vs. restriction fragment length polymorphisms (RFLP's):

Total nuclear DNA was extracted from immature ears of the 200 plants scored in the gs1 bm2 vs. Phi1 Acp4 testcross (MNL 61:86). Southern blots of the DNA restricted with HindIII and EcoRI will be probed with a series of 1L markers kindly provided by T. Helentjaris of NPI and T. Murphy of Northrup King. This laboratory will also be creating a set of publicly available RFLP markers. This is in response to a request to the USDA, ARS by the mapping subcommittee of the Maize Genetics Cooperation.

Segmental trisomics:

A set of 32 segmental trisomics involving 1L is being prepared according to the method described by Birchler (Genetics 94:687, 1980). These will permit mapping of isozyme and RFLP loci to chromosome segments.

Inversions:

Crosses have been made to map the following inversions in relation to 1L markers: Inv1a (1S.30, 1L.50); Inv1c (1S.35, 1L.01); Inv1d (1L.55, 1L.92); Inv1L-5131-10 (1L.46, 1L.82). I would appreciate any information about these inversions.

Production of genetic stocks:

The following stocks have been developed and are available upon request; some are still in limited quantities, however:

D8 gs1 Ts6 bm2 ms14 br2 bz2 rd1 gs1

In response to inquiries about parent lines being used by arm coordinators for mapping: I am using A632 and MO17 as parent lines for crosses and backcrossing. A632 has been a very good line to work with in Raleigh; Mo17 is more difficult; W23 is also good here; M14 is not; B73 is susceptible to every disease known to mankind and rather late for my purposes; Oh43 has trouble germinating.

[Mpl1, Miniplant, dominant, possibly allelic to D8, approx. 4 units distal to lw1, is described by Harberd et al. in this issue; Virginia Walbot reports that probes specific to the bz2 gene do not cross-hybridize to DNA preps from the an1-bz2-6923 stock available from the Stock Center (personal communication). This supports the hypothesis that an1-bz2-6923 is a deletion mutant induced by radiation (MNL 30:100, 1956, 31:140, 1957)].

Paul Sisco

CHROMOSOME 3S

Cg1 and a mutation resembling Cg1 are located on 3S. Earlier reports suggesting that Cg1 was not on 3S were in error because stocks were contaminated with Tp1.

Scott Poethig

CHROMOSOME 5S

Eighty F2 families from a2-m5/a2, wx1-m8/wx1(?), segregating Spm, were screened for new variants. New phenotypes appeared in eighteen families as follows: virescent 3, adherent 3, dwarf 2, crinkly leaf 3, pale green leaf 4, asymmetric leaf blade 2, narrow leaves 2, albescent 1, leaf lesions 1. The F1 had no lesions of this sort (see last year's report). There is evidence that the En at a1 transposes preferentially to loci 6-20 map units from a1 (P. A. Peterson, Theor. Appl. Genet. 40:367, 1970). Presumably some of these new loci will be near a2. They will be crossed to appropriate translocations and linkage testers in the summer of 1987.

New mapped locus: an11 (E. Coe, this News Letter) is about 10 units distal to a2.

I have been using Mo17 for mapping of hcf factors and will do the same for these new loci.

Mary Polacco

CHROMOSOME 8

In this issue we report that de*-1386A is distal to j1.

M. G. Neuffer

CHROMOSOME 9L

In this News Letter: Coe reports bk2 (5) v30 (19) Wc1 (16) Bf1. Behrendsen et al. report sh1 (24) wx1 (11) NPI121-Css (sucrose synthase-2), and Wright et al. report NPI332-Pep (PEP carboxylase) is also on 9L (see RFLP working maps). Neuffer and Beckett report wlu4 (was wl*-41A) on 9L, and Neuffer et al. report dominants Zb8 (was Atc1) and Les8 (was Les*-2005) on chromosome 9 (near wx1 per Bird and Neuffer, MNL 59:42); G6 (was G*-1585) is 18 units from wx1 and presumably on 9S inasmuch as T8-9Sb hyperploids (+/+/G6) are green.

Allelism tests among established (named) and new factors have been set up, and crosses for mapping are advancing to analysis this year.

I have been using W23 as a standard for recombination analysis whenever possible; otherwise, marker stocks of antique parentage or of F1 hybrids, W23xK55 background.

Ed Coe

CHROMOSOME 10S

No new information has been received, but Larry Beach from Pioneer Hi-Bred is close to having some major quantities of linkage information that should help to place accurately several of the genes on 10S. We anticipate much of this to be completed this summer. Once we have this information we will compile all the information available for 10S.

We are moving 10S mutants into B73. Some mutants have A632 in their background and that will be our second choice. Some of the linkage information will be derived from lines that originated from the Stock Center, inbred line/purity unknown.

Marc C. Albertsen

MAIZE GENE LIST AND WORKING MAP

An updated version of the gene list that was published in Genetic Maps, Volume III, S.J. O'Brien, ed., National Cancer Institute NIH is presented on the following pages. A total of 635 entries are in the list, with 575 separate unit factors identified. Of these 575, 464 have at least been located to chromosome, leaving 111 unplaced. A list of these unplaced factors is below. If you are working on any of these and have information as to their location (even just to chromosome), please let me know. If you are not, and are looking for work, why not map a few ?? I have also included a list of 263 factors which are not listed in the stocklist of the Coop (isozymes were not included in this list). I have supplied this list to Earl Patterson for his correction, as I am sure a number are available from the Coop and just not listed. If you have recently named a gene and not sent seed to the Coop, here is your reminder !!

Immediately following the gene list is the newly revised and updated working linkage map. The same format as in previous years was followed and I refer you to MNL 59:110 for an explanation of how the map is constructed. There are two additions to the map this year that need some explanation. The physical map of each chromosome is drawn immediately to the left of the core map. The length of each arm is in proportion to the ratio of the length of that arm to the length of chromosome 1 (lengths taken from the table in Maize for Biological Research, p.52). The length of chromosome 1 was assigned the same length as the genetic map. The centromeric region of each chromosome is aligned in both maps and the breakpoints for the B-A translocations identified where known. It is anticipated that additional cytological features will be added to the physical map as well as additional correlations with the genetic map.

Also included this year is the restriction fragment polymorphism map for each chromosome. These maps were kindly provided by Tim Helentjaris and co-workers at Native Plants, Inc. and used following only minor revisions. No attempt to correlate the molecular map with the other maps has been made, pending map revisions in the near future. All locus numbers to the right of the vertical line are NPI loci (and would be refered to as NPI#, see p. 49). Gene symbols within brackets are isozyme or morphological loci mapped relative to the RFP loci. The numbers immediately to the left of the vertical line are map distances, in centimorgans, between the tick marks. The UMC#'s to the left of these are additional loci identified in my lab. It is anticipated that within the next few months a much better RFP map will be available and lead to a better correlation of the various maps.

All genes from the linkage map are referenced in the symbol index at the end of the newsletter in order to provide a possible means for locating a particular gene in the linkage map. I appreciate any and all comments and corrections regarding the map.

Dave Hoisington

LIST OF THOSE FACTORS NOT LOCATED TO CHROMOSOME

Aco2	Amy1	Ce1	dv1	E10	Lcs1	me11	Mv1	oro2	pi1	Px8	Sod1	Tpi1	ws2
Aco3	aph1	cf12	dy1	g15	Lct1	mg1	o9	Orom1	pi2	Px9	Sod3	Tpi2	y11
Aco4	bs1	Cg2	E2	g19	lct2	ms20	o10	pam1	Px1	rgo1	Sod4	tpm1	y12
Acp2	btn1	c1h1	E5-I	g120	Lfy1	Ms21	o11	pam2	Px2	Rs1	Sup1	tr1	zb1
Adr1	bu1	cto1	E5-II	Ht2	loc1	ms22	o12	pd1	Px4	se1	sy1	ub1	zb2
afd1	bv2	db1	E6	Ht3	lty1	ms23	o13	Pdf1	Px5	sen4	Ta1	Ufo1	zn2
agt1	Cat3	dsy1	E7	is1	lty2	ms24	ora2	pe1	Px6	sft1	Thc1	v13	zpg1
Amp4	Cdh1	dsy2	E9	l4	Mc1	ms28	ora3	pg13	Px7	Sg1	t11	ws1	

LIST OF THOSE FACTORS NOT IN THE COOP STOCKLIST

afd1	cf12	dek5	dek23	g14	hcf13	hcf50	Lcs1	lty2	ms24	orp1	rd2	sft1	v24	wt2
agt1	Cg2	dek6	dek24	g15	hcf15	hcf101	Lct1	lw3	ms28	orp2	Rf3	Sks1	v25	wyg1
a1h1	c1h1	dek7	dek25	g17	hcf18	hcf102	lct2	lw4	Ms41	pam1	Rf4	Sn1	v26	y3
an11	C1t1	dek8	dek26	g18	hcf19	hcf316	Les3	ma11	ms43	pam2	Rgd2	Spc1	v27	y11
aph1	cp1	dek9	dek27	g19	hcf21	hcf323	Les4	Ma12	Msc1	pb1	rgo1	spc2	v28	y12
Asr1	cp2	dek10	dek28	g119	hcf23	hcf408	Les5	Mc1	Msc2	pd1	R11	spc3	v29	yd2
ats1	cr1	dek11	dek29	g120	hcf26	hm2	Les6	me11	Mut	Pdf1	Rp1	spt1	v30	ys2
baf1	ct1	dek12	dek30	g121	hcf28	Hsf1	Les7	mep1	Mv1	pe1	Rp5	spt2	Vsr1	Ysk1
Bif1	ct2	dek13	dep1	grt1	hcf31	Ht2	Les8	mg1	nec4	pg13	Rp6	Sup1	w2	zb7
brn1	cto1	dek14	dsy1	gs3	hcf34	Ht3	Les9	mmm1	nec5	pg15	Rpp9	sy1	w16	Zb8
bs1	d2	dek15	dsy2	gt1	hcf36	ij2	Les10	Mp11	nec6	pg16	Sdw1	tb1	w17	zpg1
btn1	d3	dek16	Dt5	hcf1	hcf38	is1	Lfy1	Mr	nec7	Ph1	se1	td1	wgs1	
bu1	da1	dek17	g2	hcf2	hcf41	113	ln1	ms1	o12	pi1	sen1	Thc1	whp1	
bv2	db1	dek18	G6	hcf3	hcf42	115	loc1	ms3	ora2	pi2	sen2	t11	wlu1	
Bx1	dek1	dek19	ga2	hcf4	hcf44	116	lp1	ms20	ora3	ppg1	sen3	T1r1	wlu2	
bz2	dek2	dek20	ga7	hcf5	hcf46	117	lte1	Ms21	orol	pro1	sen4	tpm1	wlu3	
c2	dek3	dek21	ga8	hcf6	hcf47	118	Lte2	ms22	oro2	py2	sen5	tr1	wlu4	
Ce1	dek4	dek22	ga10	hcf12	hcf48	119	lty1	ms23	Orom1	pyd1	sen6	Ufo1	Wrk1	

LINKAGE MAP OF CORN (MAIZE) (*Zea mays* L.) (2N = 20)
February 1987

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The following list, arranged by gene symbol, identifies the unit factors for which stocks are available in the Maize Genetics Stock Center (Department of Agronomy, University of Illinois, Urbana, Illinois 61801), those for which variants exist in generally available strains (e.g. isozyme variants), and those upon which current or recent research studies have been published or have been reported in the Maize Genetics Cooperation News Letter. The information tabulated includes the chromosome (L=long arm, S=short arm) and map position or approximate location, the name and phenotype, availability from the Stock Center (S), a photograph (P) in The Mutants of Maize (Neuffer, M.G., et al. 1968. Crop Sci. Soc. Amer., Madison, Wis.), and references to the original descriptions.

Databases are being compiled in our laboratory for recombinational information involving traditional loci as well as RFP's. This information will be available upon request.

The authors greatly appreciate the corrections supplied by fellow maize cooperators and encourage all those interested in maize genetics to make suggestions and/or corrections to this list.

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REFERENCE
a1	3L-141.0	anthocyaninless: colorless aleurone, green or brown plant, brown pericarp with P1-RR	S	P	73
a2	5S-35	anthocyaninless: like a1, but red pericarp with P1-RR	S	P	140
a3	3L-125	anthocyanin: recessive intensifier of expression of R1 and B1 in plant tissues	S	P	179
Ac		activator: designator for autonomous transposable elements; regulates Ds transposition and dissociation; ex. Ac9 designates element isolated from wx1-m9		P	198
Ac2		activator: similar to Ac			60
Aco1	4S	aconitase: electrophoretic mobility; monomeric			344
Aco2	-	aconitase: electrophoretic mobility			344
Aco3	-	aconitase: electrophoretic mobility			344
Aco4	-	aconitase: electrophoretic mobility; monomeric			344
Acp1	9	acid phosphatase (was Ap1, Acph1, Phos): electrophoretic mobility; dimeric			114
Acp2	-	acid phosphatase (was Ap2): electrophoretic mobility; monomeric			114
Acp4	1L-176	acid phosphatase: electrophoretic mobility			150
ad1	1L-108	adherent: seedling leaves, tassel branches, and occasionally top leaves adhere	S	P	153
Adh1	1L-128	alcohol dehydrogenase: electrophoretic mobility; hybrid bands occur; null allele is known; dimeric	S		299
Adh2	4S-46	alcohol dehydrogenase: electrophoretic mobility; dimeric; null allele is known			297
Adk1	6S-0	adenylate kinase: electrophoretic mobility; plastidial			345
Adr1	-	alcohol dehydrogenase regulator			164
ae1	5L-57	amylose extender: glassy, tarnished endosperm; high amylose content; starch branching enzyme	S	P	343
afd1	-	absence of first division: first meiotic division replaced by mitosis			110
agt1	-	ageotropic: primary root unresponsive to gravity			66
al1	2S-4	albescent plant: erratic development of chlorophyll; pale yellow endosperm	S	P	255
alh1	1L-near bm2	histone Ia (was H1a): electrophoretic mobility			329
alpha		A1 locus component (see beta): determines reduced aleurone and plant color, brown pericarp			168
am1	5S-20	ameiotic: meiosis fails, sporogenous tissue degenerates	S	P	246 269
Amp1	1L-near f1	aminopeptidase: electrophoretic mobility; monomeric; cytosolic			244
Amp2	1-near hm1	aminopeptidase: electrophoretic mobility; monomeric			244
Amp3	5S-near a2	aminopeptidase: electrophoretic mobility; monomeric			244
Amp4	-	aminopeptidase: electrophoretic mobility; monomeric			244
Amy1	-	alpha amylase: electrophoretic mobility; monomeric			38
Amy2	5S-near Mdh5	beta amylase: electrophoretic mobility; monomeric			37
an1	1L-104	anther ear: andromonoecious dwarf, intermediate stature; few tassel branches; responds to gibberellins	S	P	70 80
anl1	5S-near lu1	anthocyaninless lethal: colorless aleurone; small kernels; embryo lethal			44
aph1	-	aphid resistance			36
ar1	9L-62	argentia: virescent seedling, greens rapidly	S	P	84
as1	1-56	asynaptic: synaptic failure of meiotic prophase chromosomes	S	P	15
Asr1	4S-19	absence of seminal roots			209
Atc1		(see Zb8)			
ats1	8	atrazine susceptible: lacks glutathione S-transferase			117
B1	2S-49	colored plant: anthocyanin in major plant tissues; some alleles affect aleurone and embryo color	S	P	76
B chr		B chromosome: supernumerary chromosome		P	263
ba1	3L-94	barren stalk: ear shoots and most tassel florets missing	S	P	126
ba2	2-near ts1	barren stalk: like ba1, but tassel more normal	S		126
baf1	9S-near w11	barren stalk fastigiate (was ba*-s): ear shoots missing; tassel branches erect			45
bd1	7L-109	branched silkless: branched ear and tassel; silks absent	S	P	156
beta		A1 locus component (see alpha): determines aleurone and plant color, red pericarp			168
Bf1	9L-137	blue fluorescent: homozygous seedlings (homozygous or heterozygous anthers) fluoresce blue under ultraviolet; anthranilic acid present	S	P	335
bf2	10L-30	blue fluorescent: similar to Bf1 in expression; shows earliers, stronger seedling fluorescence than Bf1	S		2
Bg		Bergamo: regulatory element mediating o2-mr			284
Bh1	6L-50	blotched: colored patches on colorless (c1) aleurone	S	P	75
Bif1	8	barren inflorescence (was B1f*-1440): florets missing from ear and tassel			234
bk2	9L-82	brittle stalk: brittle plant parts after 4-leaf stage	S	P	167
bm1	5S-41	brown midrib: brown pigment over vascular bundles of leaf sheath, midrib, and blade	S	P	83
bm2	1L-161	brown midrib: like bm1	S		35
bm3	4-near su1	brown midrib: like bm1 (C.R. Burnham, unpublished)	S		162 219
bm4	9L-141	brown midrib: like bm1	S		32
Bn1	7L-71	brown aleurone: yellowish brown aleurone color	S		160
br1	1L-81	brachytic: short internodes, short plant; no response to gibberellins	S	P	152 155
br2	1L-near hm1	brachytic: like br1	S		170
br3	5	brachytic: like br1	S		310
brn1	3S-near cr1	brown aleurone: small, brown, defective kernel, brown embryo; seedling lethal			282
bs1	-	barren sterile			202
bt1	5L-42	brittle endosperm: mature kernel collapsed, angular, often translucent and brittle	S	P	190 347
bt2	4S-67	brittle endosperm: like bt1; ADP glucose pyrophosphorylase electrophoretic mobility (G.F. Sprague, unpublished)	S		336
btn1	-	brittle node			151
bu1	-	leaf burn: leaves show burning, sometimes horizontal bands, accentuated by high temperature			98
bv1	5L-47	brevis plant: short internodes, short plant	S		172
bv2	-	brevis plant: plant height 30-50% of normal			256
Bx1	4S	benzoxazin: blue color reaction of crushed root tip with FeCl3, indicating cyclic hydroxamates present			52
bz1	9S-31	bronze: modifies purple aleurone and plant color to pale or reddish brown; anthers yellow-fluorescent; UDPG-flavonol 3-O-glucosyl transferase	S	P	268
bz2	1L-106	bronze: like bz1; anthers not fluorescent			241
C1	9S-26	colored aleurone: c1=colorless; C1-I=dominant colorless	S	P	68
c2	4L-117	colorless: colorless aleurone, reduced plant color			42
Car1	1S	catalase regulator: enzyme activity level increased			290
Cat1	5S-near Mdh5	catalase: electrophoretic mobility; tetrameric			18
Cat2	1S	catalase: electrophoretic mobility; null allele is known; tetrameric			287
Cat3	-	catalase: electrophoretic mobility; null allele is known; tetrameric			289
Cdh1	-	cinnamyl alcohol dehydrogenase: electrophoretic mobility			92
Cel	-	curled entangled: rolled leaves tend to be entangled			39 247
cf12	-	complementary to f12			245
Cg1	3S-37	corngrass: narrow leaves, extreme tillering	S	P	309
Cg2	-	corngrass: like Cg1; mutable			184
Ch1	2L-155	chocolate pericarp: dark brown pericarp	S	P	5
Cin		Cinteotl corn insert: repetitive sequences dispersed in the genome			302
cl1	3S-52	chlorophyll: white to green seedlings, depending upon Clm1; pale yellow endosperm	S		82
clh1	-	histone Ic: electrophoretic mobility			329
Clm1	8	modifier of cl1: greens cl1 seedlings; does not restore endosperm carotenoids	S		82
Clt1	8	clumped tassel (was C1t*-985): variable dwarfing, developmental anomalies			101 233
cm1	10L-near R1	chloroplast mutator: like ij1	S		331
cms-C		cytoplasmic male sterility: female transmitted male sterility, C type; restored by Rf4			16
cms-S		cytoplasmic male sterility: female transmitted male sterility, S type; restored by Rf3			147 149
cms-T		cytoplasmic male sterility: female transmitted male sterility, Texas type; restored by Rf1 Rf2			147 149
cp1	7S-near vp9	collapsed: endosperm collapsed and partially defective			182
cp2	7S-near vp9	collapsed: endosperm rough, collapsed, partially defective; seedling very light green with darker streaks			237
cr1	3S-14	crinkly leaves: plant short; leaves broad, crinkled			77
Css1	9	sucrose synthase: sucrose synthase-2 of embryo and other tissues			195
ct1	8	compact plant: semi-dwarf plant			225
ct2	1S	compact plant: semi-dwarf plant with club tassel			104

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REFERENCE
ctDNA		chloroplast DNA: sequences or loci in chloroplast genome			
ctol	-	cob turned out: ear inverted to a sheet or tube, kernels internally placed; variable expression			342
Cx1	10L-near bf2	catechol oxidase: electrophoretic mobility; no hybrid bands; null allele is known			262
Cy		regulatory element mediating bz1-rcy			291
d1	3S-32	dwarf plant: plant andromonoecious, short, compact; responds to gibberellins	S	P	70
d2	3	dwarf plant: like d1			333
d3	9S-59	dwarf plant: like d1			57
d5	2S-34	dwarf plant: like d1	S		333
D8	1L-133	dwarf plant: dominant (compare Mpl1), resembles d1; not responsive to gibberellins	S	P	253
da1	9	dilute aleurone: aleurone color diluted			88
db1	-	dichotomously branching plants (=dib): variable location of dichotomy, usually at 4-8th node (possible association with aneuploidy)			201 202
dek1	1S-27	defective kernel (was clf1, gay1, clf*-792): germless, floury endosperm; anthocyanins and carotenoids absent			235 236
dek2	1L	defective kernel (was dsc*-1315A): discolored, scarred endosperm; inviable			235 236
dek3	2S	defective kernel (was gm*-1289): germless			235 236
dek4	2L	defective kernel (was clf*-1024A): like dek1			235 236
dek5	3S	defective kernel (was sh*-874A): shrunken endosperm; white seedling with green stripes			235 236
dek6	3L	defective kernel (was sh*-627D): shrunken endosperm; lethal			235 236
dek7	4S	defective kernel (was su*-211C): shrunken sugary endosperm; white seedling with green stripes			235 236
dek8	4L	defective kernel (was sh*-1156A): shrunken endosperm; lethal			235 236
dek9	5L	defective kernel (was crp*-1365): crumpled endosperm; lethal			235 236
dek10	6L	defective kernel (was cp*-1176A): collapsed endosperm; lethal			235 236
dek11	7L	defective kernel (was et*-788): etched endosperm; lethal			235 236
dek12	9S	defective kernel (was cp*-873): collapsed endosperm; lethal			235 236
dek13	9L	defective kernel (was o*-744): defective opaque endosperm; lethal			235 236
dek14	10S	defective kernel (was cp*-1435): collapsed endosperm; lethal			235 236
dek15	10L	defective kernel (was cp*-1427A): collapsed floury endosperm; lethal			235 236
dek16	2L	defective kernel (was fl*-1414): floury endosperm; lethal			303
dek17	3L	defective kernel (was cp*-230D): collapsed endosperm; lethal			303
dek18	5S	defective kernel (was cp*-931A): collapsed endosperm; lethal			303
dek19	6L	defective kernel (was o*-1296A): collapsed opaque endosperm; lethal			303
dek20	8L	defective kernel (was cp*-1392A): collapsed endosperm; lethal			303
dek21	10L	defective kernel (was msc*-1330): aleurone mosaic of reduced anthocyanins; reduced carotenoids; lethal			303
dek22	1L	defective kernel (was cp*-1113A): collapsed endoperm; lethal			41
dek23	2L	defective kernel (was dcr*-1428): defective crown; lethal			41
dek24	3S	defective kernel (was cp*-1783): collapsed endosperm; lethal			304
dek25	4S	defective kernel (sh*-1169A): shrunken endosperm; lethal			304
dek26	5L	defective kernel (cp*-1331): collapsed endosperm; lethal			304
dek27	5L	defective kernel (was cp*-1380A): collapsed endosperm; lethal			304
dek28	6S	defective kernel (was o*-1307A): opaque endosperm			304
dek29	8L	defective kernel (was cp*-1387A): collapsed endosperm; viable			304
dek30	9L	defective kernel (was fl*-1391): floury endosperm; lethal			304
dep1	6	defective pistils			203
Df		deficiency: general symbol for loss of segments of chromosome			
Dia1	2S	diaphorase: electrophoretic mobility; monomeric; cytosolic			344
Dia2	1L-near bm2	diaphorase: electrophoretic mobility; dimeric; cytosolic			344
dp1	4L-137	distal pale: seedling leaf tip virescent (E.G. Anderson, unpublished)	S		
Ds		dissociation: designator for transposable factors regulated by Ac; modifies gene function and/or chromosome breakage (termed "Ds-2"); ex. Ds2 designates element isolated from Adh1-2F11	S	P	198
dsy1	-	desynaptic: pairing incomplete			108
dsy2	-	desynaptic: like dsy1			107
Dt1	9S-0	dotted: regulated controlling element at A1; responding a1-m alleles express colored dots on colorless kernels and purple sectors on brown plants	S	P	264
Dt2	6L-44	dotted: like Dt1	S		242
Dt3	7L	dotted: like Dt1, but expression variable	S		242
Dt4	4	dotted: like Dt1, but dots chiefly on crown of kernel	S		61
Dt5	9S-near yg2	dotted: like Dt1			61
Dt6	4-near sul	dotted: like Dt1	S		321
dul	10L-28	dull endosperm: glassy, tarnished endosperm (P.C. Mangelsdorf, unpublished)	S		191
dv1	-	divergent: spindle nonconverging in meiosis in microsporocytes; male sterile	S		40
dy1	-	desynaptic: chromosomes unpaired in microsporocytes	S		224
E1	7L	esterase: electrophoretic mobility; hybrid bands occur; null allele is known			294
E2	-	esterase: presence-absence			296
E3	3S	esterase: electrophoretic mobility; hybrid bands occur			295
E4	3S-near cl1	esterase (was Est4): electrophoretic mobility; no hybrid bands; null allele is known			121
E5-I	-	esterase: duplicate factor with E5-II; electrophoretic mobility			186
E5-II	-	esterase: duplicate factor with E5-I; electrophoretic mobility			186
E6	-	esterase: presence-absence			186
E7	-	esterase: presence-absence			186
E8	3S-near g2	esterase: electrophoretic mobility; null allele is known			186
E9	-	esterase: electrophoretic mobility; null allele is known			186
E10	-	esterase: electrophoretic mobility			186
eg1	5L	expanded glumes: glumes open at right angle	S		33
Ej1		(= Isr1)			
el1	8L	elongate: chromosomes uncoiled during meiotic metaphase and anaphase; frequent unreduced gametes	S	P	269
En		enhancer: transposable element (equivalent to Spm); autonomous, regulates I transposition (e.g. at g2-m = pg-m = pg14-m)		P	251
Enp1	6L-near y1	endopeptidase: electrophoretic mobility; monomeric; null allele is known			200
et1	3L-153	etched: pitted, scarred endosperm; virescent seedling	S	P	325
f1	1L-86	fine stripe: virescent seedling, fine white stripes on base and margin of older leaves	S	P	176
Fcu		factor Cuna: controlling element of r1-cu			112
f11	2S-68	floury endosperm (= o4): endosperm opaque, soft; dosage effect	S	P	125
f12	4S-58	floury: endosperm opaque, soft (W.J. Mumm, unpublished)	S		228
f13	8L-0	floury	S		221
Flt		flint: designator for factors determining flint endosperm type			213
g1	10L-47	golden plant: seedling and plant with distinct yellow cast	S	P	70 72
g2	3S-0	golden plant (= g5 = pg14): like g1, but more extreme; sheaths whitish yellow-green			137
g5		(= g2)			
G6	9S-near 17	golden plant (was G*-1585): like g1; lighter yellowish sheaths			238
Ga1	4S-32	gametophyte factor (= ga9): Ga1 pollen grains competitively superior to ga1 on Ga1 silks	S		148
ga2	5L-55	gametophyte factor: Ga2 pollen grains competitively superior to ga2			31
ga7	3L-159	gametophyte factor: ga7 pollen from heterozygotes 10-15% functional regardless of silk genotype			266
ga8	9S-near lo2	gametophyte factor: Ga8 pollen grains competitively superior to ga8 on Ga8 silks			293
ga9		(= ga1)			
ga10	5	gametophyte factor			111
Gdh1	1L-near vp8	glutamic dehydrogenase: electrophoretic mobility			260
Gdh2	10	glutamic dehydrogenase: electrophoretic mobility			113
Ger		glucoside earworm resistance: designator for earworm resistance factors from Cateto Palha Roxa			213
gl1	7L-36	glossy: cuticle wax altered; leaf surface bright, water adheres	S	P	160
gl2	2S-30	glossy: like gl1	S	P	123

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REFERENCE
g13	4L-112	glossy: like g11	S		123
g14	4L-81	glossy (= g116): like g11 (G.F. Sprague, unpublished)			
g15	-	glossy (was g15-1): duplicate factor with g120; like g11 (G.F. Sprague, unpublished)			322
g16	3L-61	glossy: like g11 (G.F. Sprague, unpublished)	S		
g17	3L	glossy (= g112): like g11 (G.F. Sprague, unpublished)			
g18	5L-68	glossy (= g110): like g11 (G.F. Sprague, unpublished)			
g19	-	glossy: expression poor (G.F. Sprague, unpublished)			
g110		(= g18)			
g111	2S-near B1	glossy: like g11; abnormal seedling morphology	S		318
g112		(= g17)			
g114	2	glossy: like g11	S		320
g115	9L-66	glossy: like g11; expressed after 3rd leaf (G.F. Sprague, unpublished)	S	P	3
g116		(=g14)			
g117	5S-34	glossy: like g11, but semi-dwarf with necrotic crossbands on leaves	S		270
g118	8L-near f13	glossy: like g11; expression poor	S		4
g119	3S	glossy (was g1*-169): like g11; lethal			230
g120	-	glossy (was g15-2): duplicate factor with g15; like g11 (G.F. Sprague, unpublished)			322
g121	10S	glossy (was g1*-478B): like g11			230
Glu1	10L-near bf2	beta glucosidase: electrophoretic mobility; null allele is known; dimeric			261
Got1	3L-near Me1	glutamate-oxaloacetate transaminase (possibly = Ta1): electrophoretic mobility; glyoxysomal; null allele is known; dimeric			288
Got2	5L-96	glutamate-oxaloacetate transaminase: electrophoretic mobility; dimeric; null allele is known			115
Got3	5S-near a2	glutamate-oxaloacetic transaminase: electrophoretic mobility; mitochondrial; null allele is known			115
grt1	5L	green tip (was grt*-1308B): pale yellow seedling with green first leaf tip			230
gs1	1L-135	green stripe: grayish green stripes between vascular bundles on leaves; tissue wilts	S	P	75 207
gs2	2S-54	green stripe: like gs1, but pale green stripes; no wilting (G.F. Sprague, unpublished)	S	P	
gs3	6L	green stripe (was gs*-268): like gs2			230
gt1	1	grassy tillers: numerous basal branches; vegetatively totipotent in combination with id1 and pe1			300
h1	3	soft starch: endosperm soft, opaque	S		220
hcf1	2L	high chlorophyll fluorescence: affects NADP+ oxidoreductase			204
hcf2	1L	high chlorophyll fluorescence: missing cytochrome f/b6 complex			204
hcf3	1S	high chlorophyll fluorescence (= hcf9): missing PSII thylakoid membrane core complex			204
hcf4	1L	high chlorophyll fluorescence: affects CO2 fixation			205
hcf5	6S	high chlorophyll fluorescence: affects PSII reaction			206
hcf6	1S	high chlorophyll fluorescence: missing cytochrome f/b6 complex			171
hcf9		(= hcf3)			
hcf12	1L	high chlorophyll fluorescence			171
hcf13	1L	high chlorophyll fluorescence: affects CO2 fixation			171 205
hcf15	2L	high chlorophyll fluorescence: affects photophosphorylation			171
hcf18	5L-near pr1	high chlorophyll fluorescence (= hcf43): major loss of PSI; other thylakoid complexes reduced			205
hcf19	3L	high chlorophyll fluorescence: affects PSII thylakoid membrane core complex			171 205
hcf21	5L	high chlorophyll fluorescence: affects CO2 fixation, Rubisco			205
hcf23	4S	high chlorophyll fluorescence: affects photophosphorylation			171 205
hcf26	6S	high chlorophyll fluorescence: affects electron transport; virescent seedling			171 205
hcf28	10L	high chlorophyll fluorescence: affects CO2 fixation			206
hcf31	1S	high chlorophyll fluorescence: missing chlorophyll a/b binding protein			206
hcf34	6L	high chlorophyll fluorescence: affects photophosphorylation			171 205
hcf36	6L	high chlorophyll fluorescence: affects electron transport			206
hcf38	5L	high chlorophyll fluorescence: affects cytochrome f/b6 complex, alpha and beta components of CF1			171
hcf41	1L	high chlorophyll fluorescence: affects PSII thylakoid membrane core complex			171 205
hcf42	9L	high chlorophyll fluorescence: affects Rubisco			205
hcf43		(= hcf18)			
hcf44	1L	high chlorophyll fluorescence: affects PSI membrane core complex			205
hcf46	3L	high chlorophyll fluorescence			171
hcf47	10S	high chlorophyll fluorescence: affects cytochromes			206
hcf48	6L	high chlorophyll fluorescence: affects electron transport			206
hcf50	1L	high chlorophyll fluorescence: missing PSI thylakoid membrane core complex			205
hcf101	7L	high chlorophyll fluorescence (was Mu-5*): affects PSI thylakoid membrane core complex			206
hcf102	8L	high chlorophyll fluorescence: affects cytochrome f/b6 complex (D. Miles, unpublished)			
hcf316	10S	high chlorophyll fluorescence: affects chlorophyll a/b binding protein			206
hcf323	6S	high chlorophyll fluorescence: affects photophosphorylation, coupling factor			206
hcf408	6L	high chlorophyll fluorescence: affects chlorophyll a/b binding protein			206
Hex1	3S-near cr1	hexokinase: electrophoretic mobility; null allele is known; monomeric; cytosolic			346
Hex2	6L-near Pt1	hexokinase: electrophoretic mobility; null allele is known; monomeric; cytosolic			346
hm1	1L-64	Helminthosporium carbonum susceptibility: disease lesions on leaves, black masses of fruiting bodies on ears with race 1	S	P	340
hm2	9L-near bk2	H. carbonum susceptibility: like hm1; masked by Hm1			227
Hs1	7S-0	hairy sheath: abundant hairs on leaf sheath	S	P	334
Hsf1	5	hairy sheath frayed (was Hsf*-1595): pubescent sheaths and leaf margins; liguled enations at leaf margins			20
Ht1	2L-121	Helminthosporium turcicum resistance	S		130
Ht2	-	H. turcicum resistance			131
Ht3	-	H. turcicum resistance: (from Tripsacum floridanum)			132
I		inhibitor (=C1-I, inhibitor allele at C1 locus): also commonly used as a general symbol for inhibition and the controlling elements responding to En			65
id1	1L-near an1	indeterminate growth: requires extended growth and short days for flowering; vegetatively totipotent with gt1 and pe1	S		308
Idh1	8L	isocitrate dehydrogenase: electrophoretic mobility; null allele is known; dimeric			115
Idh2	6L-near wl4	isocitrate dehydrogenase: electrophoretic mobility; dimeric			115
ig1	3L-82	indeterminate gametophyte: polyembryony, heterofertilization, polyploidy, androgenesis	S		157
ij1	7L-52	iojap striping: many variable white stripes on leaves; conditions chloroplast defects that are cytoplasmically inherited	S	P	136
ij2	1L	iojap striping: like ij1; chloroplast inheritance unknown			230
in1	7S-20	intensifier: intensifies anthocyanin pigments	S	P	90
Inv		Inversion: general symbol for inversion of a segment of chromosome	S	P	
is1	-	cupulate interspace			95
Isr1	10L-near R1	inhibitor of striate (was Ej1): reduces expression of sr2 and other leaf-striping factors	S		158
j1	8L-42	japonica striping: white stripes on leaf and sheath; not expressed in seedling	S	P	72
j2	4L-106	japonica striping: extreme white striping of leaves, etc. (R.A. Emerson, unpublished)	S	P	81
K		knob: general symbol for constitutive heterochromatic elements			
K3L	3L-107	knob: constitutive heterochromatic element			59
K10	10L-near sr2	abnormal-10: heterochromatic appendage on long arm of chromosome 10; neocentric activity distorts segregation of linked genes	S	P	181
Kn1	1L-near Adh1	knotted: scattered proliferation of tissue at vascular bundles on leaf	S	P	29
Krn		kernel row number: designator for factors determining kernel row number			213
l1	10L-near R1	luteus: yellow pigment in white tissue of chlorophyll mutants w1, j1, ij1, etc.	S	P	174 175
l4	-	luteus: lethal yellow seedling	S	P	142
l6	9S-near bz1	luteus: like l4 (W.H. Eyster, unpublished)	S		
l7	9S-42	luteus: yellow seedling and plant	S		88
l10	6L-19	luteus: like l4	S		277
l11	6S	luteus: yellow seedling with green leaf tips	S		8
l12	6L-16	luteus: like l4	S		53

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REFERENCE
113	10L-91	luteus (was 1*-59A, 1*-Neuffer2): like 14			192
115	6L-30	luteus (was 1*-Blandy3, 1*-Brawn): like 14			281
116	1S	luteus (was 1*-515): like 14; leaves bleach to paler yellow in patches			230
117	1L	luteus (was 1*-544): like 14; leaves with lighter yellow crossbands			230
118	2L	luteus (was 1*-1940): like 14			230
119	10S	luteus (was 1*-425): like 14			230
lal	4S-55	lazy plant: prostrate growth habit	S	P	143
Lcl	10L-65	red leaf color: red color in leaf surface	S		21
Lcs1	-	thylakoid membrane polypeptide: electrophoretic mobility			216
Lct1	-	thylakoid membrane polypeptide: electrophoretic mobility			216
lct2	-	thylakoid membrane polypeptide: presence-absence			216
Les1	2S-near wt1	lesion (was Les*-843): large necrotic lesions resembling disease lesions formed by fungal infections on susceptible lines	S		231
Les2	1S-near sr1	lesion (was Les*-845A): small white lesions resembling disease lesions formed by fungal infections on resistant lines	S		231
Les3	10	lesion: like Les1			7
Les4	2L	lesion (was Les*-1375): late expression of large necrotic lesions			128
Les5	1S	lesion (was Les*-1449): like Les2			128
Les6	10S	lesion (was Les*-1451): like Les4			128
Les7	1L	lesion (was Les*-1461): late expression of small chlorotic lesions			128
Les8	9S	lesion (was Les*-2005): late expression of small, pale green lesions			128
Les9	7L-near ral	lesion (was Les*-2008): late expression of small necrotic lesions			128
Les10	2-near v4	lesion (was Les*-A607): like Les1			129
Lfy1	-	leafy: increased number of leaves			301
lg1	2S-11	liguleless: ligule and auricle missing; leaves upright, enveloping	S	P	70 71
lg2	3L-93	liguleless: like lg1, less extreme	S	P	24
Lg3	3-57	liguleless: dominant, no ligule; leaves upright, broad, often concave and pleated	S	P	248
lil	10L-near bf2	lineate leaves: fine, white striations on basal half of mature leaves	S	P	50
lls1	1S	lethal leaf spot: chlorotic-necrotic lesions resembling Helminthosporium carbonum infection	S		341
lnl	6	linoleic acid: lower ratio of oleate to linoleate in kernel			54
lo2	9S-50	lethal ovule: ovules containing lo2 gametophyte abort	S		224
loc1	-	low oil content in kernel: associated with albino seedlings			257
lpl	4	lethal pollen: lpl pollen fails in competition with Lpl			223
lte1	2-near fl1	latente: heat tolerance			210
Lte2	10L-near g1	latente: heat tolerance			211
lty1	-	light yellow endosperm			63
lty2	-	light yellow endosperm			63
lul	5S-29	lutescent: pale yellow green leaves	S		306
lw1	1L-near Adh1	lemon white: white seedling, pale yellow endosperm	S		338
lw2	5L-near pr1	lemon white: like lw1	S	P	338
lw3	5L-near v2	lemon white: duplicate factor with lw4; like lw1			338
lw4	4-near zb6	lemon white: duplicate factor with lw3; like lw1			338
lyl		(= psl-lyc)			
mal1	9	multiple aleurone layering: recessive interacts with two complementary dominants Mal2 and an unnamed factor, giving multiple cell layers			208
Mal2	4	multiple aleurone layering: (see mal1)			208
Mcl	-	mucronate: opaque endosperm			285
Mdh1	8	malate dehydrogenase: electrophoretic mobility; null allele is known; mitochondrial			239
Mdh2	6L-near w14	malate dehydrogenase: electrophoretic mobility; null allele is known; mitochondrial			239
Mdh3	3L-138	malate dehydrogenase: electrophoretic mobility; null allele is known; mitochondrial			239
Mdh4	1L-near an1	malate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic			239
Mdh5	5S-17	malate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic			239
Me1	3L-117	malic enzyme: electrophoretic mobility			115
mei1	-	meiosis: chromosomes sticky in metaphase I; male sterile			106
mep1	5L	modifier of endosperm protein: affects quantities of Prot2 protein forms			298
Mer		Maya earworm resistance: designator for earworm resistance factors from IAC Maya			212
mg1	-	miniature germ (replaces mg of Wentz): germ 1/4 to 1/3 of normal; viable			165
mi1	1	midget plant: small plant (H.S. Perry, unpublished)	S		
mmm1	1L-near an1	modifier of mitochondrial malate dehydrogenases: mobilities			239
mn1	2-near fl1	miniature seed: small, somewhat defective kernel; fully viable	S	P	183
mn2	7	miniature seed: small kernel, loose pericarp; extremely defective but will germinate (R.J. Lambert, unpublished)	S		
Mp		modulator of pericarp: transposable factor affecting P1 locus; parallel to Ac-Ds			26
Mpl1	1L-near Adh1	miniplant: dominant and monoecious, intermediate dwarf; probable allele of D8; not responsive to gibberellins (M. Freeling, unpublished)			119
Mr	9S-near 17	mutator of R-m: transposable factor, regulates R1-m mutation		P	243
Mrh		mutator: controlling element of al-m-rh			271
ms1	6L-near si1	male sterile: anthers shriveled, not usually exserted			311
ms2	9L-64	male sterile: like ms1	S		86 88
ms3	3	male sterile: like ms1			86 88
ms4		(= pol)			
ms5	5-near v3	male sterile: anthers not exserted	S		13
ms7	7L-near ral	male sterile: like ms5	S		13
ms8	8L-28	male sterile: like ms5	S	P	13
ms9	1S-near P1	male sterile: like ms5	S		13
ms10	10L-near bf2	male sterile: like ms5	S		13
ms11	10	male sterile: like ms5	S		13
ms12	1	male sterile: like ms1	S		13
ms13	5S	male sterile: like ms5	S		13
ms14	1-near as1	male sterile: like ms5	S		13
ms17	1S-23	male sterile: like ms1	S		78
ms20	-	male sterile			88
Ms21	-	male sterile: pollen grains developing in presence of Ms21 are defective and nonfunctional if sks1, normal if Sks1			169 292
ms22	-	male sterile			348
ms23	-	male sterile: allelic to ms*-Bear7			348
ms24	-	male sterile			348
ms28	-	male sterile: anaphase I disturbed			106
Ms41	4L	male sterile (was Ms*-1995): like ms1			238
ms43	8L	male sterile: anaphase I irregular			106
Msc1	1L	mosaic (was Msc*-791A): aleurone mosaic for anthocyanin color			238
Msc2	5S	mosaic (was Msc*-1124B): aleurone mosaic for anthocyanin color			238
Mst1	10L-67	modifier of R-st: affects expression of R1-st	S		6
mtDNA		mitochondrial DNA: sequences or loci in the mitochondrial genome			
Mu		mutator: freely transposable element; Mu1 designates element isolated from Adh1-S3034			280
Mut	2S-near g12	mutator: controlling element for bz1-m-rh			272
Mv1	-	resistance to maize mosaic virus I ("corn stripe")			22
na1	3L-105	nana plant: short, erect dwarf; no response to gibberellins	S	P	135 173
na2	5S-near bt1	nana plant: like na1 (H.S. Perry, unpublished)	S		
NCS1	-	nonchromosomal stripe: maternally inherited light green leaf striping			307
NCS2	-	nonchromosomal stripe: maternally inherited pale green and depressed striping; mitochondrial	S		43

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REFERENCE
NCS3	-	nonchromosomal stripe: maternally inherited striations, distorted plants; mitochondrial	S		43
nec1	8L-near f13	necrotic (was nec*-6697, sienna*-7748): chlorotic seedling that stays rolled, wilts and dies	S		192
nec2	1S-34	necrotic (was nec*-8147): green seedling develops necrotic lesions at 2-3 leaf stage lethal (E.G. Anderson, unpublished)	S		
nec3	5-near bt1	necrotic (was nec*-409): seedling emerge with tightly rolled leaves that turn brown and die without unrolling; manually unrolled leaves tan with dark brown crossbands	S		229
nec4	2S-near d5	necrotic (was nec*-516B): seedling yellow, leaf tips necrotic			127
nec5	4L	necrotic (was nec*-642A): pale green seedling becoming necrotic; dark brown exudate			230
nec6	5S-near a2	necrotic (was nec*-493): like nec3			230
nec7	5L	necrotic (was nec*-756B): seedling becoming necrotic in crossbands			230
nl1	10L-near bf2	narrow leaf: leaf blade narrow, some white streaks (R.A. Emerson, unpublished)	S	P	
NOR	6S	nucleolus organizer: codes for ribosomal RNA	S		196
NPI		Native Plants, Inc.: designator for loci defined by restriction fragment polymorphisms			
o1	4L-near g13	opaque endosperm: endosperm starch soft, opaque (W.R. Singleton and D.F. Jones, unpublished)	S		
o2	7S-16	opaque endosperm: like o1; high lysine content (W.R. Singleton and D.F. Jones, unpublished)	S	P	
o4		(= fl1)			
o5	7L-near ra1	opaque endosperm: like o1; virescent seedling	S		276
o6		(= pro1)			
o7	10L-87	opaque: like o1; high lysine content	S		214
o9	-	opaque endosperm: crown opaque and light in color, frequently with a cavity; base or abgerminal side of kernel often corneous	S		222
o10	-	opaque endosperm: like o1	S		222
o11	-	opaque endosperm: thin, opaque, somewhat shrunken kernels with greyish cast	S		222
o12	-	opaque endosperm: thin, etched or scarred kernels, variable in size; plants chlorophyll deficient and small, with pollen but few ears			222
o13	-	opaque endosperm: opaque, etched kernels with rim of corneus starch on abgerminal side	S		222
Og1	10S-16	old gold stripe: variable bright yellow stripes on leaf blade	S	P	179
ora2	-	orange endosperm			62
ora3	-	orange endosperm			63
oro1	6S	orobanche: yellow to tan necrotic with cross-banding when grown under light-dark cycle; some chlorophyll with Oroml			192
oro2	-	orobanche: like oro1			192
Oroml	-	orobanche modifier: partially corrects bleaching of oro1			192
orp1	4S	orange pericarp (duplicate factor with orp2): pericarp orange over orp1 orp2 kernels			232
orp2	10L	orange pericarp (duplicate factor with orp1)			232
oy1	10S-12	oil yellow: seedling oily greenish-yellow	S	P	87
P		plant color component at R1: anthocyanin pigmentation in seedling leaf tip, coleoptile, anthers			326 327
P1	1S-26	pericarp color: red pigment in cob and pericarp	S	P	69 180
pam1	-	plural abnormalities of meiosis: desynchronized meiotic divisions and premeiotic mitosis; plants male sterile, incompletely female sterile			109
pam2	-	plural abnormalities of meiosis: like pam1			107
pb1	6L-near y1	piebald leaves: very light, irregular green bands on leaf		P	58
pb4	6L-near y1	piebald leaves: like pb1	S		58
pd1	-	paired rows: single vs. paired pistillate spikelets; pd1 is found in teosinte also			166
Pdf1	-	thylakoid membrane polypeptide: dominant increase in electrophoretic mobility			217
pe1	-	perennialism: vegetatively totipotent in combinations with gt1 and id1			300
pg11	6L-38	pale green: duplicate factor with pg12; seedling light yellowish green; mature plant pale and vigorous	S	P	267
pg12	9-61	pale green: duplicate factor with pg11	S		267
pg13	-	pale green: seedling light yellowish green; stunted growth			305
pg14		(= g2)	S	P	251
pg15	1S	pale green (was ppg*-340B): seedling light yellowish green; bleaches to near white in patches			230
pg16	1L	pale green (was pg*-219): seedling light yellowish green			230
Pgd1	6-near rgd1	6-phosphogluconate dehydrogenase: electrophoretic mobility; null allele is known; dimeric			115
Pgd2	3L-near ts4	6-phosphogluconate dehydrogenase: electrophoretic mobility; null allele is known; dimeric			115
Pgm1	1L-near Prot1	phosphoglucomutase: electrophoretic mobility; null allele is known; monomeric			115
Pgm2	5S-0	phosphoglucomutase: electrophoretic mobility; null allele is known; monomeric			115
Phl	4S-0	pith abscission: cob disarticulation			96
Phi1	1L-149	phosphoexose isomerase: electrophoretic mobility; null allele is known; dimeric; cytosolic			115
pi1	-	pistillate florets: duplicate factor with pi2; secondary florets develop ("Country Gentlemen" or "Shoe Peg" expression) in pi1 pi2 ears			133
pi2	-	pistillate florets duplicate factor with pi1			133
Pl1	6L-49	purple plant: sunlight-independent purple pigment in plant	S	P	76
pm1	3L-near ts4	pale midrib: midrib and adjacent tissue lighter green	S	P	25
Pn1	7L-112	papyrescent glumes: long, thin papery glumes on ear and tassel	S	P	97
pol	6S-4	polymitotic (= ms4): repeats 2nd meiotic division	S	P	11
ppg1	5L	pale pale green (was cb*-199A): white seedling with faint green; white necrotic crossbands			230
pr1	5L-67	red aleurone: changes purple aleurone to red	S	P	68
pro1	8L-near f13	proline requiring (= o6): crumpled opaque kernel; green stripe lethal seedling			99
Prot1	1L-121	protein: embryo protein mobility variations			298
ps1	5S-39	pink scutellum: viviparous; endosperm and scutellum pink, seedling white with pink flush	S	P	317
Pt1	6L-60	polytypic ear: proliferation of pistillate tissue to produce irregular growth on ear and tassel	S	P	226
Px1	-	peroxidase: no hybrid bands; null allele is known			118
Px2	-	peroxidase: electrophoretic mobility			185
Px3	7L-near Pn1	peroxidase: electrophoretic mobility			185
Px4	-	peroxidase: electrophoretic mobility; null allele is known			185
Px5	-	peroxidase: presence-absence			185
Px6	-	peroxidase: presence-absence			185
Px7	-	peroxidase: electrophoretic mobility; null allele is known			185
Px8	-	peroxidase: electrophoretic mobility			23
Px9	-	peroxidase: electrophoretic mobility; null allele is known			23
py1	6L-69	pigmy plant: leaves short, pointed; fine white streaks	S	P	333
py2	1L	pigmy: like py1			230
pyd1	9S-near yg2	pale yellow deficiency: pale yellow seedling; deficiency for short terminal segment of chromosome arm			197
R1	10L-61	colored: red or purple color in aleurone and/or anthers, leaf tip, brace roots, etc.	S	P	68
ra1	7L-32	ramosa: ear branched, tassel conical	S	P	13 102
ra2	3S-near Cg1	ramosa: irregular kernel placement; tassel many-branched, upright (R.A. Brink, unpublished)	S	P	
ra3	4	ramosa: (R.A. Brink, unpublished)	S		
rd1	1L-near Adh1	reduced plant: semi-dwarf plant	S		225
rd2	6L	reduced plant: like rd1, but not as extreme			105
rDNA		ribosomal DNA: rDNA5.8S, rDNA18S and rDNA25S located in NOR on 6S; rDNA5S on 2L near Ht1			
rDt		receptor of Dotted			315
Rf1	3S-near Lg3	fertility restorer: restores fertility to cms-T; complementary to Rf2	S		146
Rf2	9-near wx1	fertility restorer: see Rf1	S		67
Rf3	2L	fertility restorer: restores fertility to cms-S			30
Rf4	2	fertility restorer: restores fertility to cms-C			116
Rg1	3-59	ragged leaves: chlorotic tissue between veins of older leaves, causing holes and torn appearance	S	P	27
rgd1	6-8	ragged seedling: seedling leaves narrow, thread-like, have difficulty in emerging	S	P	161
Rgd2	5	ragged leaves (was Rgd*-1445): leaves narrow and distorted; tillering			234
rgo1	-	reversed germ orientation: embryo faces base of ear; variable frequency, maternal trait			283
rhm1	6-near rgd1	resistance to Helminthosporium maydis: chlorotic-lesion reaction with race O	S		312
Ril	4S-27	rind abscission: cob disarticulation			96

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REFERENCE
Rp1	10S-0	resistance to Puccinia spp.		P	187 188
Rp3	3-near g16	resistance to Puccinia spp.	S		349
Rp4	4S-24	resistance to Puccinia spp.	S		349
Rp5	10S-near Rp1	resistance to Puccinia spp.			286
Rp6	10S-near Rp1	resistance to Puccinia spp.			349
Rpp9	10S-near Rp1	resistance to Puccinia polysora			339
Rs1	-	rough sheath: extreme ligule disorganization	S		159
rs2	1-near as1	rough sheath	S		159
rt1	3S-near Cg1	rootless: secondary roots few or absent	S	P	139
S		seed color component at R1: anthocyanin pigmentation in aleurone			326
Sad1	10L-near bf2	shikimate dehydrogenase: electrophoretic mobility; monomeric; plastidial			344
Sdw1	8	semi-dwarf plant (was Sdw*-1592): shortened internodes, erect leaves			19
se1	-	sugary-enhancer: high sugar content with su1; light yellow endosperm; freely wrinkled in Il1677a			89
sen1	3	soft endosperm: duplicate factor with sen2; endosperm soft, opaque			328
sen2	7	soft endosperm: duplicate factor with sen1			328
sen3	1	soft endosperm: duplicate factor with sen4; like sen1			328
sen4	-	soft endosperm: duplicate factor with sen3			328
sen5	2	soft endosperm: duplicate factor with sen6; like sen1			328
sen6	5	soft endosperm: duplicate factor with sen5			328
sft1	-	small flint type: ears on sft1 plants produce only small flint endosperms; +/sft1 ears are normal			64
Sg1	-	string cob: reduced pedicels	S	P	94
sh1	9S-29	shrunken: inflated endosperm collapses on drying, forming smoothly indented kernels; sucrose synthase-1 of endosperm; homotetramer	S	P	134
sh2	3L-141.2	shrunken: inflated, transparent, sweet kernels collapse on drying, becoming angular and brittle; ADPG pyrophsophorylase reduced	S	P	189
sh4	5L	shrunken: collapsed, chalky endosperm	S		337
si1	6L-20	silky (=ms-si): multiple silks in ear; sterile tassel with silks	S		91
sk1	2S-56	silkless ears: pistils abort, no silks	S		145
Sks1	2L-near v4	suppressor of sterility: pollen grains developing in presence of Ms21 are defective and nonfunctional if sks1, normal if Sks1			169 292
sl1	7L-50	slashed leaves: leaves slit longitudinally by necrotic streaks	S		123
sm1	6L-59	salmon silks: silks salmon color with P1-RR, borwn in P1-WW	S	P	1
Sn1	10L-near R1	scutellar node color			100
Sod1	-	superoxide dismutase: electrophoretic mobility; dimeric; plastidial			9
Sod3	-	superoxide dismutase: tetrameric; mitochondrial			9
Sod4	-	superoxide dismutase: electrophoretic mobility; dimeric; cytosolic			9
Spc1	3L-near ig1	speckled (was Spc*-1376, Les*-1376): brown speckling on leaves and sheath at flowering; supporting tissues weak			234
spc2	1L	speckled (was spc*-262A): green seedling with light green speckles			230
spc3	3L	speckled (was pg*-553C): green seedling with dark and light green speckles			230
Spm		suppressor-mutator: transposable element (equivalent to En); autonomous, regulates transposition of element at al-m1, etc.			199
spt1	2L	spotted (was spt*-464): pale green seedling with dark green spots			230
spt2	4S	spotted (was pg spt*-1269A): like spt1			230
sr1	1S-0	striate leaves: many white striations or stripes on leaves (A.M. Brunson, unpublished)	S		
sr2	10L-95	striate leaves: white stripes on leaf and sheath	S	P	144
sr3	10S	striate leaves: virescent and striate to striped	S	P	104
Ss2		(see Css1)			
st1	4S-62	sticky chromosome: small plant, striate leaves, pitted kernels resulting from sticky chromosomes	S	P	14
su1	4S-66	sugary: endosperm wrinkled and translucent when dry; sweet at milk stage	S	P	51
su2	6L-58	sugary: endosperm glassy, translucent, sometimes wrinkled	S		88
Sup1	-	suppressor: modifies o2 kernels to semi-transparent			193
sy1	-	yellow scutellum			316
T		reciprocal translocation: general symbol for exchange of parts between two nonhomologous chromosomes	S	P	
Ta1	-	transaminase (possibly = Got1): electrophoretic mobility; hybrid bands occur			185
tb1	1L-near Adh1	teosinte branched: many tillers; nodes with slender branches ending in unbranched tassel			34
td1	5-near bt1	thick tassel dwarf: (E.G. Anderson, unpublished)			
te1	3	terminal ear: stalked ear appendages at tip; varying to infolded ears	S		194
Thc1	-	thiocarbamate sensitive: sensitive to Eradicane			252
tl1	-	tasselless			202
Tlr1	1L	tillered (was Tlr*-1590): extreme tillering			238
Tp1	7L-46	teopod: many tillers, narrow leaves, many small partially podded ears, tassel simple	S	P	178
Tp2	10L-45	teopod: like Tp1	S	P	250
Tpi1	-	triose phosphate isomerase: electrophoretic mobility; dimeric; plastidial			344
Tpi2	-	triose phosphate isomerase: electrophoretic mobility; dimeric; plastidial			344
Tpi3	8	triose phosphate isomerase: electrophoretic mobility; monomeric; cytosolic			344
Tpi4	3L-near Rg1	triose phosphate isomerase: electrophoretic mobility; monomeric; cytosolic			346
tpm1	-	thylakoid peptide modifier: dominant decrease in electrophoretic mobility			215
tr1	-	two-ranked ear: distichous vs. decussate phyllotaxy in ear axis			166
ts1	2S-74	tassel seed: tassel pistillate and pendant; if removed, small ear with irregular kernel placement develops	S		74
ts2	1S-24	tassel seed: like ts1, but branches pendant rather than whole tassel	S	P	74
ts4	3L-65	tassel seed: tassel compact, upright, with pistillate and staminate florets	S	P	254
Ts5	4S-53	tassel seed: nearly normal tassel with scattered, short silks	S		79
Ts6	1L-158	tassel seed: tassel pistillate to mixed, compact; ear with irregular kernel placement	S	P	240
Tu1	4L-101	tunicate: kernels enclosed in long glumes; tassel glumes large, coarse	S	P	48 49
ub1	-	unbranched: tassel with one spike	S	P	237
Ufo1	-	unstable factor for orange: anthers, silks, and most other plant parts orange with P1-WR or P1-RR; growth retarded			332
UMC		University of Missouri, Columbia: designator for loci defined by restriction fragment polymorphisms			
Uq		ubiquitous: controlling element mediating al-ruq			93
v1	9L-63	virescent: yellowish white seedling, greens rapidly	S	P	56
v2	5L-107	virescent: like v1, but greens slowly	S	P	72
v3	5L-45	virescent: light yellow seedling, greens rapidly	S	P	56
v4	2L-83	virescent: like v2	S	P	56
v5	7S-24	virescent: like v1, but older leaves have white stripes	S	P	56
v8	4L-near Tu1	virescent: like v2	S		57
v12	5L-near ys1	virescent: like v3	S		255
v13	-	virescent: first leaf with green tip; greens slowly	S		255
v16	8L-14	virescent: like v2	S		255
v17	4	virescent: like v1, but greening from base to tip	S		255
v18	10	virescent: like v1	S		255
v21	8L	virescent (was v*-25): grainy virescent, greening from tips and margins inward	S		17
v22	1L-near an1	virescent: like v1 (E.G. Anderson, unpublished)	S		
v23	4-near su1	virescent: like v1 (E.G. Anderson, unpublished)	S		
v24	2L	virescent (was v*-424): like v1			230
v25	1S	virescent (was v*-17): greenish white seedling; greens from base upward			230
v26	2S	virescent (was v*-453): yellowish white seedling with green leaf tip and midrib			230
v27	7L	virescent (was v*-590A): like v1			230
v28	9S	virescent (was v*-27): like v1			230
v29	10L	virescent (was v*-418): grainy virescent			230

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REFERENCE
v30	9L-87	virescent (was v*-8587): like v1			44
va1	7L-near ij1	variable sterile: male sterile with some fertile anthers	S		12
Vg1	1L-85	vestigal glume: glumes very small, cob and anthers exposed	S	P	319
vp1	3L-near ts4	viviparous: embryo fails to become dormant; chlorophyll and carotenoids unaffected; anthocyanins in aleurone suppressed	S		85
vp2	5S-38	viviparous: embryo fails to become dormant; white endosperm, white seedling; anthocyanins unaffected	S	P	85
vp5	1S-1	viviparous: like vp2	S	P	273
vp7		(= ps1)			
vp8	1L-154	viviparous: embryo fails to become dormant; chlorophyll and carotenoids unaffected; small, pointed-leaf seedlings	S		274
vp9	7S-25	viviparous (also known as y7): like vp2	S	P	274
Vsr1	10L	virescent striped (was Vsr*-1486): virescent seedling; greens to white and yellow striped plant			238
w1	6L-near w14	white: white seedling	S		70 71 175
w2	10L-77	white: white seedling; endosperm pitted and spotted			177
w3	2L-111	white: like vp2	S	P	177
w11	9S-54	white: like w1	S		57
w14	6L-78	white: like w1	S		53
w15	6L-13	white: like w1	S		53
w16	7L-near vp9	white			218
w17	7S-near Hs1	white			218
Wc1	9L-107	white cap: kernel with white crown and pale yellow endosperm	S		163
wd1	9S-near yg2	white deficiency: white seedling: deficiency for distal half of first chromomere of short arm	S	P	197
wgs1	5L	white green sectors (was sct*-206B): white seedling with green sectors			230
whp1	2L	white pollen: duplicate factor with c2 for yellow pollen and for anthocyanins			46
wi1	6L-near y1	wilted: chronic wilting, delayed differentiation of metaxylem vessels	S		259
wlu1	3L	white luteus (was w1*-28): pale yellow seedling			230
wlu2	7L	white luteus (was w1*-543A): like wlu1			230
wlu3	8L	white luteus (was w1*-203A): like wlu1			230
wlu4	9L	white luteus (was w1*-41A): like wlu1			230
Wrk1	3S	wrinkled kernel (was Wr*-1020): kernels small and wrinkled			238
ws1	-	white sheath: light yellow leaf sheaths; duplicate factor with ws2	S		154
ws2	-	white sheath: see ws1	S		154
ws3	2S-0	white sheath: white leaf sheath, culm, husks	S	P	265
wsp		weak striped plant: maternally inherited pale striping			28
wt1	2S-60	white tip: tip of first leaf white and blunt	S		324
wt2	4S	white tip (was cb*-10): seedling with white leaf tip and crossbands on first 2 leaves			230
wx1	9S-56	waxy: amylopectin (stained red by iodine) replaces amylose (blue staining) in endosperm and pollen; starch-granule-bound NDP-starch glucosyl transferase	S	P	47
wyg1	7L-near ra1	white yellow green			218
y1	6L-17	white endosperm: reduced carotenoid pigments in endosperm; some alleles affects green pigments in seedlings	S	P	51
y3	2S-near al1	white endosperm: like y1			249
y7		white endosperm (also known as vp9, z1)			
y8	7S-18	white endosperm: pale yellow endosperm	S		141
y9	10S-24	white endosperm: pale yellow endosperm, slightly viviparous; green to pale green seedlings and plants	S		279
y10	3L	white endopserm: pale yellow endosperm; white seedling	S		275
y11	-	white endopserm: pale yellow endosperm; green seedling			323
y12	-	white endosperm: like y11			323
yd2	3L-near lg2	yellow dwarf			278
yg1	5L-near v2	yellow-green: yellow-green seedling and plant	S		83
yg2	9S-7	yellow-green: like yg1	S	P	138
ys1	5L-75	yellow stripe: yellow tissue between leaf veins, reflects iron deficiency symptoms	S	P	10
ys2	1S	yellow stripe: yellow tissue between leaf veins			258
ys3	3L-near Rg1	yellow stripe: like ys1	S		350
Ysk1	4-near su1	yellow streaked (was Ysk*-844): longitudinal yellow streaks top 3rd of mature leaves			234
z1		(= y7-z)			
zb1	-	zebra crossbands: yellowish crossbands on older leaves	S		55
zb2	-	zebra crossbands: crossbands on seedling leaves	S		330
zb3	5L-near v2	zebra crossbands: yellowish crossbands on older leaves (M. Demerec, unpublished)	S		
zb4	1S-19	zebra crossbands: regularly spaced crossbands on earlier leaves; enhanced by cool temperatures	S	P	122
zb6	4-79	zebra crossbands: regularly spaced crossbands on earlier leaves; enhanced by cool temperatures	S		124
zb7	1L	zebra crossbands (was zb*-101): lighter green crossbands on seedlings; glossy			230
Zb8	9-near wx1	zebra crossbands (was Atc1, C1*-1443): yellow-green crossbands on older leaves; strong anthocyanin expression in leaf tip and blade			234 238
Zer		Zapalote Chico earworm resistance: designator for earworm resistance factors from Zapalote Chico			212
zn1	10L-26	zebra necrotic: necrotic tissue appears between veins in transverse leaf bands on half-grown or older plants	S	P	120
zn2	-	zebra necrotic: like zn1	S		103
Zp		zein polypeptide: designator for loci determining zein polypeptides			313 314
zpg1	-	zebra-stripe pale green			63

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(gene symbol(s) referenced included within brackets)
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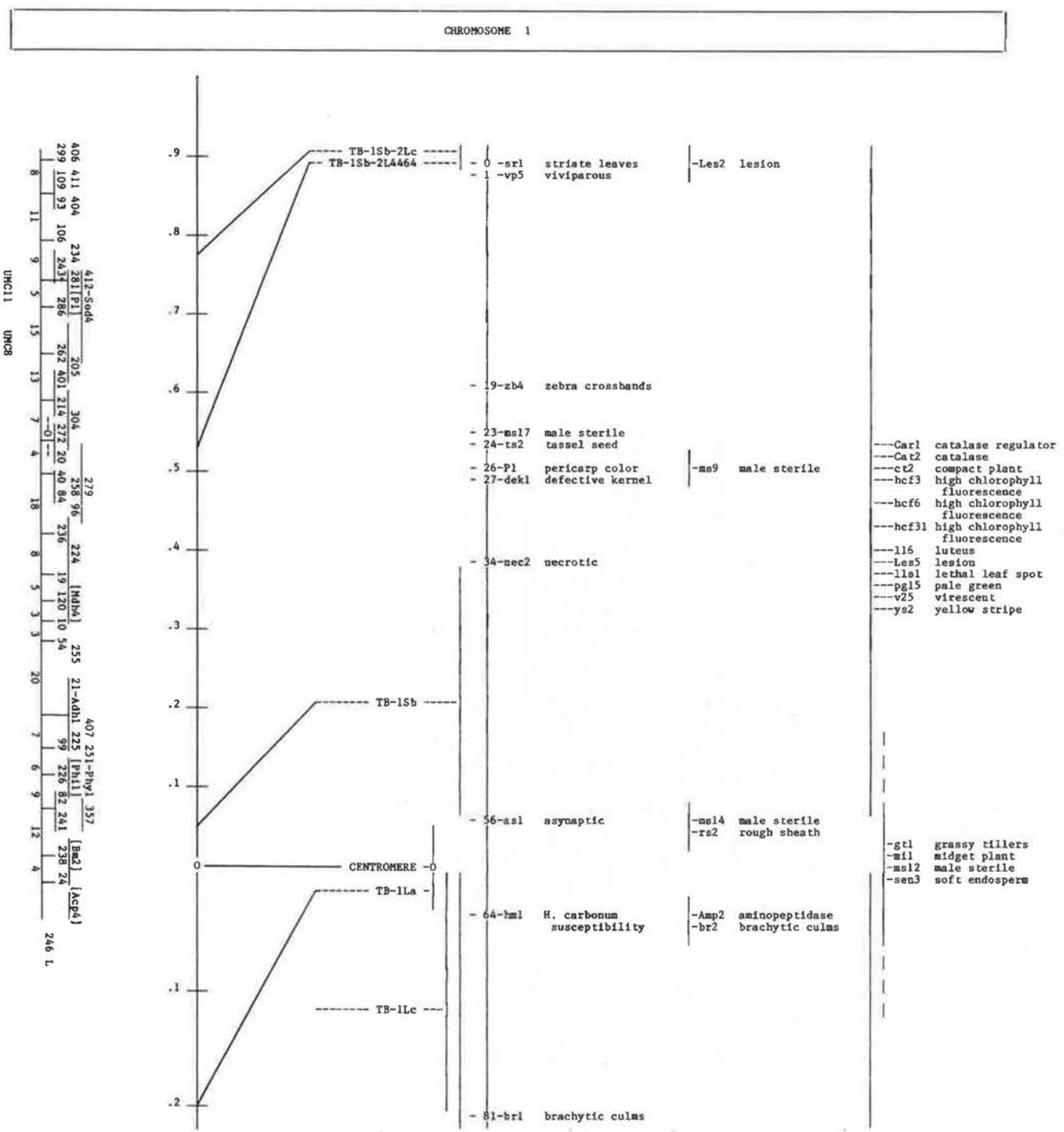
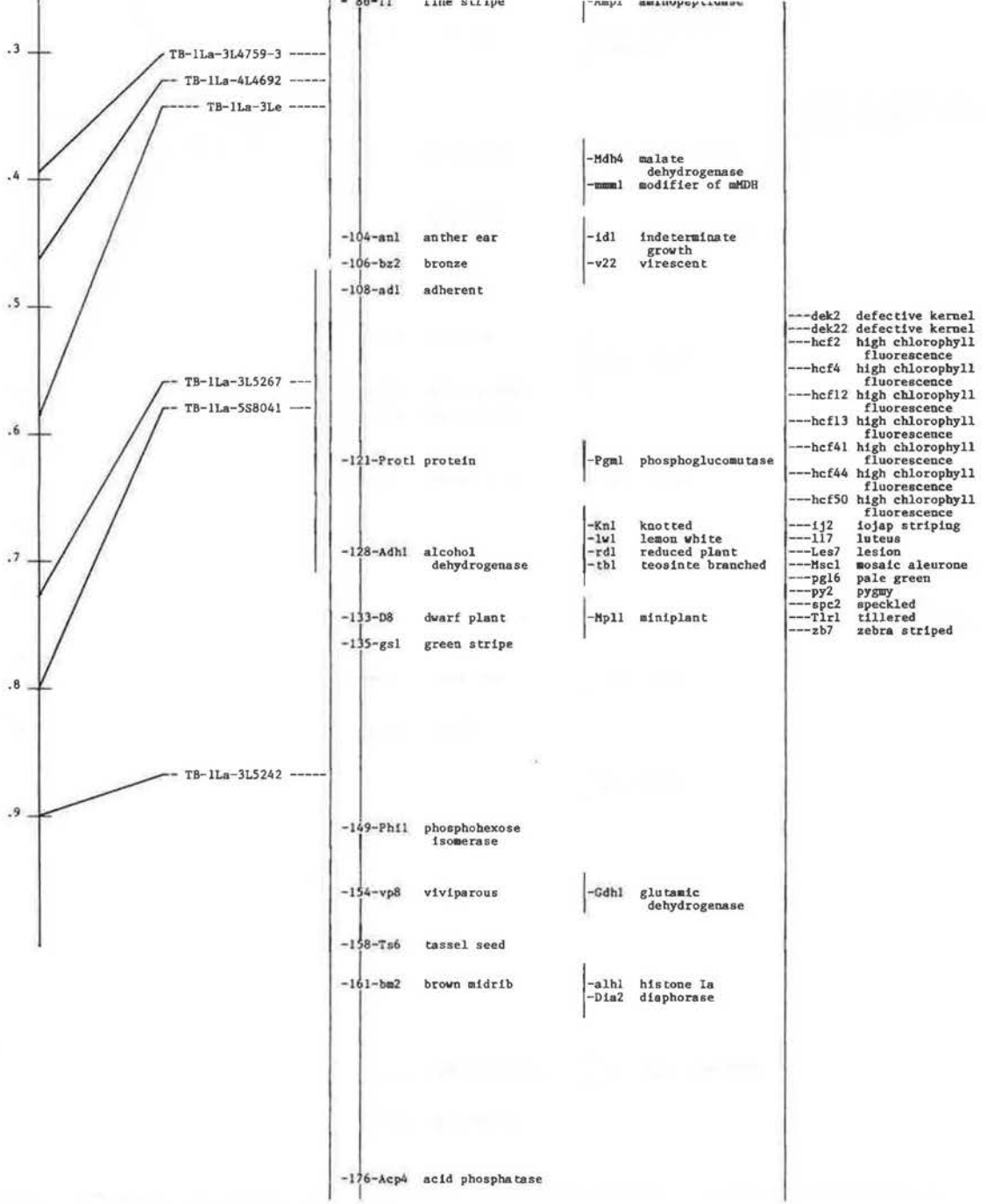

CHROMOSOME 1
TB-1Sb-2Lc
TB-1Sb-2L4464
0 -srl striate leaves
1 -vp5 viviparous
19-zb4 zebra crossbands
23-ms17 male sterile
24-ts2 tassel seed
26-P1 pericarp color
27-dek1 defective kernel
34-nec2 necrotic
TB-1Sb
56-as1 asynaptic
CENTROMERE -0
TB-1La
64-hm1 H. carbonum susceptibility
TB-1Lc
81-brl brachytic culms
-Les2 lesion
-ms9 male sterile
-ms14 male sterile
-rs2 rough sheath
-Amp2 aminopeptidase
-br2 brachytic culms
---Car1 catalase regulator
---Cat2 catalase
---ct2 compact plant
---hcf3 high chlorophyll fluorescence
---hcf6 high chlorophyll fluorescence
---hcf31 high chlorophyll fluorescence
---l16 luteus
---Les5 lesion
---lls1 lethal leaf spot
---pgl5 pale green
---v25 virescent
---ys2 yellow stripe
-gt1 grassy tillers
-mi1 midget plant
-ms12 male sterile
-sen3 soft endosperm
UMC11
UMC8
412-Sod4
[Pi1]
[Mdh4]
21-Adh1
[Phi1]
251-Phy1
[Bz2]
[Acp4]
246 L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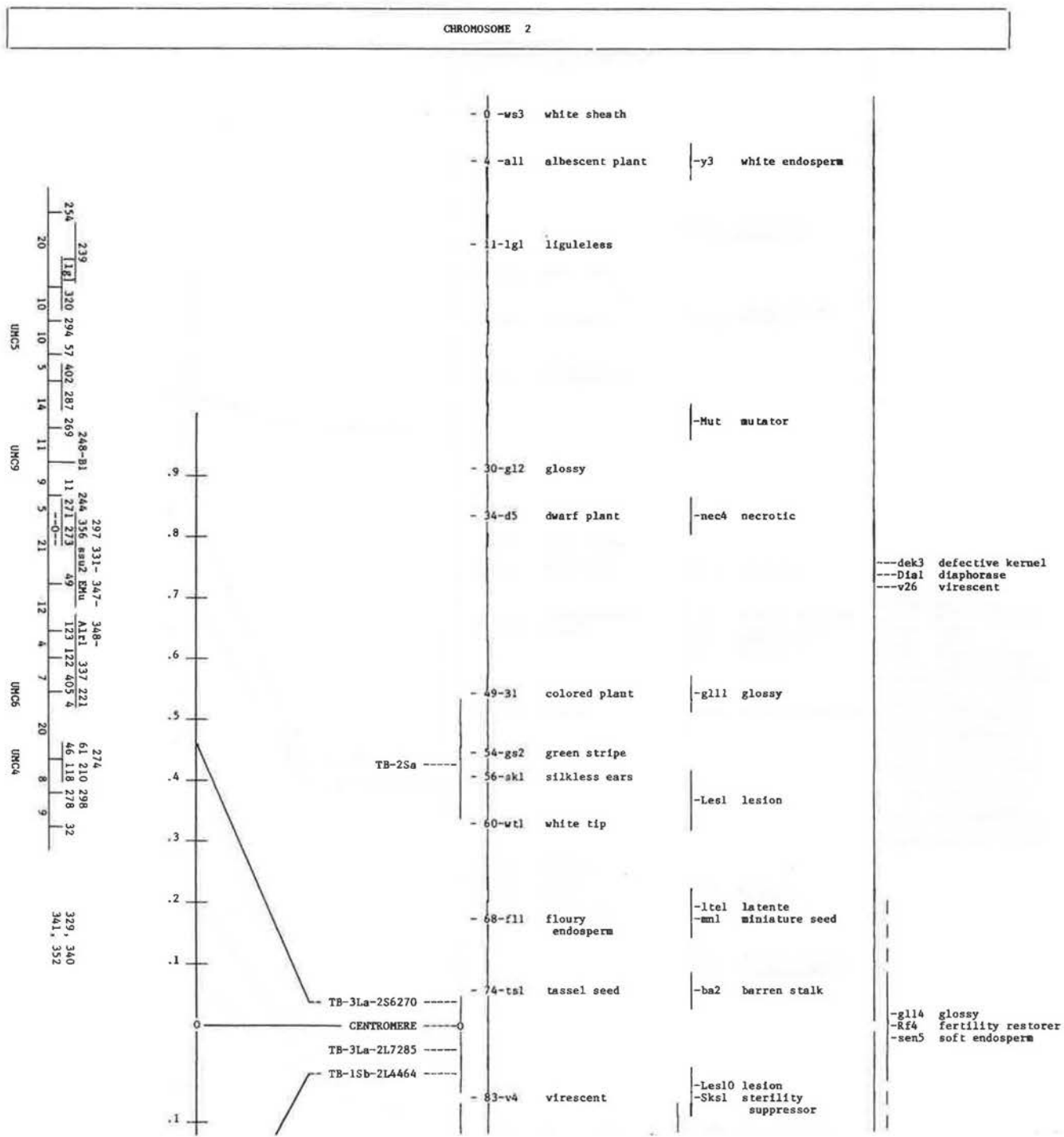
TB-1La-3L5267
TB-1La-5S8041
TB-1La-3L5242
-104-anl anther ear
-106-bz2 bronze
-108-ad1 adherent
-121-Prot1 protein
-128-Adh1 alcohol dehydrogenase
-133-D8 dwarf plant
-135-gs1 green stripe
-149-Phi1 phosphohexose isomerase
-154-vp8 viviparous
-158-Ts6 tassel seed
-161-bm2 brown midrib
-176-Acp4 acid phosphatase
-Mdh4 malate dehydrogenase
-mmm1 modifier of mMDH
-id1 indeterminate growth
-v22 virescent
-Pgm1 phosphoglucomutase
-Kn1 knotted
-lw1 lemon white
-rd1 reduced plant
-tb1 teosinte branched
-Mp11 miniplant
-Gdh1 glutamic dehydrogenase
-alh1 histone Ia
-Dia2 diaphorase
---dek2 defective kernel
---dek22 defective kernel
---hcf2 high chlorophyll fluorescence
---hcf4 high chlorophyll fluorescence
---hcf12 high chlorophyll fluorescence
---hcf13 high chlorophyll fluorescence
---hcf41 high chlorophyll fluorescence
---hcf44 high chlorophyll fluorescence
---hcf50 high chlorophyll fluorescence
---ij2 iojap striping
---l17 luteus
---Les7 lesion
---Msc1 mosaic aleurone
---pg16 pale green
---py2 pygmy
---spc2 speckled
---Tlr1 tillered
---zb7 zebra striped


CHROMOSOME 2
0 -ws3 white sheath
4 -al1 albescent plant
-y3 white endosperm
11-lg1 liguleless
-Mut mutator
30-gl2 glossy
34-d5 dwarf plant
-nec4 necrotic
---dek3 defective kernel
---Dia1 diaphorase
---v26 virescent
49-31 colored plant
-gl11 glossy
54-gs2 green stripe
TB-2Sa
56-sk1 silkless ears
-Les1 lesion
60-wt1 white tip
68-fl1 floury endosperm
-lte1 latente
-mn1 miniature seed
74-ts1 tassel seed
-ba2 barren stalk
TB-3La-2S6270
CENTROMERE
TB-3La-2L7285
TB-1Sb-2L4464
-gl14 glossy
-Rf4 fertility restorer
-sen5 soft endosperm
83-v4 virescent
-Les10 lesion
-Sks1 sterility suppressor
UMC5
UMC9
UMC6
UMC4
239
lg1
320
294
57
402
287
269
248-B1
11
271
273
--O--
244
356
mzu2
EMu
49
297
331-
347-
348-
Air1
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122
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61
210
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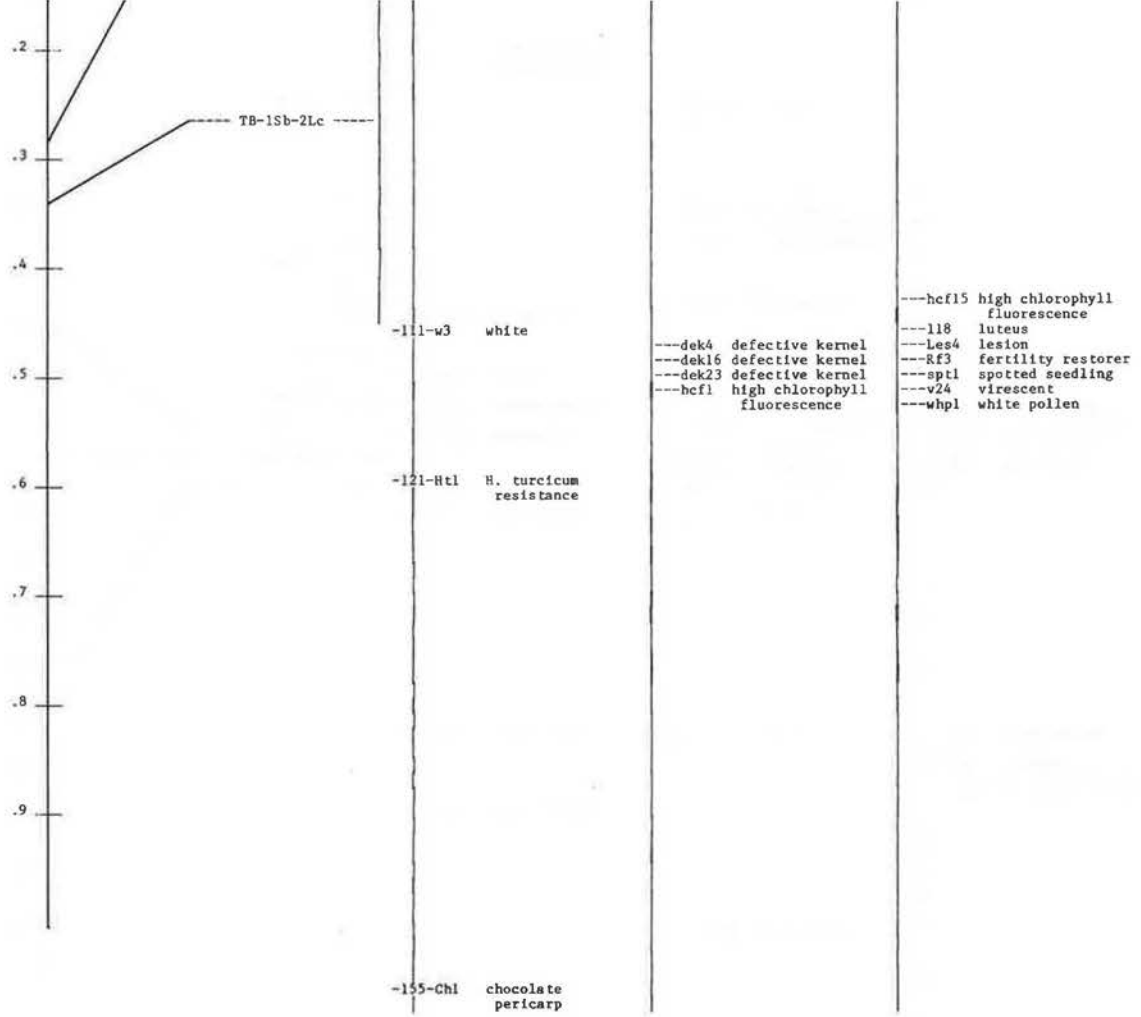
329, 340
341, 352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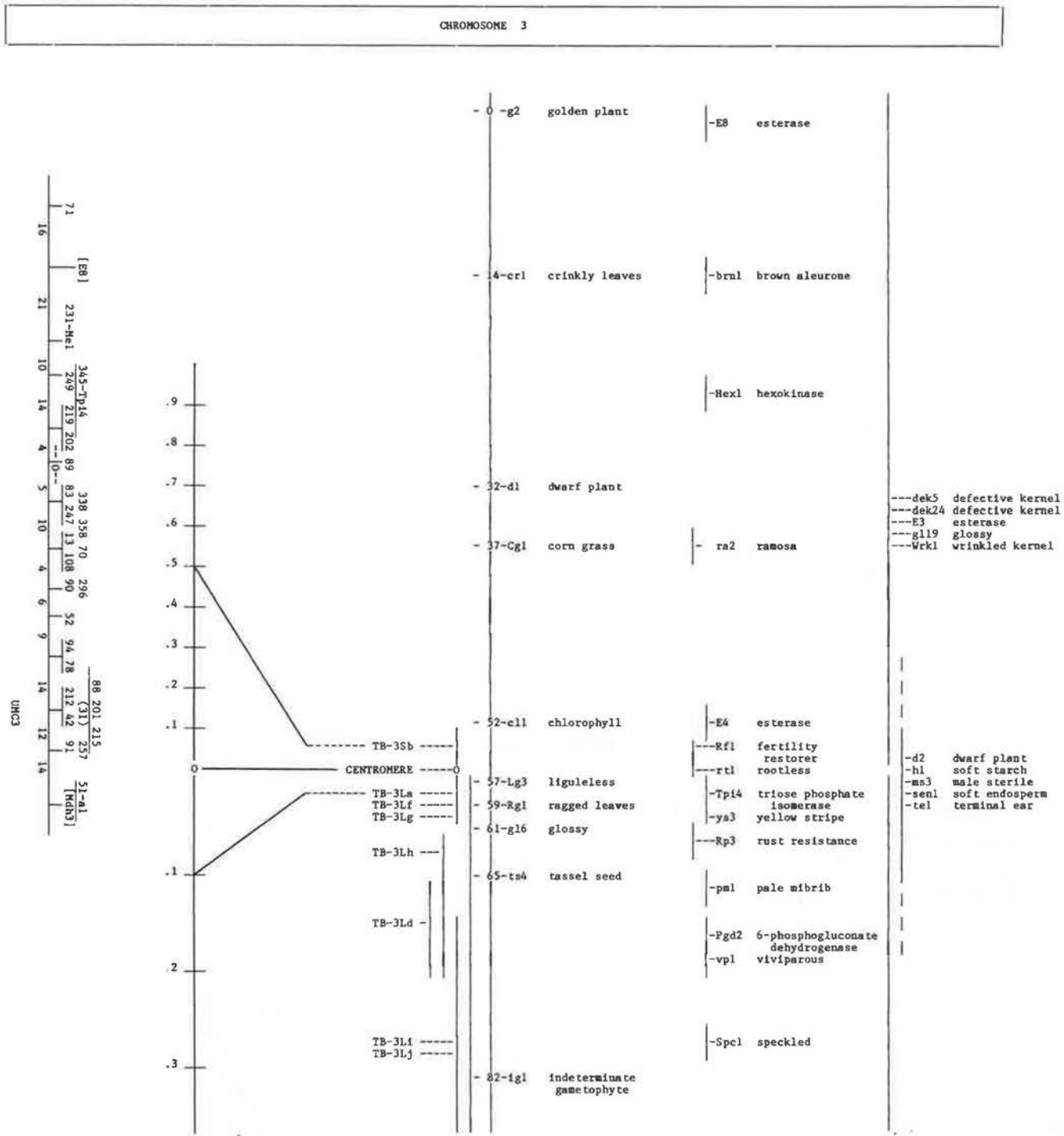
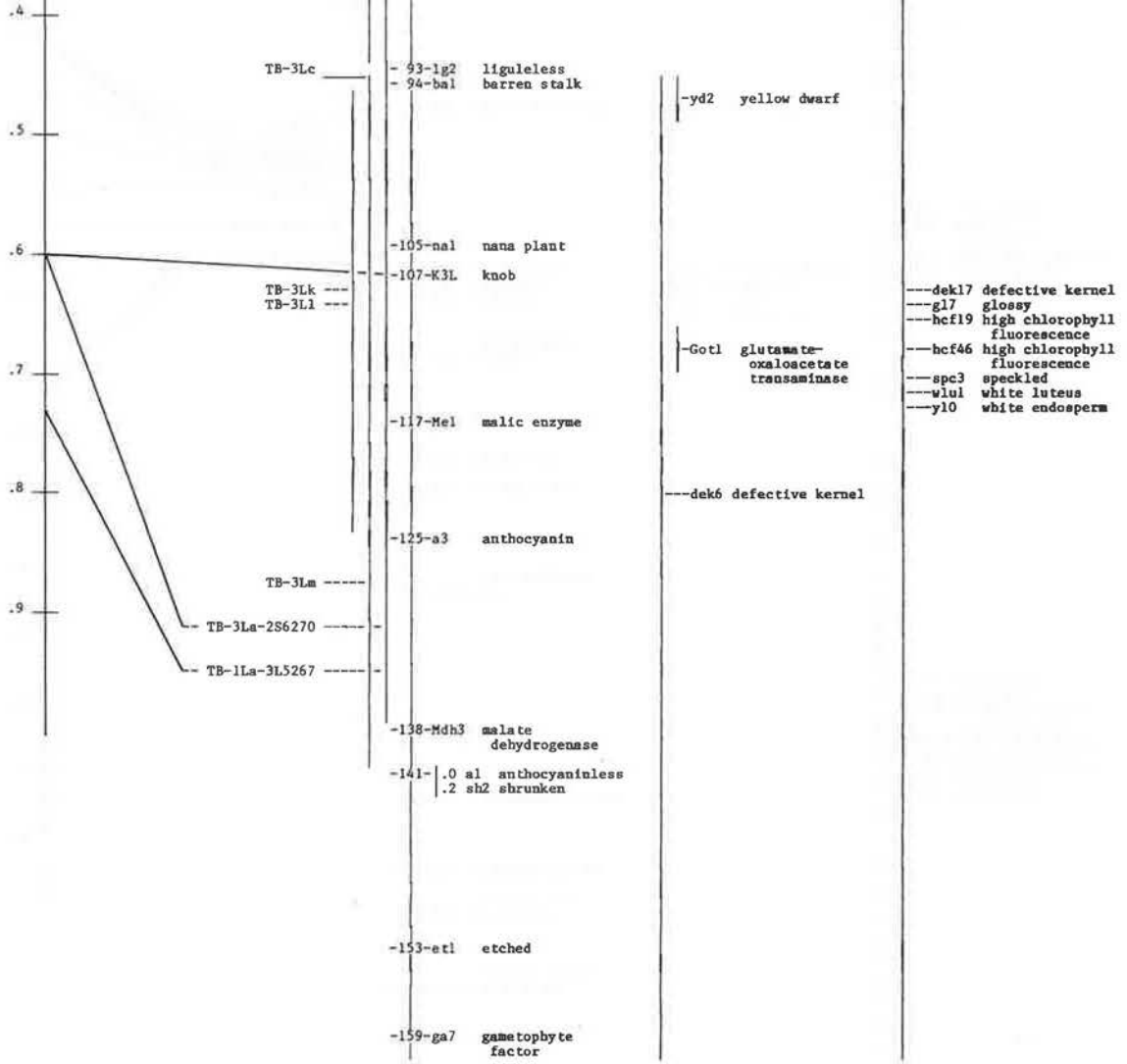
TB-1Sb-2Lc
-111-w3 white
-121-Htl H. turcicum resistance
-155-Chl chocolate pericarp
---dek4 defective kernel
---dek16 defective kernel
---dek23 defective kernel
---hcf1 high chlorophyll fluorescence
---hcf15 high chlorophyll fluorescence
---l18 luteus
---Les4 lesion
---Rf3 fertility restorer
---spt1 spotted seedling
---v24 virescent
---whp1 white pollen


CHROMOSOME 3
UMC3
[E8]
71
231-Mel
345-Tpi4
249
219
202
89
83
247
13
108
90
296
52
94
78
212
42
91
88 201 215
(31) 257
51-a1
[Mdh3]
TB-3Sb
CENTROMERE
TB-3La
TB-3Lf
TB-3Lg
TB-3Lh
TB-3Ld
TB-3Ll
TB-3Lj
0 -g2 golden plant
14-crl crinkly leaves
32-d1 dwarf plant
37-Cg1 corn grass
52-cl1 chlorophyll
57-Lg3 liguleless
59-Rg1 ragged leaves
61-gl6 glossy
65-ts4 tassel seed
82-ig1 indeterminate gametophyte
-E8 esterase
-brnl brown aleurone
-Hex1 hexokinase
ra2 ramosa
-E4 esterase
-Rf1 fertility restorer
-rtl rootless
-Tpi4 triose phosphate isomerase
-ys3 yellow stripe
-Rp3 rust resistance
-pm1 pale midrib
-Pgd2 6-phosphogluconate dehydrogenase
-vp1 viviparous
-Spc1 speckled
---dek5 defective kernel
---dek24 defective kernel
---E3 esterase
---gl19 glossy
---Wrk1 wrinkled kernel
-d2 dwarf plant
-h1 soft starch
-ms3 male sterile
-sen1 soft endosperm
-tel terminal ear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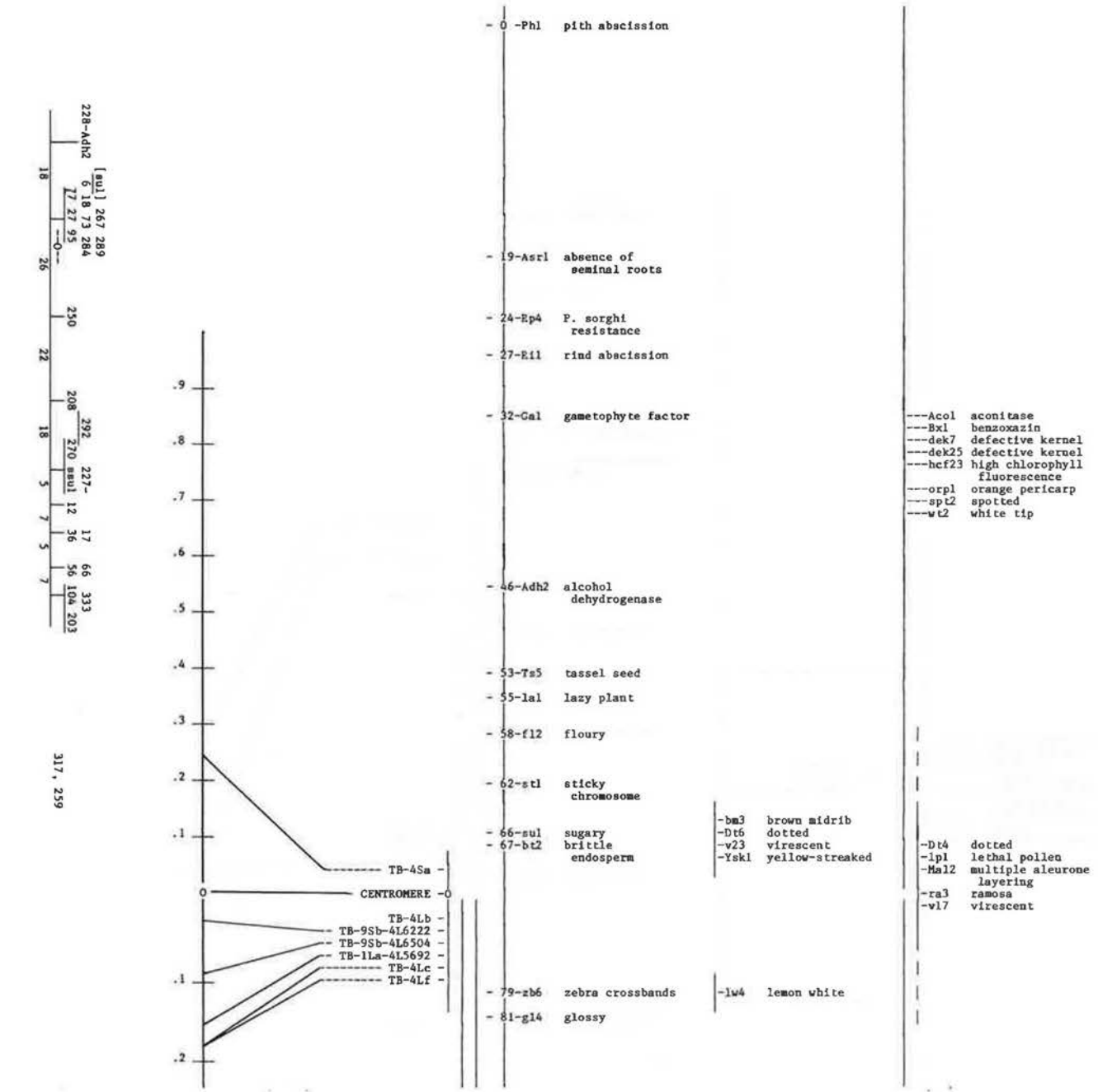
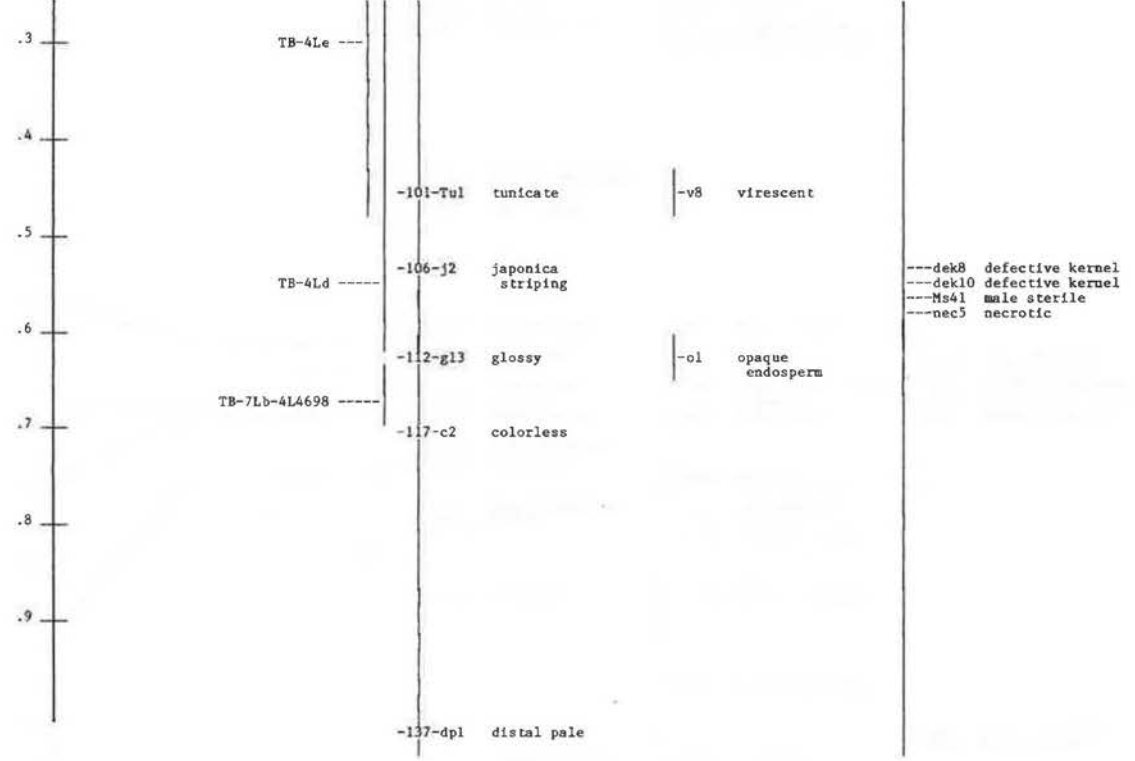
TB-3Lc
- 93-lg2 liguleless
- 94-ba1 barren stalk
-105-na1 nana plant
-107-K3L knob
TB-3Lk
TB-3Ll
-117-Me1 malic enzyme
-125-a3 anthocyanin
TB-3Lm
TB-3La-2S6270
TB-1La-3L5267
-138-Mdh3 malate dehydrogenase
-141-.0 a1 anthocyaninless
.2 sh2 shrunken
-153-et1 etched
-159-ga7 gametophyte factor
-yd2 yellow dwarf
-Got1 glutamate-oxaloacetate transaminase
---dek6 defective kernel
---dek17 defective kernel
---g17 glossy
---hcf19 high chlorophyll fluorescence
---hcf46 high chlorophyll fluorescence
---spc3 speckled
---wlu1 white luteus
---y10 white endosperm

CHROMOSOME 4

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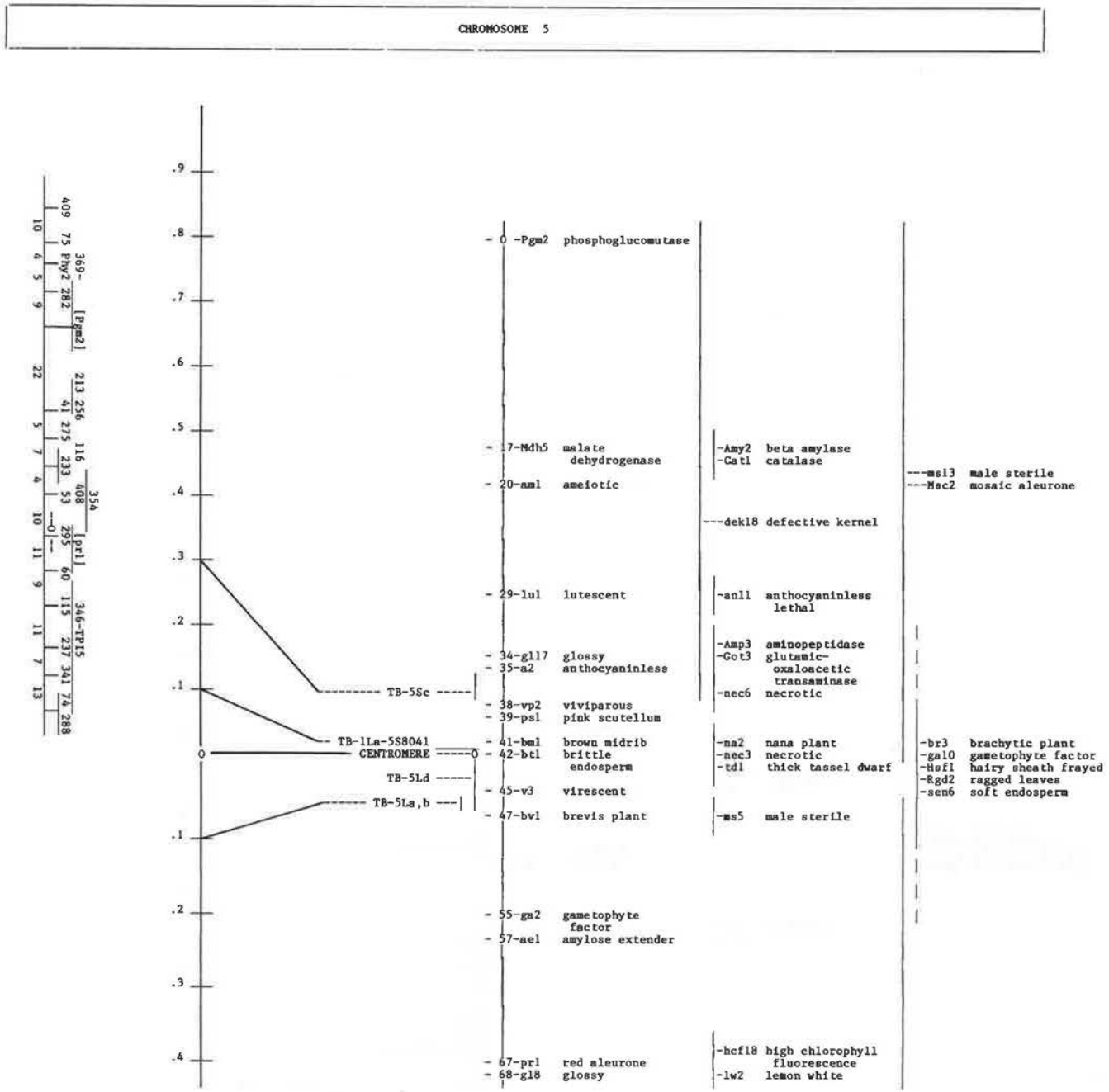
TB-4Le ---
TB-4Ld -----
TB-7Lb-4L4698 -----
-101-Tu1 tunicate
-106-j2 japonica striping
-112-gl3 glossy
-117-c2 colorless
-137-dp1 distal pale
-v8 virescent
-o1 opaque endosperm
---dek8 defective kernel
---dek10 defective kernel
---Ms41 male sterile
---nec5 necrotic


CHROMOSOME 5
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0
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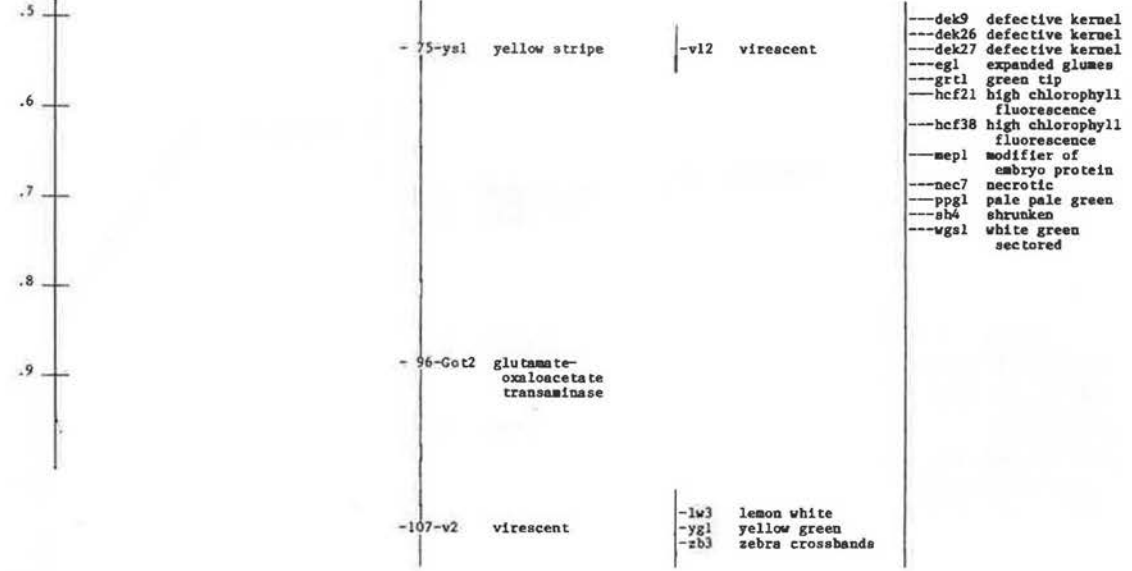
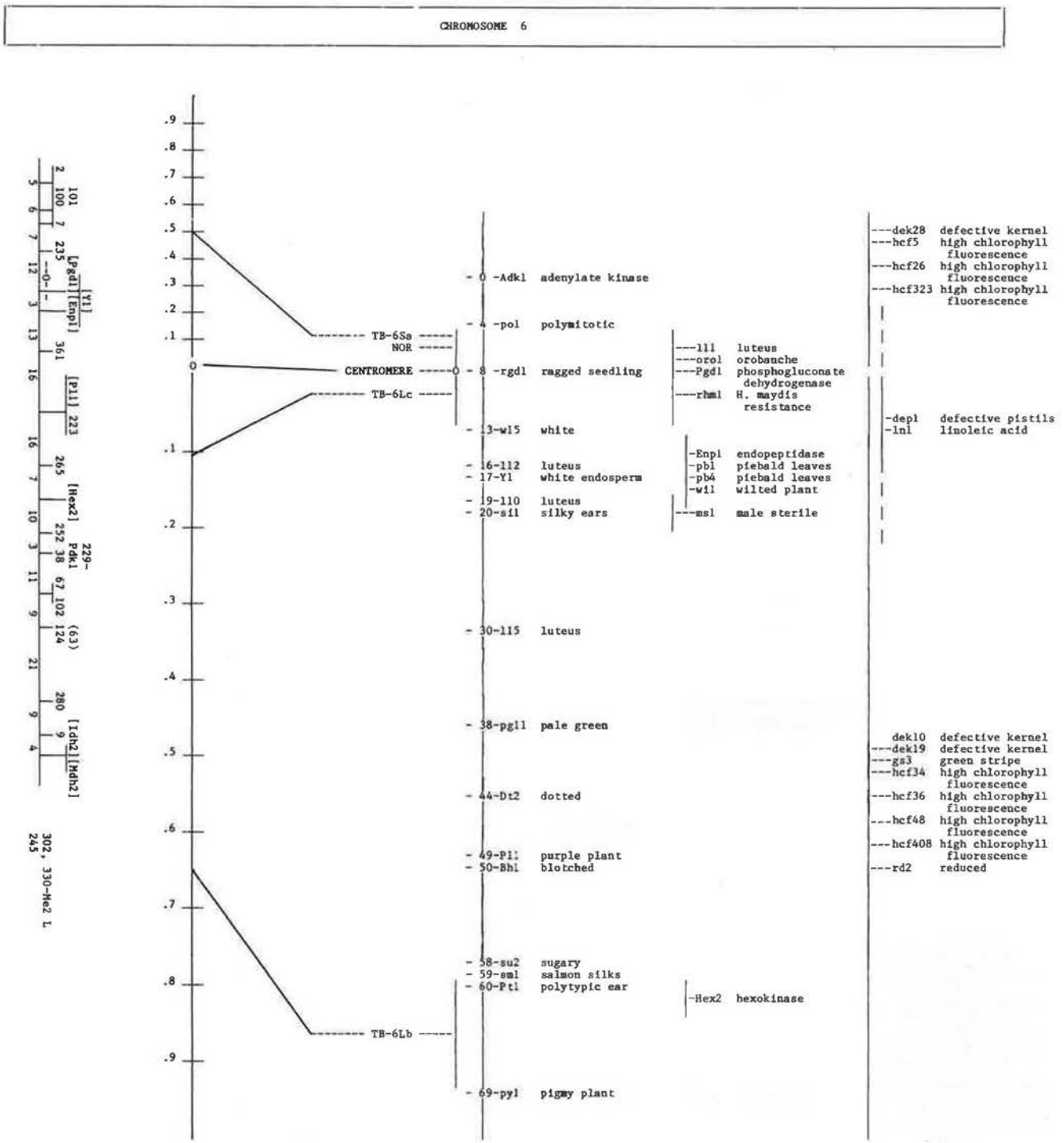
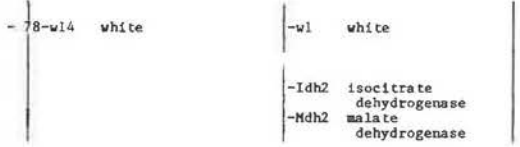
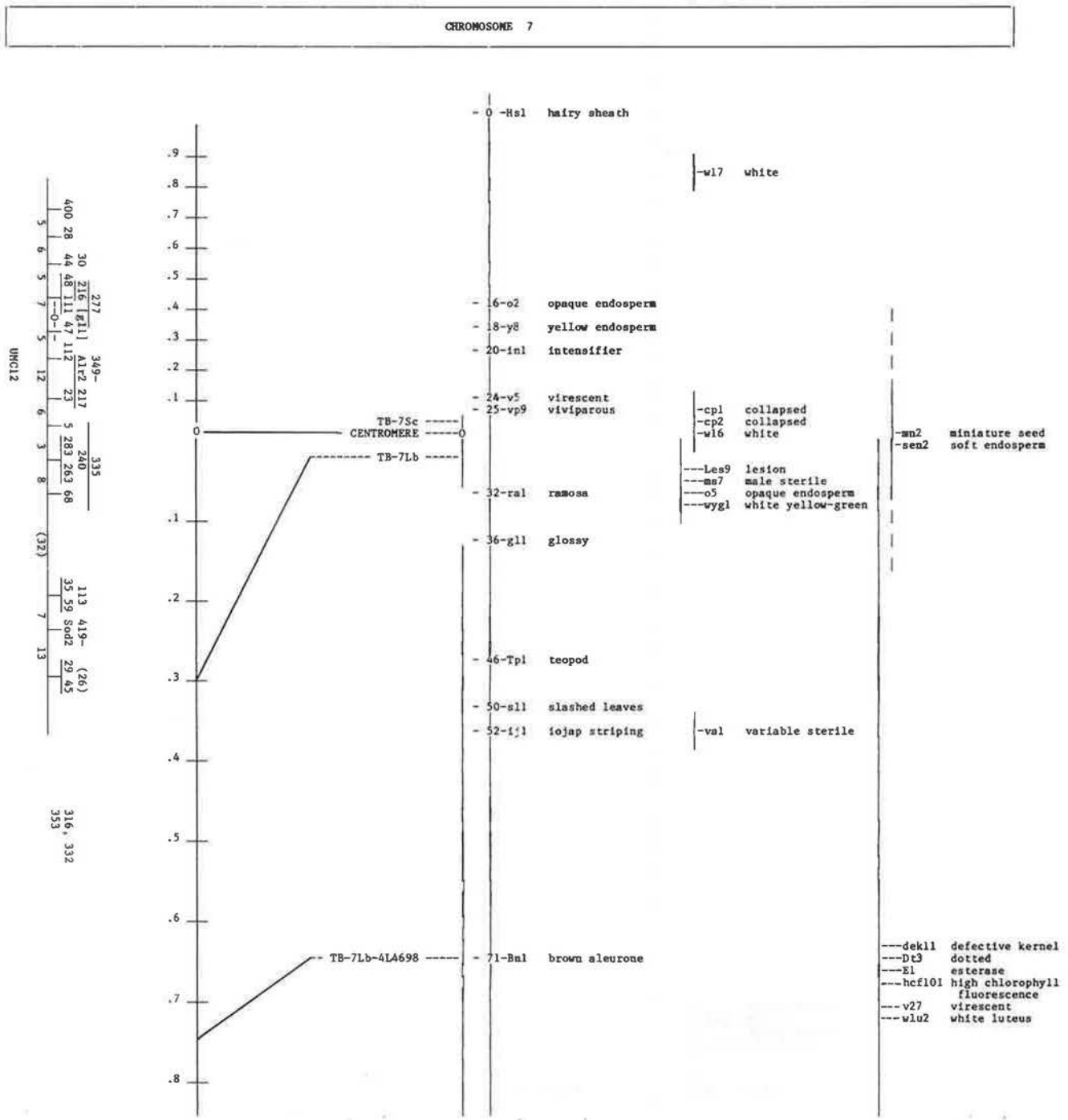
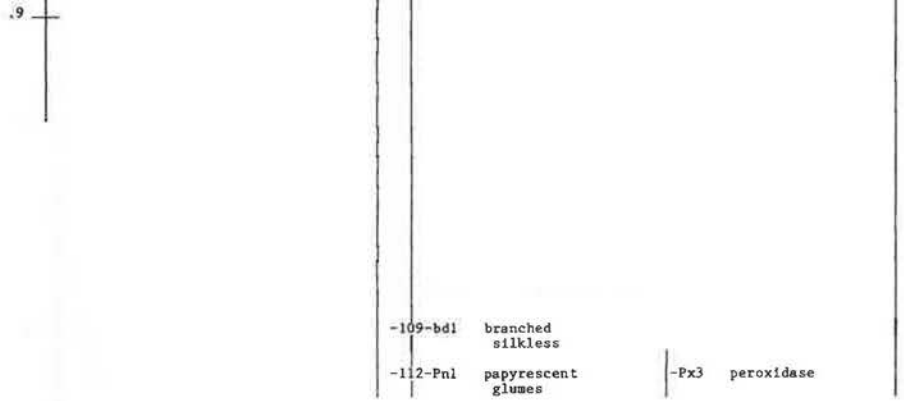
- 0 -Pgm2 phosphoglucomutase
- 17-Mdh5 malate dehydrogenase
- 20-aml ameiotic
- 29-lul lutescent
- 34-gl17 glossy
- 35-a2 anthocyaninless
- 38-vp2 viviparous
- 39-psl pink scutellum
- 41-bml brown midrib
- 42-btl brittle endosperm
- 45-v3 virescent
- 47-bvl brevis plant
- 55-ga2 gametophyte factor
- 57-ael amylose extender
- 67-prl red aleurone
- 68-gl8 glossy
-Amy2 beta amylase
-Cat1 catalase
---dek18 defective kernel
-anl1 anthocyaninless lethal
-Amp3 aminopeptidase
-Got3 glutamic-oxaloacetic transaminase
-nec6 necrotic
-na2 nana plant
-nec3 necrotic
-tdl thick tassel dwarf
-ms5 male sterile
-hcf18 high chlorophyll fluorescence
-lw2 lemon white
---msl3 male sterile
---Msc2 mosaic aleurone
-br3 brachytic plant
-ga10 gametophyte factor
-Hsfl hairy sheath frayed
-Rgd2 ragged leaves
-sen6 soft endosperm
TB-5Sc
TB-1La-5S8041
CENTROMERE
TB-5Ld
TB-5La,b
369-
[Pgm2]
Phy2
prl
TP15
10 4 5 9 22 5 7 4 10 11 9 11 7 13
409 75 282 213 256 41 275 116 233 408 354 53 295 60 115 346 237 341 74 288

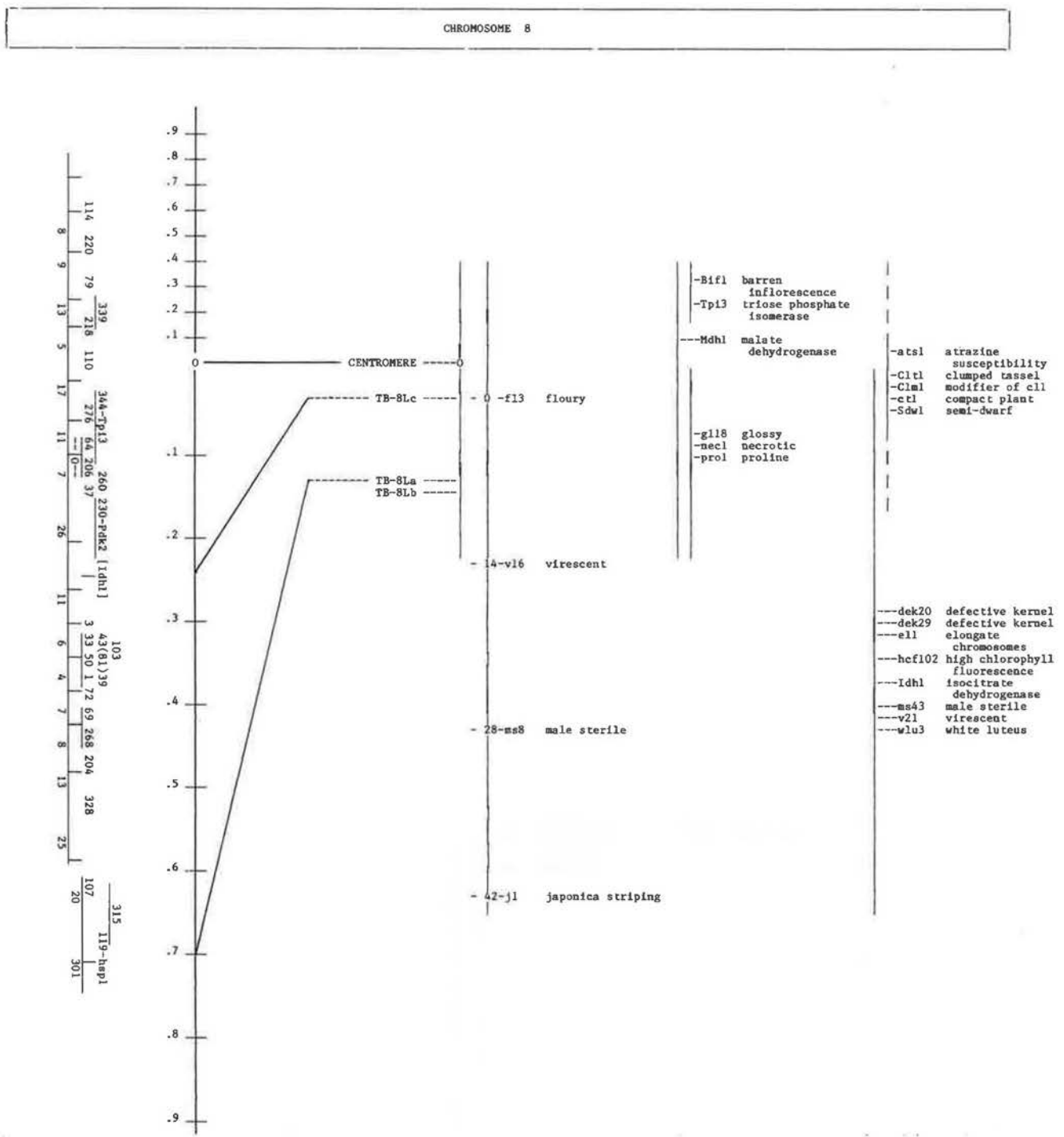

.5
.6
.7
.8
.9
- 75-ys1 yellow stripe
- 96-Got2 glutamate-oxaloacetate transaminase
-107-v2 virescent
-v12 virescent
-lw3 lemon white
-yg1 yellow green
-zb3 zebra crossbands
---dek9 defective kernel
---dek26 defective kernel
---dek27 defective kernel
---egl expanded glumes
---grtl green tip
---hcf21 high chlorophyll fluorescence
---hcf38 high chlorophyll fluorescence
---mep1 modifier of embryo protein
---nec7 necrotic
---ppg1 pale pale green
---sh4 shrunken
---wgs1 white green sectored

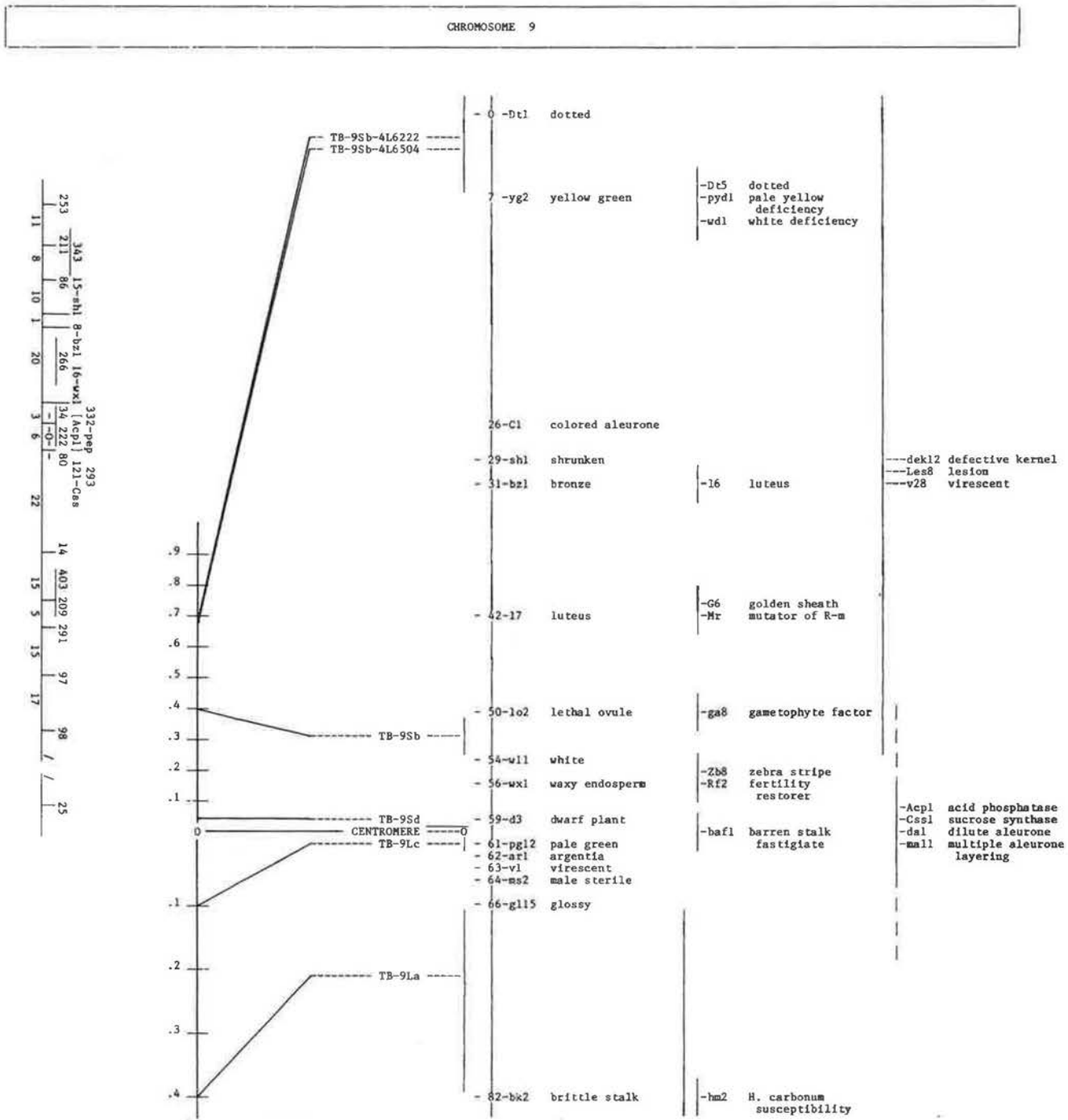

CHROMOSOME 6
.9
.8
.7
.6
.5
.4
.3
.2
.1
0
.1
.2
.3
.4
.5
.6
.7
.8
.9
TB-6Sa
NOR
CENTROMERE
TB-6Lc
TB-6Lb
- 0 -Adk1 adenylate kinase
- 4 -pol polymitotic
- 8 -rgd1 ragged seedling
- 13-w15 white
- 16-l12 luteus
- 17-Y1 white endosperm
- 19-l10 luteus
- 20-sil silky ears
- 30-l15 luteus
- 38-pgl1 pale green
- 44-Dt2 dotted
- 49-P1l purple plant
- 50-Bhl blotched
- 58-su2 sugary
- 59-sml salmon silks
- 60-Ptl polytypic ear
- 69-pyl pigmy plant
---l11 luteus
---orol orobanche
---Pgd1 phosphogluconate dehydrogenase
---rhml H. maydis resistance
-Enp1 endopeptidase
-pb1 piebald leaves
-pb4 piebald leaves
-wil wilted plant
---ms1 male sterile
-Hex2 hexokinase
---dek28 defective kernel
---hcf5 high chlorophyll fluorescence
---hcf26 high chlorophyll fluorescence
---hcf323 high chlorophyll fluorescence
-dep1 defective pistils
-lnl linoleic acid
dek10 defective kernel
---dek19 defective kernel
---gs3 green stripe
---hcf34 high chlorophyll fluorescence
---hcf36 high chlorophyll fluorescence
---hcf48 high chlorophyll fluorescence
---hcf408 high chlorophyll fluorescence
---rd2 reduced
[Y1]
[Pgd1] [Enp1]
[P11] 223
[Hex2]
Pdk1
(63)
[Idh2] [Hdh2]
101
100
235
361
265
252
38
67
102
124
280
9
229-
302+
245
330-Me2 L

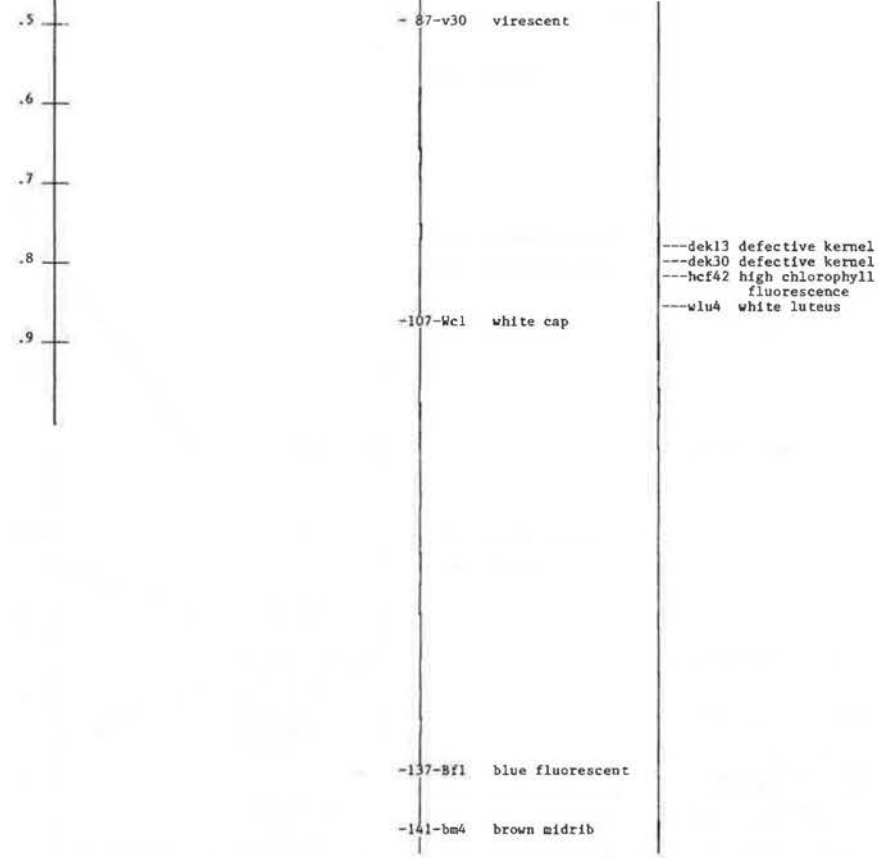

- 78-w14 white
-w1 white
-Idh2 isocitrate dehydrogenase
-Mdh2 malate dehydrogenase

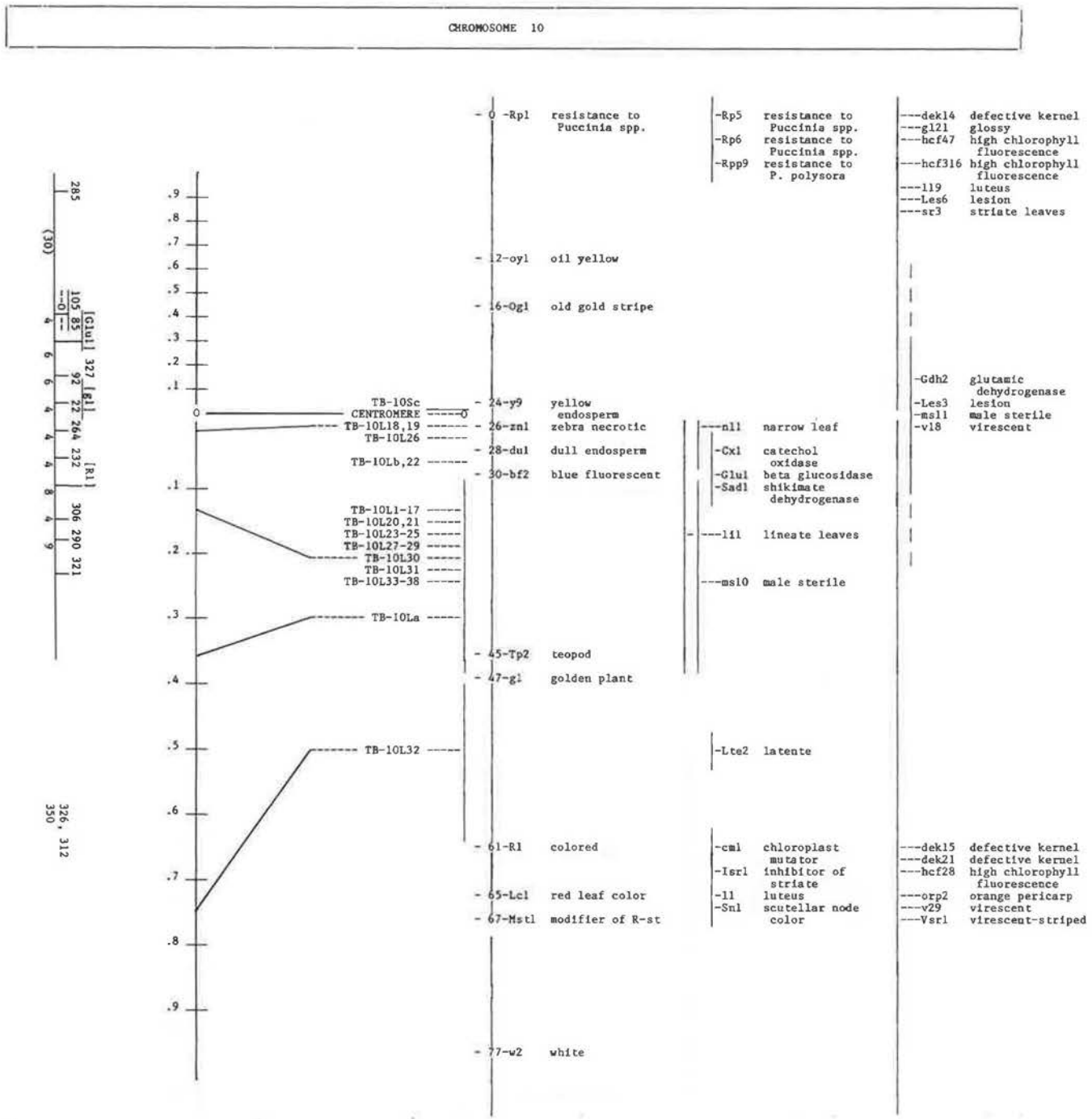

CHROMOSOME 7
- 0 -Hsl hairy sheath
-wl7 white
- 16-o2 opaque endosperm
- 18-y8 yellow endosperm
- 20-in1 intensifier
- 24-v5 virescent
- 25-vp9 viviparous
-cp1 collapsed
-cp2 collapsed
-wl6 white
-mn2 miniature seed
-sen2 soft endosperm
TB-7Sc
CENTROMERE
TB-7Lb
---Les9 lesion
---ms7 male sterile
---o5 opaque endosperm
---wyg1 white yellow-green
- 32-ra1 ramosa
- 36-gl1 glossy
- 46-Tp1 teopod
- 50-sl1 slashed leaves
- 52-ij1 iojap striping
-val variable sterile
TB-7Lb-4L4698
- 71-Bm1 brown aleurone
---dek11 defective kernel
---Dt3 dotted
---E1 esterase
---hcf101 high chlorophyll fluorescence
---v27 virescent
---wlu2 white luteus
UMC12
400 28 30 44 48 216 [gl1] 111 47 112 Alr2 217 23 349-
277
5 6 5 7 7 5 12 6 3 8 (32) 7 13
5 283 263 68 240 335
113 35 59 Sod2 29 45 419- (26)
316, 332 353
.9 .8 .7 .6 .5 .4 .3 .2 .1 0 .1 .2 .3 .4 .5 .6 .7 .8


.9
-109-bd1 branched silkless
-112-Pn1 papyrescent glumes
-Px3 peroxidase


CHROMOSOME 8
.9
.8
.7
.6
.5
.4
.3
.2
.1
0
CENTROMERE
TB-8Lc
TB-8La
TB-8Lb
-f13 floury
-14-v16 virescent
-28-ms8 male sterile
-42-j1 japonica striping
-Bif1 barren inflorescence
-Tpi3 triose phosphate isomerase
---Mdh1 malate dehydrogenase
-g118 glossy
-nec1 necrotic
-pro1 proline
-ats1 atrazine susceptibility
-Clt1 clumped tassel
-Clm1 modifier of clt1
-ct1 compact plant
-Sdw1 semi-dwarf
---dek20 defective kernel
---dek29 defective kernel
---el1 elongate chromosomes
---hcf102 high chlorophyll fluorescence
---Idh1 isocitrate dehydrogenase
---ms43 male sterile
---v21 virescent
---wlu3 white luteus
114 220 79 339
8 9 13 5
218 110
17
344-Tpi3
276 64 206 260
11 7 26
230-Pdk2 [Idh1]
37
11
103
3 33 50 1 72 69 268 204 328
6 4 7 8 13 25
43(61)39
315
107 119-hsp1
20 301


CHROMOSOME 9
TB-9Sb-4L6222
TB-9Sb-4L6504
TB-9Sb
TB-9Sd
CENTROMERE
TB-9Lc
TB-9La
0 -Dtl dotted
7 -yg2 yellow green
26-C1 colored aleurone
29-shl shrunken
31-bzl bronze
42-17 luteus
50-lo2 lethal ovule
54-wl1 white
56-wx1 waxy endosperm
59-d3 dwarf plant
61-pgl2 pale green
62-arl argentia
63-vl virescent
64-ms2 male sterile
66-gl15 glossy
82-bk2 brittle stalk
-Dt5 dotted
-pyd1 pale yellow deficiency
-wd1 white deficiency
-l6 luteus
-G6 golden sheath
-Mr mutator of R-m
-ga8 gametophyte factor
-Zb8 zebra stripe
-Rf2 fertility restorer
-baf1 barren stalk fastigiate
-hm2 H. carbonum susceptibility
---dek12 defective kernel
---Les8 lesion
---v28 virescent
-Acp1 acid phosphatase
-Css1 sucrose synthase
-dal dilute aleurone
-mall multiple aleurone layering
15-sh1
8-bz1
16-wx1
[Acp1]
121-Ces
332-pep
293
343
211
86
266
34
222
80
403
209
291
97
98
25


.5
.6
.7
.8
.9
- 87-v30 virescent
---dek13 defective kernel
---dek30 defective kernel
---hcf42 high chlorophyll fluorescence
---wlu4 white luteus
-107-Wc1 white cap
-137-Bf1 blue fluorescent
-141-bm4 brown midrib


CHROMOSOME 10
- 0 -Rp1 resistance to Puccinia spp.
-Rp5 resistance to Puccinia spp.
-Rp6 resistance to Puccinia spp.
-Rpp9 resistance to P. polysora
---dek14 defective kernel
---gl21 glossy
---hcf47 high chlorophyll fluorescence
---hcf316 high chlorophyll fluorescence
---l19 luteus
---Les6 lesion
---sr3 striate leaves
- 12-oy1 oil yellow
- 16-Og1 old gold stripe
-Gdh2 glutamic dehydrogenase
-Les3 lesion
-msl1 male sterile
-v18 virescent
TB-10Sc
CENTROMERE
- 24-y9 yellow endosperm
TB-10L18,19
TB-10L26
- 26-zn1 zebra necrotic
---nl1 narrow leaf
- 28-du1 dull endosperm
-Cx1 catechol oxidase
TB-10Lb,22
- 30-bf2 blue fluorescent
-Glu1 beta glucosidase
-Sad1 shikimate dehydrogenase
TB-10L1-17
TB-10L20,21
TB-10L23-25
TB-10L27-29
TB-10L30
TB-10L31
TB-10L33-38
---lil lineate leaves
---ms10 male sterile
TB-10La
- 45-Tp2 teopod
- 47-gl golden plant
-Lte2 latente
TB-10L32
- 61-R1 colored
-cm1 chloroplast mutator
-Isr1 inhibitor of striate
---dek15 defective kernel
---dek21 defective kernel
---hcf28 high chlorophyll fluorescence
- 65-Lc1 red leaf color
-l1 luteus
---orp2 orange pericarp
- 67-Mst1 modifier of R-st
-Sn1 scutellar node color
---v29 virescent
---Vsr1 virescent-striped
- 77-w2 white
[Glu1]
[gl1]
[R1]
285
(30)
105 85
--0 --
327
92
22 264
232
306 290 321
326, 312
350
.9
.8
.7
.6
.5
.4
.3
.2
.1
0
.1
.2
.3
.4
.5
.6
.7
.8
.9

- 87-o7	opaque endosperm		
- 91-113	luteus		
- 95-sr2	striate leaves	-K10	abnormal-10

COMPILED BY

DAVID A HOISINGTON
UNIVERSITY OF MISSOURI
FEBRUARY, 1987

The Physical and Genetic Map of the Mitochondrial Genome from the Wf9-N Fertile Cytoplasm

The restriction data (18) representing the single circular species of approximately 570 kb are unchanged from the data in MNL 60. The origin for all coordinate data is taken as the first base of the SstII recognition site, which is nearly coincident with a SmaI site (13,18) in the 5270 bp repeat (repeat-1) adjacent to the R1-homologous sequence. The major large repeated DNA sequence elements have been numbered in a clockwise direction and any future reference to the repeats will refer to the number rather than the size. Features such as the position of episome-related sequences, chloroplast-DNA related sequences and genes are given in the accompanying Figures and Tables.

In order to keep the map up to date, it would be appreciated if previously unidentified genes or other sequences could be sent directly to D. M. Lonsdale (Plant Breeding Institute), so that their position can be localised.

The data base containing all this information is available upon request from: Tony P. Hodge or David M. Lonsdale, Department of Molecular Genetics, Plant Breeding Institute, Maris Lane, Trumpington, Cambridge CB2 2LQ.

Map data for repeated sequences or genes.

(A) Repeats

Start	End	No.	Size	Reference
566.07	2.69	1	5270bp	13
245.67	250.94	1	5270bp	13
6.96	7.96	2	1kb*	
495.90	496.90	2	1kb*	
15.90	17.14	3	677bp	31
536.42	537.66	3	677bp	31
37.52	51.52	4	14kb*	
112.46	126.46	4	14kb*	
58.25	68.25	5	10kb*	
161.09	171.09	5	10kb*	
452.03	464.20	6	12kb*	
518.93	530.99	6	12kb*	

*Sizes estimated from restriction data.

(B) Genes

Start	End	Gene	Reference
298.84	301.49	ATP 9	11
311.60	312.80	COB	9
320.75	327.75	ND-1	2,8
320.75	327.75	S13	2
353.60	355.20	COX I	15
398.42	401.97	26S	7,27
417.83	417.96	5S	5,27
418.07	420.04	18S	6,27
427.89	450.69	COX III	30
454.21	452.68	ATP A-1	3,14
521.11	519.58	ATP A-2	3,14
537.70	539.32	COX II	12
556.74	561.04	ATP 6	10

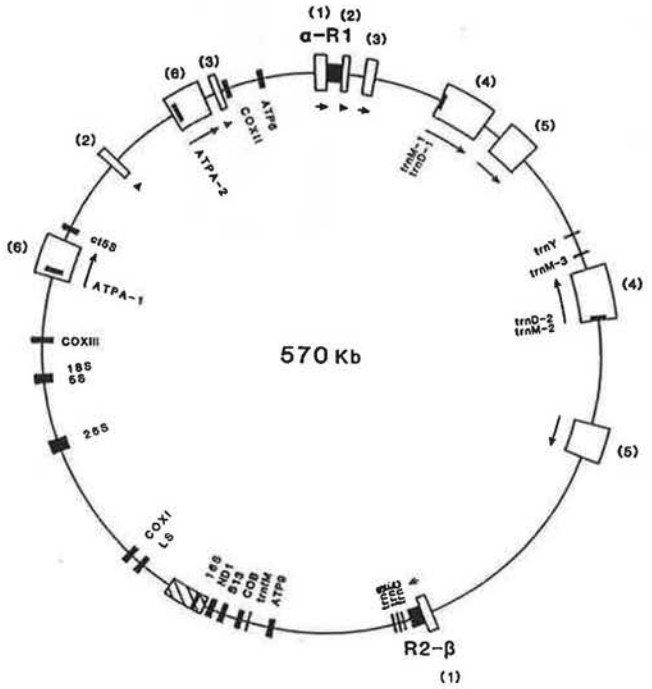
(C) Defined Sequences

Start	End	Sequence	Reference
349.65	348.30	LS	19
329.50	328.01	ct 16S	28
468.44	472.54	ct 5S	4,17
2.50	8.85	R1	13,16,20
250.75	256.20	R2	13,16,20

tRNA Coding Sequences

tRNA	Code	Coordinates	Reference
$tRNA_1^{Met}$	trnM-1	37.52 - 38.30	25
$tRNA_1^{Asp}$	trnD-1	38.30 - 39.26	26
$tRNA^{Tyr}$	trnY	92.73 - 103.05	22
$tRNA_3^{Met}$	trnM-3	105.4 - 108.6	24
$tRNA_2^{Asp}$	trnD-2	124.72 - 125.68	26
$tRNA_2^{Met}$	trnM-2	125.68 - 126.46	25
$tRNA^{Cys}$	trnC	258.4 - 266.5	29
$tRNA^{Phe}$	trnF	258.4 - 266.5	21
$tRNA^{Ser}$	trnS	258.4 - 266.5	29
$tRNA^{fMet}$	trnfM	308.9 - 320.7	24, 25

It cannot be assumed that these are functional genes. Omitted from this list are trnI, trnV and trnL (coordinates 327.4-336.6), which are known to be part of chloroplast sequences in the main genome (4,17,28). Also in this category are trnW and trnP, which are located on the 2.1/2.3 kb linear plasmid (1,23,29).



The above diagram is a circular representation of the data given in the accompanying tables. Repeats 1 to 6 (open boxes) and their relative orientation (arrows) are shown. The positions of known sequences (black boxes) including the 12 kb chloroplast sequence (hatched box) are labelled. The sequences flanking repeat-1 have been labelled α, β, R1 and R2 according to notation used previously (13).

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VII. RECENT MAIZE PUBLICATIONS

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Author and Name Indexes	
Nos. 3 through 43	Appendix to No. 44, 1970
Nos. 44 through 50	No. 50, pp. 157-180
Nos. 51 to date	Annual in each issue
Mailing List	In this issue
Stock Catalogs	
Genetic Stocks	Annual in each issue
Translocations	No. 55
Symbols	
Nos. 12 through 35	Appendix to No. 36, 1962
Nos. 36 through 53	No. 53, pp. 153-163
Nos. 54 to date	Annual in each issue
Rules of Nomenclature	No. 49, pp. 3-4
Cytogenetic Working Maps	No. 52
Gene List	In this issue
Working Linkage Maps	In this issue
News Letter Files (History)	No. 50
	No. 52
Reproductions	
1923	No. 52
1929	No. 53
1929-30	No. 54
1932	No. 56
1932-33 (MNL No. 3)	No. 57

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An interregional agronomic evaluation of inbred lines in five maturity groups has been carried out cooperatively and has been compiled by Larry Darrah. Copies are available at cost; please see page 52.