MAIZE GENETICS COOPERATION

NEWS LETTER

62

March 31, 1988

Department of Agronomy
and
U.S. Department of Agriculture
University of Missouri
Columbia, Missouri

TABLE OF CONTENTS

Gifts to the endowment fund for the News Letter are currently being received in the mail at press time. The developing support from individuals and organizations is greatly appreciated.

Donors will be listed in the 1989 issue.

I. FOREWORD

"The research newsletter is particularly suitable for information not usually suitable for scientific journals: continuing updating of gene symbols and nomenclature, location of mutants, strains and stocks... Short technical notes, short research findings, observations, new ideas and compilations of data are also suitable." --H. V. Wyatt, Research Newsletters in the Life Sciences: their scope, advantages and future, 1986.

By sharing our technical data, short notes, short findings, observations, compilations and new ideas here, we contribute to the advancement of biology and to the power of shared technical knowledge. We have known the advantages of a newsletter for more than a few years, and the expanding impact of Zea genetics in plant biology is legendary. The information here is shared with the specific understanding that it is unpublished information, not to be cited in publications without the consent of the authors.

Notes for the next issue (Number 63, 1989) should be in my hands by January 1, 1989. Short, informal notes including explicit data are preferable to narratives, rationales, references, and elaborate interpretations. Text of the note should communicate simply and efficiently, and should be double-spaced for editing. Tables, Figures and Charts must be compact, ready for direct copying by the camera. Electronic versions on floppy disk should be supplied if possible.

Subscription to the News Letter (or change of address) can be initiated with the form in the back of this issue. Gifts and lifetime subscriptions are being received to underwrite costs to the extent possible. Subscription letters and letters requesting gifts were sent so recently that at press time many potential respondents may not have received a letter. Consequently the list of donors is limited to the earliest recipients and earliest respondents; we are all grateful for the support of our colleagues, and of organizations with which we have common interests.

Dave Hoisington assembled and developed the impressive gene list and linkage maps in this issue, using his knack for encompassing the status of each research area. His advice and ideas for the News Letter, and many computer trickeries, make all the editing work feasible.

Shirley Kowalewski, Lou Martin Butler, Sherri Harris, Susan O'Dell and Mary Brazil skillfully edited and refined the copy and the proofs, kept office tasks in order, and helped in screening the literature. Kathy Chappell, Chris Browne and Mary Ann Steyaert helped with many vital tasks. Bryan Bailey, Jack Beckett, Chang-deok Han, Scott Johnson, G. Madhavi Reddy, and Allen Wright proofed copy and gave other aid. At University Printing Services, Yvonne Ball and Dale Kennedy and their staff again efficiently and carefully made sure that the job was done promptly and well.

Chloroplast maps, generously volunteered and impressively prepared by Steven Rodermel and Lawrence Bogorad, are included this year. For mitochondria, according to David Lonsdale, no new update is warranted, but please see the proposed nomenclature for mitochondria offered by David Lonsdale and Christopher Leaver; evaluations and comments are invited.

Ed Coe

II. REPORTS FROM COOPERATORS

ALBANY, CALIFORNIA
Plant Gene Expression Center, USDA - ARS

New ear phenotype recovered in *Mu* field - "fasciated ear"

In an ongoing project with Michael Freeling to recover new transposon-induced mutants, a novel ear phenotype was found in a *Mu*-containing family. The mutation is recessive and is now in its third generation of crosses. The ear begins development as an unbranched structure, but subsequently the apex divides to form first and second order branches (Fig. 1). The resulting ears are small, rounded and hollow with a proliferation of silks. The ears can be pollinated (though it is difficult to keep them shootbagged) and kernels form on the outer and inner surfaces of the ear. Segregating within the mutant-ear population is an unbranched tassel phenotype. Three of 7 branched-ear individuals had unbranched tassels out of 60 total, whereas there were no unbranched tassels on plants with unbranched ears.

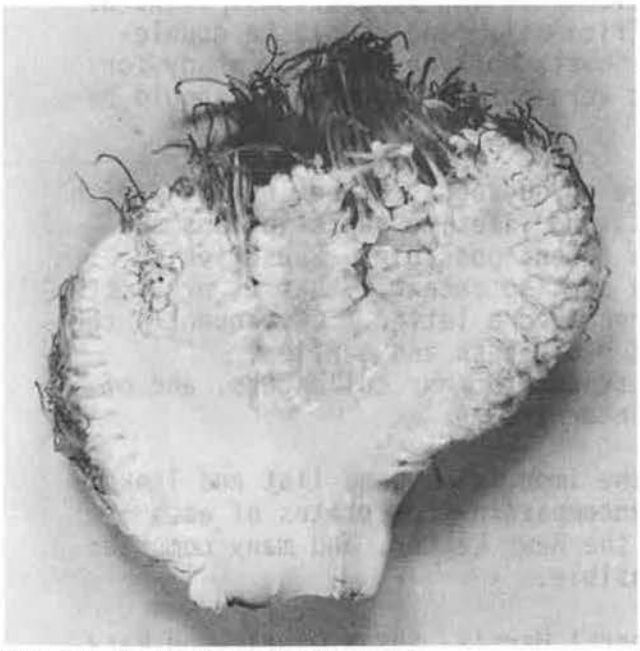


Figure 1. Section through mutant ear.

The only description in the literature, to our knowledge, that resembles this ear phenotype is *club* (N.H. Nickerson and E.E. Dale, Ann. Mo. Bot. Gard. 47:227, 1955). *Club* is described as "apparently caused by one or more incomplete divisions of the growing point when the ear is partly formed, followed by simultaneous development of each new point into a more or less independent ear tip". However, while the tassel of *club* is described as exceedingly thick due to shortened internodes, the tassel of this new mutant is of normal thickness. J.H. Kempton (U.S. Dept. Agr. Bull. 97, 1921) describes a number of branched ear abnormalities, including one entitled "bearsfoot". Our interpretation of Kempton's figures is that the branching in these various abnormal ears occurs at the base of the ear rather than at later occurring divisions of the apex.

Sarah Hake and Bruce Veit

AMES, IOWA
Iowa State University

Penetrance and expressivity of twin ears

Twin ears were first observed in an S5 progeny of BSAA *o2* and an S2 progeny of BS10(FR)C2 (MNL 58:21). Progenies were continued in the breeding nurseries by self-pollination and ears were selected from plants with the twin-ear expression. In 1985 and 1986, data were collected from 25-plant nursery rows to obtain estimates of penetrance and expressivity of 65 progenies derived from the original two ears. The original twin ears were at the top-ear node, but twin-ear expression also was observed at a lower frequency at the 2nd and 3rd nodes. In addition to recording the relative penetrance in 1986, ears were harvested from all pollinated plants within the nursery rows to determine the relative expressivity of twin-ear expression. Data collected in 1985 and 1986 are summarized here along with six selected progenies to illustrate the differences between years for penetrance and for expressivity in 1986.

			Expressivity			
	Penetrance(%)		Seed number		Seed weight	
	1985	1986	Twin	Single	Twin	Single
Average (all progenies)	83	79	401	234	100	55
Range (all progenies)	64-100	56-100	384	738	78	118
Selected progenies						
1673-3031A	100	86	415	—	104	—
1678-3042A	76	81	578	—	100	—
1686-3062B	76	100	184	—	51	—
1691-3065A	100	95	363	—	96	—
1694-3067B	93	100	332	—	82	—
1698-3074A	95	95	359	—	103	—
Average	83	79	379	—	89	—

The frequency of penetrance at the top-ear node averaged 83% in 1985 and 79% in 1986. Some progenies had 100% expression for twin ears, but none expressed 100% twin ears in both years; 1691-3065A had 100% expression in 1985 and 95% in 1986, whereas 1694-3067B had 93% expression in 1985 and 100% in 1986. Twin-ear expression at the second node was less than the top node in both years with an average twin-ear expression of 32% in 1985 and 54% in 1986. The frequency of twin ears at the second node was 22% greater in 1986 vs. a 4% lower frequency at the top node.

Data were collected for each of the twin ears for seed number and weight and for the one-ear segregates to estimate expressivity. Each pair of twin ears was visually designated as largest and smallest of the two ears. Average numbers of seeds for each ear of the pair were 219 and 182, or 401 seeds for 91 pairs of twin ears; the 27 one-eared segregants averaged 234 seeds per ear. The twin-eared plants had 71.4% more seeds than the one-eared plants. Average total seed weight for the twin-eared plants (100.1g) was 81.8% greater than for the one-eared plants (55.4g). If total seed weight is divided by total seed number to estimate the average seed size, average weight of each

seed was 0.25g for the twin-ear plants vs. 0.24g for the one-ear plants. The twin-ear plants, therefore, had greater numbers of seeds that were similar in size to the seeds of one-eared plants.

Testcrosses of 41 twin-eared plants, used as females, were produced in 1986 with lines (B77, B79, B88, B90, B91, and Mo17) that did not exhibit twin ears. Only three twin-ear plants were observed in the testcrosses and all occurred in testcrosses with Mo17. Crosses of twin-eared progenies with other twin-eared progenies, however, tend to increase the frequency of twin ears. Twin ear seems to be a threshold trait that may be difficult to stabilize. Although some progenies have a high frequency of expression in different years, it may be difficult to transfer to other lines. Greatest utility of twin-ear expression may be for genetic studies in which multiple pollinations on a single plant are desired.

Arnel R. Hallauer

a-m877527 - a putative *Cy*-regulated unstable allele

A newly arisen unstable *a* allele has been confirmed. This allele arose as a single kernel exception. The pattern of mutability is of a late medium-fine quality, our classification 7-b (Reddy and Peterson, MGG, 1983). As evident in the tabulation, there is a high frequency of spotting, indicative of many regulatory elements or autonomous control of mutability.

Origin: Colored *A A*. Male parent: *a et*. Exception: spotted. Cross of exception: *a-m/a et* × *a et/a et*. Progeny:

	Colored	Spotted	Colorless	T	% sp
87 7527 × 0170	0	178	134	312	57.05
87 7527 × 0170	0	135	99	234	57.9

Tests for system relation are in progress.

Peter A. Peterson

c2-m85-2 and *c2-m85-3* - two unstable *c2* alleles

These two alleles arose as single-kernel independent events on separate ears in a TEL population that contains a number of elements (Schnable and Peterson, Maydica, 1986). The ratios in testcrosses are indicative of independent control.

Origin: *C2/C2*. Male parent: *c2/c2*. Exception pattern: 5-6 b-c, 3-bc. Cross of exception: *c2-m/c2* × *c2/c2*. Progeny:

	Colored	Spotted	Colorless	Total
c2-m85-3 87 6750 × −1E × 6114	2	65	328	395
c2-m85-2 87 6750y/tiller [×]	25	57	132	214

System relationships are in progress.

Peter A. Peterson

bz-m877803 - a putative *En*-related *bz*-unstable

A single kernel exception showing full colored spots on a bronze background, pattern—very-high, late (our classification 8a-b), arose on an ear that was *Bz/Bz*. The segregation pattern is indicative of a two-element control.

Origin: *Bz/Bz*. Male parent: *C sh bz wx*. Exception: spotted. Cross of exception: *bz-m/bz sh* × *bz sh*. Progeny:

	Round			Shrunken	
	Colored	bz-spotted	bz no sp.	bz sp.	bz-no sp.
87 7803 × 0180	2	184	141	3	292

Peter A. Peterson

a2-m668291 - linkage of *Cy* with *pr*

The mutability of the *a2-m668291* unstable allele is controlled by *Cy*. In a testcross of this allele with *a2 bt*, mutability (presence of *Cy*) was linked with *pr*. Cross: *A2 Pr/a2-m pr Cy* × *a2 bt pr/a2 bt pr*.

		Spotted		
	Colored	pr	Pr	Colorless
87 3113-21/2902	246	155	3	114
Expected if *Cy* independent		114	44	

This would make *Cy* distal to *pr*.

Peter A. Peterson

A-m(r)-Cuna

The *A-m(r)-Cuna* allele is a fully colored allele of the *A* locus and the resident allele of the Cuna (Colombia) accession where *Fcu* was uncovered (Gonella and Peterson, Genetics, 1977). This allele is partially suppressed by *En* and in the presence of *En*, expresses darker spots on a dark background aleurone coloration. (The intensity of color is darker than the *a-m-15719A-1* coloration in the absence of *En* but clearly as dark as the *a-pale-m(r)* allele in the presence of *En* (Nowick and Peterson, MGG, 1981).

The presence of dark spots is indicative of the presence of an *I* element that responds to *En*, but unlike the *a-m-15719A-1* allele, it is not suppressed in coloration. Thus, either the orientation or other feature of the *I* element is responsible for the non-suppressive feature of this allele.

Peter A. Peterson

Developmental pattern of spikelets

The maturation of microspores in the upper and lower florets of each maize spikelet follows a developmental pattern. There is a highly positive correlation between the two florets of the same spikelet such that the developmental stage of the upper floret can be predicted from the observed stage of the lower floret of the same spikelet by the use of the regression equation.

Microsporogenesis (MSG) (meiosis and pollen maturation) progressed at a faster rate in the upper floret than in the lower floret when the tassel was young, but not as rapidly as in the lower floret when the tassel was fully mature. Thus, the time interval separating the upper and lower florets of the same spikelet varies depending on tassel maturity.

The time intervals that separate the upper and lower florets of the same spikelet during MSG is longest with undomesticated Colombian varieties (5.3 days) and shortest in hybrids (3.0 days)

Sun-Yuan Hsu, Yih-Ching Huang
and Peter A. Peterson

Distribution and retention of the *Uq* mobile element in important breeding populations

The Iowa State Corn Breeding Program here pursued

several strategies to enhance their corn breeding populations. Three of the populations are: BSSS(R), BSSS(S) and BS13(HT) (Hallauer and Miranda, ISU Press, 1981). These have been continued for several cycles, each of which was canvassed for the presence of *Uq*.

The following has been determined:

BSSS(R), from a high of nearly 50% of the plants carrying *Uq* at the C0 cycle, gradually decreased to near zero between cycles 6 and 11.

BSSS(S), from a 30% low in C1, increased to near 80% by C5.

These studies are in their initial stages but do indicate a change in the presence of *Uq* occurring during population development.

Lisa Lorenzen[1], Jean Cormack[2] and Peter A. Peterson
[1]NSF Undergraduate Research; [2]ICI-Garst

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

We have reported that crosses between 11 inbred lines and the *c-ruq* tester only produced colorless no-spot F1 kernels, indicating a lack of any active *Uq* elements in these inbreds (MNL 61:5). In order to determine if there are any *Uq* activation events as reported by the *c-ruq* reporter allele, random samples of the colorless no-spot F1 kernels were taken from 7 inbred × *c-ruq* combinations. These kernels were grown and were tested by backcrossing to the *c-ruq* tester. The resulting BC1 progeny ears have been examined to generate the accompanying data.

Inbred	Number of F1 kernels tested[a]	Total number of colorless BC1 progeny kernels		
		no-spot	few-spot (%)	sectored (%)
B14	12	7,133	0 (0)	0(0)
B37	11	7,761	0 (0)	0(0)
B73	11	7,756	2 (0.026)	1(0.013)
B70[b]	29	11,903	2 (0.017)	3(0.025)
B70	21	9,880	0 (0)	0(0)
C123[b]	24	12,692	2 (0.016)	1(0.008)
C123	11	5,864	2 (0.034)	0(0)
187-2[b]	17	9,161	1 (0.011)	1(0.011)
187-2	24	14,687	2 (0.014)	0(0)
C103	9	3,845	1 (0.026)	1(0.026)

[a]F_1 genotype: c /c-ruq(no Uq);
F_1 phenotype: colorless no-spot ;

[b]the seedling of the inbred has been treated with 30 μM 5-aza-2'-deoxycytidine

Activation of *Uq* elements is seen as sectors of spotting in the BC1 progenies of 5 inbreds (B70, B73, C103, C123 and 187-2) with frequencies ranging from 0.8×10^{-4} to 2.5×10^{-4}. No such sectors were observed in the BC1 progenies of the other 2 inbreds (B14 and B37). It is noted that this sectoring of *c-ruq/Uq* spots occurred in BC1 progenies of 3 inbreds after a 30 uM 5-aza-2'-deoxycytidine treatment, but not in the BC1 generation of their untreated sibs.

This study also indicates a lack of active *Uq* elements in the genome of the inbred B45, which has a *C/C* genotype (data not shown). In addition, the BC1 progenies of B45 and 2 other maize lines, M14 (*wx*) and M14 (*Wx*), are all colorless, completely lacking single-spots or sectors of spots (data not shown).

Yong-Bao Pan and Peter A. Peterson

Putative WSMV-induced mutants: *Uq-861058A, ba*-861059B*

In this experiment, the wheat stripe mosaic virus (WSMV) was used to inoculate 34 maize seedlings at the 3 to 4 leaf stage. All seedlings were grown from *a-ruq/Uq* sectored kernels (MNL 60:5-6, 1986). The plants were kept in a growth chamber with a set temperature of 21 C for the development of virus infection. After transplanting into the field, 3 plants showed clear symptoms of the virus infection. These plants were numbered 861058A, 861058D and 861059B, respectively. All the plants (both infected and non-infected) were crossed reciprocally with the *a-ruq* tester. Examination of the resulting progeny ears indicated the following:

1) Plant 861058A produced only colorless kernels in reciprocal crosses. However, in further testing these colorless kernels by crossing by *a-o sh2*(no *Uq*) tester, 5 out of 14 tested kernels from the ear of 861058A/*a-ruq* produced low-spotted kernels at frequencies ranging from .11 to .32. In contrast, none of the 42 tested kernels from 3 ears of *a-ruq*/861058A segregated for spotted kernels. The recovered *Uq* activity may be induced by WSMV infection. We designate this putative mutant *Uq-861058A* and are currently testing this new *Uq* element.

2) Plant 861059B, when selfed, produced 54 defective kernels in addition to 302 colorless normal kernels. It produced 97 colorless normal kernels when crossed onto an *a-ruq* tester plant. The defective kernels germinated very poorly. Only 3 plants were grown from 39 defective kernels. All were of dwarf and late type. On the other hand, 29 plants were grown from 30 colorless normal sibs. Seven plants, however, produced no ear at all. It seems, therefore, that a barren-type mutant gene has been induced by WSMV infection, and that this mutant gene is recessive. We designate this putative mutant *ba*-861059B*. It should also be mentioned here that another set of 29 plants grown from the colorless normal kernels of the ear *a-ruq*/861059B did not segregate for barren-type plants. No *Uq* activity has been seen in the progeny of this mutant.

3) Plant 861058D was male sterile and had a dwarf phenotype. Although 4 colorless kernels were set from the cross of 861058D × *a-ruq* tester, they all failed to germinate.

Yong-Bao Pan and Peter A. Peterson

Test for presence of transposable elements in populations

A routine procedure (crosses with reporter alleles) was followed to survey the transposable element content in 11 populations. If the reporter allele is unstable in the BC1 generation (a variegated or spotted phenotype), then the population contains at least one active copy of the corresponding transposable element. On the other hand, stability of the reporter allele would indicate a lack of the corresponding transposable element. The results of this study are summarized in the Table.

It can be noted that both *Uq* and *Mrh* are active in the Rhoades and Dempsey *bz-mut*, *Mut* population. The *Uq* element is also active in Rhoades and Dempsey's Hi-loss B's population, but this activity is not detectable in 431-related Mutator populations (populations 1 through 7). However, population 11, which was derived from a cross

Maize population	Reporter allele used	Number of gametes tested	Number of BC1 ears	Corresponding transposable element content[1] Uq	Mch	En	Cy	Dt	Source
1. 431-434	c-ruq	4	15	-					Mutator population
2. 431-435	"	6	17	-					"
3. 431-436	"	6	14	-					"
4. 431-437	"	4	14	-					"
5. 431-B-2	"	6	25	-					"
6. PI-431	a-ruq	6	74	-					"
7. spotted 431	a-ml	3	18			-			E. Coe
(c2/c2,in/in)	a-m(r)/a-ml	8	40			-			
	a-ruq	6	66	-					
	a-mdt	3	22					-	
8. a su pr y wx	bz-rcy	1	15				-		Coop
9. Hi-loss B's	c-ruq	2	20	*					Rhoades &
	bz-rcy	1	14				-		Dempsey
10. bz^{mut},Mut line	a-m(r)h	3	20		*				Rhoades &
	a-ruq		15	*					Dempsey
	c-ruq		36	*					
11. C2/c2,in/in, Whp/Whp,Clsh2, cl-spots(1b)	a-mdt	4	30					*	derived from a cross between populations 1 → 6 and population 7
	a-ruq	3	32	*					
	a-m(r)/a-ml	2	14			-			

[1] a "+" indicates the presence of at least one active element; a "-" indicates the absence of any active element.

between populations 1 through 6 and population 7, apparently has an active *Uq* element. This population also has an active *Dt* element whose activity is not detectable in spotted 431 (population 7). Two other elements, *En* and *Cy*, are found to be either absent or inactive in the two populations tested. The content of these two elements in other populations has not yet been tested.

Yong-Bao Pan and Peter A. Peterson

Six putative germinal events of *Uq* activation

By using both the *a-ruq* and the *c-ruq* reporter alleles, we have demonstrated that none of the maize inbreds B70, C103, C123 and 187-2 contains an active *Uq* element in its genome (MNL 60:5-6; 61:5). On the other hand, activation of a quiescent *Uq* element has been observed as a sector of *a-ruq*/*Uq* spotting in the aleurone tissue of progeny kernels of the 4 inbreds (Pan and Peterson, Genetics, submitted, 1987).

Putative Uq element	Original State	Test Cross[a]	State upon Test[b]	Sectored (%)	Total # of kernels
1. Uq-866201U-1	2a	self (1)	2a(.032)	0	440
		by a-ruq(2)	no spotted	.0058	669
2. Uq-866201U-2	2a	self (1)	2a(.12) 4ab(.006?)	.0033	597
		by a-ruq(1)	no spotted	.0082	367
		on a-ruq(1)	no spotted	.0029	336
3. Uq-866201X (mn-866201X)	3bc	by a-ruq(1)	3bc but not evenly dispersed, also miniature (.436)[c]	?[e]	330
4. Uq-866201Y	no record	self (1)	high-coarse:[d] with colorless sectors(.011); without(.31)	?	367
		by a-ruq(1)	" with(.075); without(.17)	?	53
		on a-ruq(1)	" with(.11); without(.11)	?	295
5. Uq-866201A	"	self (1)	2-3b/9a[f] not evenly dispersed(.172)	0	209
		by a-ruq(1)	2-3b/9a(.075)	0	348
		on a-ruq(1)	1b/3-4a(.067)	0	135
6. Uq-866201B	"	by a-ruq(1)	3-4b/9-10a(.13) 6-7b/10a(.32)	0	421
		on a-ruq(1)	3-4b/9-10a(.25) 6-7b/10a(.21)	0	217

[a] Numbers in parenthesis are numbers of progeny ears;
[b] numbers in parenthesis are frequencies of spotted kernesl out of the number of kernels analyzed;
[c] see Pan and Peterson, 1987, MNL 61:6;
[d] in addition, frequency of pale-round kernels:self (.16); by a-ruq(.13); on a-ruq(.068); frequency of spotted pale-round kernels:self (.033); by a-ruq(.019); on a-ruq(0);
[e] difficult to determine under the unique state;
[f] 2-3b/9a indicates spots of 2-3b in a 9a of 1-cell-spotted background. The same indication applies to other expressions followed.

We report here 6 putative germinal events of *Uq* activation recovered as single spotted kernels in a total of 163,205 colorless BC2 progeny kernels. Each of these *Uq* elements has unique features in terms of both spotting pattern and spotting frequency when interacting with the standard *a-ruq* allele (Table). Further tests have indicated that these features of the 6 putative *Uq* elements are inherited (data not shown). Other tests are in progress to determine 1) the genetics of the 6 putative *Uq* elements, and 2) the relation of each of the 6 putative *Uq* elements to the standard *Uq*/*ruq* system.

Yong-Bao Pan and Peter A. Peterson

mn-866248U:* progeny test

We have reported the coinduction of *Uq* activity and the *mn*-866248U* (miniatures) mutant (MNL 61:6) by 5-aza-2'-deoxycytidine treatment. Our preliminary data in that report indicated that the *mn** mutant is dominant and is not male transmissible. It also cosegregates with the activated *Uq* element. To confirm these initial findings, we have carried out 2 generations of progeny tests. Colorless normal kernels and spotted miniatures of 5 arbitrarily defined classes (a, b, c, d and e) were collected from the ear 866248U/*a-ruq* and were grown into plants. The plants were crossed reciprocally to *a* and *c-ruq* testers. In addition, crosses of a few miniature-derived plants on two other genotypes (*r-g* tester and inbred Hy) were also made to test the male transmission of the mutant gene. The spotted miniatures and the mottled progenies were further tested in another generation. The results of the progeny test are summarized in the Table and the Figure.

Progen Type[2]	by a sh (no Uq) mn	by a sh (no Uq) Uq	by c-ruq mn	by c-ruq Uq	on a-ruq mn	on a-ruq Uq	on c-ruq mn	on c-ruq Uq
spotted miniature(1)	30/30	30/30	19/19	?	0/33	0/33	0/42	?
colorless normal	0/10	0/10	0/4	?	0/6	0/6	0/11	?
mottled(1)	0/34	0/34	/	/	0/33	0/33	/	/
spotted miniature(2)	35/35	35/35	/	/	0/43	0/43	/	/
mottled(2)	/	/	0/7	0/7	/	/	/	/

[1] Values below the bar represent the total number of ears analyzed, whereas values above the bar are the number of ears that segregate miniature kernels or Uq activity.

[2] Sources for the progeny types tested:
spotted miniature(1) and colorless normal were from the ear 866248U/a-ruq;
mottled(1) was from the cross of r-g tester X spotted miniature;
spotted miniature(2) was from the cross of spotted miniature x a sh(no Uq) tester;
mottled(2) was from the cross of inbred Hy x spotted miniature.

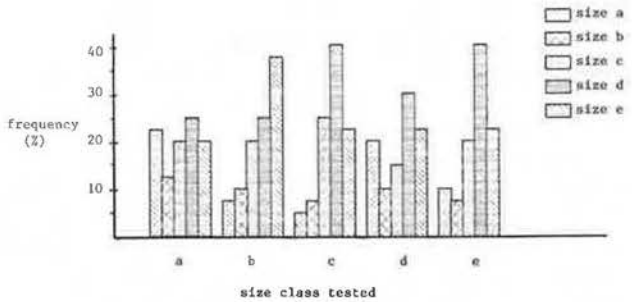


Figure. The 5 size classes (a, b, c, d, and e) of spotted miniatures produced all 5 classes of offspring when crossed by *a sh2* (no *Uq*) tester (or when selfed; data not shown here). However, the frequency for each class varies within as well as among the 5 size classes tested.

The data in the Table clearly prove the following points: 1) The activated *Uq* is associated with the miniature character; 2) whenever the miniatures are the pollen parent, the *mn** and the activated *Uq* both are lost from the progeny population; and 3) all progeny ears segregate for half normal and half miniature kernels when the spotted miniature class is crossed by a *c-ruq* tester (test for *Uq* segregation in this type of cross is in progress). Examination of pollen samples from the colorless normal class and the spotted miniature class has shown no morphological difference (data not shown). However, significant differences in their germination ability may account for the non-male-transmission of *mn** (data not shown). In addition, the difference in reducing kernel size has been proved to be due to the expressivity of this mutant gene, since each of the 5 size classes is segregating for all 5 classes of progenies in a maternal cross (Figure).

Based on these results, we propose that *mn-866248U* is caused by insertion of the activated *Uq* into a locus that plays an important role in pollen grain and endosperm development. We also strongly believe that once the *Uq* sequence becomes available, cloning and molecular characterization of this maize developmental gene will turn into a reality.

Yong-Bao Pan and Peter A. Peterson

Mitotic blocking agents for suspension cultures of Black Mexican Sweet cell lines

Colchicine has been used extensively since 1937 to block mitosis and induce polyploidy in a large number of plant species (A.F. Blakeslee, Compt. Rend. Acad. Sci. 205:476, 1937). The original studies of mitotic blockage were performed on tissues in planta, but, with the development of methods for culturing plant cells and the increasing use of these cells for research, attention has turned to the use of colchicine as a mitotic blocking agent in cell suspension cultures. Colchicine treatment gives satisfactory mitotic arrest in wheat and poppy suspension cultures. In the latter, the frequency of mitotic cells approaches 45% when the suspension is growing optimally (Hadlaczky et al., Planta 157:278, 1983).

Colchicine treatment, alone or in conjunction with hydroxyurea or physical methods, has recently been used to produce mitotic blocks in Black Mexican Sweet (BMS) suspension cultures (A.S. Wang et al., Plant Science 46:53, 1986). In those experiments the colchicine concentration used was 0.5mM (0.02%). This colchicine treatment alone doubled the frequency of mitotic figures (mitotic index: MI) compared to the untreated control, but the maximum yield was 9%. Hydroxyurea pretreatment did not increase this frequency. However, a 4-day subculturing routine combined with the selection of large aggregations of cells (280μm or more) raised the mitotic index of the untreated control to 10%, and the further addition of 0.02% colchicine again doubled the frequency of mitotic cells so that a final yield of 23% was obtained. These results clearly illustrate the effect of culture conditions and health of the treated cells upon the success of a mitotic blockade, but, again, colchicine treatment only doubled the residual number of mitotic cells. The physical methods described for this combined procedure have proved difficult to replicate and are, therefore, not easily useful for routine preparations of large numbers of mitotic cells.

Because the optimal concentration of colchicine for mitotic arrest varies widely between different plant species (e.g., 1.25mM for poppy and 2.5mM for wheat, Hadlaczky et al., 1983), we have determined the effective concentration of colchicine for mitotic blockade in BMS suspension cultures. We also report excellent mitotic inhibition obtained with micromolar concentrations of amiprophos methyl (APM), a tubulin-specific phosphoric amide herbicide (Morejohn and Fosket, Science 224:874, 1984). Trifuralin, on the other hand, was unsuccessful as a mitotic blocking agent although the herbicide is known to have a colchicine-like effect on mitosis in tobacco callus cultures (Young and Camper, Pest. Biochem. Phys. 12:117, 1979) and to depolymerize microtubules in cotton root cells (Hess and Bayer, J. Cell Sci. 24:351, 1977).

Table 1. Mitotic indices after treatment of maize suspension cultures with colchicine, APM, and trifuralin.[a]

Colchicine (mM)	(%)	% Mitotic Cells	APM (μM)	% Mitotic Cells	Trifuralin (μM)	% Mitotic Cells
0	0	4	0	3.0	0	3.2
0.5	0.02	3-5	1	3.1	25	2.1
2.5	0.1	3-5	5	5.9	50	1.8
5.0	0.2	13	10	5.3	100	1.9
12.5	0.5	25	50	20.1		
			75	17.0		
			100	13.0		
			500	0[b]		

a. Each mitotic index is based upon 1,000 cells, and each experiment had two or three repetitions.
b. Complete cell lysis.

Mitotic indices obtained after treatment with colchicine, APM, and trifuralin are shown in Table 1. Inhibition of mitosis by colchicine is concentration-dependent; 0.02% colchicine per se is insufficient for repeatable mitotic blockage, while treatment for 21 to 28h with 0.5% reliably arrests 25% of the cells in mitosis, almost all in metaphase. This mitotic arrest is also time-dependent, since in other experiments 0.5% colchicine applied for 10h yields only 6.8% mitotic cells. The effective blocking concentration of colchicine for maize cells (MI 25%) is 5 times that required for poppy cultures and 10 times that required for suspension-grown wheat cells. This result is not surprising in view of the previously mentioned species differences. This high-level colchicine requirement in mitotic blockade of maize cells is not understood. It could reflect either very low affinity of maize tubulin for colchicine or faulty entry of colchicine into the cell due to low affinity binding of the drug to transport molecules.

APM treatment produces a good mitotic blockage at low drug concentrations; 50 μM applied for 20 to 28h arrests 20% of the cells in metaphase. This result indicates that APM is effectively transported into the cell and that it is efficiently bound by maize tubulin. Trifuralin, however, does not induce any mitotic arrest although the concentration range tested was broad, 25 to 100μM.

Table 2. The effect of metaphase-blocking concentrations of APM on growth of BMS cell cultures.

APM	Initial growth lag	Population Td (d 4-7)
0	0	35h
25 μM	4d	32h
50 μM	4d	91h
75 μM	No growth by seven days	

Some experiments require reversible mitotic blockades, for example diploidization of haploid cultures or cell-cycle studies. For this work it is desirable to use drugs relatively nontoxic to the cells at blocking concentrations. Since APM is a newly reported mitotic inhibitor for maize cells, we have made some preliminary studies of the toxicity of this compound. Growth data are shown in Table 2. Suspension cultures were inoculated with cells (0.004 g/ml) from populations in exponential growth, and several concentrations of APM were added at the time of subculture. Increments in fresh weight were determined in 2 to 3 replicate flasks at 2, 3, 4, 5, 6 and 7 d. Added 25, 50, and 75μM APM induces a strong lag in population growth for 4 days after cell transfer although the control (0 drug) doubled each 35h in this time period. This lag in population growth is only partially accounted for by mitotic arrest, since after 3 days the MI in cultures with 50μM APM was still only approximately 20%. Therefore, we must assume that other cells in the population were probably temporarily arrested by unknown toxic effects of the drug. After 4 days, populations treated with 25μM APM abruptly resumed growth and attained a population doubling time (Td) like controls. Cultures with 50μM APM had a Td of 91h and cultures with 75μM APM continued in arrest. When cells from 6-day populations were stained with fluorescein diacetate, those obtained from cultures with APM were at least 50% viable, as compared with the control. And, after 6 days of growth, the MI of cell populations treated with 50μM APM was only 1.5 percent.

These data show that APM is almost certainly cytotoxic, but not completely lethal, at the concentrations used for mitotic blockade applied to continuously growing cultures. Resumption of growth after a strong 4-day lag suggests metabolism or breakdown of APM. The drop in mitotic index by 6 days may indicate escape of metaphase cells from the block. Because this drug is such an effective mitotic inhibitor at low concentrations, we plan some further experiments which will investigate the toxicity of APM during 28h pulse treatments, which are sufficient for good metaphase blockade.

J. Stadler, J. Rugemer, R.L. Phillips and M. Leonard

A chloramphenicol-resistant variant cell line selected by long-term enrichment selection in Black Mexican Sweet (BMS) suspension cultures

The first mitochondrial mutation isolated in cell cultures of higher organisms was obtained in mouse cell lines treated with increasing concentrations of chloramphenicol for 2.5 months (Bunn et al., Proc. Natl. Acad. Sci. USA 71:1681, 1974). In animals, chloramphenicol is an inhibitor of mitochondrial protein synthesis. A long selection period is necessary because of resident populations of multiple mitochondria per cell. In plants, attempts to obtain mutants resistant to inhibitors of organellar protein synthesis are complicated by the presence of two organellar genomes, those of the plastids and the mitochondria, sensitive to a similar range of drugs. Since most of the products of the plastid genome are components of the photosynthetic apparatus (or of the protein synthesizing apparatus required to make components of the photosynthetic apparatus), plastid gene expression may not be necessary for the heterotrophic growth of cultured cells. It should therefore be possible to select directly for mitochondrial resistance in cell cultures. In a first effort to obtain chloramphenicol-resistant, mitochondrial variants in corn, we have treated BMS cells with the drug for several months. BMS wild-type cultures are completely inhibited and eventually killed by 20 μg/ml chloramphenicol, although some slow population doubling (Td = 144h) occurs at 10 μg/ml. Selection was begun at the latter concentration, and after 5 months we obtained a line of cells able to grow slowly (Td = 72h) in 20 μg/ml drug.

Analysis of the mitochondria of the resistant cell line indicates that:

1. Their protein synthesizing apparatus is functional. Protein synthesis by mitochondria isolated from the resistant cell line is, however, as sensitive to chloramphenicol as protein synthesis by mitochondria from the sensitive cells; the dependence of inhibition on chloramphenicol concentration is similar for mitochondria from the two types of cell. Thus resistance is apparently not due to an alteration to the mitochondrial protein synthesizing apparatus.

2. The steady-state levels of mitochondrial gene products (relative to nuclear gene products) are lower in mitochondria of the resistant cells grown in the presence of chloramphenicol than in wild-type cells grown in nonselective conditions. Thus chloramphenicol affects gene expression in the resistant cells. These preliminary results suggest that chloramphenicol has harmful effects on the resistant cells, but they are altered in a way that increases their tolerance of these harmful effects. Alternatively, the drug may be partially excluded, so that the resistant cells are affected similarly to sensitive cells grown at lower concentrations.

J. Stadler and E. Hack

Successful transfer of the chloramphenicol transferase gene to Black Mexican Sweet (BMS) protoplasts by polyethylene glycol

Polyethylene glycol (PEG) technique has great promise for the transfer of genes into cereals and other graminaceous monocots, which are not readily transformed by *Agrobacterium tumefaciens*. This method is a desirable alternative to microinjection and electroporation since it is quick, inexpensive, reliable, and relatively nontoxic to transformed cell populations. In our laboratory, protoplast viability after PEG treatment is 57 to 77%.

The method developed by Krens for PEG transfer of genes to tobacco (Krens et al., Nature 296:72, 1982) has subsequently been modified by several laboratory groups (I. Potrykus, Plant Cell and Tissue Culture Vol. III, 289-302, 1987). One experimental variation of Krens' method, suitable for use with P.S. Chourey's BMS cell line, was recently sent to us by S. Howell, University of California, San Diego. We have made further adaptations of this technique so that we can now obtain excellent gene transfer to protoplasts of BMS-M, a fast-growing subline of BMS derived by C.E. Green and kindly given to us by D.A. Somers, University of Minnesota.

For PEG gene transfer experiments we use 5×10^6 protoplasts (S.R. Ludwig et al., Theor. Appl. Genet. 71:344, 1985) resuspended in 0.75ml of 0.2M mannitol and 80mM $CaCl_2$ (pH 5.6). Twenty to 50μg plasmid DNA and 50μg sonicated calf thymus DNA are added to the cells and

mixed thoroughly. Cells and DNA are then combined with 0.75ml 50% PEG 8000 solution, made in Krens' F medium, pH 7.2. The PEG solution is added slowly, a drop at a time, and the mixture is then incubated at 28 C for 30min without shaking. At the end of incubation the PEG-cell mixture is diluted slowly from 25% PEG to 2% PEG with F medium, in the manner of Krens. Cells are then collected by gentle centrifugation and resuspended in growth medium with 8% (439mM) mannitol.

To test our methods we have transformed BMS-M protoplasts with p CaMv I_1 CN. In this vector, kindly provided by M. Fromm (Genes and Development, in press), the chloramphenicol transferase (CAT) gene is regulated by the cauliflower mosaic virus (CaMV) 35s promoter and the enhancer activity of an *Adh* intron fragment (I_1). Transferred protoplasts were incubated for 40h before assay for CAT activity (Fromm, Proc. Natl. Acad. Sci. USA 82:5824, 1987).

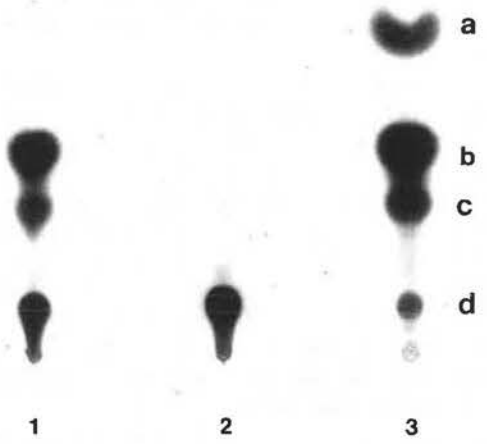


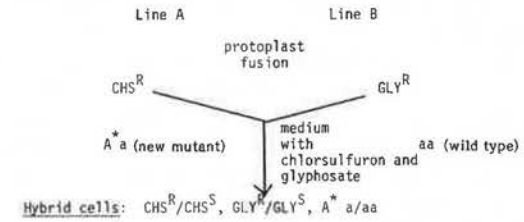
Figure 1. Expression of CAT genes transferred into BMS-M protoplasts by PEG treatment. Lanes:1, 5×10^6 PEG-treated protoplasts with 50μg pCaMVI$_1$ CN DNA; 2, 5×10^6 PEG-treated protoplasts with no added DNA; 3, 1000 units of chloramphenicol transferase from *E. coli* plus 0.1 uCi ^{14}C chloramphenicol. a, 1,3-diacetyl chloramphenicol. b, 3-acetylchloramphenicol. c, 1-acetylchloramphenicol. d, ^{14}C chloramphenicol.

Fig. 1 shows the results of one experiment. In PEG-treated cells transformed with 50μg p CaMV I_1 CN DNA, the conversion of chloramphenicol to 1-and 3-acetate chloramphenicol was 37%. This represents a 520-fold increase over background. We obtain similar results when only 20μg DNA is used in the transformation.

M. Antonelli, M. Moreno and J. Stadler

Selection by gradual enrichment for putative dominant mutations in Black Mexican Sweet (BMS) cell cultures

We have selected several variant subcultures of a fast-growing BMS cell line (BMS-M, generously supplied by D.A. Somers, University of Minnesota). Our goal is to obtain dominant markers which will be useful in protoplast hybridization experiments to test the dominance of new, previously untested mutants. We have so far obtained BMS variant lines which are chlorsulfuron resistant (CHS^R), glyphosate resistant (GLY^R), and lysine/threonine resistant (LT^R). If hybridization tests, or gene transfer, should prove that one of these new marker phenotypes is dominant, then we intend to derive other, more experimental mutations in this cell line. In that case, for example, the dominance of the new mutations could always be ascertained by the following cell hybridization and analysis:



Genetic analyses of chlorsulfuron-resistant, glyphosate-resistant, and lysine/threonine-resistant mutants obtained in cell cultures of tobacco and *Arabidopsis*, petunia, and maize, respectively, have shown that the lesions obtained were dominant (R. Chaleff and Ray, Science 223:1148, 1984; Haughn and Somerville, Mol. Gen. Genet. 204:430, 1986; K.A. Hibberd et al., Planta 148:183, 1980). Although a variety of genetically based mechanisms might yield BMS-M cells which are resistant to each of these cytotoxic substances, these published experiments demonstrate that dominant mutations of the type we desire are possible to obtain.

Chlorsulfuron is very toxic to BMS-M suspension cultures, and all cell division is halted after 5 days treatment with 10^{-6}, 10^{-7} and 10^{-8} M. Growth is significantly retarded by 10^{-9}M (Fig. 1). Normal population

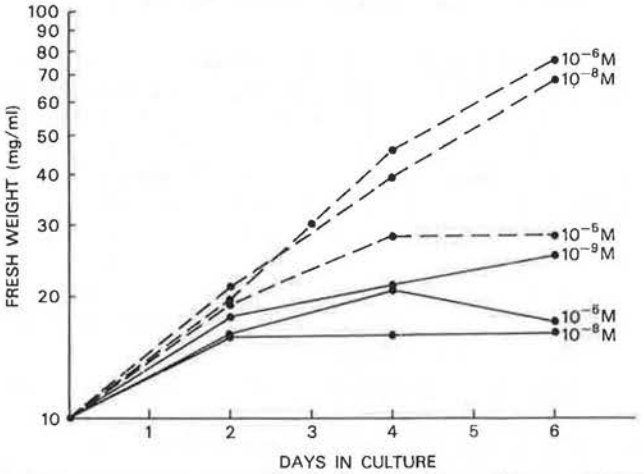


Figure 1. Growth of resistant (---) and sensitive (—) BMS cell lines in the presence of various concentrations of chlorsulfuron.

doubling time (Td = 32 to 36h) is obtained when 10^{-10} to 10^{-12} M chlorsulfuron is present in these cultures. Enrichment selections were begun by treating the cells with the partially toxic drug concentration, 10^{-9} M. Serial, 7 day passages (1:1, 1:3, or 1:5 splits) on 10^{-9} M chlorsulfuron continued for 8 weeks, and then rapid increases in drug concentration to 10^{-8}, 10^{-7}, 5×10^{-7}, and 10^{-6} M were made at 8, 11, 12, and 14 weeks after initiation of the selection. A stable, variant BMS subline (CHS^R-1) resistant to 10^{-6} M chlorsulfuron was isolated after 15 weeks. This variant line has a 36h Td at 10^{-6} M (Fig. 1) and is therefore more than 100-fold resistant to this drug. This level of drug resistance is similar to that obtained in *Arabidopsis* (Haughn and Somerville, 1986). CHS^R-1 has been cloned by protoplasting and plating in agar, and subclones will be used in all further analysis.

A glyphosate-resistant subline (GLY^R-1) was selected in a similar manner in 22 weeks. Gradual enrichment selection was begun with applications of 0.25 mM glyphosate; this concentration is mildly inhibitory (50% growth inhibition by 7 days). GLY^R-1 is now tolerant to 1.0mM, and the population fresh weight increases 5-fold in 7 days in the presence of herbicide. Wild type BMS-M is completely restricted in growth by 0.5, 1.0, and 2.0 mM glyphosate, so the increase in glyphosate resistance obtained so far is 2- to 4-times background. The sensitivity of these corn cells to glyphosate is much greater than that of petunia cells. In the latter cultures, 1.0 mM is the lowest completely toxic concentration (Steinrucken et al., Arch Biochem. Biophys. 244:169, 1986). GLY^R-1 is being tested for evidence of gene amplification (D.M. Shah et al., Science, 233:478, 1986).

Lysine/threonine-resistant variant cell lines are easy to obtain by gradual enrichment with incremental additions of 1.0 mM to 4.0 mM amino acid over a period of 3 months.

J. Stadler, M. Leonard and H.C. Huang

Mutator system in embryogenic cultures

Tissue culture allows an important extension of studies of certain in planta phenomena. These methods have been used in a limited way to analyze the activity of Activator and Enhancer transposable element systems in maize endosperm cultures (A.R. Reddy and P.A. Peterson, Maydica 22:125, 1977; Culley, VI International Congress Plant Tissue Culture p. 220, 1986), and more extensively in the analysis of transgenic expression of *Ac* activity in tobacco cells (B. Baker et al., Proc. Natl. Acad. Sci. USA 83:4844, 1986). Research to date on the Mutator system has centered primarily upon the analysis of differentiated maize tissues in planta, including leaves, seedlings, immature ears, and kernels, although endosperm cultures derived from lines with *Mu*-element-containing mutable alleles have recently been established in this laboratory. Endosperm callus, however, grows relatively slowly, making it difficult to accumulate quantities of material sufficient for extensive molecular study. In contrast, Type II embryogenic callus lines of maize (C.L. Armstrong and C.E. Green, Planta 164:207, 1985) are reliably fast-growing and allow for continued analysis of an expandable pool of genotypically identical tissue as well as the possibility of plant regeneration. To study Mutator systems in tissue culture we have, therefore, established Type II callus lines from immature F1 embryos of crosses of mutagenically active Mutator stock (Mu^2) with inbred lines A188 and H99.

We report our initial molecular analysis of *Mu* elements in 13 independently derived embryogenic callus cultures. Active Mutator lines of maize in planta are characterized by their ability to generate new mutants at 30 to 50 times the spontaneous level, and by somatic instability at Mutator-induced mutant alleles. To date, mutagenically active Mutator lines with fewer than 10 *Mu* elements per diploid genome have not been observed (J.L. Bennetzen et al., Mol. Gen. Genet. 208:45, 1987), and stable maintenance of a high *Mu*-element copy number from one generation to another is thought to be a feature of an active Mutator system (V. Walbot and Warren, Mol. Gen. Genet., in press, 1987). Mutator activity also correlates with lack of modification at the *Hin*fI restriction sites which lie within the inverted terminal repeats of *Mu* elements (V.L. Chandler and V. Walbot, Proc. Natl. Acad. Sci. USA 83:1767, 1986). Because of the correlation of these parameters with Mutator activity, we have investigated *Mu* copy number and *Hin*fI-site modification at both early and late times in culture. *Mu* modification was assessed by genomic digests with the methylation sensitive enzyme *Hin*fI (S. Hake and V. Walbot, Chromosoma 79:251, 1980), which recognizes sites in the terminal inverted repeats of this element. Subsequent hybridization of unmodified transposons with a *Mu1* probe results in discrete 1.3 and 1.6 kb bands which represent the *Mu1* and *Mu1.7* elements, respectively. However, similar treatment of DNA which contains *Mu* elements modified at *Hin*fI sites results in a ladder of bands larger than 1.3 kb. *Mu*-element copy number was determined by comparison with plasmid copy-number reconstructions or by analysis of the genomic location of all elements present.

All progeny embryos from each inbred/Mutator F1 ear were cultured. Four and 6 weeks after initiation it was possible to identify individual embryos with good Type II embryogenic callus. To establish individual embryo lines these were selected for transfer to fresh medium, and all other calli and tissues were pooled and frozen at -20 C. Thereafter, in each individual line Type II callus with the best embryogenic phenotype was transferred and propagated, and all nonselected tissues were stored separately at -20 C for subsequent molecular analysis. In this manner, all tissue descending from each individual embryo population was saved in the fresh or frozen state (Fig. 1). DNA isolated from sib embryos pooled at 4 and 6 weeks (T_2 and T_3) was analyzed molecularly to enable the earliest possible characterization of *Mu* elements in an embryogenic callus culture.

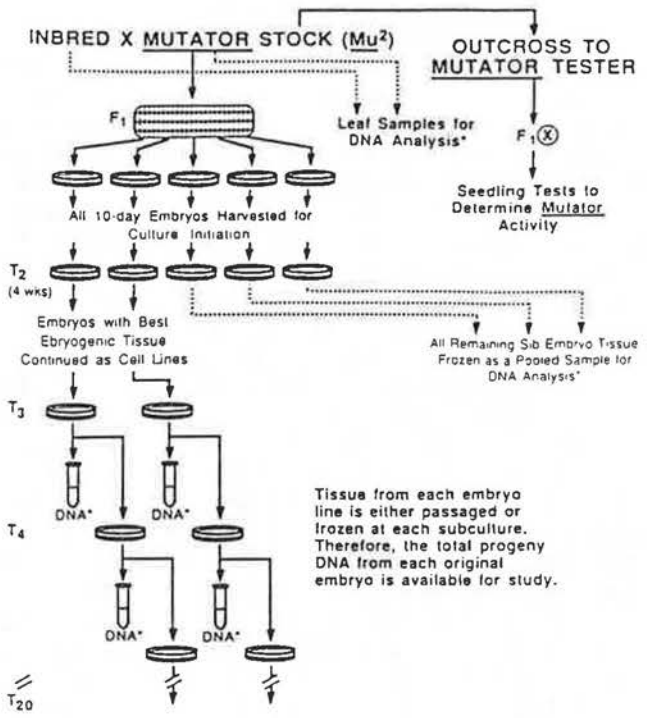


Figure 1. Scheme for propagation of embryogenic cell populations and collection of samples for molecular analysis.

Table 1. Mu element modification in parent plants and offspring cultures.

	Mu^2 parent:		Mu-element modification of F_1's				
	New Mutant Frequency	Modification	Pooled Sib Embryo Call:	Individual Embryo Callus Lines	Later Time Points		
			(4-6 wk)	(3 mo)	(7 mo)	(9 mo)	(19 mo)
H99/Mu^2:							
1.	?[a]	?		607-4A (+)[b]		(+)	
				607-4B (-)[c]		(-)	
				607-4C (-)		(-)	
2.	0	?	657-2 (+)	657-2A (+)			
				657-2C (+)			
3.	4%[e]	(-)	647-8 (+/-)	647-8A (+/-)[d]			
				647-8B (+/-)			
A188/Mu^2:							
4.	7%	?		3579-3A	(-)		(-)
				3579-16C	(-)		(-)
5.	30%	(-)	643-9 (-)	643-9B (-)			
6.	12%	(-)	646-8 (+/-)	646-8B (-)			
				646-8C (+/-)			
				646-8D (+)			

a. ?. No data available.

b. +. All Mu elements in this sample were modified the HinfI sites.

c. +/-. Some of the Mu elements were modified and some unmodified at HinfI sites.

d. -. No Mu element modification was detected.

e. Percent of new seedling mutants in F_2 (Robertson 1981b).

To determine the effects of culturing on *Mu*-element copy number and modification state, it is necessary to make comparisons with parental plant controls. Leaf samples taken at the 4- to 7-leaf stage from Mutator parents were used for molecular analysis of *Mu*-element copy number and *Hin*fI-site modification. In addition, Mu^2 parent plants were tested genetically for Mutator activity (D.S. Robertson, Mol. Gen. Genet. 183:51, 1981).

Because plant progeny which result from outcrosses of active Mutator stock have *Mu* elements which are unmodified at the *Hin*fI sites (J.L. Bennetzen, Mol. Gen. Genet. 208:45, 1987), it was expected that embryogenic cultures established from the F1 progeny of an outcross between an active Mutator line with A188 or H99 would have similarly unmodified *Mu* elements. Our results show, however, that the state of *Mu*-element modification varies in the F1 cultures examined even though the Mu^2 parent of the culture was mutagenically active (Table 1, lines 3, 4, 5, and 6). Among the six A188-derived embryogenic lines examined, one culture has *Mu* elements uniformly modified at the *Hin*fI sites, four have *Mu* elements which are unmodified at these sites, and one has a population of *Mu* elements with a mixed state of modification. A similar variation occurs when H99 is the inbred parent. Three of the 7 H99/Mu^2 F1 cultures have *Mu* elements which are uniformly modified at the *Hin*fI sites, 2 have only *Mu* elements which are unmodified at these sites, and 2 have both modified and unmodified elements. It should be noted, however, that one Mutator parent of 2 *Hin*fI -modified cultures from H99 was not mutagenically active (Table 1, line 2). Control callus cultures, generated by crossing H99 with inactive Mutator parents (Mu^{16} and Mu^{32}) show the complete *Mu* element-*Hin*fI site modification expected in the outcross progeny of Mutator lines previously made inactive by repeated intercrossing.

Comparisons of the molecular profiles of early pooled samples of sib embryos with related F1 embryo lines show that the *Mu*-modification state in the individual lines was always like that seen in the early pooled samples (Table 1). This indicates that the unexpected modification of *Mu*-element *Hin*fI sites, when seen, was an event which probably occurred early in culture, before 4 weeks.

Two A188/Mutator callus lines have been in continuous culture for almost 2 years (3579-3A and 3579-16C, Fig. 2). Analysis of the DNA isolated from tissue harvested after 7 and 19 months in culture shows no modification at the *Mu*-element *Hin*fI sites in either line. Two sibling H99/Mu^2 lines were also examined molecularly after 3, 6, and 9 months in culture (607-4A and 607-4B, Fig. 2). One line was completely and stably modified at the *Mu*-element *Hin*fI sites over this time period, while the other was stably unmodified. The basic stability of the state of *Hin*fI-site modification in each line examined suggests that continued culturing does not reverse modification patterns that are established early in culture.

The fact that *Mu* elements in approximately half of the cultures examined did exhibit *Hin*fI site modification is surprising and suggests that it may be associated with the establishment of the maize embryos in culture. Culturing of plant tissues has been shown to result in increased variability (Larkin and Scowcroft, Theor. Appl. Genet. 60:197, 1981), with generalized changes in the methylation of genomic DNA thought to be due to tissue culture stress (Brown and Lorz, in Somaclonal Variations in Crop Improvement, 1986). Additional analysis of these cultures with other methylation-sensitive restriction enzymes and probing with developmentally active and inactive genes will be used to test the specificity of the modification seen.

All callus lines studied contain approximately 15 to 25 *Mu* elements and these are also stably retained. Lines which have unmodified *Mu* elements in this high copy number are potentially active Mutator systems. Transposition of *Mu* elements in these cultures is being tested in experiments designed to fix such genomic events in callus-

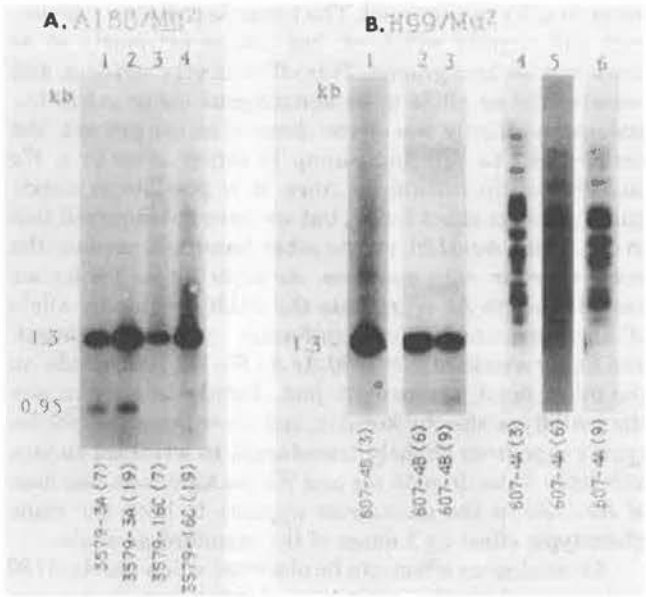


Figure 2. *Hin*fI digestion of genomic DNA from A188/*Mu*^{2} and H99/*Mu*^{2} embryogenic cultures (number in parentheses indicates months in culture). DNA samples (5-8 ug) were digested with *Hin*fI, electrophoresed through 0.8% agarose gels, transferred to a nylon membrane, and hybridized to a *Mu1* probe. A. DNA from A188/*Mu*^{2} callus lines 3579-3A (lanes 1 and 2) and 3579-16C (lanes 3 and 4) isolated after 7 and 19 months in culture. B. DNA from H99/*Mu*^{2} callus lines 607-4B (lanes 1-3) and 607-4A (lanes 4-6) isolated after 3, 6, and 9 months in culture.

line subpopulations. Our laboratory has also established embryogenic cultures from plant lines which have *Mu* elements at specific mutable alleles (*wx*, *bz1*, and *a1*), and *Mu* element excision from these loci in culture will be investigated. These studies should further determine the potential usefulness of the Mutator system in culture.

Martha James and J. Stadler

A putative Mutator-induced dominant amylose-extender mutant allele, *Ae-5180*

Few, if any, dominant mutations induced by controlling elements have been reported in the literature, and to date none have been characterized molecularly. A dominant virescent mutable mutant has been reported by Peterson, but it apparently had no interaction with the *En* (*Spm*) system, in which stocks it arose. In addition, a dominant dappled aleurone mutant arising in a Mutator population has been described by Stinard and Robertson (MNL 61:7), but has not yet been proven to have been induced by the Mutator system. In this paper we describe a dominant amylose-extender mutant which arose in a Mutator population. If this mutant proves to have been induced by a *Mu* insert, it will provide an excellent opportunity to characterize a dominant mutation at the molecular level.

In our 1984 *y1 wx gl8/Y1 Wx Gl8 Mu* isolation plot, a single glassy kernel was observed in a population of 388,688 kernels. We planted this kernel in 1985, and selfed and outcrossed the resulting plant to a standard starchy line (Standard Q60). The selfed ear segregated for a sugary kernel type in an approximate 1:2 ratio of starchy to sugary kernels. The outcross ear segregated in an approximate 2:1 ratio of starchy to sugary kernels. Thus, it appeared that our mutant (which we designated *Su*-5180*) was dominant, but was transmitted in a reduced frequency since the ratios observed differ significantly ($p<0.001$) from the expected values of 1:3 and 1:1 in the self and outcross respectively (Table 1). Further tests (described below) showed that *Su*-5180* is fully female transmissible, but frequently shows reduced transmission through the male.

Table 1. Counts of starchy and sugary kernels on the self and outcross ears of the original *Ae-5180* mutant plant grown in 1985. The outcross was made to a standard starchy line.

	Selfed Ear			Male Outcross Ear		
Plant No.	Starchy	Sugary	% Sugary	Starchy	Sugary	% Sugary
5180-1	123	248	66.5	215	103	32.4
	1:3 Chi-square = 18.1916 ($p<0.001$)			1:1 Chi-square = 39.4865 ($p<0.001$)		

We tested *Su*-5180* for allelism with *su1*, *su2*, *du*, and *ae*, since these mutants have a sugary or glassy phenotype similar to that of *Su*-5180*. Selfs of (*Su*-5180/su1*), (*Su*-5180/su2*), and (*Su*-5180/du*) segregated for fully starchy kernels as well as the parental types and their presumed combinations. Thus, we concluded that *Su*-5180* was not allelic to *su1*, *su2*, or *du*. The selfs of (*Su*-5180/ae*), however, produced only sugary or glassy kernels, suggesting that *Su*-5180* is a dominant mutant allele of *ae*. Backcrosses of (*Su*-5180/ae*) by *ae ae* stocks also produced ears with only sugary or glassy kernels, confirming the allelism of *Su*-5180* with *ae*. We now designate our dominant amylose-extender mutant *Ae-5180*.

The expression of *Ae-5180* in our standard backgrounds varies from slightly shrunken and tarnished to wrinkled sugary to brittle. This variability occurs whether *Ae-5180* is present in the endosperm in 1, 2, or 3 doses. We have made F1 reciprocal crosses of *Ae-5180* to several different inbred lines, and observed that the phenotype of the F1 kernels seems to show a maternal effect. When our standard lines carrying *Ae-5180* are used as the female parent in the inbred crosses, the F1 kernels exhibit the variability in phenotype noted above. However, when the inbreds are used as females, the expression of *Ae-5180* is consistent for a given inbred, but varies depending on which inbred is the female parent. The inbred B73 shows the most extreme expression, the mutant kernels having a glassy, near-brittle appearance. The inbreds Mo17, Oh43, N25, and A636 show good expression, with mutant kernels that are sugary and slightly wrinkled. The inbreds M14, W22, B37, A632, B76, and H99 show fair expression, with smooth, slightly translucent mutant kernels. Mutant kernels can barely be distinguished in crosses to Tama Flint, having a weakly translucent, frosted appearance. We anticipate that after *Ae-5180* has been backcrossed into these inbreds for several generations, the above phenotypes will be consistently expressed.

In order to further characterize *Ae-5180*, and to generate potentially useful stocks, we conducted linkage tests of *Ae-5180* with a series of waxy translocations, and the chromosome 5 linkage markers *gl8* and *a2*. The 3 T5-9 waxy translocations that we selected all showed linkage with *Ae-5180* (Table 2). The tightest linkage (p = 13.4 cM) was with T5-9c, which has a breakpoint in the short arm of chromosome 5 close to the centromere. This is not surprising, since *ae* is on the long arm of chromosome 5, about 15 cM from the centromere. We might have expected even closer linkage of *Ae-5180* with *wx* T5-9(4790), which has a breakpoint at 5L.34, especially since *ae* had been

Table 2. Linkage data for *Ae-5180* to *wx* translocations.

Testcross: (Ae-5180 Wx / Ae wxT) X wx wx

wxT	Ae-5180 Wx	Ae wx	Ae-5180 wx	Ae Wx	% Recomb.
5-9c	335	288	29	67	13.4 ± 1.3
5-9(4790)	391	325	84	131	23.1 ± 1.4
5-9a	354	324	179	165	33.7 ± 1.5

Breakpoints: 5-9c 5S.07 9L.10
5-9(4790) 5L.34 9L.45
5-9a 5L.69 9S.17

Table 3. Linkage data for *Ae-5180* to *gl8*.

Testcross: (Ae-5180 gl8 / Ae gl8) X Ae Ae gl8 gl8

Reg	Genotype		No.	Totals
0	Ae-5180	+	700	
	+	gl8	973	1673
1	Ae-5180	gl8	103	
	+	+	136	239

% Recombination Ae-5180 -- gl8 = 12.5 ± 0.8

Table 4. Linkage data for *a2* to *Ae-5180*.

Testcross: a2 a2 Ae Ae X (a2 Ae-5180 / A2 Ae)

Reg	Genotype		No.	Totals
0	a2	Ae-5180	899	
	+	+	1028	1927
1	a2	+	155	
	+	Ae-5180	135	290

% Recombination a2 -- Ae-5180 = 13.1 ± 0.7

previously shown by J.N. Jenkins (Ph.D. thesis, Purdue, 1960) to be very close to the T4-5c breakpoint at 5L.27. Instead we found linkage of p = 23.1 cM. Such a high percent recombination with waxy could be at least partially explained by the chromosome 9 breakpoint of *wx* T5-9(4790), 9L.45, being at least 10 cM, and perhaps as many as 26 cM, from the waxy locus on the chromosome 9 linkage map. Crossovers occurring in the region between *wx* and the chromosome 9 breakpoint would increase the apparent linkage distance between *ae* and *wx* in this particular translocation. It is frequently difficult to correlate linkage maps with cytological maps with any degree of precision. The important point to note is that *Ae-5180* does indeed show linkage with translocations involving chromosome 5. One other point of interest is that the parental and crossover classes in the *wx* translocation linkage date are not well-balanced. There seems to be about an equal number of plump (*Ae*) and sugary (*Ae-5180*) kernels in each set of translocation data, but for each translocation there seems to be an excess of *Wx* kernels over *wx* kernels.

Our linkage data for *Ae-5180* and *gl8* (p = 12.5 ± 0.8 cM, Table 3) are in close agreement with the value of 11 cM reported on the 1987 linkage map. There appears to be an abundance of starchy (*Ae*) kernels over sugary (*Ae-5180*) kernels, but this is due to the lower percent germination of the sugary kernels when we were seedling-testing for *gl8*. The linkage data for *a2* and *Ae-5180* (Table 4) yield a value (13.1 ± 0.7 cM) that is somewhat less than the reported value for *a2* to *ae* of 22 cM, but is in agreement with a 2-point test made by Jenkins, who found a value of 14 cM for *a2* to *ae* in a backcross test.

We have crossed *Ae-5180* into homozygous *wx* stocks in order to observe whether *Ae-5180* shows the same phenotypic interaction with waxy as does the standard *ae* allele. Kernels that are homozygous for the standard *ae* allele and homozygous for *wx* are smaller, more translucent, and have finer wrinkling than kernels that are homozygous for *ae* in a *Wx* background. The latter kernels are translucent and slightly wrinkled, but not as extremely so as those in a *wx* background. This effect is very striking, and requires the *ae* allele to be homozygous (*ae ae ae*) in the endosperm. If only one or two doses of *ae* are present, the kernels will be full and plump in either a *wx* or a *Wx* background (in certain *wx* lines, it is possible to distinguish a dosage effect for *ae*, but we have not observed this in our lines). *Ae-5180*, on the other hand, can produce the same effect in only one dose. *Ae-5180 Ae Ae wx wx wx* kernels (where *Ae* represents the wildtype starchy allele of amylose-extender) are uniformly small, translucent, and finely wrinkled. *Ae-5180 Ae Ae Wx Wx Wx* kernels, on the other hand, are usually just slightly smaller in size than wildtype starchy kernels, and show the wider phenotypic range from slightly translucent to wrinkled-sugary to brittle. Thus, in both *wx* and *Wx* backgrounds, one dose of *Ae-5180* in the endosperm appears to have the same phenotypic effect as 3 doses of the standard *ae* allele.

An analogous effect can be observed when the *Ae-5180* and standard *ae* alleles are crossed into a homozygous *su1* background. It has been reported (H. Kramer et al., Agron. J. 48:170) that *ae* and *su1* have an interaction such that kernels homozygous for both mutants are plump, but highly translucent. This unique phenotype is different from that of either homozygous *su1* (wrinkled-sugary) or homozygous *ae* (slightly translucent, slightly wrinkled). We have produced plants that are homozygous *su1*, but heterozygous for *Ae-5180*, and outcrossed them to homozygous *su1* testers. The resulting ears segregate 1:1 for wrinkled-sugary kernels (*Ae Ae Ae su1 su1 su1*) and plump, translucent kernels (*Ae-5180 Ae Ae su1 su1 su1*). Thus, in a homozygous *su1* background, one dose of *Ae-5180* in the endosperm seems to have the same phenotypic effect as 3 doses of the standard *ae* allele. It should be noted that the phenotypic interactions described for *Ae-5180* with *wx* and *su1* are based on the physical appearance of the kernels, and have not been studied yet biochemically.

In a homozygous *wx* background, pollen that carries the *ae* allele can be readily distinguished from pollen that carries the wildtype *Ae* allele by a differential iodine staining technique (C.W. Moore and R.G. Creech, Genetics 70:611). This technique relies on overstaining the pollen grains with iodine, which is taken up by amylose but not amylopectin, followed by heat destaining. The *Ae wx* pollen grains, which contain amylopectin but no amylose, stain red. The *ae wx* pollen grains, which contain starch that has at least some characteristics of amylose, stain dark blue. Using this technique, we found that pollen from *Ae-5180 Ae wx wx* plants had both dark blue and red staining pollen grains in a 1:1 ratio. Thus it seems that *Ae-5180 wx* pollen also stains dark blue, opening up opportunities to use differential staining to study reversion rates of *Ae-5180* and to do fine-structure mapping of *Ae-5180* with respect to other *ae* alleles.

As mentioned previously, *Ae-5180* frequently shows a reduced frequency of male transmission in competition with the wildtype *Ae* allele. In order to study this phenomenon further, we utilized several generations of male outcrosses of heterozygous *Ae-5180 Ae* plants to standard starchy lines (*Ae Ae*) in reciprocal crosses with standard lines. All reciprocal crosses were made during the summer of 1987. For the purposes of this discussion, the term

"sugary" will be used to describe kernels showing the *Ae-5180* phenotype. In all generations, *Ae-5180* was fully female transmissible (Tables 5, 6, and 7). However, male transmission varied from generation to generation, and among sibling plants. The data in Table 5 present kernel counts from female and male outcross ears of reciprocally crossed plants grown from sugary kernels of the first generation male outcross ear. None of the male outcross ears showed a transmission of *Ae-5180* as low as that of the original outcross ear presented in Table 1 (32.4% mutant kernels), and several ears showed normal transmission.

Table 5. Counts of starchy (*Ae*) and sugary (*Ae-5180*) kernels on exact reciprocal crosses to standard of plants grown from sugary kernels of the first generation male outcross of *Ae-5180* to standard.

	Female Outcross Ears			Male Outcross Ears		
Plant No.	Starchy	Sugary	% Su	Starchy	Sugary	% Su
6093-2	161	180	52.8	213	171	44.5*
-3	256	264	50.8	345	225	39.5***
-5	195	165	45.8	235	170	42.0**
-7	216	216	50.0	259	200	43.6**
-8	204	186	47.7	176	194	52.4
6094-2	221	263	54.3	226	144	38.9***
-4	207	245	54.2	216	190	46.8
-6	272	259	48.8	202	191	48.6
-9	237	243	50.6	312	220	41.4***
-10	218	210	49.5	216	215	49.9
Totals	2183	2231	50.5	2400	1920	44.4***
	Homogeneity Chi-square = 11.26 (df = 9, N. S.)			Homogeneity Chi-square = 31.87 (df = 9, p<0.001)		

Symbols: * chi-square for 1:1 significant at $p < 0.05$
** chi-square for 1:1 significant at $p < 0.01$
*** chi-square for 1:1 significant at $p < 0.001$

Table 6. Counts of second generation male outcrosses of *Ae-5180* to standard.

	Female Outcross Ears			Male Outcross Ears		
Plant No.	Starchy	Sugary	% Su	Starchy	Sugary	% Su
6097-2	308	301	49.4	241	207	46.2
-7	214	214	50.0	231	214	48.1
-8	202	161	44.4*	246	235	48.9
-9	250	273	52.2	212	187	46.9
-11	281	274	49.4	300	282	48.5
6098-1	248	287	53.6	206	196	48.8
-3	207	248	54.5	288	265	47.9
-4	213	226	51.5	264	216	45.0*
-5	284	277	49.4	333	247	42.6***
-8	297	313	51.3	229	190	45.3
Totals	2504	2578	50.7	2550	2239	46.8***
	Homogeneity Chi-square = 12.28 (df = 9, N. S.)			Homogeneity Chi-square = 7.83 (df = 9, N. S.)		

Symbols: See Table 5.

Table 7. Counts of third generation male outcrosses of *Ae-5180* to standard.

	Female Outcross Ears			Male Outcross Ears		
Plant No.	Starchy	Sugary	% Su	Starchy	Sugary	% Su
6101-3	138	116	45.7	380	289	43.2***
-4	185	187	50.3	368	290	44.1**
-5	159	173	52.1	352	226	39.1***
-6	144	140	49.3	264	238	47.4
-11	201	196	49.8	184	144	43.9*
6102-2	229	220	49.0	302	186	38.1***
-5	167	179	51.7	200	178	47.1
-9	172	212	55.2*	401	192	32.4***
-10	272	291	51.7	381	291	43.3***
-11	218	215	49.7	257	237	48.0
Totals	1885	1929	50.6	3089	2271	42.4***
	Homogeneity Chi-square = 7.54 (df = 9, N. S.)			Homogeneity Chi-square = 86.95 (df = 9, p<0.001)		

Symbols: See Table 5.

Table 6 presents kernel counts from second generation male outcross ears. The kernels which were planted to produce family 6097 came from an ear which had 30.8% sugary kernels, and the kernels planted to produce family 6098 came from an ear that had 29.1% sugary kernels. Thus, we expected the plants in families 6097 and 6098 to inherit the system responsible for low male transmission of *Ae-5180*. As can be seen from the data in Table 6, only 2 of 10 plants showed significantly reduced male transmission of *Ae-5180*.

Table 7 presents kernel counts from third generation male outcross ears. The kernels planted to produce family 6101 came from an ear with 45.6% sugary kernels. The kernels planted to produce family 6102 came from an ear with 45.8% sugary kernels. Most of the male outcross ears of families 6101 and 6102 showed reduced transmission of *Ae-5180*, with percentages of sugary kernels comparable to those reported in Table 5. There seems to be no pattern to the reduction in male transmission of *Ae-5180* other than that it recurs even after several generations of outcrossing. Some workers (Jenkins, and others) have noted transmission anomalies associated with the standard *ae* allele. Perhaps there is some inherent biochemical effect of mutant amylose-extender alleles on the ability of mutant pollen to compete with wildtype *Ae* pollen. The degree to which the competition of mutant pollen is affected might be influenced by genetic background. This would not be without precedent, since *wx* pollen grains have been observed to germinate more slowly than *Wx* pollen grains in certain genetic backgrounds, resulting in a lowered frequency of *wx* transmission. The standard starchy lines that we use in our outcross studies have as their genetic background 4 different inbred lines. Thus, the individual plants in our male transmission studies are fairly heterogeneous. This could account for the wide variation in male transmission that we observe. As mentioned previously, we are in the process of moving *Ae-5180* into several different inbred backgrounds in order to control genetic variation in these experiments.

In order to be certain that the reduced male transmission observed in our experiments is a real effect, and not due to poor penetrance of *Ae-5180* in some of the heterozygous kernels in the male outcrosses, 100 phenotypically starchy kernels from second-generation male-outcross ears were planted, and the resulting plants selfed. Out of 76 ears obtained, none segregated for sugary kernels. Therefore, the reduced number of sugary kernels in male outcross ears is due to a real reduction in male transmission of *Ae-5180*, and not due to poor expression of the gene.

In all respects, *Ae-5180* behaves as a dominant mutant allele of amylose-extender. This gives rise to a series of questions: (1) How is a dominant mutation responsible for inducing a phenotype previously observed to be induced only by recessive mutant alleles? (2) What is the molecular nature of the wildtype, recessive and dominant amylose-extender alleles, what distinguishes them, and how are they expressed? (3) It has been suggested that *Ae* is a structural gene that codes for starch branching enzyme IIb (K. Hedman and C.D. Boyer, Biochemical Genetics 20:483). Does the existence of a dominant allele at this locus imply a regulatory role for *Ae*, or can structural genes be modified to produce a dominant mutant? (4) The *Oy* locus is another example of a gene that has both dominant and recessive mutant alleles. Will dominant mutant alleles be found for loci previously known to have only recessive mutant alleles (e.g., *y1, o2, d1*), and conversely, will recessive mutant alleles be found for loci previously known to have only dominant mutant alleles (e.g., *Kn, Tu, Pn*)? (5) Is *Ae-5180* Mutator-induced? We hope that classical and molecular studies of *Ae-5180* will provide an opportunity to answer some of these questions. We address the last question in this News Letter.

Philip S. Stinard and Donald S. Robertson

Is the dominant amylose-extender mutant *Ae-5180*, Mutator-induced?

The mutant *Ae-5180* was discovered as a single sugary/glassy kernel in a *y1 wx gl8* × *Y1 Wx Gl8 Mu* isolation plot. Fortuitously, *gl8* is located 11 cM from *ae* on chromosome 5. By seedling testing the plump non-sugary kernels from the self of the plant arising from the original mutant kernel, and scoring for *gl8* (71 *gl*, 16 *Gl*), we were able to determine that *Ae-5180* arose on the homologue of chromosome 5 that came from the Mutator parent. While this is not conclusive evidence that *Ae-5180* is Mutator-induced, it increases the likelihood of this being the case.

The best case for *Ae-5180* being Mutator-induced comes from molecular evidence. From the third generation male outcross of a heterozygous *Ae-5180* plant to standard, we selected 6 sugary kernels (*Ae-5180*/+) and 6 starchy (+/+) kernels. We grew these kernels in our 1987 summer nursery and harvested immature ears for sibling segregation analysis. We extracted DNAs, digested them with *Eco*RI (which cleaves outside the *Mu1* element), and electrophoresed them on agarose gels. In addition, *Eco*RI-digested Standard B70, Q60, and *y1 wx gl8* tester were electrophoresed on the same gel, as controls. We Southern-blotted onto nylon membranes, hybridized with the central *Mlu*I fragment of *Mu1* made radioactive with ^{32}P-dCTP by random hexamer priming, and prepared autoradiographs. We observed a segregating 6.6 Kb *Mu1*-homologous band in heterozygous mutant plants, but not in sibling non-mutant plants and controls. The number of copies of *Mu1*-homologous elements in the sibling plants based on our *Eco*RI digests is approximately 12. We will continue outcrossing our *Ae-5180* lines in order to reduce the copy number further. If the 6.6 Kb *Mu1*-homologous band that cosegregates with *Ae-5180* is the result of a *Mu* element insertion within the *Ae* gene, then this band should persist. We will also rerun these sibling segregation analyses using other restriction enzymes that cleave outside *Mu1*.

One unexpected result of this study was that our standard control lanes had a few *Mu1*-homologous bands in them; these probably represent endogenous *Mu1* -homologous sequences. The last standard to which *Ae-5180* was crossed for this analysis was Q60. An 8.6 Kb band common to all 12 sibling plants, both mutant and nonmutant, and to Q60 appeared on the autoradiograph. This demonstrates the inherent consequence of endogenous *Mu* elements in standard lines, i.e. a *Mu1*-homologous element carried by a standard could potentially result in a band that might obscure a segregating mutant band and ruin the analysis.

Philip S. Stinard and Brent Buckner

New putative Mutator-induced recessive kernel mutants with *ae*-like phenotype

The Mutator system of transposable elements has proven to be effective in the transposon-tagging of genes for cloning and molecular analysis. Since we are interested in analyzing the dominant amylose-extender mutant *Ae-5180*, we have screened a collection of 12 independent sugary/translucent kernel mutants that have arisen in our studies of the Mutator system for allelism with *ae* (Table 1). Of the 12, 7 were allelic to *ae*. It is anticipated that at least several, if not all, of these *ae* mutants will contain a *Mu* insert, and prove useful in the cloning of *Ae*. The 5 mutants that were not allelic to *ae* will be further tested for allelism with *su1, su2, sh1,* and *du*. The two mutants *su-sh*-3328* and *su-sh*-5079* are virescent, producing yellow seedlings that green as the plants mature; the virescent seedlings of *su-sh*-3328* are mutable.

Table 1. Results of allele tests of recessive *ae*-like kernel mutants with *ae*.

1987 Mutant Designation	Original Source	Source Description	Allelic to ae?	New Designation
ae*-Mu1	84-5081-3@	Stand/wx-Mus3	No	su-sh*-5081
ae*-Mu2	86-2161-2@	y1 wx gl1/Mu2	Yes	ae-Mu1
ae*-Mu3	86-2177-8@	Bf1-Mu/Stand	Yes	ae-Mu2
ae*-Mu4	86-1200-1@	Stand/y1-Mu	Yes	ae-Mu3
ae*-Mu5	86-1023-3@	Stand/yg2-Mu	Yes	ae-Mu4
ae*-Mu6	86-2162-4@	y1 wx gl1/Mu2	No	sm1-su*-2162
ae*-Mu7	85-3191-7@	Stand/y1-Mu	No	su-sh*-3191
ae*-Mu8	85-3297-28@	Stand/Mu8	Yes	ae-Mu5
ae*-Mu9	85-3238-7@	Stand/y1-Mu	Yes	ae-Mu6
ae*-Mu10	83-3328-46@	Mu2 2 X O.C.	No	su-sh*-3328
ae*-Mu11	84-5321-23@	Mu8/Stand	Yes	ae-Mu7
ae*-Mu12	84-5079-24@	Stand/wx-Mus2	No	su-sh*-5079

Symbols: su, translucent or glassy; sh, shrunken like sh1; sm1, small.

Kernels from 86-8501-1/908-4 & 910-1 were grown in our 1987 summer nursery, and the resulting plants were

Philip S. Stinard and Donald S. Robertson

A putative Mutator-induced deletion of the *A1* and *Sh2* loci on chromosome 3

Evidence has accumulated that deletions have been induced by Mutator at the *yg2* and *Bf1* gene loci (see subsequent articles in this News Letter). We now present evidence of a Mutator-induced deletion of a segment of chromosome 3 including the *A1* and *Sh2* loci.

In our 1985-86 winter nursery, we observed a 3-seed sector of yellow brittle kernels on an ear (85-86-6512-6/5513-3) from the cross of a purple aleurone Mutator stock by an *a1-Mum2 Sh2* stock heterozygous for the standard *a1 sh2* tester alleles in coupling. Since our purple Mutator stocks are homozygous for the *A1* and *Sh2* alleles, we hypothesize that the 3-seed sector arose premeiotically by the deletion of a small segment including the *A1* and *Sh2* loci (located 0.2 cM apart) on one of the chromosome 3 homologues of the purple Mutator parent. The eggs arising from the ear sector carrying this deletion were fertilized by *a1 sh2* pollen from the male parent, giving rise to the sector of 3 yellow brittle seeds. Since the male parent was heterozygous for *a1 sh2*, there is a possibility that some eggs carrying the deletion could have been fertilized by pollen carrying the *a1-Mum2* and *Sh2* alleles. Kernels arising from such a fertilization would be starchy, and either mutable or yellow stable. No such kernels were observed.

In order to analyze this putative deletion genetically, we attempted to germinate the 3 yellow brittle kernels in Petri dishes. Two of the 3 kernels molded, but the third kernel produced a small, weak seedling that grew slowly and reached silking stage about 90 days after germination. We had intended to pollinate this plant (86-8501-1) by a standard purple aleurone stock in order to separate the standard *a1 sh2* homologue from the *a1 sh2* deletion homologue, but instead pollinated by *c1 c1 r r* B79 inbreds (86-908-4 and 86-910-1) on two successive days, as these were the only plants shedding pollen late in the season. This pollination accomplished the same result, as B79 is wildtype for *A1* and *Sh2*. The ear resulting from this cross (86-8501-1/908-4 & 910-1) had normal seed set and produced about 100 kernels. We would expect about half of these kernels to be *A1 Sh2/a1 sh2*-deletion, and half to be *A1 Sh2/a1 sh2*-standard.

reciprocally crossed with a homozygous standard *a1 sh2* tester on the first ear, and pollinated by either a purple aleurone stock or an *a1 a1 et et* stock on the second ear. In addition, shedding tassel branches were sampled from as many of these plants as possible, and the pollen examined for sterility. The results of the reciprocal crosses with standard *a1 sh2* are presented in Table 1. Out of 27 pairs of reciprocals made, 11 were significantly deficient ($p < 0.001$) in the yellow brittle class when the heterozygous parent was outcrossed as a male. These 11 heterozygotes presumably carry the putative *a1 sh2* deletion, and transmit the deletion in a highly reduced frequency when the deletion pollen competes with wildtype pollen. The average frequency of transmission of *a1 sh2* through the male in these 11 plants is 18.5%, but the individual frequencies are very heterogeneous (homogeneity chi-square $p < 0.001$). The putative deletion seems to be fully transmissible through the female if one looks at individual ears, since none of the crosses of the putative deletion heterozygotes as females by standard *a1 sh2* gave rise to ears deviating significantly from 1:1 in the ratio of purple starchy to yellow brittle kernels. However, when one looks at the totals for the deficient ears, the average percent transmission of *a1 sh2* through the female is 46.4%, significantly different from a 1:1 ratio ($p < 0.001$). Thus, female transmission of the putative deletion may be very slightly reduced. The pollen samples from all putative deletion heterozygotes examined were normal, or near normal, with no evidence of semisterility. Apparently, the major effect of the deletion on the pollen is to reduce its competitive ability with wildtype pollen. Whether this is accomplished by reduced pollen germination, by slower pollen tube growth, or some other mechanism, is not known. We have examined the pollen from a known *a1 sh2* deletion heterozygote (*a1-x1*) provided by the Maize Coop, and find that it is semisterile. Perhaps the *a1-x1* deletion includes regions essential for the growth of the male gametophyte that are not deleted in our putative *a1 sh2* deletion. Cytological analysis may be able to help verify whether this is true.

Table 1. Frequencies of transmission of *a1 sh2* in reciprocal crosses with a standard *a1 sh2* tester of plants heterozygous for either the standard *a1 sh2* alleles (*A1 Sh2/a1 sh2*) or the putative *a1 sh2* deletion (*A1 Sh2/a1-sh2-Del*).

Heterozygous parent	Probable status of parent	Pollen classification	Kernels from crosses with heterozygote as female parent: Pl	yeh	%yeh	Kernels from crosses with heterozygote as male parent: Pl	yeh	%yeh
6170-1	standard	n10	153	169	52.5	173	177	50.6
-2	deletion	n	9	14	60.9	353	93	20.9**
-3	standard	n	117	117	50.0	171	167	49.4
-8	deletion	n	167	139	45.4	276	53	16.1**
-5	standard	n	ND	ND		82	80	49.4
-7	standard	n	ND	ND		179	177	49.7
6171-1	standard	n	ND	ND		227	219	49.1
-2	deletion	n10	158	128	44.6	378	65	14.8**
-4	standard	n5	230	193	45.6	170	184	52.0
-5	standard	n	86	81	48.5	257	254	47.9
-6	standard	n	265	254	48.9	273	247	47.5
-7	deletion	n	8	4	33.3	132	38	22.4**
-8	standard	n	254	193	43.2*	204	221	52.0
-9	standard	n	199	200	50.1	127	152	54.5
-10	deletion	n10	3	5	62.5	80	35	30.4**
6172-1	deletion	n	210	161	43.4	451	100	18.1**
-2	standard	ND	236	208	50.8	171	159	48.2
-3	standard	ND	120	148	54.5	285	233	45.0
-4	standard	ND	148	127	46.2	210	274	56.6*
-5	deletion	ND	187	153	45.0	89	43	32.6**
-6	deletion	ND	208	189	48.1	157	60	27.6**
-7	standard	ND	65	72	52.6	265	219	45.2
6193-1	standard	n	108	110	50.5	129	114	46.9
-2	standard	n	205	190	48.1	122	132	52.0
-3	deletion	n	225	216	49.0	429	62	12.6**
-4	standard	n	130	152	53.9	178	151	45.9
-6	deletion	n	187	168	47.3	316	75	19.2**
-7	deletion	n	ND	ND		385	65	14.8**
Total deletion			1358	1173	46.4**	3082	689	18.5**
Total standard			2316	2246	49.2	3223	3160	49.5

Symbols: n, normal pollen; n5, n10, mostly normal pollen, with 5% and 10% abortive pollen grains, respectively
Pl, purple starchy kernels
yeh, yellow brittle a1 sh2 kernels (Crossover kernels were very few, and are not included in these data. There were no crossovers observed in the putative deletion heterozygotes.)
ND, no data available
* chi-square for 1:1 ratio significant at p=.01 level
** chi-square for 1:1 ratio significant at p=.001 level

Crosses of putative deletion heterozygotes by etched stocks gave rise to only non-etched kernels. Thus, the putative deletion does not include the etched locus.

We have not analyzed the putative deletion molecularly, but we hope that this may soon be accomplished by either ourselves or others. Should this prove to be a deletion, and should a copy of a Mutator element be found at the site of the deletion, this would shed further light on the role of Mutator in inducing deletions. Furthermore, since we have sibling kernels from the ear on which the putative deletion was observed, one could investigate molecularly the possible mechanisms for the formation of the deletion (e.g. pre-meiotic unequal sister chromatid exchange or somatic crossing over, occurring by slant pairing of homologous Mutator elements on either side of the *A1* and *Sh2* loci).

Philip S. Stinard and Donald S. Robertson

A putative Mutator-induced deficiency linked to *A1* on chromosome 3

In our 1985 summer nursery, we crossed a purple aleurone Mutator (Pl *Mu*) plant by a Mutator-induced *a1* mutable stock (*a1-Mum3*) as part of our ongoing Mutator inbreeding experiments. Kernels from this cross (85-6153-2/6152-5) were planted in our 1986 selfing block, and the resulting plants self-pollinated. Of 44 selfs, 15 ears segregated for ratios of purple to mutant kernels that deviated greatly from the expected 3:1 ratio. In each instance, the purple class was deficient, resulting in ratios as low as 1:4 (Table 1). These same ears were also semisterile. We hypothesized that the low frequency of transmission of *A1* in these selfs was due to a deficiency linked to *A1* that is not transmissible through the male, and probably only poorly transmitted through the female. The purple kernels appearing on the deficient selfed ears were either the result of crossovers with the chromosome 3 homologue not carrying the deficiency, or the result of reduced female transmission of the deficiency. The deficiency arose in the Pl *Mu* parent (or its progenitor) on one of the chromosome 3 homologues, and was transmitted to its progeny in a reduced frequency (15/44). In order to study this phenomenon further, we planted more kernels from 85-6153-2/6152-5 in our 1987 crossing block, and reciprocally crossed the resulting plants with *a1* testers. We also planted purple kernels from the deficient selfed ears, and reciprocally crossed the resulting plants with *a1* testers.

Table 1: Frequencies of purple and *a1* mutant kernels on *A1*-deficient ears from the selfs of plants grown from the seeds of 85-6153-2/6152-5.

Plant number	Pl	a1	%Pl
1260-5	11	65	14.5**
-6	38	98	27.9**
-12	181	234	43.6**
-13	63	131	32.5**
-24	59	219	21.2**
-26	88	159	35.6**
-27	55	193	22.2**
-28	108	168	39.7**
-31	27	113	19.3**
-35	13	49	21.0**
-36	28	85	24.8**
-38	50	69	42.0**
-39	53	52	50.5**
-42	12	45	21.1**
-44	9	11	45.0*
Totals	795	1687	32.0**

Symbols: * chi-square for 3:1 ratio significant at p=.01 level
** chi-square for 3:1 ratio significant at p=.001 level

Table 2: Frequencies of transmission of *A1* in reciprocal crosses with an *a1* tester of plants grown from the seeds of ear 85-6153-2/6152-5.

Plant number	Probable genotype	Pollen classification	Kernels from female outcross: A1	a1	%A1	Kernels from male outcross: A1	a1	%A1
6167-1	A1/a1	n	ND	ND		67	86	43.8
-2	A1/a1	n	61	52	54.0	223	223	50.0
-3	A1/a1	n	261	245	51.6	17	14	54.8
-4	A1-def/a1	s-	5	34	12.8**	7	80	8.0**
-5	A1-def/a1	s-	31	40	43.7	19	315	5.7**
-6	A1/a1	n	37	54	40.7	106	112	48.6
-7	A1/a1	n	65	66	49.6	134	122	52.3
6168-1	A1-def/a1	s-	19	65	22.6**	15	135	10.0**
-5	A1/a1	n	231	233	49.8	305	280	52.1
-6	A1/a1	n	ND	ND		264	267	49.7
-7	A1/a1	n	291	284	50.6	266	258	52.7
Total Deficient			55	139	29.1**	41	530	7.2**
Total Standard			946	938	50.3	1404	1362	50.8

Symbols: n, normal pollen; s-, nearly semisterile pollen
ND, no data available
* chi-square for 1:1 ratio significant at p=.01 level
** chi-square for 1:1 ratio signigicant at p=.001 level

Table 3: Frequencies of transmission of *A1* in reciprocal crosses with an *a1* tester of plants grown from purple seeds produced by the self-pollinations of plants heterozygous for the *A1*-linked deficiency (*A1-def/a1*).

1986 parent	Plant number	Probable genotype	Pollen classification	Kernels from female outcross: A1	a1	%A1	Kernels from male outcross: A1	a1	%A1
1260-13	6177-2	A1/a1	n	ND	ND		107	135	44.2
	-3	A1-def/a1	s	122	207	37.1**	20	208	8.8**
	-5	A1-def/a1	s-	36	75	32.4**	24	406	5.6**
	-6	A1-def/a1	s25	2	3	40.0	54	345	13.5**
	-7	A1-def/a1	n15	1	1	50.0	17	113	13.1**
	-8	A1-def/a1	n-	7	23	23.3*	47	268	14.9**
1260-24	6178-2	A1/a1	n	31	40	43.7	71	68	51.1
	-3	A1/a1	n	10	8	55.6	141	158	47.2
	-4	A1-def/a1	ND	47	130	26.6**	22	93	19.1**
	-5	A1-def/a1	n	79	115	40.7*	18	147	10.9**
	-6	A1/a1	n-	139	139	50.0	119	126	48.6
	-7	A1-def/a1	s25	73	102	41.7	26	227	10.3**
	-8	A1/a1	n	235	244	49.1	ND	ND	
	-10	A1-def/a1	s-	ND	ND		7	66	9.6**
1260-26	6179-6	A1-def/a1	s-	88	160	35.5**	2	36	5.3**
	-7	A1/a1	n	20	17	54.1	76	61	55.5
	-8	A1-def/a1	s-	ND	ND		5	32	13.5**
	-9	A1/a1	n	ND	ND		148	153	49.2
	-10	A1-def/a1	s-	15	27	35.7	1	22	4.3**
1260-27	6180-4	A1-def/a1	ND	115	186	38.2**	ND	ND	
	-6	A1/a1	n	109	128	46.0	219	228	49.0
	-7	A1/a1	n	281	260	51.9	179	173	49.6
Total deficient				585	1029	36.2**	283	1963	11.0**
Total standard				825	836	49.7	1051	1102	48.8

Symbols: n, normal pollen; n-, nearly normal pollen; n15, mostly normal pollen, with 15% abortive pollen grains
s, semisterile pollen; s-, nearly semisterile pollen; s25, 25% pollen sterility
ND, no data available
* chi-square for 1:1 ratio significant at p=.01 level
** chi-square for 1:1 ratio signigicant at p=.001 level

Of 11 plants grown from the kernels of 85-6153-2/6152-5, only 3 carried the deficiency (Table 2). Male transmission of the *A1* allele in reciprocal crosses with *a1* testers was greatly reduced (7.2%), but female transmission was reduced as well (29.1%). Pollen shed from anthers collected from deficient plants was semisterile, probably reflecting gametophytic lethality associated with pollen carrying the deficiency.

Of 22 plants grown from the purple kernels of deficient selfed ears, 13 carried the *A1*-linked deficiency (Table 3). Again, both male and female transmission of the *A1* allele was reduced in reciprocal crosses with *a1* testers (11.0% and 36.2% respectively), and pollen produced by these plants was semisterile (with the exception of one which had only 15% abortive pollen). The 9 plants from this set that did not carry the deficiency (as evidenced by the equal transmission of *A1* and *a1* through both the male and the female) probably reflect crossovers occurring in the parent during either mega- or microsporogenesis that placed *A1* in coupling with non-deficient chromosome 3 segments.

We made crosses of deficient plants by homozygous etched stocks, but recovered no etched kernels, indicating that the deficiency does not include the etched locus.

It is possible to derive both a percent recombination of *A1* with the deficiency, and a rate of female transmission of the deficiency, if one makes the following assumptions: (1) the deficiency is not male transmissible; (2) the percentage recombination between *A1* and the deficiency is the same in both the male and the female; (3) the deficiency is transmitted through the female with the same frequency in both the parental and crossover classes. With these assumptions, and combining the data for the reciprocal crosses of deficient plants in Tables 2 and 3, one arrives at a percent recombination of *A1* with the deficiency of 10.2 $\pm$ 0.6, and a female transmission rate of 31.7% (i.e. in a plant heterozygous for the deficiency, 31.7% of the ovules will carry the deletion). This female transmission frequency agrees well with the percentage of the offspring of 85-6153-2/6152-5 that carry the deficiency (18/55 = 32.7%). One can use the calculated percent recombination and female transmission frequency to deduce expected frequencies of purple and mutant kernels in selfed ears of deficiency heterozygotes. The expected frequency of purple kernels in such selfs, 42.0%, differs significantly from the average frequency of purple kernels observed in the selfed plants from Table 1 (32.0%), but comes close to several individual values.

We plan to conduct further experiments with other linkage markers to determine whether the deficiency is proximal or distal to *A1*. We will also try to recover the crossover class that places the deficiency in coupling with *a1*. If this deficiency is Mutator-induced, it should also be amenable to sibling segregation analysis to determine whether a Mutator element co-segregates with the deficiency.

Philip S. Stinard and Donald S. Robertson

Dappled-1, a dominant aleurone developmental mutant, is located on 5L

In the 1987 issue of the Maize News Letter (MNL 61:7-9), we reported on the genetics of a dominant aleurone developmental mutant, Dappled-1 (*Dap1*), that arose in one of our Mutator stocks. This mutant, which is expressed only in outcrosses as a female, yet is both male and female transmissible, produces a phenotype of irregular patches of normal, pigmented aleurone cells surrounded by colorless and morphologically abnormal aleurone cells. We now present linkage data that suggest that *Dap1* is located on the distal half of the long arm of chromosome 5.

During the summer of 1986, we crossed heterozygous Dappled plants by a comprehensive series of waxy translocations. This past summer, we backcrossed the semisterile progeny by a homozygous purple *wx R-scm2* line and scored the resultant ears for the Dappled and waxy traits. The only translocations that showed linkage of Dappled with waxy were *wx* T5-9 (4790) and *wx* T5-9a (Table 1). The closest linkage (about 3 cM) is with T5-9a, with breakpoints at 5L.69 and 9S.17. Since none of the other waxy translocations [other than T5-9(4790)] showed linkage with dappled, this gene must be located on chromosome 5. The linkage data for T5-9(4790), with breakpoints at 5L.34 and 9L.45, show much looser linkage of Dappled with waxy (about 39 cM), putting Dappled much closer to the T5-9a breakpoint at 5L.69. We are currently making crosses to test linkage of Dappled with *pr, v12, yg1,* and *v2*, all on the long arm of chromosome 5.

To date, we have not been able to isolate a homozygous

Table 1. Translocation backcross data for linkage of Dappled-1 with *wx* T5-9(4790) and *wx* T5-9a.

Female Parent	Trans-location	Pl wx	Dap Wx	Pl Wx	Dap wx
6131.0-1	5-9(4790)	25	31	22	13
6131.0-5	" "	16	27	15	14
Totals	" "	41	58	37	27

% Recombination = 39.3 ± 3.8

Female Parent	Trans-location	Pl wx	Dap Wx	Pl Wx	Dap wx
6130.1-1	5-9a	107	86	4	0
6130.1-2	"	76	105	4	2
6130.1-3	"	73	61	6	0
6130.1-4	"	138	115	5	2
Totals	"	394	367	19	4

% Recombination = 2.9 ± 0.6

Dappled line. With the use of *wx* T5-9a, we should be able to place Dappled in close coupling with *wx*, and use this close coupling to isolate a homozygous *wx* line that is presumably homozygous for Dappled. Based on our previous unsuccessful attempts to isolate a homozygous Dappled line, we would predict that such a homozygote will be inviable.

Philip S. Stinard

Dappled-1 shows incomplete penetrance

Dappled-1 expresses itself only when transmitted through the female of heterozygous plants (homozygous Dappled lines have not yet been isolated). However, the frequency of phenotypically Dappled kernels observed on segregating ears is somewhat less than the expected 50% (average percent Dappled observed = 31.25%, see MNL 61:7). This frequency varies from the base to the tip of the ear, with a higher frequency of Dappled kernels appearing at the base.

To determine whether incomplete penetrance of Dappled accounts for the deficiency of phenotypically Dappled kernels, we selected 25 fully colored purple kernels from the base, and 25 from the tip of 6 different segregating ears of Dappled heterozygotes that had been pollinated by purple aleurone lines (these ears were included in our 1986 report), and grew them in our 1987 selfing block. The resulting ears were scored for the presence of Dappled.

We found that a small percentage of purple kernels did indeed give rise to heterozygous Dappled plants, and that this percentage was lower for kernels from the base (4.20% Dappled) than for kernels from the tip (15.75%, Table 1).

Table 1. Phenotypes of plants grown from purple non-sectored kernels taken from the base and tip of heterozygous Dappled ears.

1985 source ear	1987 selfing family no.	Base kernels Pl	Base kernels Dap	Tip kernels Pl	Tip kernels Dap
634-8/834-6	2120	22	3	18	4
635-3/833-3	2121	24	0	13	5
633-6/833-4T	2122	22	2	21	3
633-1/833-2	2123	22	1	18	7
634-7/833-2T	2124	24	0	18	0
634-6/834-9	2125	23	0	19	1
Totals		137	6	107	20

Contingency chi-square for base vs. tip kernels = 9.0303, $p < 0.01$

However, these values were not great enough to account for the deficiency of Dappled kernels observed on our 1985 ears (Table 2). When adjustments are made for the full colored purple kernels that are really heterozygous Dappled, the average proportion of Dappled kernels on these ears is 37.82%, still significantly less than 50%. Thus, even accounting for reduced penetrance, we can still conclude that Dappled is transmitted at a reduced frequency through the female. We estimate that the average penetrance of Dappled is 75.7%, although this seems to vary from ear to

Table 2. Observed, and adjusted* percent Dappled kernels on heterozygous Dappled plants crossed as females by purple aleurone stocks.

1985 female	Observed % Dappled Base	Tip	Total	Adjusted % Dappled Base	Tip	Total	Percent Penetrance Base	Tip	Total
634-8	44.29	27.42	38.66	50.71	40.86	45.09	87.3	67.1	76.9
635-3	43.68	17.60	28.75	43.68	40.34	41.77	100.0	43.6	68.8
633-6	32.80	31.92	32.25	38.40	40.38	39.64	85.4	79.1	81.3
633-1	40.00	12.80	22.56	42.86	37.20	39.23	93.3	34.4	57.5
634-7	11.11	35.50	27.93	11.11	35.50	27.93	100.0	100.0	100.0
634-6	6.58	35.10	25.55	6.58	38.81	27.75	100.0	91.7	92.1
Totals	33.56	25.63	28.61	36.24	38.77	37.82	92.6	66.1	75.7

* The adjusted values are the values reported in our 1986 Newsletter article adjusted for the percentage of phenotypically wildtype, but genetically Dappled kernels reported in Table 1.

ear, and may vary in different genetic backgrounds.

When conducting detailed linkage studies, the incomplete penetrance of Dappled necessitates growing the phenotypically wildtype kernels of backcross ears in an open pollination block, and scoring the ears of these plants for Dappled. However, even if this is not done, approximate linkage data can still be obtained by scoring the kernels at the base of the ear, where the penetrance of Dappled is highest.

Philip S. Stinard and Donald S. Robertson

Two-point linkage data for *brn1* to *ra2, g2,* and *d1*

We report 2-point linkage data for *brn1* to *ra2* (Table 1), *g2* to *brn1* (Table 2), and *brn1* to *d1* (Table 3). Linkage

Table 1. Linkage data for *brn1* to *ra2*.

Testcross: (brn1 Ra2 / Brn1 ra2) X Brn1 Brn1 ra2 ra2

Reg	Genotype		No.	Totals
0	brn1	+	181	
	+	ra2	170	351
1	brn1	ra2	69	
	+	+	59	128

% Recombination brn1 -- ra2 = 26.72 ± 2.02

Table 2. Linkage data for *g2* to *brn1*.

Testcross: G2 G2 Brn1 Brn1 X (G2 brn1 / g2 Brn1)

Reg	Genotype		No.	Totals
0	g2	+	282	
	+	brn1	302*	584
1	g2	brn1	43*	
	+	+	53	96

% Recombination g2 -- brn1 = 15.82 ± 1.99

* The values for the *brn1* classes were not used in calculating the % recombination because classification for *g2* in seedlings grown from *brn1* kernels is uncertain. Had these values been included, the % recombination for *g2—brn1* would be 14.12 ± 1.34.

Table 3. 1987 linkage data for *brn1* to *d1*.

Testcross: Brn1 Brn1 D1 D1 X (brn1 D1 / Brn1 d1)

Reg	Genotype		No.	Totals
0	brn1	+	82	
	+	d1	93	175
1	brn1	d1	24	
	+	+	24	48

% Recombination brn1 -- d1 = 21.52 ± 2.75

tests were set up as modified backcrosses. Tassels were scored for *ra2* just prior to bagging. The trait *brn1* was scored on the ears of selfed plants. The traits *g2* and *d1* were scored in sandbench seedling tests of selfed ears. The trait *g2* could not be reliably scored in seedling tests of selfed ears where it was in coupling with *brn1*. The pale green seedling phenotype of *brn1 brn1* plants masked the expression of *g2*, and crossovers placing golden plants into the yellow class of kernels from these ears were too infrequent to allow reliable classification. Therefore, data from the selfed ears segregating *brn1* were not used in calculating the percent recombination for *g2* to *brn1*.

If one combines our 1986 linkage data (MNL 61:6) with this year's data (Table 3), one obtains a map distance of 19.69 ± 1.56 cM for *brn1* to *d1*. This value places *brn1* about 2 cM distal to *cr1* on chromosome 3. If one combines our *g2* - *brn1* map distance (15.82 ± 1.99 centimorgans) with our *brn1* - *d1* distance, one obtains an approximate *g2* - *d1* distance of 35.5 cM. This would place *g2* 3.5 cM further out on 3S than indicated on the 1987 linkage map. Good 3-point linkage data will be required to confirm this.

Philip S. Stinard

Putative Mutator-induced *a2* mutants with colored scutellum

From our 1979 *a2 bm bt* purple Mutator plot, we isolated 3 stable pale purple *a2* mutant kernels (*a2-Mus1*, *a2-Mus2*, and *a2-Mus3*). We extracted the homozygous *a2-Mus* mutants, crossed these mutants to purple Mutator lines, and selfed the F1 to see whether somatic mutability could be induced in the *a2-Mus* alleles. We did not recover somatic mutability, but we did observe the segregation of pale purple kernels with full purple scutella. We checked the pedigree, and found that the purple Mutator lines which the mutants were crossed into carried the *R-scm2* allele. In our 1987 crossing block, we planted pale purple kernels (*a2-Mus* aleurone phenotype) with purple scutella from segregating selfed ears, and selfed and outcrossed the resulting plants to *a2* testers (*a2 bt*). For all 3 *a2-Mus* mutants, selfed ears were recovered that were homozygous *a2-Mus*, and either homozygous or segregating (3:1 purple:colorless) for scutellum color (two ears from our *a2-Mus3* selfs segregated for both the *a2* mutant and scutellum color, and were presumably the result of heterofertilization events, but other selfed ears of this mutant were homozygous *a2-Mus*.) For *a2-Mus2* and *a2-Mus3*, the outcrosses to *a2* testers produced ears with all pale purple kernels, and either all or half of the kernels had purple scutella. No crosses of *a2-Mus1* to *a2* testers set seed.

These data suggest that the *A2* locus may have separate components responsible for color in the aleurone and in the scutellum. Chen et al. (Genetics 116:469) have described an organ-specific Mutator-induced *Adh1* mutant that has a *Mu* insert in the promoter region of the *Adh1* gene. This appears to be an example of a gene that has a compound regulatory region, with sequences in the promoter that are required for the expression of the gene in certain tissues. Perhaps *A2* is regulated in an analogous manner.

Alternatively, the *A2* coding region could contain tissue-specific components for scutellum and aleurone color. A *Mu* insert in one of these components could disrupt transcription or tissue-specific splicing of RNA transcripts in one tissue, but not the other. Molecular analysis of the *a2-Mus* mutants could provide valuable insight into the regulation and expression of the *A2* gene, and perhaps other genes, in maize.

We are moving our *a2-Mus* mutants into a *B Pl* background to observe whether purple plant color is expressed in homozygous *a2-Mus* plants. If the *a2-Mus* mutants behave as do previously reported *a2* mutants, these homozygous *a2-Mus* plants should be brown, with some tissue deterioration. If the putative scutellum color component also allows the expression of plant color, then purple plants will be observed.

Not all Mutator-induced *a2* mutants show scutellum color in the presence of *R-scm2* as do the 3 *a2-Mus* mutants described above. The only other Mutator-induced *a2* mutant we have in an *R-scm2* background, *a2-Mum3* (a mutable allele), has colorless scutellum. Furthermore, it is possible that the 3 *a2-Mus* alleles described above are the result of the same mutational event, since the purple Mutator parent was used as a male in the isolation plot in which the 3 *a2-Mus* mutants were isolated, and we have no way of telling whether these mutants arose independently of each other, or whether they arose from a tassel sector. Thus it is possible that *a2* mutations that express scutellum color are a very rare event. We would be happy to share our *a2-Mus* mutants with anyone wishing to work with them.

Philip S. Stinard and Donald S. Robertson

Progress toward the cloning of genes responsible for carotenoid biosynthesis

The leaves of all green plants contain carotenoid pigments, one function of which is to protect chlorophyll pigments from photooxidation. Yellow-seeded corn also contains carotenoid pigments in the endosperm. The carotenoid pigments in the green plant parts and endosperm are thought to be synthesized by a similar or identical biochemical pathway that is controlled by the same genes. The genetics of many mutants involved in carotenoid biosynthesis in maize has been analyzed, including *y1*, *vp5*, *vp9*, *ps*, *w3*, *vp2*, *al*, *lw1*, *lw2*, *lw3*, *lw4*, *y9*, *cl*, and *y10*. Information has been obtained about the relationship of several of these genes to proposed steps in the biosynthetic pathway of carotenoids. Kernels homozygous for these mutant alleles are pale yellow or white, and are deficient in beta-carotene. When these kernels are germinated, they give rise to pale green or albino seedlings, also deficient in beta-carotene (a notable exception is the *y1* locus, discussed below). Some of these mutants accumulate carotenoid precursors in the leaves of mutant plants (e.g. *w3*, phytotene and phytofluene; *vp5*, phytoene, phytofluene and zeta-carotene; *ps*, phytoene, phytofluene and lycopene). Accumulation of precursors has also been demonstrated in endosperms of some of these mutants. We are attempting to clone several of the genes responsible for carotenoid biosynthesis in corn.

The gene product of the *Y1* locus is thought to be a regulatory element in the carotenoid biosynthetic pathway in maize. We have analyzed 28 families segregating for plants heterozygous for different putative *Mu*-induced *y1* mutations (*Y1/y1-Mum*). DNA of 40 such heterozygous plants selected from these families was isolated. The approximate number of *Mu1*-homologous elements in each plant was established by Southern blot analyses. The plants with low *Mu* copy numbers (6 to 12) were outcrossed to a white endosperm stock homozygous for the standard *y1* allele, producing seeds of two types: yellow (*Y1/y1*), and white (*y1/y1-Mum*). DNA was isolated from plants grown from each type of seed, digested with restriction endonucleases that cleave outside the *Mu1* element, electrophoresed, Southern blotted and hybridized with *Mu1* DNA. The *Eco*RI hybridization profile of the outcross progeny of one heterozygous plant, 2045-10, contained a 5.9 Kbp band that appeared in all plants derived from white seeds, but was absent in all plants derived from yellow seeds. It is

expected that the *y1* gene, carrying a *Mu* element insertion, would segregate exclusively with mutant white-seeded individuals. DNA from a single white-seeded progeny of plant number 2045-10 was digested with *Eco*RI, and the DNA fragments between 5.5 and 6.0 Kbp in length were isolated. This fraction of DNA was shown to contain a 5.9 Kbp *Mu1*-hybridizing fragment. No other *Mu1*-homologous DNA fragments were present. The size-fractionated DNA was ligated into *Eco*RI-digested lambda-gt10, packaged, and transfected into *E. coli*. A single *Mu1*-hybridizing plaque was isolated. This clone was purified and shown to contain a *Mu* element equivalent in size to *Mu1* as well as another sequence(s) repetitive in both the clone and the maize genome. To demonstrate that the cloned DNA is homologous to the 5.9 Kbp *Eco*RI fragment shown to segregate with the white endosperm phenotype, a nonrepetitive sequence from the cloned DNA will be used as a hybridization probe of the *Eco*RI Southern segregation analysis. To identify a nonrepetitive sequence, the DNA is being subcloned and restriction mapped.

In addition to our efforts to clone the *Y1* gene, we have also transposon tagged other genes involved in the carotenoid biosynthetic pathway. Putative *Mu*-induced mutants allelic to *y9, w3, vp5, vp9* and *ps* have been isolated. Plants that are recessive for any one of these loci have known patterns of accumulation of carotenoid precursors. We have not yet begun a molecular analysis of these mutants. We also have numerous additional *Mu*-induced white-kernel/albino-seedling mutants that have not yet been tested for allelism.

Some white endosperm inbred lines (e.g., A188) are homozygous for a dominant white endosperm gene as well as for *y1*. Mutator stocks homozygous for a dominant white endosperm gene (nonallelic to *y1*) have been produced. These stocks will be used next summer for isolating a *Mu*-induced mutant at this locus. We are also in the process of testing our dominant white stocks for allelism with the dominant white cap allele (*Wc*).

Brent Buckner and Donald S. Robertson

The *y1-wmut* allele of *y1* does not interact with the *Ac Ds* and *En Spm* controlling element systems

We have over 300 putative Mutator-induced mutations of the *y1* locus; however, mutants of the *y1* locus induced by other transposable element systems would be useful in our attempts to isolate the *y1* gene (see previous report in this issue). We have stocks of a mutable *y1* allele, *y1-wmut*, that was first isolated at the California Institute of Technology by E. G. Anderson. This allele is a temperature-sensitive pastel allele similar to *y1-8549*, but it is unstable and regularly produces large revertant sectors in both endosperm and plant tissue. This behavior suggests that this allele of *y1* may have been induced by the insertion of a transposable element. We have crossed this mutant to *Ac* and *En Spm* tester stocks to determine if this mutant is related to one of these controlling element systems. We find that this mutant does not activate either controlling element system.

Brent Buckner and Donald S. Robertson

Are deletions of the *y1* locus viable?

Genetic (Robertson and Stinard, Genetics 115:353, 1987) and molecular (L.P. Taylor and V. Walbot, EMBO J. 4:869-876, 1985) evidence indicates that Mutator can induce deletions. In our laboratory, we have used Mutator to induce deletions at the *yg2, wd, Bf1* and *a1 sh2* loci. We have over 300 putative Mutator-induced mutants at the *y1* locus. Preliminary genetic studies of these *y1* mutations suggested that some of them may have been the result of small or large deletions involving the *y1* locus. These putative deletions were selected on the basis of one of two criteria: 1) The absence of pastel seedlings when kernels from self pollinated ears of *y1-Mu/y1* plants were germinated at 37 C; and 2) a low frequency of white seeds on ears of self pollinated *Y1/y1-Mu* plants. The first criterion for selecting deletions seemed reasonable because most *Mu*-induced *y1* mutant seedlings are pastel when grown at 37 C. If a large deletion, that could not be transmitted through the male, was induced by *Mu* involving the *y1* locus, self pollinated ears of *y1-Mu/y1* plants would have only kernels of the genotypes *y1-Mu/y1* or *y1/y1* neither of which would give seedlings of pastel phenotype. The second criterion was chosen because a *Mu*-induced small deletion with reduced male transmission would result in a low frequency of white kernels on self pollinated ears of *Y1/y1-Mu* plants. Other explanations are possible for the occurrence of plants with these patterns of inheritance, but it seemed that these two criteria were the most reasonable ones to use for selecting deletions, if they were being induced.

Using the above criteria, we have selected putative *y1* deletion plants and crossed them to stocks homozygous for *ms1* or *si* to produce plants heterozygous for the putative *y1* deletions and *ms1* or *si* in repulsion. The *ms1* and *si* loci are very closely linked to *y1*. Therefore, if the *Mu*-induced *y1* mutations are deletions, and if the deletions include the *ms1* or *si* loci, the heterozygotes with *ms1* or *si* might uncover one or the other of these alleles and exhibit the male sterile or silky phenotype. We have studied 3 different putative deletions as heterozygotes and found none with the male sterile or silky phenotype:

1986-87 planting nos.	No. of mature plants screened *Ms1*	*Si*
8679,8680	29	9
8681,8682	19	58
8703,8704	36	68

In a similar analysis, stocks heterozygous for three different putative deletions of the *y1* locus were pollinated by plants known to be heterozygous for *l10* or *l*-4920*. Both of these luteus seedling mutants are within two crossover units of *y1*. Therefore, a deletion involving the *y1* locus might also delete one or the other, or both, of these seedling mutant loci. If a deletion included these loci, and if it was female transmissible, some of the progeny of this cross would be expected to exhibit the luteus phenotype in the seedling. For one putative deletion 5 plants were crossed by *l*-4920* heterozygotes and one plant by a *l10* heterozygote. No luteus seedlings were observed in the progeny of these crosses. For the second and third putative deletions, 3 plants and 1 plant respectively were crossed by *l*-4920* heterozygotes. Again, no luteus seedlings were observed in the progeny of these crosses. Thus we have not yet identified any *Mu*-induced deletions involving the *y1* locus.

To further test the possibility of obtaining deletions for the *y1* locus, we will attempt to generate *y1* deletions from plants heterozygous for 2 reciprocal translocations involving chromosome 6 but with breakpoints bracketing the *y1* locus. If such a deletion is transmissible, some of the meiotic products of the heterozygous reciprocal translocation plants will be missing the region between the 2 chromosome 6 breakpoints (i.e., the *y1* region). These plants will be pollinated by homozygous *y1* testers. If the eggs carrying the deletion are viable, white kernels should be observed in these crosses. If this region is essential for the viability of the female gametophyte, no white seeds will be observed. This winter, T6-9(6270) (6L.19), T6-9(6019) (6L.27) and T6-9(8906) (6L.27) will be crossed with T6-9e (6L.18). The latter translocation is known to have its breakpoint proximal to *y1*. T6-9(6270) has a breakpoint very close to T6-9e and indeed may not be distal to *y1*, but if it is, it will generate the shortest deletion possible involving this locus. T6-9(6019) and T6-9(8906) should have breakpoints that are distal to *y1*.

The inability (to date) to find deletions for the *y1* locus is intriguing since many of the apparent mutants we have recovered at the *yg2* and *Bf1* loci are deletions. If the gene product of the *y1* locus is a regulatory element in the beta-carotene biosynthetic pathway, it could be that deletions that include this locus are not viable. This is supported by the fact that no white endosperm albino mutant alleles of the *y1* locus have ever been found, in contrast to all other mutants (with the exception of *y9*) in the carotenoid biosynthetic pathway. If the gene product of *Y1* is involved in regulating not only the carotenoid pathway, but other pathways as well that are essential for viability, null alleles at this locus would not result in viable kernels or seedlings.

Brent Buckner, Philip S. Stinard
and Donald S. Robertson

Mutator 1:1 ratios: An alternative explanation?

In MNL 61:11-13, we reported on the occurrence of 1:1 (mutable: nonmutable kernels) ears in crosses of Mutator-induced mutable *a1* mutants (*a1-Mum*). These ratios were found in about half the crosses of plants from stable *a1-Mum* kernels to plants from non-*a1-Mum* kernels segregating on the same 1:1 outcross ear, from which the stable kernels came. It was proposed that the 1:1 ratios were due to the presence, in the heterozygous condition, of a regulator of somatic mutability in half of the plants from non-*a1-Mum* kernels. Further evidence that a regulator was involved was obtained when plants from mutable kernels found on 1:1 ears were outcrossed to a standard *a1 sh2* stock. These crosses in turn produced 1:1 ears.

A possible alternative explanation for these ratios would be the segregation of a second aleurone gene, such as *r* or *c*. For example, if the original 1:1 ear resulted from the cross of a plant that had the genotype *R/r; a1-Mum/a1 sh2* × *r/r; a1 sh2/a1 sh2*, the crosses as outlined above would have given the observed results. In such a situation, the stable seeds from 1:1 ears will be *r/r; a1-Mum/a1 sh2*, and one half of the non-*a1-Mum* kernels would be *R/r; a1 sh2/a1 sh2* and one half would be *r/r; a1 sh2/a1 sh2*. Plants from the *R/r; a1 sh2/a1 sh2* kernels crossed with plants from the stable (*r/r; a1-Mum a1-Mum*) kernels will give ears with 1:1 ratios due to the segregation of the *r* alleles. Also, a plant from a mutable kernel (*R/r; a1-Mum/a1 sh2*) from a 1:1 ear when crossed to a *r/r, a1 sh2/a1 sh2* tester would give a 1:1 ratio.

Unfortunately, such an explanation for these ratios is a possibility in some of our stocks and must be ruled out before we can say conclusively that we are dealing with a regulator of somatic mutability (an autonomous element). Our *a1 sh2* tester stocks were all descended from *a1 sh2* kernels isolated from self pollinated purple aleurone/*a1 sh2* F1 plants, which segregated in 3:1 ratios for purple:*a1 sh2* kernels. However, later tests indicated that some, *but not all*, of our *a1 sh2* stocks used before 1985 were segregating for a second aleurone gene. How this second gene gained entry into our stock is unknown. Mutation is a possible, but unlikely, source. A more likely source would be contamination from a generous border of Illini Extra Sweet sweetcorn that surrounds our experimental plots each year to protect them from the ravages of a large raccoon population, which invades our field from an adjacent wildlife preserve.

We have evidence, however, which indicates that some 1:1 ratios are real. The 1:1 ratios reported in Table 1 of last year's article could not be the result of the presence of a second recessive aleurone gene. Plants from stable kernels of the 85-86 crosses were crossed to homozygous purple aleurone stocks, and the plants from the resulting kernels (50 from each cross planted) were self pollinated. In all cases, there was no evidence of a second gene segregating. The outcrosses of plants from mutable kernels (i.e., the 1986 crosses, mistakenly indicated as 1985 in the heading of Table 1, MNL 61:11-13, 1987) to plants from standard *a1 sh2* were made to a newly isolated *a1 sh2* stock, eliminating the possibility of a second recessive aleurone gene being present. Thus these 1:1 ratios are real. Further tests have also been made with some of the crosses reported in Table 2 from last year's article. A plant from a stable kernel, which was found on the ear from which the kernels for families 2274 and 2295 came, was outcrossed to a homozygous purple aleurone stock. One half of the plants from approximately 100 kernels of these outcrossed ears, when self pollinated, segregated for *a1* and *r*, while the remaining plants only segregated for *a1*. Similar outcrosses of plants from two stable kernels from the ear providing kernels for families 2275 and 2300 were made. All plants of one of these outcrosses segregated for only *a1* when self pollinated, while for the second outcross half of the plants segregated for *a1* and *r* and half segregated only for *a1*. Thus none of the 3 plants, which were derived from stable kernels, tested to be *r r* as required for stable kernels if the segregation of *r* is responsible for the 1:1 ratio. In addition, mutable kernels from 1:1 ears of families 2274 (2295), and 2275 (2300), when crossed to *a1 sh2* tester, frequently produced 1:1 ratios. The *a1 sh2* stocks used in these crosses were newly isolated from purple aleurone crosses and thus would not be carrying another recessive aleurone gene.

The above results suggest that at least some, and perhaps all, of the 1:1 ratios reported last year were real and not due to the segregation of a second aleurone color gene. However, to confirm this conclusion, stable kernels from the 1:1 ears of families 86-2274, 86-2275, 86-2280, 86-2281 and 86-2282, listed in Table 2 of the 1987 report, have been planted in the winter nursery and will be

crossed with *a2, c1, c2* and *r* testers to establish, with certainty, the aleurone genotype of each of these lines.

We are also testing the Illini Extra Sweet line with the aleurone testers to establish its aleurone genotype to determine if it was the possible source of the second recessive aleurone gene found in some of our older *a1 sh2* stocks.

Donald S. Robertson

Additional evidence that *Mu* Mutator activity can occur during the development of the embryo

In the following article, evidence is presented suggesting that *Mu* Mutator activity occurs in the male gametophyte. In one of our 1986 reports (MNL 60:12-14), evidence was presented that Mutator activity could occur in early developmental stages of the embryo. The present report will provide some additional support for *Mu* activity occurring in the development of the embryo.

In most of our tests for Mutator activity, the putative *Mu* plant is self-pollinated and outcrossed to a standard (non-*Mu*) plant that has been selfed on the second ear. The kernels from both self-pollinated ears are seedling tested to ensure that no seedling mutants are segregating in either parent. In spite of these precautions to ensure that both the parents are not segregating for a mutant, occasionally in a *Mu* by standard cross about half of the selfed ears segregate for a seedling mutant of a given phenotype. This would be expected if one of the parents carried a recessive seedling mutant. However, sometimes the test of the selfed ear from both parents indicates that this is not the case (i.e., neither parental selfed ear segregates for a mutant). In the early stages of our study of the Mutator system, we assumed such anomalous results were due to bookkeeping errors or numbering errors in the field. However, after observing similar situations over a period of several years, we began to consider other possible explanations. One possibility is that during the development of the kernel that gave rise to the *Mu* parent a mutational event occurred in the cell lineage of the tassel after it had separated from the cell lineage of the ear. An alternative explanation is that a mutation occurred in the cell lineage of the ear of the standard parent after it had separated from the cell lineage of the tassel. However, because mutations are extremely rare in standard stocks, while a high mutation frequency is characteristic of Mutator plants, it is much more likely that the mutation occurred in the Mutator parent. How can the hypothesis of a mutation in only the tassel lineage be tested? If this hypothesis is correct, the self-pollinated ear of the Mutator parent would not segregate for the mutant, but at least some of the kernels on this ear would be expected to be heterozygous for the same mutant as that observed in the self-pollinated ears of the *Mu* by standard cross. If the mutation occurred early in the tassel cell lineage, all tassel tissue would be heterozygous for the mutant and thus half of the kernels on the selfed ear of the Mutator parent would be heterozygous. If, however, the mutation event was somewhat later in the tassel cell lineage, then not all of the tassel could be made up of cells heterozygous for the mutant and thus half of the kernels on the selfed ear of the Mutator parent would be heterozygous. If, however, the mutation event was somewhat later in the tassel cell lineage, then not all of the tassel could be made up of cells heterozygous for the mutant. In such a situation, less than half of the kernels on the selfed ear of the *Mu* parent would be heterozygous for the mutant. Last spring when it was time to get material out for planting, we searched for past Mutator tests that had half, or a significant number, of the outcross progeny segregating for a given mutant phenotype not seen in the progeny of selfed ears of either parent. Unfortunately, in most cases of this type the selfed ear of the *Mu* parent either did not have enough kernels left for a test, or ears had been discarded (a practice necessary to provide storage space). We did, however, find one instance suitable for analysis. The outcross progeny of *Mu* parent 84-2015-1 produced 47 plants, of which 14 (30%) segregated for yellow-green seedlings when self-pollinated. The other 33 plants produced selfed ears that did not segregate for this trait. The self-pollinated ears of the *Mu* and standard parents of this cross did not segregate for this mutant. In 1986, kernels from the selfed ear of the *Mu* parent were sown and the plants self-pollinated. Although the selfed *Mu* parent ear did not segregate for yellow-green seedlings, selfed plants from the kernels produced on this selfed ear did segregate for yellow-green seedlings. Of the 83 plants tested, 24 (29%) segregated for this mutant. This is about the same percentage as that observed in the selfed outcross progeny. When these two results are compared by a contingency chi-square test, they are found not to be significantly different. Thus, results expected if a mutation had occurred solely in the tassel cell lineage are realized. It could be suggested, however, that there is a female gametophytic lethal tightly linked to the yellow-green locus. However, if that were the situation, why do less than half of the outcross and selfed progeny carry the yellow-green mutant? The fact that the frequencies of heterozygous mutant plants are closer to a 2:1 or 3:1 ratio than a 1:1 ratio supports the suggestion that a mutation giving rise to a tassel sector is more reasonable. Another alternative explanation might be that the tightly linked gametophytic lethal mutant is not only completely lethal in the female gametophytes but is also partially lethal in the male gametophytes. Thus no mutants will be seen in the self-pollinated *Mu* progeny but the mutant allele would be transmitted to less than half of the outcross progeny.

Both of these hypotheses are unlikely because the first ear of the *Mu* plant, which was outcrossed, had a normal seed set and none of the self-pollinated plants from this outcross progeny segregated for the yellow-green mutant. The fact that the *Mu* parent had a normal seed set rules out the possibility of a tightly linked female lethal mutant functioning in the gametophyte or earlier in the development. If that were the case something less than a normal seed set would be expected. The fact that the progeny of the first ear also did not segregate for the yellow-green mutant confirms that the mutation occurred solely in the tassel.

We will be analyzing other transmission patterns of this type for what they can tell us about *Mu* activity in the developing sporophyte.

Donald S. Robertson

Further evidence for Mutator activity in the male gametophyte

In the 1985 and 1987 News Letters (MNL 59:14-15,

1985; 61:10-11, 1987), we presented evidence suggesting that Mutator (*Mu*) was producing mutations in the male gametophyte. This evidence came from reciprocal crosses of homozygous *Y1 Wx Gl1 Mu* and *y1 y1 wx wx gl1 gl1* non-*Mu* plants. In such crosses, significantly more white kernels were found when the *Mu* parent was crossed as a male than when *Mu* plants were crossed as females. In addition, a significant portion of the kernels from the crosses of *Mu* plants as males were discordant (i.e., endosperm *y1 y1 y1*/embryo *Y1 y1*) whereas when *Mu* plants were the female parent, few if any discordant kernels were observed. The discordant kernels were suggested to be the result of a mutation in the male gametophyte in which one sperm carried the mutant *y1-Mu* allele while the other sperm carried a nonmutated *Y1* allele. The discordant seeds resulted when the *y1-Mu* sperm fertilized the polar nuclei and the *Y1* sperm the egg. Because of the manner in which the female gametophyte develops, this class of discordant seeds is expected rarely if at all as a result of mutations occurring in the gametophyte.

If the discordant seeds are due to mutations in the male gametophyte, there should be a second class of discordant kernels when *Mu* is used as a male parent (i.e., *Y1 y1 y1* endosperm: *y1 y1-Mu* embryo). Unfortunately this class of discordant kernels, if it occurs, cannot be recognized phenotypically. However, such discordant kernels should give rise to plants which, when self pollinated, will have homozygous white endosperm ears. Thus to determine if such kernels occur, large numbers of yellow kernels from the *y1 y1 wx wx gl1 gl1* × *Y1 Y1 Wx Wx Gl1 Gl1 Mu* crosses must be sown, grown to maturity and the ears scored. Last summer a one acre isolation plot was sown with such kernels and the resulting plants allowed to open-pollinate. In this field half the pollen would carry *Y1* and half *y1*. Thus plants from discordant yellow kernels (i.e., with the *y1 y1-Mu* embryos) would have a 1:1 ratio for yellow versus white kernels. Most plants, which are expected not to be discordant, would have a 3:1 ratio of yellow:white kernels. Ears were scored on 37,975 plants and those that differed markedly from the expected 3:1 ratio were harvested and kernel counts made (Table 1). Half of the putative 1:1 ears were in reality not 1:1 but had on average 37% white kernels. These ears had a semisterile seed-set pattern, indicating that perhaps an ovule lethal mutant was induced on chromosome 6 approximately 28 map units removed from the *y1* locus. Six ears had 1:1 ratios but had semi-sterile seed set patterns. The plants bearing these ears could have come from discordant kernels which carried some unlinked factor responsible for 50% ovule abortion (e.g., translocation), or they may have had an ovule lethal tightly linked to *Y1*. Only one ear had the normal seed set and 50% white kernels expected from a yellow discordant kernel. If the plant that bore this latter ear came from a yellow discordant kernel, the frequency of this class of discordant kernels was 2.63×10^{-5}. If all of the 7 ears listed in Table 1 were borne on plants from discordant kernels, the frequency of such kernels was 18.43×10^{-5}. In last year's News Letter report (MNL 61:10-11), 34.29% of the white endosperm mutant kernels were found to be discordant. If this is the true frequency of this class of discordant kernels among white mutant kernels, 46 of the 135 kernels found in the test summarized in last year's Table 1 would be expected to be discordant. Thus the frequency of white-endosperm discordant seeds in the 275,623 kernel population scored (1987, Table 1) would be 16.69×10^{-5}. Because the yellow kernels tested this last summer came from the same population analyzed in the 1987 report (Table 1), it is possible to compare the frequencies of the two classes of the discordant seeds. If only the 1:1 ear with normal seed set is from a discordant kernel, the frequency of the two classes of discordant seeds would be significantly different at the 1% level. However, if all 7 of the 1:1 ears are from discordant kernels there is no significant difference in the frequency of the two kinds of discordant kernels. These results tentatively support the hypothesis of *Mu* activity in the male gametophyte. However, the 1:1 ears, especially the semisterile ears, could be the result of other phenomena such as a linked ovule lethal. Fortunately, it is possible to test for the basis for the 1:1 ears. As reported last year 71.94% of the *Mu*-induced *y1* mutants have pastel seedlings. If self-pollinated plants from half of the yellow kernels from the 1:1 ears segregate for white kernels, all of which produce pastel seedlings, it would be reasonable to assume that the plants with 1:1 ears came from discordant kernels with embryos of the genotype *y1 y1-Mu*. If the 1:1 ratio is due to an ovule abortion gene linked to *Y1* most, if not all, of the kernels from these ears should give rise to plants with normal seed set. Since these ears were open-pollinated, a few kernels would be expected to have been produced by pollen from the same plant. Some of this pollen should carry the ovule abortion gene linked to *Y1*.

It should be noted that *Mu*-induced mutants in the zygote would give the same results as would mutations in the male gametophyte, and the result considered in this report and the previous ones cannot distinguish between these possibilities.

Table 1. Kernel counts of ears from putative discordant seeds (*Y1 y1 y1* endosperm; *y1 y1* embryo).

Ear number	Seed[a] set	Number of yellow kernels	Number of white kernels	Total kernels	% white kernels	χ^2 significance 1:1	χ^2 significance 3:1
1	n	141	159	300	53.00		**
2	♣	123	122	245	49.80		**
3	♣	104	102	206	49.52		**
4	♣	78	72	150	48.00		**
5	♣	22	18	40	45.00		**
6	♣	41	27	68	39.71		**
7	♣	71	51	122	41.80		**
Total		580	551	1131	48.72		**
8	♣-	224	153	377	40.58	**	**
9	♣	108	54	162	33.33	**	**
10	♣	113	61	174	35.06	**	**
11	♣	112	66	178	37.71	**	**
12	♣	67	33	100	33.00	**	**
13	♣	80	46	126	36.51	**	**
14	♣	60	38	98	38.78	*	**
Total		764	451	1,215	37.12	**	**
29		270	7	277	2.53	**	**

[a] n = normal seed set
♣ = semisterile seed set
♣- = slightly less than semisterile seed set

* - significant difference at the 5% level.

** - significant difference at the 1% level.

Donald S. Robertson and Philip Stinard

Evidence for somatic crossing over in chromosome six

In the previous article, we described an open-pollinated isolation plot in which nearly all the plants were heterozygous for *Y1 y1*. One ear out of a population of 37,957 consisted of two sectors (Fig. 1). About ¾ of the ear had only yellow kernels, while the balance of the ear had a 1:1 ratio of yellow to white kernels (see kernel count, Figure 1). Such a sector could be produced if a somatic crossover had occurred in one of the primordial cells giving rise to

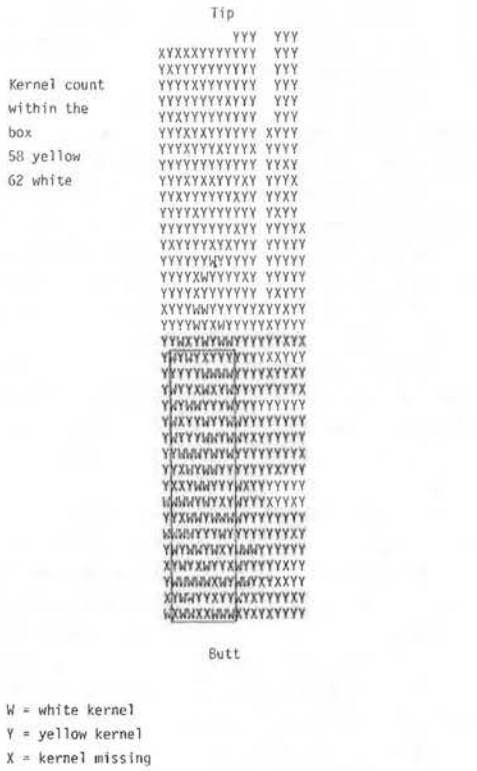


Figure 1. Ear map showing twin sectors resulting from a putative somatic crossover.

the ear, followed by mitotic segregation of the crossover chromatids. If both *y1* chromatids go to the same daughter cell, one cell lineage would be *y1 y1* (giving rise to the 1:1 sector upon open pollination), and the other cell lineage would be *Y1 Y1* (giving rise to the yellow kernel sector).

Philip Stinard and Donald S. Robertson

Reversion frequencies of *Mu*-induced waxy alleles

At last count we had 13 independent *Mu*-induced mutable waxy alleles (*wx-Mum*). For the last few years we have been developing *wx-Mum* stocks that can be used to test the frequency of germinal revertants. To do this we have produced stocks of *wx-Mum1, wx-Mum2* and *wx-Mum4* that are homozygous for *gl1* and *y1* as well (contamination markers). This past summer we planted large amounts of these multiply marked stocks in an isolation plot. The *wx-Mum* lines were used as detasseled females and *y1 y1 wx wx gl1 gl1* plants were used as the pollen source. Unfortunately a second isolation plot, in which the plants were heterozygous for *y1, wx,* and *gl1*, was planted too close to the *wx-Mum* plot for complete isolation (results from this block are described in our previous article on *Mu* activity in the male gametophyte.) By the time it was discovered that the two plots were insufficiently separated, they both had been planted. The *wx-Mum* block was planted three weeks before the other block and thus, if we were lucky, the *wx-Mum* block would have completed pollinating before the other block shed. Since contamination from the *wx-Mum* block would not affect the results of the other block, it was decided to proceed under these less than satisfactory conditions. The degree of contamination, if any, in the *wx-Mum* block could be determined by the frequency of yellow waxy kernels occurring upon *wx-Mum* ears.

For the *wx-Mum1* allele, 40,485 kernels were scored. Nine starchy kernels and 2 intermediate staining kernels (i.e., stained blue but not as rapidly as expected for a *Wx* kernel) were found. If the intermediate kernels are counted as revertants, the frequency is 2.72×10^{-4}. If the intermediates are excluded, the frequency of revertants would be 2.22×10^{-4}. However, 2 yellow *wx* kernels were observed in this population, which probably were the result of contamination. In the block from which the contaminating pollen came, *Y1 wx* pollen was produced in the same frequency as *y1 Wx* pollen. Thus 2 of the 9 revertant kernels could be expected to result from contamination. If indeed there were only 2 contaminants among the starchy seeds, the revertant frequency would be 1.73×10^{-4}. A chi-square test to determine if the 9 *Wx* kernels would all be expected to be contaminants, based on observation of the 2 known contaminants, gave a value of $X^2 = 4.4545$, which is significant at the 5% level. Thus some of the starchy kernels are probably germinal revertants, but the exact frequency is yet to be determined.

The *wx-Mum2* allele is quite different from the *wx-Mum1* allele both in its phenotype and reversion frequency. The somatic mutability pattern of *wx-Mum1* is typical of most *Mu*-induced mutants (i.e., it reverts quite late in the development of a given cell lineage, resulting in revertant sectors of one or just a few cells). The revertant sectors of *wx-Mum2* are quite variable in size, ranging from one cell to very large sectors easily visible with the naked eye. This is one of two mutable Mutator mutants with consistent large revertant patterns that we have observed; the other is *vp1-Mum2*. Dr. Vicki Chandler has seen a similar pattern in a couple of her *Mu*-induced *B-Peru* mutants.

The *wx-Mum2* allele also differs from *wx-Mum1* in that it has a much higher germinal reversion frequency. Out of a population of 642,933 kernels screened, 521 starch kernels were observed, giving a reversion frequency of 8.10×10^{-4}. The *wx-Mum2* allele also differs from *wx-Mum1* in that, with the latter allele, no sectors of revertant kernels were observed on 133 ears scored. However, from the 1,899 ears scored of *wx-Mum2*, 21 had sectors of revertant seeds, indicating that some revertant events were premeiotic. There were 13 2-seeded, 3 3-seeded, 3 4-seeded, 1 5-seeded and 1 11-seeded sectors. These counts just represent the starchy revertant kernels observed in these sectors. The starchy kernels, of course, were interspersed with waxy non-revertant seeds. Each sector is the result of one reversion event and thus each was only counted as a single kernel in the above total of 521.

In the *wx-Mum2* material, only 4 yellow waxy contaminant kernels were found (frequency = 6.22×10^{-6}). If one assumes that 4 of the white starchy kernels were contaminants rather than revertants, the reversion frequency would be 8.04×10^{-4}.

All *wx-Mum* kernels in this isolation plot came from crossing homozygous *wx-Mum y1 gl1* plants. All kernels were screened before planting to be sure they were waxy, and all seedlings were checked for glossy in the field. These points are emphasized because in the *wx-Mum2* material, 13 ears were observed that were heterozygous for starchy seeds. The frequency of such ears was 6.80×10^{-3} (13/1,912). These ears are expected if the kernels that produced these plants resulted from the functioning of a pollen grain in which a reversion had occurred in one of

the sperm but not the other. A kernel in which the egg was fertilized by a sperm with the revertant allele would have a waxy endosperm because the sperm with the non-revertant allele would have had to fertilize the polar fusion nucleus. These heterozygous ears were expected because white starchy kernels (presumably kernels with revertant alleles) were observed on some of the ears used as the source of kernels for the isolation block. For this reason, the seeds planted were carefully screened to be sure no starchy kernels were planted. Seventeen plants from starchy (presumed revertant) kernels, from the ears providing the kernels for the isolation plot, were self-pollinated this past summer. Sixteen of these selfed ears segregated for starchy kernels, as expected, and one was homozygous waxy. The kernel that gave rise to this latter plant was probably the result of a fertilization involving a pollen grain with one *wx-Mum2* sperm, which fertilized the egg, and one revertant sperm, which fertilized the polar fusion nucleus. This is the fertilization pattern reciprocal to that suggested to be responsible for the heterozygous starchy ears found in the isolation plot.

The *wx-Mum4* block yielded 2 starchy revertant seeds but 2 yellow waxy contaminant seeds were also found; thus, nothing can be said at this time about the reversion frequency of this allele.

No starchy revertant or yellow waxy contaminant kernels were found on the ears of the *y1 y1 wx wx gl1 gl1* plants of the pollen rows, which also served as controls for this experiment. There were 171,277 kernels in these rows and since each kernel is the product of 2 *y1 wx gl1* gametes this number of kernels is equivalent to testing 342,554 gametes.

The reversion frequencies for both *wx-Mum1* and *wx-Mum2* are minimal values because it is known that there were kernels with different levels of somatic mutability on the ears that provided the kernels for this test. Many of these ears had some stable derivative kernels as well. Thus all plants would not be expected to have the same frequency of reversion and some, those derived from stable kernels, might possibly have no reversions. A few kernels from all ears used as a source of kernels for the isolation plot were scored for mutability and only ears segregating mutable kernels were used. However, it was not practical to score each kernel planted or all the ears harvested for levels of mutability.

Donald S. Robertson and Philip S. Stinard

Further observations on *Mu*-induced deletions in the short arm of chromosome 9

We have previously described some Mutator-induced deletions in the short arm of chromosome 9 involving the *yg2* locus (Genetics 115:353, 1987). In order to isolate these deletions, *Yg2 Yg2 Mu* females were pollinated by *yg2 yg2* males. The yellow-green offspring recovered from this cross were pollinated by standard (*Yg2 Yg2*) pollen. To determine whether a deletion of the *yg2* locus was involved, plants from the standard cross [*Yg2 yg2-Mu(del)*] were reciprocally crossed to *yg2* testers. Isolates in which there was a deficiency of *yg2* transmission through the male, and either normal or reduced transmission of *yg2* through the female, were considered as being possible deletions. Further testing confirmed that deletions were indeed present in at least some of these isolates. Since a large control (non-*Mu*) population produced no *yg2* events, the *yg2* events occurring in our Mutator population are presumably *Mu*-induced.

McClintock (Genetics 29:478, 1944) has shown that some small deletions that include the *yg2* locus do not interfere with male transmission. If these deletions include *yg2* and a small region proximal to this locus (the *wd*, or white-deficiency deletion), then plants heterozygous for one of these deletions, when self-pollinated and seedling tested, will segregate for homozygous deficient seedlings, which have an albino phenotype. McClintock observed 2 types of ratios when she selfed heterozygous *wd* plants: 1) 3:1 ratios of green:albino seedlings, and 2) ratios greater than 3:1 of green:albino seedlings. The former are *wd* deletions with undiminished transmission through the egg and pollen, while the latter are *wd* deletions with reduced male transmission.

In 1986, plants heterozygous for 5 *Mu*-induced deletions, which had been shown by genetic tests to be the shortest of the 12 deletions published upon in our 1987 paper, were self-pollinated. One did not segregate for albino seedlings, and 4 did. Of those 4, one segregated for approximately 25% albinos and 2 segregated for less than 25% albinos.

Also in 1986, plants heterozygous for 110 different putative *yg2* mutant isolates were self-pollinated and the selfed ears seedling tested. These heterozygous plants were produced by outcrossing the original yellow-green isolates as males to standard (*Yg2 Yg2*) lines. By using crosses in this direction, all large deletions were selected against, and only small male-transmissible *wd* deletions, *yg2*-only deletions, and mutations of the *yg2* locus would be transmitted. The following patterns of segregation would be expected in the selfed ears of the isolates: 1) If an isolate segregated albino seedlings in some selfs, and yellow-green seedlings in the other selfs, then a male-transmissible *wd* deletion is present. 2) If some selfs of an isolate segregate for yellow-green mutable seedlings, and the other selfs segregate for yellow-green stable seedlings, then a mutable Mutator-induced *yg2* mutation (*yg2-Mum*) is present. 3) If all selfs of an isolate segregate for stable yellow-green seedlings, then either a *yg2*-only deletion, a stable *yg2* mutation, or a non-male-transmissible deletion is present. 4) If half of the selfs of an isolate produce only green seedlings, and the other half segregate for yellow-green seedlings, then the isolate is not a *yg2* mutant at all, and must have been misclassified during the original screening. When the data from this selfing regimen are combined with the data reported in our 1987 Genetics paper, the following results are obtained: Of the 110 isolates examined, 64 proved to involve the *yg2* locus. The remaining 46 were either not allelic to *yg2*, or the data were insufficient to determine allelism. Of the 64 proven *yg2* events, 14 (21.9%) were male-transmissible *wd* deletions; of these 14, 3 were fully male transmissible and 11 were partially male transmissible. Seven (10.9%) of the *yg2* events were larger non-male transmissible deletions including at least the *wd* locus. Twenty events (31.3%) were Mutator-induced mutable *yg2* mutations. The remaining 23 events (35.9%) fell under pattern 3 listed above; there was insufficient evidence to determine whether these events were *yg2*-only deletions, stable *yg2* mutations, or non-male transmissible deletions.

Thus, at least one third (32.8%) of the events induced at the *yg2* locus in our Mutator stocks are *wd* deletions. In

the original Mutator population that we screened, at least 64 good yellow-green seedlings were isolated out of a total of 779,213 seedlings, giving a frequency of 8.21×10^{-4}. The results reported here indicate that the frequency of *wd* deletions in this population is 2.70×10^{-5}, and the frequency of *Mu*-induced mutable mutations is 2.57×10^{-5}.

In the summer of 1987, kernels from 6 different *wd-Mu* deletion isolates were sown, and all possible intercrosses were made to test for "allelism" of the deletions. If the albino *wd* phenotype in all instances is due to the presence of a small deletion involving the chromosomal region just proximal to the *yg2* locus, all of these allelism tests should be positive. This indeed turned out to be the case. The same *wd-Mu* plants used in the allelism tests were crossed as males to *yg2* tester plants, and all crosses except one produced green and yellow-green seedlings in ratios very close to 1:1. The *wd-Mu3* line had outcrosses that were deficient in yellow-green seedlings. Out of a total of 1,180 seedlings scored, only 290 (24.6%) were yellow-green. Thus, the *wd-Mu3* deletion must be slightly larger than the other 6 *wd-Mu* deletions.

To date, our studies of *Mu*-induced events involving the *yg2* locus have demonstrated that a substantial number of these events are deletions. The deletions are not uniform in size, but can range from quite small ones, which are fully male and female transmissible, to larger ones, which have reduced or no male transmission but are fully female transmissible, to the largest class, which shows no male transmission and reduced female transmission. These results establish that Mutator can induce deletions in this region and that many different sites of breakage appear to be involved in generating these deletions. Other reports from our laboratory in this News Letter establish that Mutator can apparently induce deletions in other regions of the maize genome as well. Thus, the production of deletions seems to be a frequent and important aspect of Mutator activity.

Donald S. Robertson and Philip S. Stinard

Mu-induced deletions involving the *Bf1* locus on the long arm of chromosome 9

In 1986, we reported preliminary evidence suggesting that deletions had been induced by Mutator that include the *Bf1* locus (MNL 60:11-12). The original isolates carrying the putative deletions were of the genotype *Bf1/Bf1-Mu(del)*. All isolates were crossed by standard lines (+/+). In our 1985-86 winter nursery, kernels from 9 of these crosses of the putative deletion isolates by standard were planted, and the resulting plants reciprocally crossed with homozygous *Bf1* testers; the progeny of these reciprocal crosses were seedling tested and scored for *Bf1* (Table 1). Seven of the isolates had the pattern of segregation expected if a deletion were present (i.e., a deficiency of *Bf1* transmission in male outcrosses of putative deletion heterozygotes to *Bf1* testers). Isolates 044-9 and 045-7 showed uncertain indication of a deletion being present; isolate 044-9 showed some reduced female transmission of *Bf1* but normal male transmission, and isolate 045-7 did not produce a statistically significant sample. Two isolates showed reduced transmission of *Bf1* through both the male and female.

In our 1986 summer nursery, reciprocal crosses to *Bf1* testers were made of 19 additional putative deletion

Table 1. Classification for *Bf* in 1985-86 reciprocal crosses of *Bf1-Mu(del)*/+ X *Bf1/Bf1*.

Isolate number	Pollen sterility	Putative deletion as female: +	Bf	XBf	Putative deletion as male: +	Bf	XBf
044-4	s(pf)	48	29	37.7*	48	1	2.0**
044-9	n	83	33	28.4**	16	15	48.4
045-3	n15	24	14	36.8	59	20	25.3**
045-7	n-	14	20	58.8	23	12	34.3
046-6	s(hpf)	76	65	46.1	116	43	27.0**
050-8	n(1&s)	144	71	33.0**	251	47	15.8**
544-5	s(pf)	71	69	49.3	111	49	30.6**
547-6	n	52	68	56.7	94	46	32.9**
548-1	n-	35	39	52.7	63	28	30.8**

Symbols: * chi-square for 1:1 ratio significant at p=.05
** chi-square for 1:1 ratio significant at p=.01

Pollen sterility: n, normal pollen; n-, normal with a few abortive pollen grains; n15, mostly normal, with 15% abortive grains; n(1&s), all pollen grains filled, but half smaller than normal; s(pf), semisterile, with partially filled abortive pollen grains; s(hpf), semisterile, half of the abortive pollen grains are partially filled, half are empty.

Table 2. Classification for *Bf* in 1986 reciprocal crosses of *Bf1-Mu(del)*/+ X *Bf1/Bf1*.

Isolate number	Pollen sterility	Putative deletion as female: +	Bf	XBf	Putative deletion as male: +	Bf	XBf
044-6	n-	160	121	43.1*	221	143	39.3**
044-9	n	73	73	50.0	88	51	36.7**
045-6	n-	105	111	51.4	157	94	37.5**
045-8	s	26	28	51.9	79	53	40.2**
046-1	n-	58	45	43.7	79	9	10.2**
046-5	n	58	51	46.8	58	32	35.6**
047-7	n	25	28	52.8	24	4	14.3**
047-10	n-	23	20	46.5	95	38	28.6**
048-3	n	59	86	59.3*	96	38	28.4**
544-9	s	24	17	41.5	48	16	25.0**
545-5	s-	48	46	48.9	85	40	32.0**
546-5	n	42	37	46.8	80	31	27.9**
547-5	n	80	75	48.4	139	63	31.2**
549-5	n-	52	46	46.9	58	37	38.9*

Symbols: * chi-square for 1:1 ratio significant at p=.05
** chi-square for 1:1 ratio significant at p=.01

Pollen sterility: n, normal pollen; n-, normal with a few abortive pollen grains; s, semisterile; s-, slightly less than 50% abortive pollen grains.

isolates that had been crossed by standard. Isolate 044-9, which gave ambiguous results in Table 1, was retested as well. Fourteen of these showed reduced male transmission of *Bf1* indicative of the presence of a deletion (Table 2); of these 14, one showed reduced female transmission as well. Among this set of isolates, as well as those presented in Table 1, there did not seem to be a consistent association of pollen sterility with reduced male transmission. Thus, pollen sterility does not seem to be invariably associated with deletions involving *Bf1*.

In 1986, kernels from 8 crosses of putative deletion isolates by standard were planted and the resulting plants pollinated by TB-9Lc pollen. Each plant was also outcrossed to a *Bf1* tester to determine which ones carried the putative deletions. Approximately 50 kernels from each ear crossed by TB were sown in the seedling bench and scored for *Bf1*. Some of the TB crosses of plants heterozygous for a putative deletion segregated for a few *Bf* seedlings, and some did not (Table 3). Thus, if deletions are indeed present in all of these stocks, it seems that some deletions are viable in the hemizygous condition

Table 3. Summary of seedling tests of ears from the cross of *Bf1-Mu(del)*/+ by TB-9Lc.

Isolate number	No. of TB crosses giving no Bf seedlings	No. of TB crosses giving a few Bf seedlings
045-3	1	0
046-6	0	1
050-8	1	0
544-5	1	2
547-6	0	4
547-9	3	0
548-1	2	0

(i.e., those in which *Bf* seedlings were found), while others are not (i.e., those in which no *Bf* seedlings were found). However, more seedlings of the latter class should be tested before it can be said with certainty that these putative deletions will not survive in the hemizygous state.

Last summer, *Bf* seedlings from the TB crosses were transplanted into the field. Following are the putative deletions tested and, in parentheses, the number of plants observed for each: 046-6 (4), 544-5 (3), 547-6 (12), and 548-1 (5). In all cases, these plants had tassels that extruded few, if any, anthers. They also were later maturing, were reduced in height, and had narrow leaves. The latter 3 traits are characteristic of hypoploid TB-9Lc plants, but the lack of anther extrusion is not. It would seem that if deletions are indeed present, these deletions are missing loci necessary for anther extrusion. It was also of interest to note that all plants had green midribs. If deletions are present that include *Bf1*, they seemingly do not extend to the *bm4* locus, which is distal to *Bf1*. Thus, these would not seem to be terminal deletions. We are currently growing test crosses of our *Bf1* deletion lines by *bm4* to verify whether this is so.

Dr. Earl Patterson has recently located at least one male sterile mutant to the long arm of chromosome 9 by crossing with TB-9Lc. Crosses of this male sterile by our putative *Bf1* deletion stocks that have reduced male transmission will be made to determine whether this male sterile locus is uncovered by our deletions.

In summary, there is genetic evidence of Mutator having induced deletions which include the *Bf1* locus on the long arm of chromosome 9, some of which are lethal in the hemizygous condition, and at least some of which do not include the *bm4* locus and are thus not terminal. Those deletions tested to date also seem to involve a locus controlling anther extrusion.

Donald S. Robertson and Philip S. Stinard

BALTIMORE, MARYLAND
Carnegie Institution of Washington

Excision of *Spm* in tobacco

The ability to analyze the functions of the Suppressor-mutator (*Spm*) element by in vitro mutagenesis techniques requires that the element be able to function in a transformable cell system. It has been established that the Activator element transposes at a high frequency in tobacco cells when introduced by *Agrobacterium*-mediated transformation (Baker et al., Proc. Natl. Acad. Sci. 83:4844, 1986; EMBO J. 6:1547, 1987). We have obtained preliminary evidence that the *Spm* element also moves in tobacco cells.

To test the ability of the *Spm* element to both excise and *trans*-activate excision in tobacco, we constructed derivatives of the binary *Agrobacterium* vector pMON410, developed by Monsanto Co. (Rogers et al., Meth. Enzymol. 118:627, 1986). It carries 2 bacterial drug-resistance marker genes, both of which have been modified for expression in plant cells by the substitution of a plant promoter and the addition of a plant polyadenylation sequence. The plasmid is capable of conferring resistance to both kanamycin and hygromycin on transformed tobacco cells.

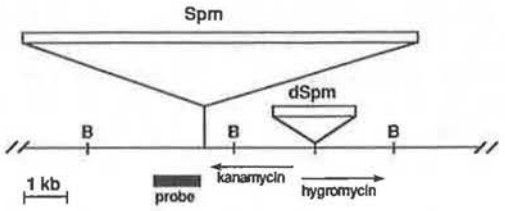


Figure 1. A diagrammatic representation of the region of the modified *Agrobacterium* Ti plasmid containing the drug-resistance markers and inserted *Spm* elements. As indicated in the text, the *dSpm* element is inserted between the promoter and the coding sequence of the hygromycin-resistance gene. The location of the sequence used as a probe is indicated by the filled block.

The pMON410 plasmid was modified by the introduction of a transposition-defective *Spm* (*dSpm*) element between the promoter and the hygromycin resistance marker (Fig. 1). The *dSpm* element was constructed by deleting the element's 2 internal *Eco*RV restriction fragments (Pereira et al., EMBO J. 5:835, 1986; Masson et al., Genetics 177:117, 1987). In addition, a complete *Spm* element was inserted at a unique cloning site near the plasmid's kanamycin-resistance marker (neomycin phosphotransferase II). The relevant parts of the plasmid's structure are shown in Figure 1.

The modified plasmid containing both the *dSpm* and *Spm* elements, and control plasmids lacking one or both *Spm* insertions, were introduced into tobacco cells by the leaf disc transformation technique (Rogers et al., 1986). Transformation was judged by the development of kanamycin resistant calli as a consequence of the introduction of the MPTII gene of the pMON410 plasmid. The ability of the plasmid-borne *Spm* elements to excise was assessed both by direct analysis of the T-DNA structure of transformed cells, and by the acquisition of hygromycin resistance by transformed cells.

Because the *dSpm* element is inserted at a site between its promoter and the hygromycin-resistance marker, the modified plasmid should not be capable of conferring hygromycin resistance on transformed cells. Control experiments with a plasmid containing the *dSpm* element but no *Spm* element established that the *dSpm* insertion disrupts expression of the hygromycin-resistance gene. If an *Spm* element residing on the plasmid can catalyze excision of the *dSpm* element, then transformed cells in which such an excision event has occurred will be resistant to both kanamycin and hygromycin.

Selection for hygromycin resistance has been applied both immediately after transformation and after the development of calli on kanamycin-containing medium. The number of hygromycin-resistant calli was the same for the experimental plasmid and the control plasmid lacking the *trans*-acting *Spm* element when hygromycin selection was applied immediately after transformation. Hygromycin resistance has been observed in calli initially selected for kanamycin resistance, but the structure of the resident T-DNA has not yet been analyzed.

However, the resident T-DNA has been analyzed in DNA extracted from several plants regenerated from kanamycin-resistant calli transformed with *Agrobacteria* carrying experimental and control plasmids. Different

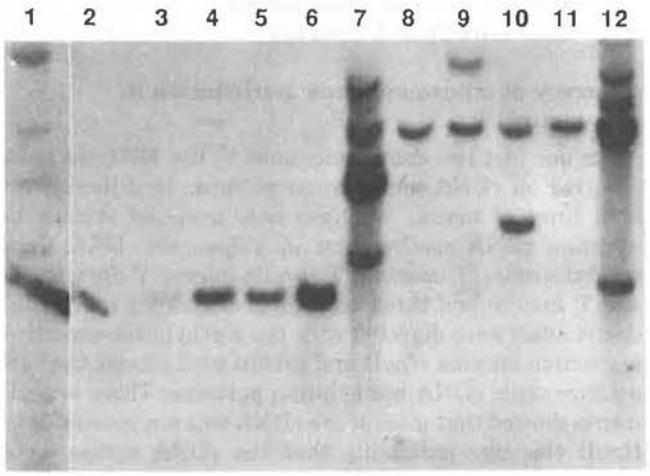


Figure 2. Blot hybridization analysis of DNA isolated from tobacco plants transformed with *dSpm*-and *Spm*-containing *Agrobacterium* plasmids. The DNAs were digested with *Bam*HI, fractionated on an agarose gel, transferred to Nytran membrane and hybridized to the ^{32}P-labeled Ti plasmid designated "probe" in Figure 1. Lanes 3 through 6 contain digests of DNA from plants transformed with a control plasmid lacking the *Spm* element. Lanes 7 through 12 contain digests of DNA from plants transformed with a plasmid containing the complete *Spm* element. Lane 2 contains a digest of DNA from an untransformed plant and lane 1 contains *Hind*III-digested phage lambda DNA.

probes of T-DNA structure were used to determine whether the intact element and the *dSpm* element were moving. The results of one such experiment are shown in Figure 2. The DNA was digested with *Bam*HI and probed with a fragment whose location is shown in Figure 1 to determine whether the complete *Spm* element was present on the plasmid.

All of the DNAs from calli transformed with a plasmid lacking the *Spm* (lanes 3-6) exhibited only a 3.5-kb *Bam*HI fragment with homology to the probe. This represents the undisrupted *Spm* insertion site. All of the DNAs from calli transformed with a plasmid containing the *Spm* element exhibit the expected 8.5kb fragment comprising part of the *Spm* element and the plasmid sequence flanking it in the original construct. In addition, the DNA samples in lanes 9 and 12 show a fragment with homology to the probe that is about 3.6kb in length. The DNA sample in lane 11 also showed a fragment of the same mobility after a longer exposure. The 3.6kb fragment is likely to represent an empty donor site from which the element has excised, since the inserted element was flanked by about 0.1kb of sequence from the maize *a* locus from which it was originally cloned.

Additional evidence that excision is occurring has been provided by comparable results of analyses of the *dSpm* insertion site (not shown). Not surprisingly, the same DNA samples that exhibit excision of the *Spm* element give evidence of *dSpm* excision. Taken together, these observations provide encouraging preliminary evidence that the *Spm* element is mobile in tobacco. Experiments are under way to clone *Spm*-containing sequences from tobacco to provide direct evidence for excision and transposition.

Patrick Masson, Kathy Toohey and Nina Fedoroff

Changes in the genetic activity of *Spm* are associated with changes in the methylation pattern of a GC-rich region at the element's 5′ end

The *Spm* element undergoes reversible changes in genetic activity. We have been investigating the genetic and molecular basis of such changes, using active and inactive derivatives of the *a-m2* allele of the *a* locus (McClintock, CIWYB 58:390, 1959; 60:421, 1961; and 61:429, 1962). The *a-m2-7991A1* allele has a full-length, autonomous *Spm* element inserted within the promoter region of the *a* gene. We have isolated derivatives of this allele, designated *a-m2-7991A1-i*, in which the element has become genetically inactive. The heritability of the inactive phase differs in the main stalk and the tillers. An inactive element remains largely inactive in the main stalk, although reactivation is sometimes observed more in the second ear than in the first ear. Reactivation of the element is frequently observed in tiller ears. An active element undergoes inactivation more frequently when transmitted through the male parent than through the female parent. These observations suggest that developmental signals in the plant affect the reversal of element activity. Moreover, an active *Spm* element activates an inactive element (McClintock, CIWYB 58:390, 1959; Masson et al., Genetics 177:117, 1987).

We have analyzed the patterns of methylation of the *Spm* element in plants with active and inactive elements. This was done by digesting genomic DNA with methylation-sensitive restriction enzymes having sites within the *Spm* element and the flanking *a* locus. Cleavage at these sites was then assessed by Southern blot hybridization analysis using both *a*-locus and *Spm* sequences as probes. Fourteen restriction sites within the element and 7 sites within the *a* locus were examined.

The results of these experiments can be summarized as follows: 1) the *a* locus sequences flanking the element are always unmethylated regardless of the activity of the *Spm* element; 2) the *Spm* element is always methylated regardless of its activity, with the exception of a region of about 600bp at the 5′ end of the element, which contains the transcription start site (Pereira et al., EMBO J. 5:835, 1986) and is extremely rich in G and C residues (subsequently referred to as the GC-rich region); 3) methylation of the GC-rich region varies with the element's activity. Within the GC-rich region, an active *Spm* element is hypomethylated, while an inactive element contains both methylated and unmethylated sites. Furthermore, the ratio of methylated to unmethylated sequences for a given restriction site varies among plants grown from kernels displaying an inactive *Spm* phenotype. An illustration of the differences in methylation pattern between the active and inactive elements is given here. Genomic DNA from plants heterozygous for the *a-m2-7991A1* allele with either an active or inactive *Spm* and the *a* allele was first digested with *Bam*H1, an enzyme which is not sensitive to methylation. The 3.8kb *Bam*H1 fragments were then purified from agarose gels following gel electrophoresis. The isolated fragments include the *Bam*H1 fragment containing the element's left end and *a* locus sequences adjacent to it (Figure 1). The purified fragments were then digested with *Ava*1, which is sensitive to methylation, fractionated on agarose gels and probed with an *a* locus fragment (see Figure 1 for location of probe). The 3.8kb

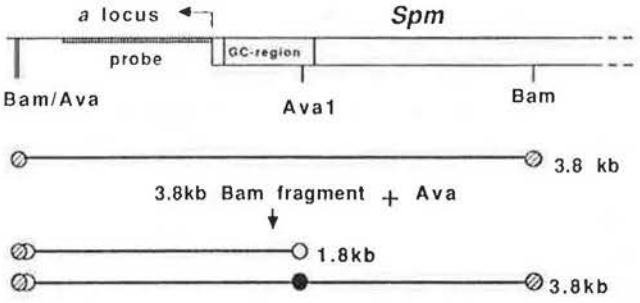


Figure 1. Diagram of the left end of the *Spm* element and adjacent *a* locus, showing relative positions of the *Bam*H1 and *Ava*I sites, and fragments expected after digestion of the 3.8kb *Bam*H1 fragment with *Ava*1. Open circles refer to unmethylated *Ava*1 sites and closed circles refer to methylated *Ava*1 sites.

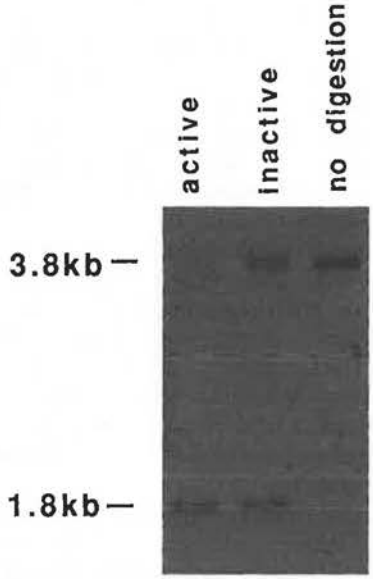


Figure 2. Autoradiogram of Southern hybridization blot after digestion of the 3.8kb *Bam*H1 fragment isolated from active and inactive elements with *Ava*1. The probe used in this experiment is shown in Figure 1.

*Bam*H1 fragment from the active *Spm* element is almost completely digested by *Ava*I, while the fragment from the inactive *Spm* element is only partially digested by *Ava*I (Figure 2). Near-complete digestion of the 3.8kb *Bam*H1 fragment by *Ava*I (and all other methylation-sensitive enzymes with sites within the GC-rich region) is observed in plants with either active elements, spontaneously reactivated elements, or elements reactivated in the presence of other active *Spm* elements (data not shown).

These genetic and molecular experiments suggest that 1) changes in the activity of the element have no effect on the *a* locus, which is always unmethylated; 2) methylation of most of the *Spm* element does not affect its activity; 3) changes in the activity of the element are associated with changes in methylation of a short region at the element's 5′ end, which has a very high GC content; 4) an active element must be largely unmethylated within the GC-rich region; and 5) reversal in phase of activity from inactive to active results in demethylation of the GC-rich region.

J. Banks and N. Fedoroff

BATON ROUGE, LOUISIANA
Louisiana State University

A survey of ribosomal gene methylation in *Tripsacum*

In our last two communications to the MNL we have reported on rDNA methylation patterns in different inbred lines of maize. We have now initiated studies to examine rDNA methylation in *Tripsacum*. DNA from several species (*T. australe, T. cundinamarce, T. dactyloides* and *T. laxum*) and three accessions of a single species (*T. dactyloides*) were digested with the methylation-sensitive restriction enzyme *Hpa*II and probed with pZmr1 to characterize their rDNA methylation patterns. These experiments showed that most of the rDNA was not accessible to *Hpa*II cleavage indicating that the rDNA arrays were completely methylated. However, a significant fraction of the rDNA (15-25%) was digested by *Hpa*II into fragments of repeat unit length (see Figure 1A; lanes 1-7). These *Hpa*II single digest patterns are similar to those of maize shown in our previous work. Some species of *Tripsacum* are heterogeneous for rDNA length. In Figure 1A (lanes 1 and 4) we can see that rDNA repeat units of two distinct lengths have been released by *Hpa*II digestion. In Figure 1B, we have mapped the distance from the conserved *Xba*I site (in the 18S gene) to the site of *Hpa*II cleavage. The 8.6 kbp band released in a *Hpa*II/*Xba*I double digest maps the site of *Hpa*II cleavage to a location within the intergenic spacer of *Tripsacum* rDNA. Considering the difference in total repeat unit length between *T. dactyloides* (9.5 kbp)

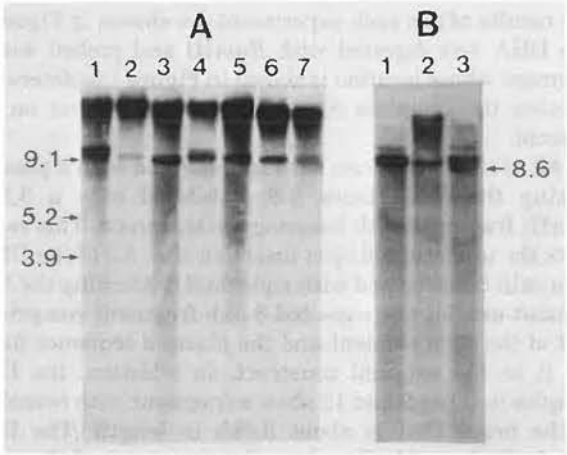


Figure 1. DNA purified from leaves of various species of *Tripsacum* was digested with appropriate restriction enzymes, electrophoresed on 0.8% agarose gels, and transferred to Zeta bind membranes by Southern blotting. In (A) we have *Hpa*II digests of DNA from five species of *Tripsacum* (including three accessions of *T. dactyloides*) probed with a full length maize rDNA probe (pZmr1). Samples loaded in lanes are as follows: (1) *T. australe* (2) *T. dactyloides* (acc. 68-50-5) (3) *T. dactyloides* (acc. 63-229) (4) *T. dactyloides* (acc. 63-39) (5) *T. cundinamarce* (6) *T. laxum (7) T. peruvianum.* In (B) we show preliminary mapping experiments for *T. dactyloides* (acc. 63-229). DNA was digested with *Xba*I (lane 1), *Hpa*II (lane 2) and *Hpa*II/*Xba*I (lane 3). A 400 base pair probe (p*XBr*1) adjacent to the conserved *Xba*I site was used to indirectly end-label the fragments produced in double digests. Fragment sizes were calculated from maize rDNA digested with *Bam*HI and detected with the probe pZmr1 (size indicated by arrows on left) and lambda DNA digested with *Hin*dIII. We wish to thank Dr. M. D. McMullen for providing the plasmid pZmr1 and Dr. D. H. Timothy for *Tripsacum* seeds.

and inbred lines of maize (9.1 kbp), this site falls in a region very near that previously observed in inbred lines of maize (R. Phillips et al., Keystone Conference, 1985; E. Jupe and E. Zimmer, unpublished observations).

Vishal Sachdev, Eldon Jupe and Elizabeth Zimmer

BERGAMO, ITALY
Istituto Sperimentale per la Cerealicoltura

Identification of a restriction DNA fragment encoding for zein polypeptides Zp20/1, Zp20/2, Zp20/3

Zein, the prolamin of corn, is a group of alcohol soluble proteins constituting about 50% of the total storage protein in maize endosperm. Zeins are resolved, in SDS-PAGE, mainly in four classes with apparent MW of 22, 20, 15 and 10 kDa. When analyzed by IEF, however, a complex protein-banding pattern is seen which is different for different inbred lines. Taking advantage of the large genetic variability existing among maize strains in the IEF and SDS polyacrylamide banding patterns of storage proteins, 20 structural loci, encoding for different zein polypeptides, have been mapped on chromosome 4 and 7 (H. Hartings et al., Genet. Agr. 38:447, 1985). Sequences of several zein genes have been identified and used as probes for the studies of their molecular organization. We have set up a strategy to associate zein polypeptides with specific DNA restriction fragments containing genes encoding for zein polypeptides. In this respect we have found, among several inbreds examined, that zein polypeptides designated Zp20/1, Zp20/2 and Zp20/3 are always simultaneously present or absent in different genotypes. The cluster of genes encoding for these polypeptides is on the short arm of chromosome 7, linked to the opaque-2 (*o2*) gene. The fact that we have never observed recombination within this cluster of genes suggested the possibility that they could be located on a single restriction DNA fragment.

In experiments involving segregation of the *o2* gene with presence or absence of the three zein polypeptides, associated with the molecular analysis of the recombinant seeds, we detected that a restriction DNA fragment of 20 kb cosegregated with the presence of these polypeptides; this fragment was absent in DNA extracted from plants lacking the three zein polypeptides. This suggested that this fragment might contain the genes encoding one or even all three polypeptides.

R. Marotta, B. Vaciago, N. Di Fonzo, F. Salamini, C. Soave, M. Motto and A. Spada

Interaction of endosperm mutants in the synthesis of storage proteins

Proteins from maize endosperm can be fractionated into various classes on the basis of their solubility. The purpose of this investigation was to determine the effects on zein synthesis and endosperm development of some regulatory loci, i.e. opaque-2 (*o2*), defective endosperm (*De*-30*), mucronate (*Mc*), and opaque-6 (*o6*) (Table). All the mutations considered significantly reduced final endosperm weight. In particular, it has also been observed that opaque-6 affects more drastically the synthesis of the zein fraction in comparison to other mutants. Moreover, in order to investigate the relationships among these regulating loci some double mutant combinations have been analyzed. The result showed that in the double mutants *o2 De-30, Mc De*-30* and *o2 o6* the percentage of the zein fractions is much lower in comparison to the level present in the single mutants. This suggests an additive effect of these genes in the deposition of the storage protein.

	Endosperm weight (mg)	Protein %	Protein fractions AG [1] %	Z_1 [2] %	Z_2 [3] %	G+R [4] %
B37 +	206	12.17	11.88	47.60	2.21	38.31
B37o2	130	9.20	17.68	26.96	3.62	51.75
B37Mc	170	11.43	12.55	41.28	2.03	44.13
B37De30	194	10.12	12.23	37.46	1.41	48.90
B37o2De30	143	7.74	31.87	10.94	0.91	56.28
B37McDe30	130	10.78	19.23	29.36	0.94	50.47
A69Y+	216	12.56	6.96	54.00	7.76	31.28
A69Yo2	142	10.80	21.75	24.61	8.89	44.75
A69Yo6	92	11.80	31.07	13.07	5.54	50.32
A69Yo2o6	66	10.15	41.85	6.45	3.16	48.53

1) albumins and globulins; 2) alcohol soluble zeins; 3) alcohol soluble zeins plus 2-mercaptoethanol; 4) glutelins plus insoluble proteins

N. Di Fonzo, E. Gentinetta, H. Hartings, M. Brembilla, M. Motto, C. Soave and F. Salamini

The b-32 protein from endosperm: nucleic acid (c-DNA) and amino acid sequences

The b-32 protein is a maize endosperm albumin that apparently is not bound to any particulate cytoplasmic structure. Its expression during development is temporarily and quantitatively coordinated with that of zein storage proteins. The b-32 protein is found either in the acidic or in the basic form in all inbreds as a gene product of two codominant alleles. It has been shown that the *o2* and *o6* mutants lack this protein. This makes the protein a candidate to be a trans-acting factor involved in the positive regulation of storage protein genes. A full length c-DNA for the b-32 mRNA has been cloned and the complete amino acid sequence of the protein derived. A lambda gt11 library from immature endosperm was screened for the expression of the b-32 protein using antibodies against the purified protein. The nucleotide sequence shows that several internal gene duplications have taken place during evolution. The protein has a length of 303 residues (MW of 31979 daltons) and its sequence shows several outstanding features: no signal peptide is detectable, indicating that b-32 protein probably is not a secretory protein; it contains 6 Trp, a residue that is absent in maize storage proteins; polar and hydrophobic residues spread out along the whole sequence; several pairs of basic residues are detectable at the N-terminal region (res 1-70); the half C-terminal region of the molecule is rich in repeats either of the same residue or of groups of two or three residues; secondary structure predictions suggest two structural domains that would fold up giving rise to a globular protein.

N. Di Fonzo, H. Hartings, M. Brembilla, M. Motto, C. Soave, F. Salamini, E. Navarro and J. Palau

Molecular cloning of *o2-m5*

The Opaque-2 (*O2*) locus of maize represents one of the repertoire of regulatory loci affecting the accumulation of zein polypeptides in developing kernels. Because the molecular cloning of the *O2* locus and the analysis of its

structure and expression may contribute to shed light on the nature of its regulatory effects, we developed an indirect strategy for cloning the *O2* locus based on its association with the transposable element *Ac*. In order to induce instability at the *O2* locus in the *wx-m7* strain two experiments were performed in the summer nursery in 1985 and 1986 by crossing homozygous *wx-m7* plants to a stable *o2* tester line. Approximately 760,000 (1985) and 250,000 (1986) F1 kernels were obtained by hand-pollination. In the two experiments 451 and 235 kernels showing somatic instability at the *O2* locus (i.e., with vitreous and opaque sectors) were identified. These kernels were sown and the resulting plants selfed. The majority of these plants segregated normal and opaque seeds in a 3:1 ratio, suggesting that the somatic instability observed in the F1 endosperms was not inherited. In fourteen cases, however, the variegated phenotype was transmitted to the next generation. The frequency of induction of mutable alleles at this locus was 1.4×10^{-5}, a value consistent with that of 1×10^{-5} reported for the induction of unstable mutants by maize transposable elements. Genetic analysis of one of these alleles, i.e. *o2-m5*, showed that it contains an autonomous *Ac* element. For the cloning of *o2-m5* allele, we used a central segment of *Ac* as a probe. We identified two cloned fragments that contained a sequence that resembles the *Ac* element in structure. A DNA sequence flanking the autonomous *Ac* insertion was found to be *o2* specific and provided a probe for the molecular analysis of the *O2* locus.

F. Salamini, M. Motto, G. Ponziani, M. Maddaloni, C. Soave, N. Di Fonzo, R. Thompson and R. Marotta

Salt tolerance: in vitro selection with regard to the regenerative potential

Selection in vitro on NaCl containing medium seems a promising approach for selecting cell lines which tolerate salt in their nutritional environment. Despite the possibly complex nature of the salt tolerance expressed by plant cells, many examples are reported in literature, on the in vitro isolation of salt tolerant cell lines with eventual recovery of regenerates displaying acquired traits of tolerance at the whole plant level (C. P. Meredith, in: J.P. Gustafson (ed) Gene Manipulation in Plant Improvement. Plenum Press, New York, 1984; Croughan et al., Env. Expt. Bot. 21:317-324, 1981). Maize is classified among the cereals which are rather sensitive to the presence of salt. Exposure of maize calli to salt stress results in drastic changes in growth and protein synthesis (Ramagopal, Plant Cell Rep. 5:430-434, 1986). Therefore, studies on in vitro screening for obtaining NaCl tolerant embryogenic lines might help in elucidating the genetic and physiological aspects of the tolerance, and possibly the recovery of the acquired trait in the regenerates.

We developed a selection scheme mainly considering the following points: a) choice of genotypes which can develop in vitro long-term highly embryogenic cultures; b) choice of material in which genetic recombination was occurring, in order to have the chance to meet *ab initio* an advantageous genetic set; c) use of somaclonal variation as a source of further genetic variability; d) the choice of a level of selection not drastically hampering the embryogenic capability of the selected cell lines in order to test the acquired tolerance in the progenies of the regenerates.

Embryogenic cultures were derived from W64A × A188 F2 immature embryos and explanted at 12 DAP on N6 basic medium (C.L. Armstrong and C.E. Green, Planta 164:207, 1985). Each cell lineage derived from an initial zygotic embryo, which stably performed as embryogenic culture along the first three months of life, was considered a somaclone, in that both its initial genetic set and the variation eventually originated during the period of culture, might account for the specific results in each case. A pool of the original material was first subjected to growth in the presence of various amounts of NaCl (0, 50, 100, 148, 198, 222 and 291 mM). Growth was seriously affected at 100 mM NaCl with a drastic effect on the somatic embryogenesis of the calli. Therefore selection was performed at 85 mM NaCl (0.5% NaCl) in which some perfectly embryogenic outgrows could be rescued on the calli at each passage and subcultured in the same conditions. Thirty-nine original somaclones were subjected to the scheme of selection described in Figure 1. Each step lasted 21 days. At the end of each subculture, embryogenic outgrows were subcultured on fresh medium. At the end of the 3rd step on 0.5% NaCl, 18 of 39 somaclones gave resulting tolerant derivatives. All the tolerant cultures were perfectly embryogenic calli of type 1. Out of these latter ones, 8 performed particularly well and were subcultured on higher dosage (128 mM NaCl), to test

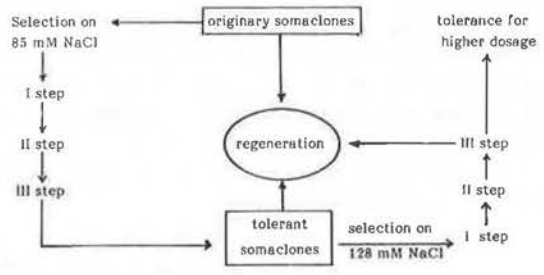


Figure 1 - Scheme of selection

Table 1 - Regenerates obtained in salt tolerant somaclones in selective and non-selective conditions

Salt tolerant somaclones (STSC)	2	3	14	15	16	19	20	21	22	23	24	27	28	29	35	37	41
N° of regenerates on MS hormone-free + 85 mM NaCl	4	2	2	4	6	0	6	5	3	0	2	2	7	3	3	0	0
N° of regenerates on MS hormone-free	6	4	3	8	6	7	9	16	6	2	4	0	5	8	3	1	5
Total n° of regenerates	10	6	5	12	12	7	15	21	9	2	6	2	12	11	6	1	5
N° of established plants in soil	8	5	4	9	11	5	8	16	7	2	5	1	10	10	3	1	3
Clones tolerant 128 mM NaCl and regenerable	n.t.	-	n.t.	n.t.	-	n.t.	++	++	n.t.	-	n.t.	n.t.	n.t.	n.t.	n.t.	n.t.	-

n.t. = non tested

whether they might be tolerant to higher levels of NaCl. Indeed, 2 clones (STSC 20 and 21) grew as much as the control maintaining a highly embryogenic phenotype. These latter ones are now being evaluated for regeneration and growth on higher levels of salt (1% NaCl). Seventeen tolerant somaclones were transferred to Murashige and Skoog (1962) hormone-free medium for plant regeneration. Regenerative conditions were both in the presence and in absence of the selective level of NaCl. Final results are summarized in Table 1. At the end of the 3rd step of selection, all the somaclones were embryogenic calli of type 1 capable of regeneration in various degrees. Twelve somaclones produced plantlets on both selective and non-selective conditions: the plantlets produced were equally efficient. Four somaclones (STSC 19, 23, 37, 41) produced plants only in non-selective conditions, while STSC 27 regenerated only in NaCl containing medium. Over the total number of regenerates obtained, a good percentage have been successfully transplanted into soil. The efficiency of establishment in pots was not apparently correlated to the conditions of regeneration. Although observations on the morphology of direct regenerates are strongly affected by epigenetic changes due to the in vitro culture itself, however, a larger root system was observed in the regenerates on NaCl containing medium, with more than 4- to 20-fold difference compared to the control. No phenotypic aberration or noticeable variation could be observed on the aerial parts, except that a few regenerates showed intense tillering. This was noticed also in regenerates from the control. Further investigations will be carried out on the progenies of these regenerates.

E. Lupotto, M. Mongodi and M.C. Lusardi

An example of genotype flexibility in tissue culture: switch from type 1 to type 2 embryogenic cultures in the inbred B79

In recent years, great attention has been paid to genotypes of maize capable of inducing in vitro friable highly embryogenic calli, termed type 2 calli. These cultures have been developed as a more promising source of material for maize genetic manipulation than the first callus cultures, which were hard and morphogenic, typically derived from most of the maize genotypes, known as type 1 cultures. So far, embryogenic friable type 2 calli have been established mostly from the inbred A188, A188 × B73 and reciprocal, under the conditions first described by C.E. Green (in: A. Fujiwara, ed., Plant Tissue Culture, Maruzen, Tokyo, 1982), essentially on N6 medium supplemented with l-proline. This genotype × culture condition appeared to be highly specific in obtaining type 2 callus cultures (D.T. Tomes and O.S. Smith, Theor. Appl. Genet. 70:505, 1985: C.L. Armstrong and C.E. Green, Planta 164:207, 1985). By using the same cultural conditions, we were capable of inducing type 1 calli from the inbred B79 and its behaviour in culture has been characterized in comparison with the most extensively studied inbred W64A. Evaluations have been made on cultures derived and propagated on Murashige and Skoog (1962) basic medium without l-proline (ZM) and N6 basic medium as recommended by Armstrong and Green (1985). Callus induction was on 2 mg/l 2,4-D and propagation on 1 mg/l 2,4-D in the light (3000 lux, day/night 16/8 hrs). Results obtained in the establishment of the primary cultures are summarized in Table 1. Only in B79-N6 derived cultures, calli essentially of type 1 displayed a more pronounced friability, thus being indicated as type 1a. In no case were type 2 calli derived.

Table 1 - Evaluations on primary and secondary cultures of W64A and B79

Genotype	N° of explants a)	Medium b)	% responding explants c)	% established cell lineages d)	Primary cultures type	Secondary cultures type
W64A	200	N6	94.4	53.8	1	-
	200	ZM	62.3	21.4	1	-
B79	130	N6	89.9	61.4	1a e)	2
	130	ZM	67.3	12.3	1	-

a) immature embryos taken at 11-12 DAP
b) N6: inorganic salts according to Chu et al. (Sci Sin 18: 659-668, 1975); B5 vitamins (Gamborg et al., Expti. Cell Res. 50: 151-158, 1968); 25 mM L-proline, 100 mg/l Difco Casamino Acids; 2% w/v sucrose
ZM: MS inorganic salts according to Murashige and Skoog (Physiol Plant 15: 473-497, 1962); KNO_3 final concentration 2500 mg/l; KH_2PO_4 final concentration 510 mg/l; vitamins and L-asparagine (13 mM) as in Green and Phillips (Crop Sci. 15: 417-421, 1975); 3% w/v sucrose
c) counted at day 30 after embryo loading
d) % calculated on the initial responding embryos as stable embryogenic lineages
e) here used to indicate an embryogenic callus type 1 with enhanced friability respect to the true callus type 1 of W64A

Primary callus cultures lasted 4-8 months on ZM medium, and 7-12 months on N6 medium, thus confirming that N6 medium may be considered a superior medium also for type 1 callus culture. Light was essential for propagation. Regenerative capability obtained on MS hormone-free medium of the type 1 cultures declined along subcultures ranging from 12-15 plantlets regenerated per gram of fresh weight tissue in the first three months, to the complete loss of regenerative capability in the subsequent 5-6 months of life. A slightly longer callus life and regenerative potential were detected on N6-propagated callus cultures.

However, during regeneration on hormone-free medium, in the case of B79-derived calli, developing plantlets underwent embryogenic callus proliferation at the coleoptilar node, a phenomenon strictly reminiscent of the

Table 2 - Regeneration efficiency in primary and secondary embryogenic cultures of the inbreds W64A and B79

Genotype	Regeneration efficiency a) per month of life on N6 medium								
month	3	4	5	6	7	8	9	10	20
W64A primary cultures	10.7 ± 2.8	11.2 ± 3.2	6.8 ± 2.9	8.1 ± 2.5	7.1 ± 2.8	6.5 ± 3.1	2.8 ± 1.9	-	-
W64A secondary cultures	-	-	-	-	-	-	-	-	-
B79 primary cultures	12.1 ± 1.8	13.4 ± 2.7	11.6 ± 0.9	12.4 ± 1.2	7.9 ± 0.5	7.5 ± 3.1	4.1 ± 1.9	1.1 ± 0.6	-
B79 secondary cultures	38.7 ± 10.1	35.4 ± 7.1	39.1 ± 6.3	35.7 ± 5.4	n.e.	n.e.	n.e.	n.e.	34.7 ± 5.8

a) expressed as number of regenerates per gram of fresh weight tissue on N6 hormone-free medium; each value in the mean of three replicates ± SD
n.e. = non evaluated

recurrent or secondary somatic embryogenesis described for a few plant species not belonging to the Gramineae (alfalfa, clover, papaya, cassava). The secondary somatic embryogenesis in B79 allowed the establishment and propagation of true type 2 embryogenic calli, as secondary cultures, on N6 medium, completely comparable to the callus cultures obtained in A188 and its derivatives. The most dramatic effect of the switch from type 1 to type 2 callus in B79 cultures was mainly reflected by two important features: a) cultures became long-term embryogenic cultures and b) the regenerative potential, calculated on the basis of frequency of regeneration per gram of fresh weight tissue, increased dramatically. All the cell lineages which underwent secondary somatic embryogenesis originating type 2 callus cultures could be propagated on N6 medium in dim light without losing their peculiar morphology. To date they have been stably propagated for almost two years. The regenerative potential of the secondary cultures is as high as four fold over the one recorded in young (3 months old) primary cultures of B79 and did not decline with time. Evaluations made recently at the 20th month of life confirmed the high regenerative capability still present in these cultures (Table 2). The peculiarity of these data assumes particular relevance since B79 is one of the most currently utilized genotypes in maize breeding programmes.

E. Lupotto and M.C. Lusardi

BLOOMINGTON, INDIANA
Indiana University

Role of the knob and the differential segment of K10-I in preferential segregation

In the 1986 MNL, we suggested that the K10 knob near the terminus of the long arm of abnormal chromosome 10 type I was no different from any other knob and that the phenomena we had associated with this chromosome, namely preferential segregation and neocentromere formation, might be determined by the more proximally situated insert of foreign euchromatin designated the differential segment. The reason for this somewhat heretical view was the finding that plants containing deficient abnormal chromosomes 10, all lacking the knob and euchromatic segments of varying lengths but retaining the differential segment, displayed considerable neocentric activity in the two meiotic divisions. Tests demonstrating that preferential segregation also occurred in such plants were obviously required to support the hypothesis. A year ago, we conducted the first of such tests, using Df K10-I/N10; K9L *yg2*/K9S *Yg2* female parents crossed by *yg2* males. Earlier (G.Y. Kikudome, Genetics, 1959), female testcrosses of K10/N10 heterozygotes, where one chromosome 9 carried the *Yg2* allele and a medium sized knob and the homolog possessed the *yg2* allele and a smaller knob, produced progenies with 65-70 percent of *Yg2* plants. However, no preferential segregation of the chromosome 9 with the larger knob was found in our data when a Df K10 chromosome, lacking the terminal knob, replaced K10. The data are presented below:

	Yg	*yg*	Total	% *yg*
Df(C)/N10 ; K9L *yg2*/K9S/*Yg2*	1157	1099	2256	48.7
N10/N10 ; K9L *yg2*/K9S/*Yg2*	334	368	702	52.4
Df(K)/N10 ; K9L *yg2*/K9S/*Yg2*	344	336	680	49.3

Since preferential segregation is dependent on crossing over and is known to vary in different environments, we repeated the test a second year. To maximize the degree of neocentromere formation, we used Df(K)/Df(K) homozygotes, heterozygous for the large knob on chromosome 9, as female parents and followed the *yg2* marker as before. Again, no indication of preferential segregation was found (48% *yg* in a population of 1635). We are forced to conclude that the K10 knob is necessary for the occurrence of preferential segregation, although neocentromeres are formed in its absence. The question then arose as to whether the 2 phenomena of preferential segregation and neocentric activity are related at all. We have always believed that anaphase I and anaphase II neocentric activity at knobbed regions of bivalents heterozygous for dissimilar sized knobs in plants with the K10 chromosome was responsible for the orientation of chromatids with the larger knob toward the outer 2 spores in megasporogenesis and the preferential inclusion of the large knobbed chromatid in the basal megaspore. A second assumption was invoked, namely that the orientation established at anaphase I should be maintained into the second meiotic division, i.e., through the intervening interphase and prophase II stages. If the differential segment alone is able to induce neocentromeres but no preferential segregation follows, perhaps the K10 knob is responsible for the maintenance of orientation. If this be true, it is a unique property shared with no other knob. To further elucidate the activity of the knob region, we have undertaken the isolation of the K10 knob from the differential segment. This involves production of breaks in the short arm of chromosome 9 and the long arm of chromosome 10 and the transfer of the K10 knob from chromosome 10 to chromosome 9. We will use the high-loss system to engineer the desired 9-10 half translocation. If we are successful and the new 9-10 chromosome is transmissible, we will test the K10 knob for its ability to induce preferential segregation and neocentromeres in sporocytes having no differential segment in chromosome 10 or elsewhere in the complement.

M.M. Rhoades and Ellen Dempsey

Effect of K10-II on preferential segregation of chromosome 9

K10-II was found by T.A. Kato (B. McClintock, T.A. Kato, and A. Blumenschein, 1981) in a strain of Mexican teosinte. Unlike the K10-I chromosome, which occurs in both maize and teosinte, K10-II has not been found in maize. We obtained a sample of the teosinte K10-II strain from Kato and have now introduced this chromosome into maize. It resembles K10-I in having an extra partially heterochromatic segment attached to the long arm of chromosome 10 but differs morphologically in several ways. In the region comparable to the differential segment of K10-I, identified in that chromosome by 3 prominent chromomeres, K10-II shows only a single chromomere in a more distal position. Instead of a single large knob, K10-II possesses 2 smaller knobs separated by a stretch of euchromatin. In spite of the morphological differences, the 2 abnormal chromosomes 10 both induce neocentromeres and both undergo preferential segregation in K10/N10 heterozygotes. In testcrosses of K10-II *R*/N10 *r* female parents, the *R* allele was recovered with a frequency of 70.8%, comparable to the 70% found with K10-I. This past

summer, we were able to show that K10-II also causes preferential segregation of chromosome 9 alleles. Female parents of K10-II/N10 ; K9L *yg2*/K9S *Yg2* constitution, along with N10/N10 controls, were testcrossed by *yg2* male parents and the following data were obtained:

	Yg2	*yg2*	Total	% *yg2*
K10-II/N10	602	1293	1895	68.2
N10/N10	366	333	699	47.6

It is evident that K10-II, like K10-I, induces preferential segregation of the marker carried by the chromosome 9 with the larger knob. K10-I possesses a third attribute, the ability to increase crossing over in sensitive regions susceptible to changes in crossover frequency. Studies are underway utilizing the *a2 bt* region of chromosome 5 to test whether or not K10-II also possesses this ability.

M.M. Rhoades and Ellen Dempsey

Structure of K10-II chromosome and comparison with K10-I

By means of a series of deficiencies lacking variable amounts of the terminal segment of the K10-I chromosome, we were able to demonstrate that the K10-I chromosome differed from normal chromosome 10 (N10) by (1) insertion of foreign chromatin (the differential segment) between the *R* and *W2* loci, (2) inversion of the *W2 07 L13* region of N10 and its insertion distal to the differential segment, and (3) addition of a large heterochromatic knob plus a terminal euchromatic tail beyond the distal *Sr2* locus. (See diagrams in Rhoades and Dempsey, Plant Genetics, Alan R. Liss, Inc., 1985).

We have recently produced a number of deficient K10-II chromosomes, selected through the high-loss system (Rhoades and Dempsey, Genetics, 1972) as having lost the *Sr2* locus, and some information about the structure of K10-II is now available. The following chart is a summary of results with 6 deficient K10-II chromosomes. Each was tested with *l13* and *w2* testers to determine if the dominant alleles of those markers were still present. In 2 cases, data on male transmission of the *r* marker on the deficient chromosome are available.

	Deficient for		Male transmission in Df K10-II *r*/N10 *R* compounds (% *r*)
	L13	*W2*	
Df(M)	no	yes	3% (Σ = 1931)
Df(N)	no	no	—
Df(O)	(no)	yes	—
Df(P)	(no)	yes	—
Df(Q)	yes	yes	—
Df(S)	(no)	yes	18.5% (Σ = 1190)

Values in parentheses need further tests. The fact that the deficient chromosomes are marked by *r* instead of *R* makes their identification difficult and has allowed us to obtain transmission data only in plants where the ratio was obviously aberrant. Other male testcrosses giving 1*R*:1*r* ratios could have involved sib plants not carrying the deficient chromosome. Aside from this difficulty, the *r* allele serves as an efficient marker for the Df chromosome since crossing over between *r* and *Sr2* in K10-II *R Sr2*/N10 *r sr2* compounds is less than 4% and a low frequency of *r Sr2* recombination should also occur in Df K10-II/N10 plants.

We can now draw some conclusions about the structure of the K10-II chromosome. Since loss of *W2* can take place while *L13* is still retained, the order of these 2 genes must be inverted in K10-II with respect to their order in N10, i.e., the order of the two genes is the same in K10-II and K10-I. All the deficient K10-II chromosomes are at least as long as N10; Df(N) was noticeably longer than N10. Therefore, the segment bearing the *W2* and *L13* loci in K10-II must lie distal to the differential segment, as is true for K10-I.

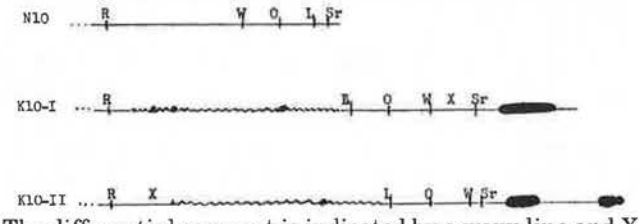
Some crossover studies with K10-I and K10-II also indicate that K10-II is more similar to K10-I in structure than to N10. The Df chromosomes shown below are all derived from K10-I.

	% *R*-Df (or *R-Sr2*) Recombination		% *R*-Df (or *R-Sr2*) Recombination
K10-I/N10	0.8	K10-II/N10	3.9
K10-I/Df(C)	7.5	K10-II/Df(C)	6.7
K10-I/Df(F)	15.0	K10-II/Df(F)	8.9
K10-I/Df(K)	19.5	K10-II/Df(K)	10.9

Both K10-I and K10-II show the greatest reduction in recombination when opposed by a N10 chromosome. Obviously, the 2 abnormal chromosomes 10 show little homology with N10 in the distal part of the long arm. The increase in recombination when K10-I is combined with deficient chromosomes of increasing length [(Df(C)<Df(F)<Df(K)] is due to the increase in homologous segments where crossing over can occur. A similar but much less pronounced increase was found with the K10-II series, indicating a lesser degree of homology between K10-II and the Df chromosomes derived from K10-I. Unfortunately, a direct comparison of the 2 abnormal chromosomes 10 by recombination studies in K10-I/K10-II compounds cannot be made because of the lack of a distal marker gene.

One obvious difference in the 2 K10 chromosomes is evident from cytology; the differential segment of K10-I (and of the Df K10-I chromosomes) possesses 3 prominent chromomeres while the differential segment of K10-II has only 1 enlarged chromomere in a more distal position. The 2 chromosomes also differ in compounds with N10 in the amount of crossing over between *R* and *Sr2*. These crossovers occur in a region from the *R* locus to the proximal end of the differential segment. The higher crossing over in K10-II/N10 plants indicates that the differential segment is located more distally in K10-II than in K10-I. Because of this change, the inverted region containing the *L O W* loci must be longer in K10-I than in K10-II by the inclusion of chromatin adjacent to those loci. (See diagram below). Another point of difference between the 2 abnormal chromosomes 10 is in male transmission; in K10-I *R*/N10 *r* pollen parents, only 42% of the functioning pollen grains carry the *R* allele. In similar crosses involving K10-II *R*/N10 *r* plants, 50.3% of the progeny from male testcrosses (Σ = 3555) have the *R* allele.

Our present interpretation of the structure of the distal region of N10, K10-I and K10-II is shown below:



The differential segment is indicated by a wavy line and X marks the chromatin adjacent to *R* which is located distally in K10-I.

M.M. Rhoades and Ellen Dempsey

Crossing over per unit chromatin

The frequency of crossing over per unit of physical length of the pachytene chromosome is not the same in all parts of the maize genome. In 1968, we studied a transposition involving the transfer of a segment consisting of about 10% of the long arm of chromosome 3 to a new location in the short arm of chromosome 9 (Rhoades, in Replication and Recombination of Genetic Material, Canberra, 1968). This segment of chromatin undergoes little if any recombination either in its original location or in its new position in chromosome 9. Comparisons of *Sh Wx* recombination in plants homozygous for the transposition with that in plants having only normal chromosomes 9 showed no differences even though the physical distance between the *Sh* and *Wx* marker genes was considerably increased in the transposition homozygotes by the inserted chromatin. Similarly, crossing over between the *Lg2* and *A* markers spanning the segment in question in normal chromosome 3 was not affected in plants homozygous for deficient chromosomes 3, in spite of a decrease in the cytological length. We concluded that "no exchanges occur in the transposed segment when it is a part of chromosome 3 or when placed in the Tp9 chromosome."

The differential segment of K10-I appears to be another example of chromatin which has a low rate of crossing over. Df(C), derived from K10-I by loss of the terminal knob and the distal euchromatic segment with the *L O W* markers, possesses the differential segment of K10-I. In Df(C)/N10 sporocytes, the 2 chromosomes 10 are of equal length. In N10/N10 plants the distance from *R* to the most distal marker, *Sr2*, is about 35 map units and a comparable genetic length might be anticipated for the region from *R* to the end of Df(C) when Df(C) is combined with a homologous Df10 chromosome, such as Df(H). This segment can be measured in the absence of a distal marker gene because Df(C) is not male transmissible. Therefore, when Df(C) *R*/Df(H) *r* plants are used as male parents in testcrosses, the percentage of *R* progeny measures the recombination from *R* to the end of Df(C). Df(H) is longer than Df(C) since it possesses the euchromatic region with the *L O W* markers in addition to the differential segment but in all other respects it is completely homologous to Df(C). Instead of the 35% *R Sr2* observed in N10/N10 plants, the frequency of recombination in Df(C)/Df(H) heterozygotes was 8.9%, of which about 3-4% can be attributed to the region from *R* to the start of the differential segment. Thus, recombination in the differential segment itself must be very low.

The absence of recombination potential in the 2 segments of chromosome 3 and chromosome 10 described above may result from the lack of Chi sites in the DNA or may be due to sequestering of these sites so they are not available to recombination enzymes. In the case of the transposed segment of chromosome 3, addition of B chromosomes to the complement caused at least a twofold increase in recombination in the *C Wx* region of chromosome 9. Perhaps the Chi sites in the transposed segment were exposed by some activity of B chromosomes. We have not yet tested the response of the differential segment to B chromosomes.

M.M. Rhoades and Ellen Dempsey

BROOKINGS, SOUTH DAKOTA
South Dakota State University and USDA-ARS

Chromosome location of peroxidase-1

The inheritance of isozyme locus peroxidase-1 (*Px1*, also called *Prx1*) was first reported by Hamill (MNL 42:36, 1968). Electrophoretic assays of F2 progeny of crosses between various inbred lines failed to reveal linkage of *Px1* with any of the following isozyme loci: *Pgd1, Idh2, Got1, Mdh2, Acp1, E1 (Est1), E4 (Est4)* and *Glu1* (A.L. Kahler, J. Hered. 74:239, 1983).

The chromosomal location of *Px1* was determined by analyzing F2 progeny of single-cross hybrids made by crossing selected inbred lines with a series of waxy-marked reciprocal translocations whose genotypes with respect to *Px1* had been determined previously. Electrophoretic procedures were as given by Kahler (Crop Sci. 23:572, 1983). Contingency chi-square tests for independent segregation and recombination values were calculated using the maximum likelihood LINKAGE-1 program of K.A. Suiter, J.F. Wendel, and J.S. Case (J. Hered. 74:203, 1983).

Two crosses, both involving *wx* T2-9d (breakpoints 2L.83, 9L.27), gave highly significant chi-square values indicating linkage and suggesting that *Px1* is on the distal portion of the long arm of chromosome 2. The data are as follows:

(*wx* T2-9d *Px1-2*/WF9 *Px1-3*) selfed:

	Px1-2/2	*Px1-2/3*	*Px1-3/3*	
+	5	78	60	n = 248
wx	88	17	0	χ^2 indep. = 171.45

(*wx* T2-9d *Px1-2*/N7A *Px1-3*) selfed:

	Px1-2/2	*Px1-2/3*	*Px1-3/3*	
+	3	64	32	n = 180
wx	68	10	3	χ^2 indep. = 122.36

Px1 was then mapped with respect to *v24* (MNL 61:50, 1987) and *pgspt*-579B*, two seedling markers provided by Dr. M. G. Neuffer and placed by him on chromosome 2L by B-A translocation tests. *v24* has not yet been mapped precisely. Neuffer (pers. comm.) reports that *pgspt*-579B* is not the same mutant as *spt1*. Although *pgspt*-579B* and *spt1* are both on 2L and have similar phenotypes, they have not been tested for allelism. S. McCormick and A. Barnason (MNL 59:91, 1985) mapped *pgspt*-579B* 36 map units distal to *v4*. Our two-point F2 tests of *Px1* with these mutants gave the following data:

(B73 *Px1-2/v24 Px1-3*) selfed:

	Px1-2/2	*Px1-2/3*	*Px1-3/3*	
+	31	77	13	n = 162
v	0	12	29	χ^2 indep. = 59.59 15.5 ± 3.1% recomb.

(B75 *Px1-2/pgspt*-579B Px1-3*) selfed:

	Px1-2/2	*Px1-2/3*	*Px1-3/3*	
+	30	70	2	n = 143
pgspt	0	5	36	χ^2 indep. = 110.92 4.6 ± 1.79% recomb.

These data confirm the location of *Px1* on chromosome 2L, but further tests are needed to determine the order of the three loci. We thank Joanne Mathees for assistance in conducting the electrophoretic assays.

R.H. Whalen and A.L. Kahler*

*Now at Garst Seed Co., Slater, Iowa

CAMBRIDGE, ENGLAND
Institute of Plant Science Research
EDINBURGH, SCOTLAND
University of Edinburgh

Mitochondrial gene nomenclature

At the conference on 'Higher Plant Mitochondrial DNA', held at Airlie House, Virginia, USA in October 1986, a discussion on the possibility of creating a standardized nomenclature for plant mitochondrial and nuclear genes encoding mitochondrial proteins and RNAs was held. This proposal details the consensus opinion of that meeting and of a number of interested individuals who responded to the circulars. An overriding aim was to retain, wherever possible, the current commonly used gene designations and the general principles of chloroplast gene nomenclature were followed (R.B. Hallick and W. Bottomley, Plant Mol. Biol. Rep. 1:38-43, 1983).

Mitochondrial proteins: The polypeptide products of both nuclear and mitochondrial genes are to be designated by capital letters, e.g.:

ATPB-1: beta-subunit of the mitochondrial ATPase.
COXII: subunit 2 of cytochrome oxidase.

Mitochondrial genes: Genes on the mitochondrial chromosome or associated with mitochondrial plasmids will take a three letter code in lower case which will be either italicised or underlined, for example:

cox *trn* *rrn* *orf* *urf*

Any suffixed descriptors will also be italicised (underlined), for example:

atpA-1 *coxII*

Nuclear genes (specifying mitochondrial components): Nuclear genes will be distinguished from mitochondrial genes by being in UPPER CASE. Again a three letter italicised or underlined code will be used, for example:

ATPB-1

The accepted convention for nuclear genes is to use numbers to identify genes with a related function or different members of the same gene family. However, we feel that where multipolypeptide complexes exist and where these have several components with unknown numbers of constituent polypeptides, for example the F0 and F1 components of ATP synthase, both letters and numbers should be used to distinguish the components of the complex whether or not the individual polypeptides of the components are mitochondrial encoded. This will allow additional polypeptides to be assigned to a complex regardless of the compartment in which it is encoded.

Mutations: In some instances mitochondrial genes specifying a gene product in one species or cytoplasm will be non-functional in another species or in another cytoplasm of the same species. It is therefore necessary to distinguish between functional and non-functional genes. The simplest way of achieving this is to provide the non-functional gene with a prefix, preferably a Greek symbol, e.g. phi (lower case).

Functional Gene	Mutant Gene
urf1	*ϕurf1*

Gene copy number: In both the nuclear and organelle compartments gene copy number may exceed one. In such instances genes will take a suffixed arabic number, for example:

atpA-1 *atpA-2* *trnS1* *trnS2*

Where the physical map of the mitochondrial genome has been determined, repeated sequences, if present, will be numbered sequentially from the origin of the map, the genes within repeats will be given the same reiteration-number as the repeat.

Species and cytoplasm designation: Species can be designated by the standard three letter code, for example:

Oenothera berteriana : Obe
Triticum aestivum : Tae
Zea mays : Zma

Many higher plant species have more than one cytoplasm. If no nomenclature exists to distinguish these then they should be distinguished by an upper case letter. In many instances cytoplasms have been described as either fertile or sterile. In these instances S and F will suffice. In maize the accepted designations of N, T, S and C will be retained. These cytoplasm descriptors will follow the three letter species code, for example:

Code	Description
Zma C	The C-cytoplasm of maize

This type of abbreviation is most often used with restriction endonucleases. Without exception these cytoplasm identifiers must precede the gene to which they may be hyphenated, for example:

T-*urf13* N-*atpA-1* N-*atpA-2* S-*pcf*

Such cytoplasm identifiers can be optional to reduce the size of the gene acronyms. Please note that the species acronym (if used) and the cytoplasm identifier are NOT to be italicised or underlined.

Genes of plasmids: Plasmids, both linear and circular, DNA or RNA are associated with some cytoplasms but not all. There is a growing body of evidence to suggest that specific plasmids are NOT associated with a particular cytoplasm. In order to account for this, we propose that a plasmid designation replaces the cytoplasm identifier. It will be necessary for the research worker to specify the cytoplasm elsewhere. Where many plasmids are associated with one cytoplasm it may be useful to identify them by mp1 : mitoplasmid 1, mp2 etc. Alternatively, trivial and commonly used designations may be retained, for example S1, S2, R2 etc. An mp prefix will assume a circular topology. Linear plasmids, not having commonly known designations, can be identified by their size in nucleotides or nucleotide base pairs and an L descriptor. Single-stranded molecules can be noted using the 'ss' abbreviation. Perhaps, fortunately, there are sufficiently few linear nucleic acid entities associated with motochondria for them to retain or be given trial designations.

Plasmid	Gene	Format
S2	*urf1*	S2-*urf1*
2.3L	*trnW-TGG*	2.3L-*trnW-TGG*

Recommended nomenclature for known mitochondrial genes

Ribosomal genes

Gene	Gene product
rrn26	26S rRNA
rrn18	18S rRNA
rrn5	5S rRNA

The designation rDNA (e.g. 26S rDNA) includes the ribosomal gene as well as the transcriptional promoters and the transcribed flanking sequences.

Transfer RNA genes—Transfer RNA genes are designated *'trn'* with the addition of the single letter amino-acid code to identify the species; isoaccepting species will be designated with the anticodon following the amino acid code. Duplicated genes, for example those associated with repeated sequences, will be identified with a reiteration-number corresponding to the reiteration-number of the repeat. Unfortunately, it is impossible to distinguish between tRNA gene duplications which have occurred due to promiscuous DNA transfer between organelles. A few of the chloroplast tRNA genes in the mitochondrial genome of maize, for example *trnW-TGG*, are transcribed, but their ability to accept amino acids and transfer these to growing polypeptide chains has never been demonstrated. Therefore, they could be considered pseudogenes and be designated as such.

Gene	Gene product	Comment
trnM or *trnM-ATG*	$tRNA^{Met}$	Elongator species
trnfM or *trnfM-ATG*	$tRNA^{fMet}$	Formyl-methionine initiator species

Where there are isoacceptors:

trnL-TTG	$tRNA^{Leu}$-CUU
trnL-CTG	$tRNA^{Leu}$-CAG

Where gene duplication has occurred:

trnL1 or *trnL1-CTA*	$tRNA^{Leu1}$-UAG
trnL2 or *trnL2-CTA*	$tRNA^{Leu2}$-UAG

The mitochondrial genetic code has one possible anomaly: CGG specifies tryptophan instead of arginine. It is therefore recommended that in this instance it be fully abbreviated as follows:

trnW-CGG	$tRNA^{Trp}$-CCG

Mitochondrial polypeptide genes: The gene designations in this section will make use of the commonly used or accepted gene designations where possible.

Ribosomal protein genes—The designations 'rps' for small subunit proteins, 'rpl' for large subunit proteins, are recommended. Where homology to an existing *E. coli* ribosomal protein exists the gene can be designated with the same number. If no homology exists then the identifier should be a letter. For example:

Gene	Gene product	Comment
rps13	RPS13	ribosomal protein S13

Polypeptides of the electron transport chain

Complex I: NADH-ubiquinone oxidoreductase—The components of this complex will be designated 'nad'. Individual genes will be given numerical identifiers, these will indicate homology or functional equivalence to the mammalian subunits. Additional genes will accept the next number of the series.

Gene*	Gene product	Mammalian gene
nad1	NAD1	*urf1*
nad2	NAD2	*urf2*
nad3	NAD3	*urf3*
nad4	NAD4	*urf4*
nad4L	NAD4L	*urf4L*
nad5	NAD5	*urf5*
nad6	NAD6	*urf6*

*These gene designations presume a mitochondrial location. To date only sequences related to *nad1* and *nad5* have been identified.

ATP synthase—The two multicomponent subunits of this complex, F_0 and F_1, will be differentiated by suffixed letters (F_1) and suffixed arabic numbers (F_0).

Subunits of the F_1 complex:

Gene	Gene product
atpA	ATPA
ATPB-1	ATPB-1
ATPB-2	ATPB-2

Subunits of the F_0 complex:

Gene	Gene product
atp6	ATP6
atp9	ATP9

Genes of other complexes, including complex III and complex IV—

Gene	Gene product	Description
coxI	COXI	subunit 1 of cytochrome oxidase
coxII	COXII	subunit 2 of cytochrome oxidase
coxIII	COXIII	subunit 3 of cytochrome oxidase
cob	COB	Apocytochrome B

Open reading frames and unidentified reading frames—Open reading frame, *orf*, is a gene for which no specific gene product has been identified. Unassigned reading frame, *urf*, is an open reading frame which is transcribed and translated into a polypeptide whose function has not been assigned. Individual *orf*'s and *urf*'s will be distinguished by the number of amino acids which are coded for by the open reading frame. An *urf* can also accept the size of the polypeptide in kilo daltons as its descriptor, for example:

Gene	Gene product	Comment
T-*urf115* or T-*urf13*	T-URF13	Gene coding for the 13 kDa polypeptide which is unique to the T-cytoplasm of maize.

Gene library and central registrar: It is hoped that during 1988 a gene library will be established at the Institute of Plant Science Research in Cambridge. The clones deposited will be freely available on request. As an adjunct to this facility it would be recommended that people wishing to designate a new gene would first check to ensure that the nomenclature is consistent with this proposal. The contents of the library and the rules of gene nomenclature should be accessible via Bitnet using the following address: Lonsdale @ UK.AC.AFRC.CAMB.

D.M. Lonsdale and C.J. Leaver

[**Ed. note:** This proposed nomenclature, like that for chloroplasts, should be considered a working framework, just as are those for nuclear genomes; readers and users are urged to deliberate on these nomenclatures and to convey suggestions and criticisms to the authors. The editor would appreciate receiving copies of correspondence, toward internally consistent evolution of all nomenclatures]

CAMBRIDGE, ENGLAND
Institute of Plant Science Research
RICHMOND, ENGLAND
Royal Botanic Gardens

Wheat × maize and barley × maize hybridization

A) Wheat × maize crosses. When hexaploid wheat is pollinated with maize, fertilization typically occurs in about 30% of florets. Fertilization has been recorded in 25

of the 26 wheat × maize genotype combinations so far studied (the one failure was in a cross where only 11 florets were examined) but, curiously, double fertilization is relatively rare and most florets in which fertilization has occurred contain an embryo but lack an endosperm, the polar nuclei remaining unfertilized. For example, when 343 florets of 'Chinese Spring' wheat fixed 48h after pollination with 'Seneca 60' maize were examined 80 contained only an embryo, 8 contained only an endosperm, while 12 had both an embryo and an endosperm, giving an overall total of 100/343 (29.2%).

Proof of successful hybridization was obtained from zygotes at metaphase since these contained the expected F1 combination of 21 wheat chromosomes and 10 very much smaller maize chromosomes. However, the centromeres of the maize chromosomes are usually very indistinct and show little affinity for spindle microtubules. As a result they are rapidly eliminated and the developing embryos have only a haploid complement of wheat chromosomes. Wheat × maize crosses are potentially an attractive system for wheat haploid production via chromosome elimination since maize is relatively insensitive to the action of dominant alleles of the so-called crossability genes in wheat (*Kr1* and *Kr2*) which normally reduce the frequency of fertilization in crosses between wheat and alien species such as rye or *Hordeum bulbosum*.

Unfortunately embryos allowed to develop on plants almost always abort before they reach a stage where they are viable in embryo culture, and only 1, which developed into a haploid wheat plant, was recovered from 2,440 florets. A sample of this size would be expected to contain 592 embryos (calculated from data on the frequency of egg-cell fertilization in florets fixed for cytological analysis 48h after pollination).

However, we have recently succeeded in recovering haploid wheat plants from wheat × maize crosses at a much higher frequency by using an alternative culture method which will allow embryos to develop in the absence of any detectable endosperm. Spikelets containing cross-pollinated florets are removed from plants 2 days after pollination, surface sterilized, and placed on Murashige and Skoog medium containing 0.1mg l^{-1} 2,4-D. After 3 weeks incubation at 20 C the florets are dissected and the embryos are transferred to embryo culture medium. A total of 47 embryos were recovered from 706 florets and of these, 31 developed into haploid plants. This was estimated to be 17.0% of the number of embryos that would have been present 48h after pollination, making this method 100 times more efficient for recovering plants than allowing embryo development in vivo.

B) Barley × maize crosses. During 1987 we also found that 'Sultan' barley × 'Seneca 60' maize crosses result in fertilization. In 100 florets fixed 48h after pollination 4 contained only an embryo, 8 contained only an endosperm, and 16 had both an embryo and an endosperm. Thus the overall frequency of fertilization (28/100) was similar to that found in wheat × maize crosses, but the frequency of double fertilization was higher so that most embryos were accompanied by an endosperm, albeit one that was highly abnormal. Again proof of hybridization was obtained from zygotes fixed 48h after pollination since these contained the expected F1 combination of 7 barley chromosomes and 10 much smaller maize chromosomes. Interestingly, maize chromosomes showed well-defined centromeres in zygotes and although they were subsequently eliminated, at least some persisted for at least 4 cell cycles. So far no plants have been recovered from barley × maize crosses but they have not yet been tried in spikelet culture.

C) Potential uses of these crosses. Wheat × maize crosses have potential for wheat haploid production, particularly for the many wheats which carry dominant alleles of the *Kr* genes, but our main interest is to transfer maize DNA into wheat by sexual hybridization. All of the wheat × maize crosses investigated so far have rapidly lost the maize chromosomes but since we know that zygotes contain a complete haploid complement of maize chromosomes it might still be possible to transfer DNA by inducing intergenomic translocations prior to elimination. At present we are trying to do this using X-ray irradiation. An obvious goal for this work is the transfer of maize transposable elements into wheat, with a view to developing a stock which could be used for 'transposon tagging' experiments. This might also be feasible for barley if plants can be recovered using the spikelet culture method.

D.A. Laurie and M.D. Bennett

CAMPINAS, SÃO PAULO, BRAZIL
Instituto Agronômico

Allometric genetics V: Completing the 5th theorem by the calculus of α and β variances

In MNL 61:32-34 we presented an universal mathematical solution for the calculus of p, recombination value, and α and β, the allometric coefficients of the effects of genes *A* and *B*. The p variance was also calculated. The purpose of this report is to obtain the variance of α and β.

The solution usually, is the Cramér-Rao inequality.

$$V_{(\tilde{\alpha})} \geq -\frac{1}{E\left(\frac{\partial^2 L}{\partial \alpha^2}\right)} = \frac{1}{E\left[\left(\frac{\partial L}{\partial \alpha}\right)^2\right]}$$

In the 2 x 2 table combining the allometric coefficiens put in the frequency expectations, the formulation is, for the F_2.

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

By the maximum likelihood method we take from the table

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

Deriving in relation to α we have:

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

Only the signs of the 3rd and 4th terms change. Deriving again, these same terms change sign again and we are at a loss to decide which means which, on the inequality. A solution is found for a similar problem by R.A. Fisher (Ann. Eug. 9:50, 1939), and we reproduce his work with the adaptations required for our case.

"The variance error of any statistic, P, (he used T), explicitly expressed in terms of the observed class frequencies, and the number in the sample, is given by the general formula

$$\frac{1}{n} V(P) = S\left[p\left(\frac{dP}{\partial n}\right)^2\right] - \left(\frac{\partial P}{\partial n}\right)^2$$

where S stands for the summation over the several classes of observations, p for the probability of an observation falling in any

class, a for the frequency observed in that class and n for the size of the entire sample.

If this formula is applied to find the standard error of

$$F = \log_e(ad/bc) = \log_e\left(\frac{(2+P)^{1+\alpha+\beta} \times (P)^{1-\alpha-\beta}}{(1-P)^{1+\alpha-\beta} \times (1-P)^{1-\alpha+\beta}}\right)$$

we find for the four values of $\partial P/\partial a$

$$\frac{1}{a} \quad -\frac{1}{b} \quad -\frac{1}{c} \quad \frac{1}{d}$$

or

$$\frac{1}{(2+P)^{1+\alpha+\beta}} - \frac{1}{(1-P)^{1+\alpha-\beta}} - \frac{1}{(1-P)^{1-\alpha+\beta}} \quad \frac{1}{(P)^{1-\alpha-\beta}}$$

while if $g^{1+\alpha+\beta}$, $s^{1+\alpha-\beta}$, $s^{1-\alpha+\beta}$, $l^{1-\alpha-\beta}$ represent the proportions of a, b, c, and d surviving to be classified, the probabilities of falling in the four distinguishable classes are

$$p = g^{1+\alpha+\beta}(2+P)^{1+\alpha+\beta}, s^{1+\alpha-\beta}(1-P)^{1+\alpha-\beta}, s^{1-\alpha+\beta}(1-P)^{1-\alpha+\beta}, l^{1-\alpha-\beta}$$

$$(P)^{1-\alpha-\beta} \div \left(g^{1+\alpha+\beta} + s^{1+\alpha-\beta} + s^{1-\alpha+\beta} + l^{1-\alpha-\beta}\right)$$

Writing the expectations for the frequencies after differentiation, that is, putting

$$a = pn$$

We find

$$nV(F) = \left(g^{1+\alpha+\beta} + s^{1+\alpha-\beta} + s^{1-\alpha+\beta} + l^{1-\alpha-\beta}\right) \times \left[\frac{1}{g^{1+\alpha+\beta}(2+P)^{1+\alpha+\beta}} + \right.$$

$$\left. + \frac{1}{s^{1+\alpha-\beta}(1-P)^{1+\alpha-\beta}} + \frac{1}{s^{1-\alpha+\beta}(1-P)^{1-\alpha+\beta}} + \frac{1}{l^{1-\alpha-\beta}(P)^{1-\alpha-\beta}}\right] \quad (I)$$

Since in the equation of estimation, P is equated to

$$\log_e\left[(2+P)^{1+\alpha+\beta} \times p^{1-\alpha-\beta} / (1-P)^{1+\alpha-\beta} \times (1-P)^{1-\alpha+\beta}\right]$$

We may derive the sampling variance of our estimation of α, by dividing by the square of $\delta F/\delta\alpha$

$$\text{Now } \frac{\delta F}{\delta\alpha} = (1+\alpha+\beta)\ln(2+P)+(1+\alpha-\beta)\ln(1-P)+(1-\alpha+\beta)\ln(1-P)+(1-\alpha-\beta)\ln(P) \quad (II)$$

Hence $V_{(\tilde{a})} = V(F) \div (\delta F/\delta\alpha)^2$ " sic,(almost). (I) ÷ (II) = III

With this formula we calculated the parameters presented in Table 1, the upper of the double line on the table. By trial and error we find that for allometrical genetics the correspondence is for this case, in the lower line

$$V_{(\tilde{a})} \geq \left\{\frac{1}{\left[a(1+\alpha+\beta)\ln\left(\frac{2+P}{4}\right)\right]^2} + \frac{1}{\left[b(1+\alpha-\beta)\ln\left(\frac{1-P}{4}\right)\right]^2} + \frac{1}{\left[c(1-\alpha+\beta)\ln\left(\frac{1-P}{4}\right)\right]^2} + \right.$$

$$\left. \frac{1}{\left[d(1-\alpha-\beta)\ln\left(\frac{P}{4}\right)\right]^2}\right\}^{\frac{1}{2}} \leq \frac{V_{(F)}}{\left(\frac{\delta F}{\delta\alpha}\right)^2} \quad (IV)$$

That is, the variance of α or β is the square root of the total sum of the inverse each squared first, four terms. It is another inequality to extend the Cramér-Rao one. Note in Table 1 that by allometry the value is usually a little smaller. The discrepancies are mostly with the markers *B*, *Su*, and *Pl* due to values of $p > 0.5$ which is not congruent with the mathematical models. Comparing with p measures several-fold bigger populations are needed to measure effects of α and β.

Only the *Ts5 Tr* α effect approaches significance. It becomes more clear that the expression of *pd* and *tr* is due to a much more complex system of epistatic effects, since with so many duplicated genes it expresses itself as being always a single pair of recessive alleles with disturbed segregation, which once attained, blocks the effects of the other loci, because we always have a little less than one fourth *tr* or *pd* in F2 segregation. Within cultivated maize, these numerous genes at different loci act in a more quantitative way, the dominants adding their effects to achieve higher kernel row numbers.

Table 1. This table partially completes the analysis of Table 1 in MGCNL 61:33 giving the t values of α and β and the variance of these coefficients. In each double line the above is by Fisher's method $V_{(\tilde{a})} = V_{(F)} \div (\partial F/\partial\alpha)^2$, the lower line by our allometric method $V_{(\tilde{a})} = \left\{\Sigma_1^4 (\partial P/\partial\alpha)^2\right\}^{\frac{1}{2}}$ Most of the discrepant values are when p > 0.5 with B, Su, and Pl. Our method gives always smaller variance values, very near the above with some smaller variances.

			Pd (Fas)			Tr (Krn)			Sum	n
			t		$V_{(\alpha)}$ & $V_{(\beta)}$	t		$V_{(\alpha)}$ & $V_{(\beta)}$	$V_{(\alpha)}$ + $V_{(\beta)}$	
			α	β		α	β			
1	Fl	R	0.39	0.33	0.098	0.60	0.01	0.045	0,143	225
			0.19	0.42	0.058	0.62	0.01	0.043	0.101	
	Bm2	R	0.22	0.49	0.043	0.15	0.12	0.060	0.103	225
			0.22	0.50	0.041	0.17	0.13	0.047	0.088	
2	Lgl	R	0.21	0.62	0.042	0.13	0.02	0.046	0.088	257
			0.21	0.63	0.040	0.15	0.03	0.036	0.076	
	Gℓl	R	0.12	0.68	0.040	0.14	0.01	0.046	0.086	257
			0.12	0.71	0.037	0.17	0.01	0.032	0.069	
	B	C	0.68	0.49	0.178	0.80	0.34	0.099	0.277	115
			1.13	0.81	0.065	0.94	0.40	0.072	0.137	
	V_4	R	0.46	0.65	0.039	0.74	0.05	0.025	0.064	257
			0.45	0.64	0.041	0.61	0.04	0.037	0.078	
3	A_1	R	0.23	0.69	0.074	0.24	0.25	0.077	0.151	160
			0.23	0.71	0.070	0.28	0.29	0.057	0.127	
	Lg2	R	0.10	0.61	0.104	0.10	0.21	0.074	0.178	160
			0.09	0.58	0.117	0.11	0.23	0.061	0.178	
4	Ts5	C	0.27	0.11	0.110	1.45	0.32	0.120	0.230	93
			0.33	0.14	0.074	1.46	0.32	0.119	0.193	
	Su	R	0.68	0.43	0.122	0.79	0.49	0.091	0.213	83
			0.61	0.50	0.121	0.64	0.40	0.138	0.259	
5	Pr	R	0.10	0.10	0.107	0.17	0.55	0.145	0.252	101
			0.12	0.12	0.075	0.24	0.77	0.074	0.149	
6	Y	C	0.06	0.62	0.039	0.09	0.00	0.037	0.076	257
			0.06	0.65	0.036	0.11	0.00	0.029	0.065	
	Pℓ	R	0.45	0.12	0.076	0.47	0.64	0.102	0.178	145
			0.56	0.15	0.049	0.70	0.95	0.047	0.096	
	Py	R	0.80	0.52	0.042	1.02	0.13	0.063	0.105	300
			0.86	0.56	0.036	0.93	0.12	0.075	0.111	

Luiz Torres de Miranda,
Luiz Eugenio Coelho de Miranda and Toshio Igue

Allometric genetics and reciprocal translocation backcrosses for general mapping

In the preceding report we completed the protocol for a thorough analysis involving p, recombination values and α and β, the allometric coefficients of the effects of genes *A* and *B* and the variance of all parameters. To evaluate the potential value of the procedure, analysis was illustrated in actual numbers with data from D.L. Langham (Genetics 25:88) involving F2 data between *pd* and *tr* and standard markers in crosses of maize with Durango teosinte. Searching for another batch of data with the widest possible interest we analyse here backcross data involving translocations *T wx* reported by E.G. Anderson, et al. (MNL 39:106-109, 1965). We picked only those which are in the waxy and nonwaxy reciprocal translocations listed in MNL 61:113, which are a standard set. The results are presented in Table 1. The p values were calculated by the Product Ratio method, by maximum likelihood and by our allometric method. Our method gives p values between the others, nearer to the maximum likelihood, and the p values are always smaller in the normal cases. But most important are the even smaller variances, so that a much more exact pooling of different sets of data can be arrived at by weighting the invariances.

There is no sign of allometrical effects indicated by the t values. In other words, semi-sterility affects equally all

Table 1. In this table are analyzed the data with those *wx* T9 translocations reported by Anderson et al. in MNL 39:106-109, 1965 that are listed in the standard translocations from the Cooperation Stock Center in MNL 61:113.

Translocations	Break point 9 / other	Parental classes a / b	d / c	p % Product Ratio	max lik+ allo metry+	% $V_{(p)}$ / $V_{(p)}$	a / β	$V_{(\alpha)}$ / $V_{(\beta)}$	to / tβ
1 wx 1-94995	S.20	264	264	1.82	2.58	0.464	-0.097	0.096	0.31
	L.19	12	2		2.23	0.325	0.097		0.31
2 wx 1-98389	L.13	182	144	3.17	3.26	0.936	-0.046	0.069	0.18
	L.74	4	7		3.27	0.911	-0.115		0.44
3 Wx 3-98447	L.14	100	106	4.16	4.19	1.867	0.005	0.084	0.02
	S.44	5	4		4.18	1.866	0.034		0.12
4 Wx 3-98562	L.22	63	67	3.63	3.70	2.639	-0.004	0.151	0.01
	L.65	3	2		3.68	2.605	0.046		0.12
5 Wx 4-9e	L.26	118	117	4.00	4.08	1.597	0.023	0.078	0.08
	S.53	4	6		4.05	1.594	-0.029		0.10
6 wx 4-9g	L.27	77	83	3.81	4.19	2.404	-0.033	0.130	0.09
	S.27	5	2		4.00	2.189	0.084		0.23
7 wx 4-95657	S.25	135	104	1.44	1.65	0.668	-0.035	0.202	0.08
	L.33	1	3		1.60	0.607	-0.149		0.33
8 Wx 5-94817	S.17	80	70	39.57	39.52	9.368	-0.028	0.024	0.18
	L.69	49	49		39.61	9.391	-0.028		0.18
9 Wx 5-98386	S.13	407	391	3.15	3.51	0.410	0.045	0.031	0.26
	L.87	8	21		3.33	0.383	-0.073		0.41
10 wx 7-9a	S.07	262	276	1.27	1.47	0.265	0.074	0.098	0.23
	L.63	2	6		1.39	0.243	-0.037		0.12
11 wx 7-94363	7 ctr	182	169	1.26	1.68	0.463	0.056	0.182	0.13
	9 ctr	1	5		1.51	0.376	-0.108		0.25
12 Wx 9-108630	S.28	121	136	1.33	1.15	0.437	0.097	0.199	0.22
	L.37	0	3		1.46	0.537	-0.015		0.03

four classes a, b, c, d. The Product Ratio method exaggerates the strength of linkages, underestimating the error variances at the tight p values.

The mean of the 12 p values gives p = 5.91. In MNL 61:34-35 using our whole set of genetricks we obtained a mean p = 7.54 for 12 estimates of the standard *T wx* set, although between them only 2 are common. Of these, in 10 cases we believe that, more probably than not, the correct orientation was arrived at using information from some of our other reports. Perhaps a more realistic comparison is made by taking out 9L.10;5L.14, giving then p = 6.31 (it was deleted from the standard set). For the present set S.17;L.69 should then be taken out, and now gives p = 3.29 (it should also be discarded from the standard set). Taking into account also the common segregation for *Krn* factors, we can in many cases increase considerably the efficiency of the process of mapping as we have done in prior works. We think the correct mapping of *T wx* (and *Krn* factors) of utmost interest for greater marginal returns by getting the second approximation to the correct position in one strike.

Luiz Eugenio Coelho de Miranda
and Luiz Torres de Miranda

The mapping of ghost genes: Chromosome two *krn2(tr2)* and the Michoacán 21 latente supergene

In the MNL we have been reporting a series of tricks to detect and analyse "invisible" genes. We will try to show by cross-checking the relative effectiveness of the procedures used, summing up the available evidence for *Krn2 (tr2)*, *lte1* and *Lsc*. The independent bits of information are as follows. J.S. Rogers (Genetics 35:541) reported linkage of *lg1* with a *tr* factor. P.C. Mangelsdorf (Corn, its Origin and Improvement) reported linkage of *lg1* with a *tr* factor. W.C. Galinat (D.B. Walden, ed., Maize Breeding and Genetics pp. 99-100) reported a *tr* factor between *lg1* and the centromere. Miranda et al. (MNL 61:32), analyzing D.H. Langham's data reported a loose linkage between *lg1* and *tr* but commented that the distance was grossly overestimated by direct measurement. Miranda et al. (MNL 60:35) placed *Krn2* at 23, with B at 49. Miranda et al. (MNL 60:29) calculated the distances *Krn* 14.1 *Lsc* 32.9 *fl*, which gives on the map *Krn2* at 21, *Lsc* at 35, and *fl* at 68. Miranda et al. (MNL 58:47) placed *lte1* at 30, and *B* at 49.

By the pooled evidence there is a *tr2 (Krn2)* gene about position 22, most probably within the *lg1 gl2* span. These are also the best prospective markers for the latente-1 locus, which is at about the 30-35 position in 2S.

These results also illustrate the use of *Krn* factors in general mapping. If it were not for the eerie precision of *Krn2* mapping we would not have so much confidence in the latente gene's position.

Regarding the *tr pd* phenomenon makes one remember one Iberic proverb which, translated to English, goes more or less as "I don't believe in sorceries but it exists, exists".

Luiz Torres de Miranda
and Luiz Eugenio Coelho de Miranda

CANBERRA, AUSTRALIA
CSIRO

Genetics of instability at the rust resistance (*Rp*) locus

There are at least 14 alleles known at the *Rp* locus (resistance against *Puccinia sorghi*) located at the tip of the short arm of chromosome 10 (R.K.S. Saxena and A.L. Hooker, PNAS 61:1300, 1968). Eight are unstable, giving rise to susceptible variants designated *Rp'* alleles (T. Pryor, MNL 61:37, 1987) in crosses of the following general type:

$$\frac{Rp\text{-}x}{Rp\text{-}x} \times \frac{rp}{rp} \longrightarrow \underset{\text{Resistant}}{\frac{Rp\text{-}x}{rp}} + \underset{\text{Rare susceptible}}{\frac{Rp'\text{-}x}{rp}}$$

The frequency of the event appears to be allele specific and varies at least 100-fold between the extremes. However, the frequency with which a given allele produces susceptible variants may vary 5-10 times in different genetic backgrounds.

Allele	Frequency range of instability
Rp-g	0.002 to 0.014
Rp-d	0.0001 to 0.0002

The *Rp'* alleles do not revert or alter to a form that confers resistance. No second-time resistant variants were observed in 11,716 progeny of *Rp-g'* or from 30,357 *Rp-d'* F1 test progeny, which were all susceptible.

No known transposable element system is associated with this event and the following crosses were designed to investigate the genetic nature of the event at the *Rp* locus that gives rise to this high frequency of susceptible *Rp'* variant alleles. All progeny were screened with the rust race R-1, which recognises the *Rp-g* and *Rp-d* alleles but is virulent (compatible) on the alleles *Rp-c* and *Rp-m* (Conversely the presence of the *Rp-c* and *Rp-m* alleles can be recognised with race R-2-1, which is virulent on *Rp-d* and *Rp-g*).

Cross 1: To measure and confirm instability of *Rp-g*, *Rp-g*/*Rp-g* × *Rp-m*/*Rp-m*:

$\frac{Rp\text{-}g}{Rp\text{-}m}$	$\frac{Rp\text{-}g'}{Rp\text{-}m}$	*Frequency*
322	2	0.006

Cross 2: To measure linkage of *Rp* to *Oy* (Oil yellow seedling), *Rp-g* +/*Rp-c Oy* × *Rp-m*+/*Rp-m*+:

Rp-g +	*Rp-g Oy*	*Rp-c* +	*Rp-c Oy*	
Rp-m+	*Rp-m* +	*Rp-m* +	*Rp-m* +	T
65	10	6	64	150

Recombination *Rp* to *Oy* = 11%

Cross 3: To measure recombination in the region *Rp* to *Oy* during the production of *Rp'* alleles, *Rp-g* +/*Rp-d Oy* × *Rp-m* +/*Rp-m* + (25 ears):

	Resistant			
	Green		Oil Yellow	
Proposed genotypes	*Rp-g* + / *Rp-m* +	*Rp-d* + / *Rp-m* +	*Rp-g Oy* / *Rp-m* +	*Rp-d Oy* / *Rp-m* +
	1,969		1,985	

Susceptible		Oil Yellow	
Green	Oil Yellow	lethal	T
Rp-g' + / *Rp-m* +	*Rp-g' Oy* / *Rp-m* +	? / *Rp-m* +	
28	0	1	3,982

Unlike previous crosses with the *Rp* alleles homozygous, this cross demonstrates that the event producing the *Rp'* instabilities occurs in heterozygotes. In the above analysis the proposed genotypes have been assigned on the assumption that in this small population it was the *Rp-g* allele which gave rise to the *Rp'* variants. The observed frequency of *Rp'* variants is 28/3,982 or 0.007, which is not substantially different from 0.006 observed in the control population (Cross 1) designed to measure the stability of *Rp-g* in a related background during the same crossing season. This is considerably higher than that expected from *Rp-d*. However, differences in genetic background are probably too large to conclude whether these values are meaningful. Another consideration is whether our expectation in Cross 3 should have been ½ that of Cross 1 since there is only one *Rp-g* allele.

All of the susceptible seedlings in Cross 3 were green, yet from the estimate of recombination *Rp* to *Oy* in Cross 2 in a sample of size 28 one would expect 3.08 recombinants with the *Oy* marker (based on 11% recombination). This suggests that the event at *Rp* giving rise to the *Rp'* variants may inhibit crossing over in an adjacent region. By analogy to the rosy locus analysis in *Drosophila* (A.J. Hilliker and A. Chovnick, Gen. Res. 38:281, 1986) this might indicate a crossing over event rather than gene conversion, but certainly doesn't eliminate other mechanisms such as intrachromosomal events. Since the event occurs in homozygotes an unequal crossover would be necessary to generate a novel form. The observed numbers are small (at the borderline of significance) and will be increased. The cross will be repeated with the *Oy* marker linked to the *Rp-g* allele with the expectation that the *Rp'* variants should be oil yellow. Ideally a distal marker for a three point testcross is required but this is not yet possible for the *Rp-g* allele.

Tony Pryor

Transposition of *Ac* in tobacco plants

The transposable element *Ac* has been used in maize to identify and clone specific genes (Fedoroff et al., PNAS 81:3825, 1984). The presence of the element within a mutated gene provides a tag by which the gene may be recovered for molecular analysis. Baker et al. (PNAS 83:4844, 1986) have shown that *Ac* will transpose in tobacco callus, thus extending the range of species in which *Ac* may be useful as a gene tag. Since many of the mutations which may be obtained by this method require whole plant screening, we have analyzed the behavior of *Ac* in tobacco plants regenerated directly after transformation with *Ac*.

The *Ac* element from *wx-m9* (Fedoroff et al., Cell 35:325, 1983) was inserted into the binary *Agrobacterium* vector pGA482 (An. Plant Physiol. 81:86) and transferred by leaf disk transformation (Horsch et al., Science 227:1229, 1985) into Wisconsin 38 tobacco. Thirty-five kanamycin resistant plants were obtained, 23 of which exhibited Southern hybridization patterns consistent with at least one unrearranged copy of *Ac*. This was assessed by the presence of a 7.5kb *Bgl*II fragment consisting of the 4.5kb *Ac* in a 0.25kb *Pst*I *wx* gene fragment (Fedoroff et al., 1983) and pUC8, a 2.7kb cloning vector (Vieira and Messing, Gene 19:259, 1982).

When *Ac* transposes from the initial site of insertion, a 3.0kb *Bgl*II fragment is created. This fragment was detected at a copy number of 0.5 to 1.0 copies per cell in 9 plants, and was just detectable (<0.1 copy) in a further 11 plants. Three plants showed no evidence of *Ac* transposition.

In order to determine whether the frequency of *Ac* transposition is related to the level of *Ac* transcription, we isolated RNA from 19 transformants with different *Ac* transposition frequencies and determined the level of *Ac* transcription by S_1 analysis (Weaver and Weissman, Nucl. Acids Res. 7:1175, 1979). All plants which showed transposition also showed transcription; however, no correlation could be made between the level of transcription and the relative intensity of the 3.0kb band. *Ac* transcripts were detected in 2 plants which were negative for *Ac* transposition, while one plant which was negative for transposition had no detectable transcription.

We are currently analyzing DNA from shoots which have undergone sequential regenerations from the same transformed root stock to determine whether the frequency of transposition is constant with time or if it changes during extended residence of the element within a genome.

B.H. Taylor, E.J. Finnegan and E.S. Dennis

CHAPEL HILL, NORTH CAROLINA
University of North Carolina
JOHNSTON, IOWA
Pioneer Hi-Bred International
URBANA, ILLINOIS
University of Illinois

Somatic instability of a mutable allele of *B-Peru* in different inbred lines

We are attempting to isolate the *Rf3* gene using transposon tagging; our plan is to use an *Rf3* homozygote containing a transposable element system as male parent onto *cms-S rf3 rf3* plants. The resultant rare sterile or semisterile plant is the potential recipient of an *Rf3* locus with an inserted transposable element. While making the stock constructions for these experiments, we noticed differences in the level of somatic instability of the mutable allele in a number of inbred backgrounds.

We used a mutable allele of *B-Peru* isolated by V. Chandler and V. Walbot. Although this allele, *b-Peru-mu5*, was derived from Robertson's Mutator material, it

contains neither a *Mu1* nor a *Mu1.7* element at the *B* locus (V. Chandler, MNL, this issue). It should be noted that expression of the somatic instability of this allele is very stable in backcrosses to *b r-g* tester stocks in a W23/K55 hybrid background (V. Chandler).

Our first crosses were onto B37 *cms-S* plants; the result was that very few ears exhibited the expected spotted kernel phenotype. In subsequent seasons we performed similar crosses onto 16 other inbred lines with *cms-S* cytoplasm. Four to eight ears from each cross were analyzed. Three basic classes of F1 progeny resulted. In class I, all F1 ears had only rare (2-3 per ear) spotted kernels. The inbred lines that gave this result were Oh51A, W64A, Tr, and Mo17. Class II progeny contained two populations of ears; those that had only rare spotted kernels and those with close to 100% spotted kernels. These inbred lines were 38-11, N28, K55, R138, B37 and B84. The class III result was 100% spotted kernels on all F1 ears. The inbred lines giving this result were WF9, B73, M14, Va26, SK2 (all with pale spots) and R802A and Oh545 (deeply pigmented spots). The results were unaffected by the cytoplasmic type, e.g. four different *cms-S* cytoplasms in the N28 background gave the same result, and B37 *cms-S* and N (normal) maternal parents gave the same result. It should be noted that in all of our crosses the mutable parent was the male; it would be interesting to see the results of the reciprocal crosses considering the differential expression of somatic instability reported with a *bz2-mu1* allele using reciprocal crosses (V. Walbot, Genetics 114:1293, 1986).

Our results suggest that different inbred lines contain factors which influence the expression of somatic instability of the *b-Peru-mu5* allele. The most puzzling is the class II result, in which two types of ears were produced in the F1. We will test the heritability of this result in future studies.

P. Bedinger, M. Albertsen, S. Gabay-Laughnan
and J.R. Laughnan

CHESTNUT HILL, MASSACHUSETTS
Boston College

Meiotic chromosome fusions in the progenies of anther culture-derived plants

Chromosome fusions in prophase I of meiosis were observed in the haploids, aneuploid and dihaploid of the anther culture-derived R0 plants (first generation) (Ting, Maydica, 1985). In order to know more about this phenomenon, meiotic studies were carried out on the R1 and R2 plants of King Huang-13. All of these plants were dihaploids. It was interesting to find that chromosome fusions during the first meiotic prophase were not unlike what were reported previously. For certain sporocytes, the 10 bivalents were fused into a big chromatin mass. For the others, the 10 bivalents might form 3 or 4 separate groups. Identification of the individual bivalents in any of the groups was impossible. It was certain, however, that no multiple translocations were involved. At harvest, seedsets of all the above plants studied were close to 100%. Hence, it is reasonable to conclude that the fusions did not affect plant fertility.

Y.C. Ting and Stephen Schneider

Selection for stable inbreds from the progenies of anther culture-derived plants

The first generation (R0) of King Huang-13 showed great variability in plant height, despite the uniform appearance of all the other identifiable characters (Ting, Maydica, 1985). After 2 years of self-fertilization and selection for vigorous and tall plants, the progeny plants became uniform in height. If the segregation in height of the R0 plants was due to a tissue culture activated *Ac* element, it was probably lost in the subsequent generations. Since the selection was for normal, tall plants, those without *Ac* were perhaps favored.

In contrast to the above, selections for dwarf plants and self-pollinations of dwarf plants were also attempted. However, seedsets of all of them were very poor. A further study on the segregation for plant height of these progeny plants was impossible.

Y.C. Ting and Stephen Schneider

Effect of gibberellic acid on growth and regeneration of callus

During last fall, our callus lines SAN_1, SDN_1 and 81-B_5B were treated with varying amounts of gibberellic acid (GA) ranging from 0.1 to 5.0mg/l. The GA was mixed with regeneration media having 2% sucrose. Each treatment had at least 5 replicates and 3 controls. It was found that cultures of SAN_1 and SDN_1 containing 1 to 2 mg/l GA produced more prolific growth with higher chloroplast content than those with the other treatments. In addition, these cultures also had some regenerations. A few of the calli had 2 to 4 somatic embryos. These embryos, however, did not continue development and differentiation. Most of them stopped at the leaf primordium stage. For callus line 81-B_5B, no positive responses were observed.

Y.C. Ting and Stephen Schneider

COLD SPRING HARBOR, NEW YORK
Cold Spring Harbor Laboratory

Extrachromosomal *Mu* elements

In a recent study we showed that the *Mu1* and *Mu1.7* elements of Robertson's Mutator lines can be detected as extrachromosomal DNA circles in active *Mu* lines. (V. Sundaresan and M. Freeling, PNAS 84:4924, 1987). We have now found that several other *Mu1* homologous elements appear also to be present as extrachromosomal DNA circles based on hybridization to isolated circular fractions in Southern blots. A library of the extrachromosomal circular DNA from an active *Mu* line has been constructed and three clones that exhibited *Mu1* homology have been characterized. They were found to contain inserts of 2 kb, 1.4 kb and 0.9 kb, respectively, that exist as multi-copy sequences in the chromosome. The structures of the cloned sequences suggest that they carry the inverted repeat termini of *Mu1* with internal sequences non-homologous to *Mu1*. Elements with this structure resemble the "endogenous *Mu*" elements that have been observed in both *Mu* and normal lines (C. Rivin, et al.; MNL 61:54, 1987).

V. Sundaresan

Temperature effect on leaf striping in *Mu* lines

Many researchers have observed variable clonal striping on leaves within lines of *Mu* plants (D.S. Robertson, MNL 55:2, 1981; M. Freeling, Ann. Rev. Plant Phys. 35:277, 1984). These variable clonal stripes are a type of dominant somatic leaf striping seen in active *Mu* plants. Most of the stripes are small, although occasionally a white or yellow clonal stripe may be wide enough to cover from ⅛ to ¼ of a single leaf. In general, inactive derivatives of *Mu* lines do not exhibit such clonal striping (D.S. Robertson, MNL 57:4, 1983; V. Sundaresan, unpublished data). If the DNAs from the striping sectors and from the normal green tissue from the same leaf are examined by Southern blot analysis, differences in the pattern of *Mu*-hybridizing restriction fragments are observed (M. Alleman, Ph.D. Thesis, U. of California, Berkeley, 1984). These observations suggest that the clonal stripes result from the activity of the *Mu* elements. Robertson (op. cit.) has observed that there is considerable variability in the striping exhibited within the same *Mu* lines when grown at Berkeley or at Ames, with the striping observed at Berkeley being significantly greater. He has suggested that this difference might result from the lower temperatures in Berkeley. Further investigation showed that lower temperatures did in fact cause increased striping in *Mu* plants, (D.S. Robertson, MNL 56:2, 1982); however, this appeared to be true for some normal plants as well, and therefore it was not clear that *Mu* activity was influenced by low temperature. In this investigation, we re-examine the effect of temperature on *Mu* activity. In the experiment, three identical growth chambers were set up at three different temperatures and plants were grown in the chambers at mean temperatures (day + night/2) of 19 C, 24 C, 29 C. The warmest chamber was actually started at 34 C for two days after planting, and then turned down in order to avoid heat damage to the seedlings. There were a total of twelve maize lines used, including 282 plants that were distributed among the three temperatures; this included active *Mu* lines from Freeling (*Mu*, 1s2p) and Robertson (Pl *Mu*) stocks, and inactive derivatives of these lines that served as controls. All of the active lines displayed a high degree of clonal striping at the coldest and warmest temperatures but striping at the intermediate temperature was infrequent. By contrast the inactive *Mu* lines exhibited little or no striping at any temperature. The actual breakdown of the number of plants that had clonal striping at each of the temperatures is shown in Table 1.

Table 1. Ratio of striped plants to total plants.

Line	Activity*	Pedigree	19°C	24°C	29°C
A	+	Pl *Mu*	2/5	0/5	2/5
B	-	*Mu*, 1s2p	0/13	0/13	2/39
C	+	*Mu*, 1s2p	6/8	2/9	3/5
D	+	*Mu*, 1s2p	4/15	1/16	5/14
E	+	*Mu*, 1s2p	1/5	0/6	0/3
F	-	Pl *Mu*	1/3	1/5	0/6
G	-	Pl *Mu*	0/4	0/4	0/4
H	-	*Mu*, 1s2p	0/4	0/4	0/5
I	+	Pl *Mu*	2/4	0/4	0/4
J	+	*Mu*, 1s2p	3/6	0/4	0/6
K	+	Pl *Mu*	1/5	0/6	2/6
Total for Active *Mu* lines			19/48	3/50	12/43
Total for Inactive *Mu* lines			1/24	1/26	2/54

* + = active *Mu*
- = inactive *Mu*

From the data, which include all plants tested in 1987, there was only one obvious trend, which was the consistent appearance of clonal stripes in active *Mu* plants in the warmest and coldest chambers. These results, though not complete, suggest that Mutator activity is greater at temperature extremes. The results obtained in this study could be viewed in the light of McClintock's hypothesis of "genome shock". McClintock (Science 226:792, 1983) has suggested that the activity of transposable elements could be regulated by stresses to the organism and that such stresses could serve as a mechanism for the reorganization of the genome. An increase in Mutator activity when plants are grown under sub-optimal conditions would be consistent with this hypothesis.

Kevin Nelson Heller

A change in state of the *Ac*-induced *P-VV* allele is associated with inversion of *Ac*

The maize *P* locus controls pigmentation of the pericarp and glumes of the cob. The *P-VV* allele, which gives variegated pericarp and cob, comprises the transposable element *Ac* situated at the *P* locus. I have isolated an allele termed *P-OVOV* (orange variegated pericarp and cob) derived from *P-VV*. The *P-OVOV* allele was recovered from a sector of about 30 kernels with orange variegated pericarp on an ear with otherwise ordinary variegated pericarp, from a plant of *P-VV/P-WR* genotype. *P-VV* specifies colorless pericarp with red sectors, whereas *P-OVOV* specifies orange pericarp with many dark red sectors, and some colorless sectors. Thus, *P-OVOV* represents a change in state of the *P-VV* allele.

Two hypotheses have been proposed to explain the molecular basis for changes in state. The composition hypothesis attributes changes in state to changes within the transposable element, such as internal deletions or DNA modification. The position hypothesis proposes that the element is unchanged, but that it has moved to a new position within the locus. The *P-OVOV* allele was studied to determine the basis of this change in state.

The genetic properties of the *P-OVOV* allele were investigated in progeny of the cross *P-OVOV/P-WR* × *P-WW*. For each progeny plant, ear phenotype was scored by inspection, and the presence or absence of *Ac* was determined in testcrosses. The results are in the following table:

	Ear phenotype			
	WR	OVOV	RR	Other
Ac +	2	109	5	2
Ac −	124	0	5	1
Totals:	126		122	

Three points can be drawn from the data. First, the number of *P-WR* ears (126) is approximately equal to the combined number of *P-OVOV*, *P-RR*, and variant ears (122). This shows that *P-OVOV* segregates as an allele of *P-WR;* the *P-OVOV* phenotype does not depend on unlinked modifying factors. Second, *Ac* activity is tightly linked to *P-OVOV*. 109 plants producing *P-OVOV* ears carried *Ac*; there were no examples of plants producing *P-OVOV* ears which did not carry *Ac*. Third, the *P-OVOV* allele is itself unstable, as evidenced by the *P-RR* and "other" ears among the progeny. These variants are presumed to arise from mutation of *P-OVOV*, because the *P-WR* allele used in this cross is stable.

Has the *Ac* element at the *P* locus undergone a compositional change in the mutation of *P-VV* to *P-OVOV*? This possibility was tested by comparing the abilities of the *Ac* elements associated with the *P-VV* and *P-OVOV* alleles to *trans*-activate a *Ds* element. No differences in the *Ds*-response were seen, suggesting that the *trans*-acting functions of *Ac* are unchanged. These genetic results are similar to those reported by F.A. Valentine (Orange variegated, a mutant originating from variegated pericarp in maize. Unpublished Ph.D. thesis, Univ. Wisconsin Library, Madison, 1957).

Southern analysis of DNA from the progenitor *P-VV* allele and the *P-OVOV* allele shows that the *Ac* element associated with *P-OVOV* has remained within an 850bp genomic DNA fragment at the *P* locus. Interestingly, the *Ac* element is in an inverted orientation in *P-OVOV* compared to *P-VV*.

The genetic and molecular data are consistent with the following explanation for the origin and behavior of the *P-OVOV* allele. The progenitor *P-VV* allele comprises the *Ac* element inserted in a particular orientation at the *P* locus. The *P-OVOV* allele may have arisen by an event in which *Ac* has inverted, or flipped, from its orientation in *P-VV* to the opposite orientation. Inversion might have occurred by 1) short range transposition and reinsertion in an inverted orientation, 2) a specialized inversion mechanism of *Ac*, or 3) inversion mediated by sequences just outside *Ac*. In the inverted orientation, the *Ac* insertion may partially suppress *P* expression, resulting in a lowered level of *P* function, less pigment formation, and thereby an orange pericarp color. *Ac* can excise from *P-OVOV* to restore a *P-RR* gene, producing the red sectors on *P-OVOV* kernels and *P-RR* germinal revertants. The colorless sectors may result from other mutagenic *Ac* activities, such as imprecise excision, formation of deletions, or transposition of *Ac* to a new site within the *P* locus, where it may partially or completely suppress *P* expression.

These results suggest that the change in state of *P-VV* to *P-OVOV* may be due to inversion of *Ac*. A definitive explanation of how this inversion alters *P* expression awaits further information on the structure of the *P* gene.

Thomas Peterson

COLLEGE STATION, TEXAS
Texas A&M University

Regulation of gene expression by abscisic acid

The multiple pleiotropic effects associated with viviparous mutants in maize (D.S. Robertson, Genetics 40:745, 1955) suggest that these mutations affect expression of other genes, but the manner by which they do this has not been obvious. The xanthophyll-deficient mutants *vp2*, *vp5*, *vp9*, *ps*(=*vp7*) and *w3* all appear to have lesions in structural genes involved in carotenoid biosynthesis, and all of these mutants have similar pleiotropic effects, including reduced levels of the phytohormone abscisic acid (ABA).

The *vp* mutant has normal xanthophyll synthesis, and ABA levels in *vp*/*vp* embryos are in the normal range. It also has multiple pleiotropic effects but, with the exception of vivipary, the pleiotropic effects of *vp* appear to be different from those of the xanthophyll-deficient mutants.

We have previously shown that ABA, if applied at the appropriate time, can induce embryo dormancy in xanthophyll-deficient mutants. Initiation of embryo dormancy is very specific for stage 4 of embryogenesis about 15 days after pollination (DAP). Thus, if ABA is directly involved in the regulation of gene expression, it seems probable that some of these regulatory events should occur at about the same time that embryo dormancy is inducible by ABA.

Kernel sectors from the inbred Tx5855 develop normally when cultured in vitro, and they produce sufficient ABA to induce embryo dormancy. Both xanthophyll and ABA synthesis is blocked if sectors are cultured on medium containing fluridone. These kernels are viviparous and embryos contain near-zero levels of ABA. We incubated both types of kernel sectors for 24h on media containing ^{35}S-Met on a daily basis from 12-19 DAP, extracted proteins from embryo and endosperm, and compared flurograms of SDS-PAGE gels. No differences were observed in 12-15 DAP embryo or endosperm between normal and fluridone-treated kernels. However, in 16-19 DAP kernels several differences were detected in both embryo and endosperm tissue for newly synthesized proteins in the 32 to 45 kD range. These differences became more obvious as the kernels aged. None of these bands have been identified yet.

Scutellum tissue of normal maize kernels has essentially no lipase activity at any stage of kernel development. However, scutellum of both *vp* and *vp5* has detectable lipase activity at 16 DAP and high activity at 25 DAP. Activity is about 3 times as high in *vp* as in *vp5* at 25 DAP. The *vp* mutant has normal ABA levels, but it is ABA insensitive, while ABA levels in 25 DAP field-grown *vp5* are about 50% of normal. This suggests that both the *vp* gene product and ABA are required to suppress lipase activity during kernel development, and that the effect of ABA may be concentration-dependent.

Although these experiments are not definitive, they suggest that ABA has a direct or indirect effect on the expression of specific genes at specific stages of maize kernel development.

D.J. Hole, D.G. Bai, F. Fong, C.W. Magill
and J.D. Smith

Anatomy of the transfer cell region in corn

There is no vascular connection between the maternal plant and developing caryopses of corn. All nutrients entering the seeds are believed to pass through, by symplastic or apoplastic methods, a specialized group of cells called transfer or basal endosperm cells. Even though these cells are of primary importance in the process of grain filling, surprisingly little is known about their biochemical and physiological processes. During ongoing studies in our laboratories concerning the physiology of grain filling, it became apparent that the few previously published descriptions of transfer cells in corn were not adequately describing what we were seeing in our own anatomical investigations.

Sections were made, for examination under light and electron microscopes, of various stages of caryopsis development of Tx5855, starting at four days post pollination and proceeding until development of a black layer. Samples of grains at 23 days post-pollination were chosen to illustrate the structure of mature, functioning transfer cells. The samples were examined in two planes of section, one along the long axis of the cells and referred to as longitudinal, the other perpendicular to the long axis and referred to as cross section.

Figure 1. This transmission electron micrograph shows transfer cells in a longitudinal section, crushed nucellar cells, and pedicel parenchyma from a corn caryopsis 23 days post pollination. Note the basal transfer cells have extensive cell wall proliferation at the base and along the sides (unlabeled arrows) and that the centers are filled with cytoplasm. This gives these cells a goblet-like appearance (TC1, TC2). Other transfer cells are present in this layer that do not have the goblet appearance because the section plane is off median (TC3).
TC: Transfer cell; CC: crushed cells; PP: pedicel parenchyma

These two planes of section showed that the typical transfer cell had an extensive network of cell wall ingrowths, all of which were bounded by plasma membrane. In longitudinal sections (Fig. 1) the transfer cells had the greatest cell wall proliferation in the basal portions of the cells and progressively less toward the apices. The central regions of the cells were devoid of cell wall material and were filled with cytoplasm containing normal organelles including numerous vesicles and occasional crystals. The overall appearance of these cells was goblet-shaped.

In cross section the extension of the ingrowths from the primary cell wall could be easily observed (Fig. 2). One of the most striking features found in this plane was that wall ingrowths of contiguous cells often arose from a common site on the primary wall. In the areas between the ingrowths, numerous plasmodesmata were present.

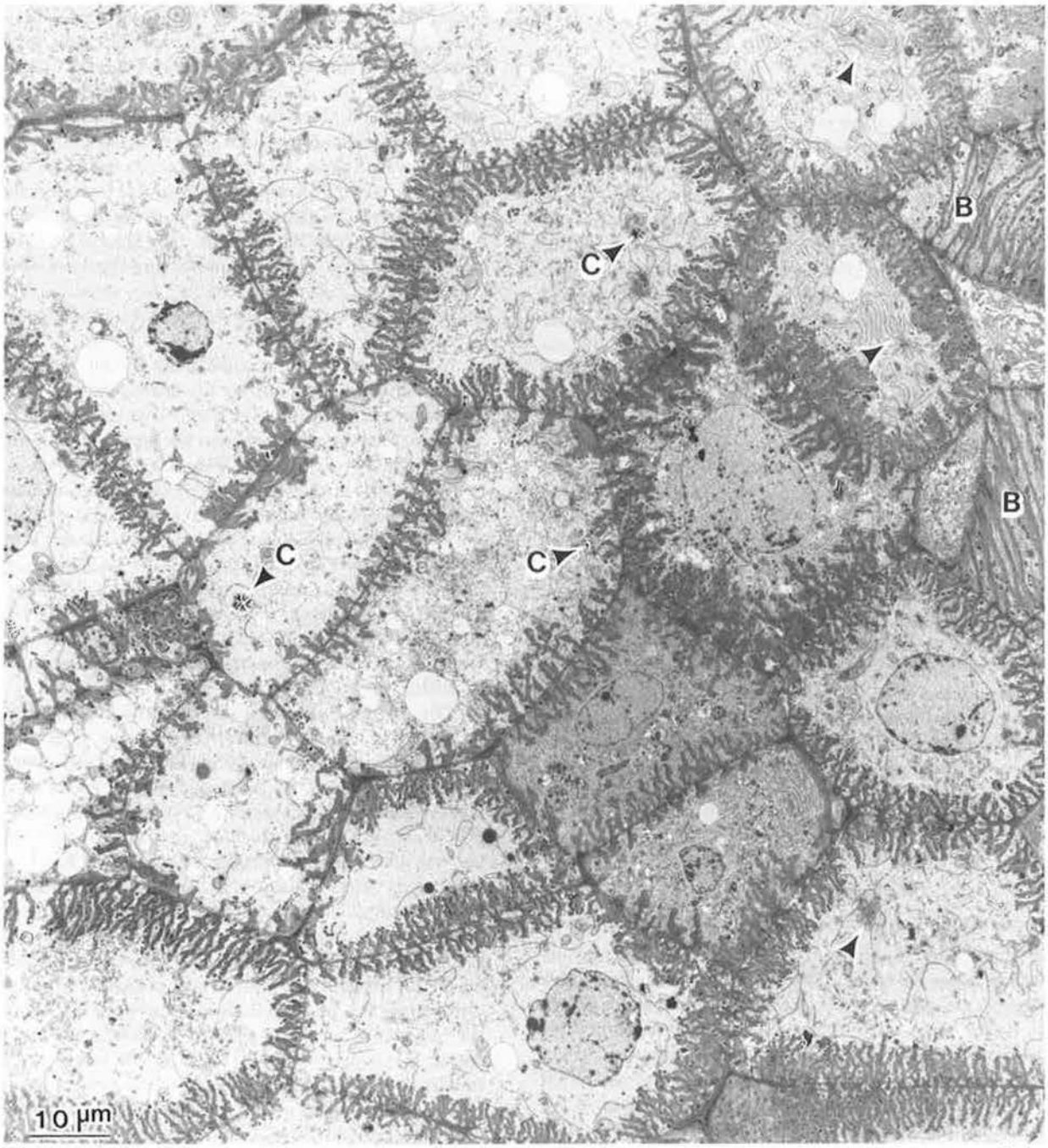


Figure 2. This transmission electron micrograph shows transfer cells in cross section. Note that most of the contiguous cells have wall extensions originating at common loci. This plane of section is probably through either the upper portion of basal transfer cells or through second level transfer cells. The basal portions of two second level cells are present (B). Some of the cells contain an unusual aggregation of endoplasmic reticulum (unlabeled arrows) and plastids with crystalline inclusions (arrows labeled C).

The most basal transfer cells had the greatest amount of cell wall proliferation. Evidence of wall extensions could be found in cells approximately 6 layers deep in the endosperm. Each successively deeper cell layer had less proliferation until there was a complete transition to typical endosperm cells. There was also a transition area at the lateral edges of the transfer cell zone where it meets the aleurone layer.

An hypothesis currently under our consideration is that the production of the transfer cell zone is initiated by a diffusible promoter, possibly a hormone. The primary effect of this substance is in the most basal endosperm cells. These are the first non-maternal cells that assimilates come in contact with. As the promoter substance diffuses inward it may have progressively less effect, accounting for the eventual transition from transfer to endosperm cells. The production of the aleurone layer is presumably also under hormonal control. The competition between the initiating substances for the aleurone and transfer cell zones might account for the transition zone where these tissues meet.

Ronald W. Davis, B. Greg Cobb
and J.D. Smith

COLOGNE, WEST GERMANY
University of Cologne

Analysis of the *sh-m6258* allele

sh-m6258 is one of the mutable *sh* alleles which arose when B. McClintock analysed transposition events of the *Ds* element located between *C-I* and *Sh*. Biochemical analysis of the *sh-m6258* allele shows that the *Sh* gene is interrupted by a long insertion of at least 45 kb. Cloning of part of this insertion including the 5′ and 3′ junction between *Sh* and insert DNA detects *Ds* elements on both sides of the insert. Structurally this insertion in *sh-m6258* is similar, though not identical, to the 30 kb insert found in the *sh-m5933* allele. Interestingly, the *sh-m5933* allele has the same genetical progenitor as the *sh-m6258* allele.

On one side of the insert of *sh-m6258* we find a single *Ds* element of approximately 2 kb. The other side carries a double *Ds* structure at the junction with the *Sh* DNA. This structure comprises one complete 2 kb *Ds* element and one half *Ds* element. The same construct was found on one side of the above-mentioned *sh-m5933* insert. This indicates to us that both alleles were formed by the same mechanism.

Cloning and partial DNA sequencing of a revertant allele of *sh-m6258* (isolated by N. Fedoroff, Baltimore) reveals the presence of a 2 kb *Ds* element at the location of the long insert described above. We are presently sequencing the *Ds* parts of the *sh-m6258* allele in order to understand what kind of excision or other rearrangement gave rise to the remaining *Ds* element in the revertant allele. The insertion site is located in the last but one intervening sequence of the sucrose synthase(*Sh*) gene. Thus, the 2 kb insert in the revertant does not necessarily interfere with gene expression.

Ingrid Pahl, Maria Durany
and Hans-Peter Döring

Analysis of *Ac* sequences required for transposition

We have been analyzing the sequences of the autonomous transposon *Ac* required for transposition in transgenic tobacco plants. We recently reported (Baker et al., MNL 1986; Baker et al., EMBO J. 6:1547, 1987) a phenotypic assay which allowed the selection of *Ac* excision events in tobacco. This assay involved the construction of an NPTII gene inactivated by the insertion, in vitro, of a copy of *Ac*. This inactivated gene was inserted into the T-DNA of *A. tumefaciens* and transferred to tobacco protoplasts. The presence of transformed tobacco calli resistant to the antibiotic kanamycin indicated *Ac* excision from the NPTII gene, restoring activity of the gene. We have now used this assay to study *Ac* transposition by inserting a series of in vitro constructed derivatives within the NPTII gene and determining whether they could excise. A preliminary report of these experiments was given by Coupland et al., MNL 1986.

The first type of *Ac* derivatives have suffered deletions within the DNA which encodes the long (600-700 b) untranslated leader of the *Ac* transcript. Two of these deletions extended from *Ac* coordinates (we number *Ac* from the *Bam*HI end, i.e. reversed coordinates compared to Müller-Neumann et al., Mol. Gen. Genet. 198:19, 1984) 356 bp to 920 bp (in plasmid pKU31) and from coordinates 245 bp to 736 bp (in plasmid pKU33). Both of these derivatives yielded Km^R calli at a frequency equal to or higher than that of *Ac* itself (in plasmid pKU3). As the deletions remove almost all of the untranslated leader, we conclude that this unusual feature of the *Ac* transcript is not required for expression of a product required for transposition. Indeed the deletions remove approximately 14% of the *Ac* element, which still transposes autonomously.

However, a deletion which removes the DNA encoding approximately 27 amino acids from the 5′-end of the open reading frame (in plasmid pKU9) prevents transposition. However, this deleted element does transpose in tobacco cells already containing *Ac*, indicating the deletion derivative could be complemented by an intact *Ac*, so the deletion must remove sequences required for expression of a *trans*-acting *Ac* product.

In order to confirm that the 3.5 kb *Ac* transcript encodes a product required for transposition, we cloned the open reading frame (ORF) of the transcript, present in a cDNA clone constructed from the partial clones of Kunze et al. (EMBO J. 6:1555-1563, 1987), between a plant promoter and a plant polyadenylation signal. The combined cDNA clone contained the entire ORF and 160 bp of the leader. The cDNA was cloned in the sense (in pKU38) and the nonsense (in pKU39) orientations. These constructions were then transferred to tobacco protoplasts which had previously been transformed with a T-DNA (from plasmid pGV3850::pKU4) which contained an *Ac* Δ element incapable of autonomous transposition stably inserted within an NPTII gene.

The *Ac* Δ element had lost the entire internal *Hin*dIII fragment of *Ac*. We have three lines of evidence to indicate that expression of the ORF in tobacco protoplasts allowed excision of the *Ac* Δ element: (i) A low number of Km^R calli were recovered after transformation of the pGV3850::pKU4 transformed protoplasts with pKU38, but not after transformation with pKU39. Km^R ought to occur only after excision of *Ac* Δ. (ii) The T-DNAs of pKU38 and pKU39 carry a Hygromycin resistance gene as well as the *Ac* ORF. Hy^R calli which had inherited the T-DNA of pKU38 or pKU39 were selected and then tested for NPTII expression by an in situ gel assay. None of 6 calli containing the pKU39 T-DNA expressed NPTII. Four of six calli containing the pKU38 T-DNA expressed NPTII. Again the presence of the ORF in the sense orientation results in NPTII expression. (iii) Southern analysis of DNA isolated from one Hy^R $NPTII^+$ callus transformed with the pKU38 T-DNA and the pGV3850::pKU4 DNA revealed a 2.9 kb *Eco*RI-*Hin*dIII fragment which hybridized to an NPTII gene probe. This fragment is expected only after excision of the *Ac* Δ element from the NPTII gene. This confirmed that NPTII expression in the callus was a consequence of *Ac* Δ excision.

The ORF must therefore encode a product required for *Ac* excision, and it becomes increasingly likely that this product is the only *Ac* product required for transposition, that is the transposase.

In addition we have tried to identify the sequences required at the ends of the *Ac* element for transposition. This analysis is most advanced at the end encoding the 5′-end of the transcript. The deletion of *Ac* sequences between coordinates bp 44 and bp 92 (in plasmid pKU19) and between bp 75 and bp 181 (in plasmid pKU28) prevent transposition even if *Ac* is present in the same cell. We conclude that these sequences are required for recognition

of the ends of the element by an *Ac* encoded and/or host proteins prior to excision. An active end of *Ac* clearly constitutes more than 75 bp, as pKU19 will not transpose, but less than 245 bp, as pKU33 excises autonomously. We have constructed a series of deletions progressing outwards from 245 bp towards the end of the element and have tested their ability to excise in tobacco protoplasts containing *Ac*. These experiments are in progress, but for full activity more than approximately 150 bp are apparently required at the 5'-end of the *Ac* element.

A similar series of deletions at the 3'-end are currently being transformed into tobacco protoplasts.

In addition we have constructed a plasmid which contains an *Ac* element in which the 3'-end of the element has been replaced by approximately 480 bp from the 5'-end. This element therefore contains a functional 5'-end at both ends. This element was unable to transpose in tobacco protoplasts containing *Ac*.

These experiments pose two problems: how does the transposase or other host proteins recognize, but distinguish between, the two ends of *Ac*, and secondly how does *Ds1* transpose when it only contains very short sequences of less than 20 bp at the ends which are directly homologous to the ends of *Ac*? We are currently pursuing these problems.

George Coupland, Christiane Plum, Barbara Baker, Jozef Schell and Peter Starlinger

In situ studies

We are studying the expression of the waxy and shrunken genes in developing maize kernels by in situ hybridization, and the expression of both sucrose synthase genes by in situ immunofluorescence. Regarding the expression of the sucrose synthase, we see a tissue-specific expression of the two sucrose synthase molecules. Sucrose synthase 2 appears earlier, and is strongly represented in the aleurone and subaleurone cell layers. This enzyme is seen later and in small amounts in the endosperm (as judged by its appearance in *sh bz-m4* mutants). Sucrose synthase 1 is found mainly in the endosperm. More of the enzyme is seen in the apex than in the basal parts, the enzyme thus forms a gradient, which is also seen at the mRNA level by in situ studies.

Both techniques are used to detect sectors of revertant cells after the excision of *Ac* or *Ds* elements from mutated genes. The reversion events of *sh-m5933* occur only after 30 days and are predominantly single cell sectors in the interior of the endosperm. The reversion of *wx-m7* leads to larger sectors that appear earlier and are distributed more evenly over the kernels. The reasons for this seeming discrepancy are being investigated.

Manfred Heinlein, Ralf Link and Peter Starlinger

Molecular analysis of the *P* locus

We have continued our work on the structure of the *P* gene. We have isolated a 14 kb *Eco*RI clone extending from the *Eco*RI site in *Ac* to an *Eco*RI site located in the direction of the 5'-end of the *Ac* transcript. Near the end of this clone, a 1.2 kb *Sal*I fragment is located that is seen in Southern hybridizations of genomic DNA of *P-vv, P-rr* and a revertant from *P-vv* to *P-rr*.

From a *P-rr* library, two *Mbo*I clones of 13.5 and 14 kb length were isolated that overlap with the DNA sequence located beyond the 3'-end of *Ac* on the original 8 kb *Sal*I fragment. This clone does not contain any further sequences hybridizing to the three *Sal*I fragments of 1.2 kb, 3.0 kb and 3.4 kb length hybridizing to the DNA flanking the *Ac* insertion in *P-ww*.

A cDNA was cloned from a library constructed from pericarp RNA of *P-rr* kernels 24 days after pollination. It is 503 bp in length and is supposed to represent the 3'-terminus of (a) larger transcript(s). It is nearly 100% homologous to a genomic region located 2 kb 3' of the *Ac* insertion site. The direction of transcription is determined by this cDNA clone.

Using the cDNA sequence in a single strand specific M13 vector as a probe in the RNA analysis of different *P* alleles, two large *P-rr* and *P-revertant* specific transcripts of 7 kb and 12 kb in size were detected. These are not found in *P-vv* and *P-ww* derived RNA. The two transcripts were also detected with a single strand specific probe derived from a 400 bp fragment located directly 5' of the *Ac* insertion site. The same probe also detects an 8 kb and very weak >12 kb transcript in the RNA of *P-vv*. These transcripts were also detected by a single strand specific probe derived from *Ac*.

They represent chimeric molecules consisting of *Ac*- and *P*-derived sequences The direction of transcription of the *P* gene and *Ac* is identical in *P-vv*. The data are in agreement with the hypothesis that the gene is terminated at the location of the polyadenylation of the *Ac* transcript (Kunze et al., EMBO J. 6:1555, 1987). In support of this hypothesis, the transcripts from *P-vv* do not hybridize to the cDNA clone.

It is noteworthy that this result excludes the 1.2 kb *Sal*I band located on the other side of *Ac* from these transcripts and that no other transcripts hybridizing to this DNA have yet been detected.

Christa Lechelt and Peter Starlinger

Studies on the *Ac* protein

We have continued our studies on the *Ac* protein. We detect a specific reaction in Western blots with a protein (apparent molecular weight of 110 kD) with two antisera prepared against fusion proteins containing separate segments from the long ORF of *Ac* in *E. coli*. We detect the same protein in *Spodoptera frugiperda* cells infected with a Baculovirus vector containing a cDNA copy of the *Ac*-coding region. In the insect cells, the protein is 500-1000 times more abundant than in maize cells. Degradation of the protein leads to a pattern of peptides reacting with our antisera. Those degradation bands visible in the maize extracts can also be seen in the degradation pattern in the protein from the insect cells. This supports the hypothesis that the reactive protein in maize cells that is found in nuclei from *Ac*-carrying, but not from *Ac*-free cells, is indeed the *Ac* protein.

The predicted molecular weight of this protein is only 92,000. However, it is known that other nuclear proteins also migrate more slowly than expected on the basis of their predicted molecular weights.

Heidi Fusswinkel, Markus Müller-Neumann, Charlotte Both, Walter Doerfler and Peter Starlinger

In vitro analysis of protein - DNA - interactions between the sucrose synthase gene promoter and nuclear factors

The shrunken gene on chromosome 9, coding for the enzyme sucrose synthase, is regulated during plant development. The transcription rate is specific for different organs of the plant and the gene is inducible by anaerobic stress (Springer et al., 1986). We suppose that nuclear factors should bind to cis-acting sequences in the vicinity or within the gene to regulate the level of transcription.

In gel retardation experiments, using crude nuclear extracts of immature kernels and different promoter fragments, spanning the region between -1592 and +393 (with regard to the transcription start), we have observed multiple protein-DNA interactions. Two major binding activities (called MNP 1 and MNP 2 for maize nuclear protein) seem to recognize multiple binding sites distributed over the 1.5 kb upstream region of the shrunken gene, which can be concluded from competition experiments with heterologous unlabelled promoter fragments and is supported by DNase I footprint experiments or sequence homologies. At the moment we are trying to purify these factors by affinity chromatography.

Jörg Schürmann, Boris Springer, Thomas Lugert, Regina Bellmann and Wolfgang Werr

Quantitative analysis of the sucrose synthase gene promoter by transient gene expression in protoplasts

The shrunken gene, encoding the enzyme sucrose synthase, has been cloned and characterized in detail (Werr et al., EMBO J. 4:1373, 1985). The expression of this gene is regulated on the transcriptional level. High transcript levels accumulate in the endosperm of developing kernels. Reduced levels of transcript are detected in roots, leaves and shoots of etiolated seedlings. The transcription rate of this gene can be increased 10-20 fold upon anaerobic stress (Springer et al., Mol. Gen. Genet. 205:461, 1986).

To analyse the shrunken gene promoter for cis-regulatory elements, we established a transient gene expression system in protoplasts of a suspension cell line (BMS, kindly provided by Horst Lörz). By Northern experiments we verified that the internal sucrose synthase gene is expressed in this cell line. The level of transcript can be increased 10-15 fold by anaerobic treatment. Therefore this cell line is suitable to test for the influence of promoter deletions on the transcription rate of the plasmid pSKAN 1 (Werr and Lörz, Mol. Gen. Genet. 202:471, 1986). This plasmid contains a 2 kb promoter fragment of the shrunken gene including the transcription start fused to the bacterial NPTII-coding region of Tn 5. A set of deletion mutants, shortening the 2 kb promoter fragment from its 5'-end towards the transcription start, was constructed and transfected in maize protoplasts. NPTII levels were measured, differences in the efficiency of individual transfections were estimated by cotransfection with a 35s CaMV-CAT gene and used to correct NPTII levels. The functional map so far obtained indicates that multiple regulatory sequences, spread over the entire 2 kb promotor fragment of the sucrose synthase gene, can influence transcription in a positive and negative way.

We will also use this system to identify regulatory element(s) involved in anaerobic induced or tissue specific expression of the sucrose synthase gene.

Christoph Maas and Wolfgang Werr

COLOGNE, WEST GERMANY
Max-Planck Institute für Züchtungsforschung

Cloning of the transposable element *Mpi1* from *c2-m3*

The cloning of the *c2* locus (U. Wienand et al., Mol. Gen. Genet. 203:202, 1986) has enabled us to characterize molecularly the transposable element insert at the *c2-m3* allele. This transposable element is not capable of activating receptors of other known transposable element systems like *En* (*Spm*), *Dt, Uq, Cy, Mrh, Bg* and *Fcu* (B. McClintock, personal communication; P. Schnable, personal communication; P.A. Peterson, MNL 58:3, 1984).

Southern analysis of DNA carrying the wildtype, *c2-m3* and *c2-m3* revertant alleles using various *c2* specific probes demonstrated that this element (here termed *Mpi1*) is 9kb in size and is integrated in the intron of the *c2* locus. Eight kb of the *Mpi1* were cloned and analyzed (attempts to clone the right side of the element were unsuccessful). The left end of the element starts with the sequence CACTA (characteristic of the CACTA-family of elements, *En1, Tam1, Tam2, Tgm1;* A. Pereira et al., EMBO J. 5:835, 1986; U. Bonas et al., Mol. Gen. Genet. 194:138, 1984; K. Upadhyaya et al., Mol. Gen. Genet. 199:201, 1985; L.O. Vodkin et al., Cell 34:1023, 1983) and includes many direct and indirect repeats within the first 300bp.

Transcript analysis of wildtype and *c2-m3* polyA+ RNA (25 days after pollination) using a 5kb and a 3kb *Eco*RI fragment of *Mpi1* revealed a 2kb-long *Mpi1*-specific mRNA hybridizing to the 3kb fragment.

The *Mpi1* element is a member of a family of low repetitive elements: only 10-15 copies could be detected in the maize genome.

Ulrike Weydemann, Udo Wienand, Ulla Niesbach-Klösgen, Peter A. Peterson[1] and Heinz Saedler

[1]Ames, Iowa, Department of Agronomy

Molecular analysis of somaclonal variants

We have described earlier (Goebel et al., MNL 1986) the surprisingly large number of phenotypic distortions that were found in maize plants of the line A188 regenerated via somatic embryogenesis. In an attempt to characterise the molecular basis for this variation, we have undertaken a series of experiments to determine whether, for example, gene methylation status is altered as a consequence of tissue culture stress and whether somaclonal variation can be related to methylation changes.

One of the first experiments was to compare methylation status in phenotypically normal and abnormal plants. By digestion with methylation-sensitive enzymes such as *Msp*I, HpaII, *Dpn*I, *Sau*3A and *Mbo*I and subsequent probing with known gene sequences, the results showed that no correlation exists between obvious symptoms of somaclonal variation and methylation status. Plants which appear to be phenotypically normal can be shown, with different genes, to be methylation-abnormal. Conversely, dependent on the gene used, phenotypically abnormal plants can appear to be genetically identical to controls.

We further characterised plants by producing 'lines' of plants; i.e., the progeny of plants all derived from a single embryo, and subsequently compared methylation status not only between 'lines' (i.e., different embryo-derived plants) but within the progeny of the same line. Plants have been examined for up to 5 generations. Although culture conditions were identical for all the original calli, highly significant differences have been found, not only between sibling plants but between lines derived from different embryos on the same callus, as well as plants from embryos produced on different calli. The only consistent pattern appeared to be within the progeny of plants. Therefore, once the methylation status has been changed, then the alteration appears to be stable for the subsequent generations.

As methylation sequences are also assumed to be mutational 'hotspots', RFLP analysis is also being undertaken in an attempt to further characterise genetic alterations. Once again, the amount of variation was significantly much greater than would have been expected. In an attempt to determine which factors during tissue culture are responsible for inducing these mutations, different components of the procedure are being examined, both on callus cultures as well as on direct regenerants. As such, we have found that highly significant differences in genetic status can be produced by the media, some of the more widely used media being considerably more mutagenic than others. There is also evidence emerging for genetic alterations occurring during the process of regeneration from callus to plants, as opposed to induction from immature embryos.

The amount of variation that we have found so far is considerable and has led us to suspect that this may be a reflection not just of tissue culture per se, but a characteristic of the line A188. This work is now being repeated with a number of different maize lines as well as with rice, enabling us to determine what differences, if any, can be found between protoplast and callus derived plants.

Peter Brown, Elke Goebel and Horst Lorz

COLUMBIA, MISSOURI
University of Missouri and USDA-ARS

The production of the telocentrics of maize

Telocentric chromosomes are formed when unpaired centromeres (as found in trisomes) become attached to spindle fibers going to opposite poles. The method used to isolate the telocentric chromosomes of maize is to examine the self-fertilized progeny of primary trisomes for deviant genetic ratios.

A self-fertilized duplex (*A*/*A*/*a*) primary trisome has a genetic ratio of about 17 *A* : 1 *a* and that of a simple (*A*/*a*/*a*) trisome is about 2 *A* : 1 *a*. These ratios are affected by several factors; the amount of double reduction (which varies primarily with the linkage of the marker gene with the centromere), the loss of univalent chromosomes during meiosis, the transmission frequency of disomic gametes through the pollen, and the relative viability of euploid and aneuploid zygotes. Data are given in Tables 1 and 2.

A self-fertilized duplex (t*A*/*A*/*a*) telocentric trisome has a ratio of about 7 *A* : 1 *a* and a self-fertilized simplex (t*A*/*a*/*a*) has a ratio of about 1 *A* : 1 *a*. Actually there is

Table 1. Genetic ratios of self-fertilized duplex trisomes.

Trisome	Number of plants tested	Number of gametes tested	Marker gene	Percent recessives
1	2	303	bz2	5.28
2	7	618	lg	7.93
2	9	766	gl2	8.48
3	18	4,564	sh2	6.64
4	45	13,095	su	7.65
5	13	4,087	bt	8.73
6	7	1,725	y	7.94
7	12	990	gl	7.57
8	6	720	v16	9.72
9	4	752	bz	9.84
10	-	-	-	-

Table 2. Genetic ratios of self-fertilized simplex trisomes.

Trisome	Number of plants tested	Number of gametes tested	Marker gene	Percent recessives
1	3	1,011	bz2	33.14
2	28	2,856	lg	36.41
2	13	1,298	gl2	40.14
3	15	3,023	sh2	38.14
4	57	16,019	su	35.93
5	41	10,913	bt	37.18
6	13	4,124	y	36.06
7	22	2,456	gl	36.64
8	6	760	v16	35.79
9	21	5,240	bz	38.05
10	23	7,150	r	41.71

Table 3. Genetic ratios of self-fertilized simplex telocentric trisomes.

Telocentic trisome	Number of plants tested	Number of gametes tested	Marker gene	Percent recessives
t2Sa	5	652	gl2	69.78
t4Sa	12	2,735	su	65.63
t6La	39	8,493	y	71.53
t6Lb	11	1,497	y	60.65
t6Lc	20	5,309	y	71.78
t7La	2	190	gl	57.89
t10La	2	441	r	63.04

usually a large excess of *a* gametes because of the frequent loss of the telocentric chromosome due to a high univalent frequency. These ratios are affected by all the factors listed for the primary trisomes along with the results of the non-random disjunction of the trivalent—the two normal chromosomes generally disjoin and go to opposite poles. Models have been devised which account for all cytological events, but they are too long to be given here.

In practice it has been found that it is generally difficult to distinguish between *A*/*A*/*a* and t*A*/*A*/*a* plants by genetic ratios because of small family size and the variability of cytological events. Progeny testing is required.

Seven telocentrics representing five different chromosomes have been identified. Their genetic ratios are given in Table 3.

Possible simplex and duplex telocentric progenies have been obtained for the other five chromosomes and will be tested. A complete set of telocentric chromosomes in maize should be available in a few years. Once a telocentric for one of the chromosomes is obtained it is possible to obtain the telocentric chromosome for the other arm. In a telocentric trisome frequently a centromere of a normal chromosome will be unpaired and will be torn apart by spindle fibers. A telocentric for the short arm of chromosome 6 was found in the progeny of a t6L/6/6 plant. It was in a plant sacrificed for cytological studies. Thus some of the progeny of a telocentric trisome should be double telocentric disomes (tS/tL/N) which, when self-fertilized, should yield some true-breeding double dicentric disomes (tS/tS/tL/tL).

The double dicentric disomes (tS/tL/N) should have a pollen abortion rate of about 25% because of the adjacent

disjunction of tS-N-tL trivalents and is therefore recognizable. One such case has been found in the progeny of t4Sa/4/4, but it has not yet been confirmed cytologically.

G.G. Doyle

Chromosome arm locations of four mutations isolated from Mutator stocks

A number of recessive nuclear mutations have been isolated from the M2 generation following crosses between an active Mutator line (DR8091 × 7092) supplied by Don Robertson and standard inbred lines W23 and Mo17. The chromosome arm locations of two high chlorophyll fluorescent (*hcf*) mutations, *hcf3-Mu* and *hcf102* (reported as 1113-3) have been reported previously (MNL 61:44). The chromosome arm locations of two additional *hcf* mutations have been determined. *hcf104* was uncovered by TB-7Lb and *hcf113* by TB-9Sb.

Plants homozygous for *hcf104* possess a normal green color, but produce a high level of chlorophyll fluorescence when illuminated by long wave UV light. Chloroplasts isolated from mutant plants support a normal rate of electron transport through Photosystem II (PSII) but only 40% of the normal rate of electron transport through Photosystem I (PSI) and through both photosystems. The concentration of chemically oxidized minus reduced P700, the reaction center pigment of PSI, is approximately 40% of the normal concentration on a chlorophyll basis. Polyacrylamide gel electrophoresis (PAGE) of thylakoid polypeptides reveals a loss of 60% of the 110 kilodalton (kDa) chlorophyll protein complex (CP1) and its 66kDa apoprotein, both of which are associated with PSI. Twelve of 100 F1 progeny of a cross between a plant heterozygous for *hcf104* and TB-7Lb stock expressed the *hcf104* phenotype.

hcf113 mutant plants are yellow-green and also exhibit an *hcf* phenotype. Mutant chloroplasts support 58% and 76% of normal electron transport rates through PSII and PSI respectively, but only 38% of the normal rate through both photosystems. PAGE of thylakoid polypeptides isolated from *hcf113* reveals severe reductions in CP1 and its 66kDa apoprotein as well as other polypeptides associated with PSI and the b_6/f complex. These data suggest that *hcf113* has multiple effects including disruption of electron transport in at least two locations and interference with the proper assembly of the chlorophyll protein complexes. Between 9 and 20 mutant plants were identified among 100 F1 progeny of each of 3 crosses between *hcf113* heterozygotes and TB-9Sb stock.

Two other mutations recovered from these Mutator stocks were also mapped to chromosome arms. 1258-7 and 1253-6 are independent isolates arising from different male and female parents in the mutagenic crosses. Both exhibit typical glossy phenotypes. Several crosses between plants homozygous for the two mutations yielded uniformly glossy progeny. The mutation present in 1258-7 was uncovered by TB-7Lb. Allelism between the two glossy isolates and *gl1* has not been tested.

Bill Cook and Don Miles

DIMBOA production in tissue culture

Recent work by Williams et al. (Agric. Ecosystems Environ. 18:185) has indicated that resistance to several corn insect pests including the European corn borer can be identified using tissue culture material. Both antibiosis and nonpreference were indicated to be important in determining the resistance. The hydroxamic acid, 2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3-one (DIMBOA) has been shown to play an important part in resistance to the first brood ECB. This raises the question of whether or not DIMBOA is produced in tissue culture and if so at what levels.

To try to answer this question 18- to 21-day old embryos were plated out on Murashige and Skoog medium supplemented with 1mg/l 2,4-D, 4mg/l α-naphthaleneacetic acid and 0.05 mg/l 6-(γ,γ-dimethylallylamino-purine. The callus which developed was transferred and divided 4 times at 2 to 4 week intervals before being used. DIMBOA was extracted from the callus using a modification of the methods of Gutierrez et al. (J. Agric. Food Chem. 30:1258). The major breakdown product of DIMBOA, 6-methoxy-2-(3H)-benzoxazolinone (MBOA), co-extracts with DIMBOA. Both DIMBOA and MBOA were identified by HPLC methods and quantitated using standard curves based on peak height.

Both CI31A and Oh43 normally produce high levels of DIMBOA in seedlings and maintain high levels long enough to give resistance to the first brood ECB. Under the tissue culture conditions used here Oh43 produced an average of only 2.2μg DIMBOA/g tissue. The CI31A calli were quite variable, producing from 0 to 44.8μg DIMBOA/g tissue. The breakdown product, MBOA, was found at levels of 2.9 and 7.0μg/g tissue in CI31A and Oh43 respectively. The level of DIMBOA was much lower than that found in seedling tissue where DIMBOA can reach greater than 10 fold higher amounts in seedling tissue. It is noted that most of the calli had far more MBOA than DIMBOA. Under the extraction conditions used this is unexpected. In normal cells DIMBOA exists as a glucoside and MBOA is not found. It may be that what DIMBOA is produced is not sequestered normally and breaks down. A second possibility is that the MBOA comes from cells that died during culture and therefore released their DIMBOA allowing it to break down. In order to get a better picture of DIMBOA production under tissue culture conditions several other corn lines and several other tissue culture conditions will have to be tested.

B.A. Bailey, J. Bussard and R.L. Larson

Glycosylation of DIMBOA by tissue-culture-isolated enzyme

In an associated note it is reported that DIMBOA is produced in maize callus. For the majority of callus the breakdown product of DIMBOA, MBOA, was found at higher concentrations. This abnormal occurrence is unexplained. One possible explanation is that once DIMBOA is synthesized it is not properly handled by the cell. DIMBOA normally exists in the cell as a glucoside which is relatively stable. The question then arose whether the enzyme responsible for glycosylating DIMBOA might be inactive in tissue culture resulting in the observed breakdown of the aglycone to MBOA. The present study concerns the identification of glucosyltransferase activity capable of adding glucose to the carbon 2 position of DIMBOA using callus tissue.

The conditions for callus culture are reported in the associated report. Enzyme was extracted from the CI31A and Oh43 callus using 2ml of buffer (50mM HEPES, 2mM

DTE, pH 7.5) from which the particulates were removed by centrifugation for 20′ at 26,000 X G. The assay conditions included 20μg protein, 372μM DIMBOA, 1mM UDPG and 7mM $CaCl_2$ in 200μl extraction buffer (pH 8.2). The assays were incubated at 37C for 5′ and reactions were terminated by addition of 0.8ml of a 2:1 chloroform: methanol solution (1% HCl). DIMBOA glucoside produced in each assay was identified by HPLC methods and quantitated using standard curves based on peak height. The specific activity (nm/mg protein/min) was then determined for each assay.

The enzyme isolated from callus did include a significant amount of glucosyltransferase capable of adding glucose to DIMBOA. Specific activities of 84.5 and 123 were observed for CI31A and Oh43 callus respectively. The enzyme activity obtained from callus is similar to levels found in seedling tissue (manuscript in preparation). The success found in identifying DIMBOA glucosyltransferase activity suggests that other enzymes in the pathway for DIMBOA production may be studied using callus. These results also suggest that the breakdown of DIMBOA to MBOA in callus is not due to the failure of glucosyltransferase to protect the aglycone by glycosylation.

B.A. Bailey, J. Bussard and R.L. Larson

Luteus, *l*, interacts with *ij* to make the *ij* seedlings yellowish

ij-affected tissues show yellow or white color depending on genetic background. While *ij* plants of Oh51a inbred line always have yellow or yellow-striped tissues, *ij* plants with Tr, Mo17, Ky21, K55, and Wg (*R-g* conversion of W23) backgrounds have only white-striped tissues. Oh51a has been known to carry homozygous *l* (luteus) gene, and the Stock Center supplies this inbred as an *l* stock. The *l* locus is located on chromosome 10 and is 2 map units distal to the *R* locus (Emerson et al., Cornell Univ. Agri. Exp. Stn. Mem., 1935)

Coe et al. (Stadler Symp., 1982) reported that some of the maternal exceptions derived from Wg background are germless and fail to germinate. Also, heritability in backcrosses and the distribution of germless kernels in ear maps indicated the possibility of involvement of a nuclear factor or factors (very possibly *Isr*, which is tightly linked to the *R* locus) in the germlessness.

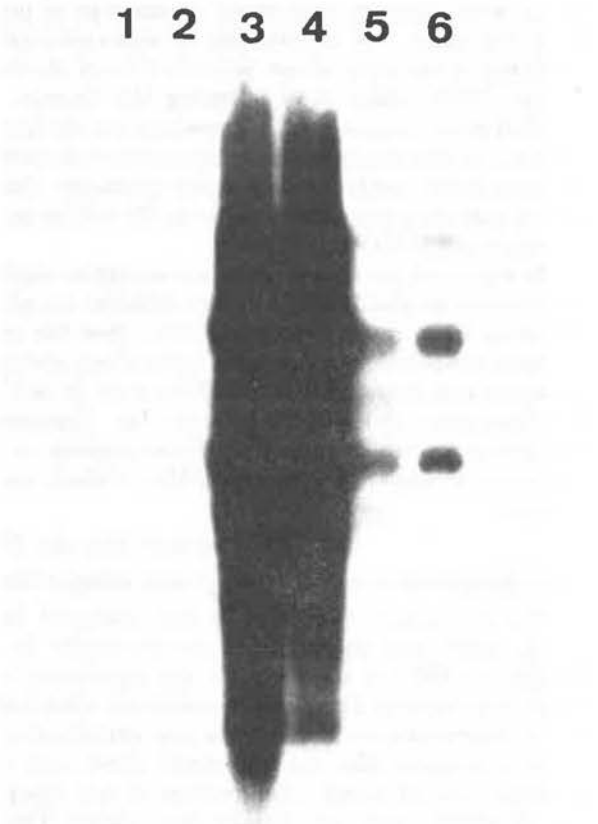
To obtain genetic evidence for *l* as a genetic factor determining yellow in *ij*, and to examine whether *R* or a gene closely linked to the *R* locus could be involved in germlessness, we selected and planted the colored kernels from the cross *ij ij* (Oh51a/Wg) × + + (Oh51a). Since the constitution of Oh51a is *c r-r*, the ratio of colored and colorless kernels is expected to be 1:3 from the progeny of the backcross. Among a total of 645 kernels examined, 198 kernels showed colored aleurone and the rest (447) of the kernels were colorless. Our previous experience indicated that Oh51a contains a dominant pale inhibitor(s) of aleurone pigment. Consequently, we could only score the colored class properly. Among the 198 colored kernels, 121 seedlings were white and 64 seedlings were green. We did not find any yellow seedlings even though a couple of yellows are expected by recombination.

We scored the kernels with no germ and those failing to germinate in both the colored and the colorless classes. Percentages of germless kernels were 6.5 and 5.2 in colored and colorless kernels, respectively. Our data do not suggest that *R* or a genic factor linked to the *R* locus is involved in germlessness of maternal exceptions. More detailed genetic work on the relation between germlessness and *R* is in progress, using the different *R* stocks kindly supplied by Dr. J.L. Kermicle (University of Wisconsin).

Chang-deok Han and Ed Coe

Plastid 23S rRNA is present in *ij* seedlings, not in maternal exception seedlings

To understand the behavior and genetics of plastids subject to the genetic lesion of the nuclear gene *ij*, we reexamined whether 23S plastid rRNA in white seedlings of *ij* and of maternal exceptions could be detected by using RNA blotting techniques. Because 23S rDNA has been reported to contain plastid-specific sequences (D.B. Stern and D.M. Lonsdale, Nature, 1982) and the plastid genome has coding capacity for some of the subunits of RNA polymerase, measuring the transcripts of 23S rDNA will be informative in understanding the capability of the transcription and translation apparatus of plastids in *ij* and maternal-exception seedlings. To minimize photodamage that usually occurs in pigment-deficient mutants, the seedlings were grown in darkness for 3 days and then in dim light (0.01 μmol/sec cm^{-2}, 28C) for 3 days. Total cellular RNAs were examined for this study. Samples from *ij* seedlings were selected that were pure white to the eye.



Northern blot analysis of total RNAs (5μg/lane). Lane 1, maternal-exception yellow seedlings (+/*ij*, *l*); Lane 2, maternal-exception white seedlings (+/*ij*, +); Lane 3, seedlings of Oh51a (+/*l*); Lane 4, seedlings of Tr (+ +); *Lane 5, ij* yellow seedlings of Oh51a (*ij l*); Lane 6, *ij* white seedlings of Oh51a/Tr (*ij* +).

The DNA probe for the 23S rRNA was kindly supplied by Dr. K. Oishi (University of Arizona). As shown in the Figure, yellow (*ij l*) and white (*ij* +) seedlings contain 23S rRNAs, but maternal-exception (+/*ij*) whites and yellows do not contain any detectable level of rRNAs. We also examined the transcripts in 6-day-old white maternal seedlings that were grown and harvested in darkness. There was no detectable signal even after long exposure of the blot. Also we found that transcripts of 6-day high light (97 μmol/sec cm^{-2}) *ij* yellow seedlings are present in as high amount as those grown in dim light. Our data extend the previous report by V. Walbot and E.H. Coe (PNAS, 1978) by showing that ribosomal RNA is actually present in white or yellow tissue of *ij* plants.

We have shown genetic evidence for reversibility of defective plastids in *ij* plants (Genetics Abstract, 1987). Once the *ij* gene back-mutates in some tissues of *ij*, *ij*-affected plastids in the clone are converted to normal green plastids. In contrast, *ij*-like patterns have never been observed in maternal-exception seedlings.

Is the detectable amount of 23S plastid rRNA in the *ij* seedlings due to low activity of expression of the plastid genome, or just due to green plastids in heteroplastidic cells of *ij*-affected tissues (Thompson et al., Amer. J. Bot. 1983)? Since there is no detectable level of plastid rRNA in maternal-exception seedlings, if two kinds of plastids (functional green and nonfunctional white or yellow plastids) are involved in organelle transmission from the *ij* female parent, maternal-exception seedlings should result from complete sorting-out of one type of plastids from the other. But distribution of maternal-exception seedlings in ear maps shows polarity (Coe et al., Stadler Symp., 1982) rather than following the lineage of *ij*-affected white tissues. Such arguments raise the question whether *ij*-affected plastids may fail to follow properly the developmental mechanism imposing positional distribution of plastids during embryogenesis. We will be examining those possibilities.

In any event, our data on maternal-exception seedlings, which show no plastid rRNA and no different morphology of leaves from green siblings, indicate that the plastid genomic compartment alone takes little, if any, part in leaf ontogeny and development. (The RNA work in this study has been done in Dr. Mary Polacco's lab. Expression of nuclear genes, responding to *ij*-affected plastids, is being examined in cooperation with Drs. Mary Polacco and Bill Patrie.)

Chang-deok Han and Ed Coe

High-germination *sh2 Ga* sweet corn composites

The *sh2* mutant discovered by E.B. Mains (J. Hered. 40:21, 1949) and characterized biochemically by J.R. Laughnan (1953) is the basis for the significant super-sweet corn industry. However, the useful *sh2* allele has two major disadvantages: it conditions poor germination and, being a recessive like *su1* (the major sweet corn gene), produces normal starch when crossed by any other corn (all of which carry the starchy *Sh2* allele). The first disadvantage was overcome by mass selection in heterogeneous populations (R.D. Bell, et al., Crop Sci. 23:461, 1983). The second can be overcome by incorporating the *Ga* factor commonly found in popcorn. For this purpose, 4 fairly separate populations of *sh2 Ga* were created using 4 disparate ears of M.S. Zuber's high-germination *sh2* composite, crossing these by a commercial yellow popcorn, crossing the 4 F1's as male to 4 different commercial *su1* sweet corn varieties, then selecting the *sh2* heterozygote and crossing these as males to 4 Missouri white *Ga* inbreds. Finally, the progenies from these crosses, which were *Ga Ga* and half heterozygous for *sh2*, were selfed and the *sh2* seed bulked, to produce 4 *sh2 Ga* composites designated *sh2 Ga* 501, 503, 504, and 505, based on the Missouri white *Ga* line used in each case.

Desirable characteristics of these separate composites were increased by planting in isolation from each other ten 50-plant rows, allowing to open pollinate, harvesting only the best ears from the middle 8 rows, and bulking the resultant seed. This was done for 7 generations. Germination was improved by overplanting under adverse conditions. *Ga* stability was improved by planting every fifth hill to a colored aleurone *Sh2 ga* stock and discarding all ears with any colored *Sh2* kernels. Disease resistance and agronomic characteristics were improved by discarding all diseased or poor plants. No selection was attempted for flavor or tenderness.

M.G. Neuffer

Rationale for seed treatment with chemical mutagens

Because maize has separate male and female germline primordia, it has been recommended in the past to use pollen treatment for effective chemical mutagenesis, which is more efficient and precise. However, in special situations (namely, when seeking dominant kernel and seedling mutants or when screening for recessive kernel and seedling mutants of a type that can easily be recognized) a minimal sample method may make seed treatment practical.

If dry seed is treated with a chemical mutagen before germination the following logic applies. Each kernel contains 4 separate primordial germcells each for the tassel and the ear. These 8 cells carry 16 genomes. Treatment of 10,000 kernels will affect 160,000 genomes and produce 80,000 recessive (50% rate), and 400 dominant mutants. Planting and selfing the resulting M1 plants will produce about 10,000 ears with the above stated mutants present in heterozygotes, but unexpressed except for dominant kernel mutants.

Dominant mutants may be identified by observation (kernel mutants) and by planting and screening as seedlings or plants. Since each kernel will represent 2 genomes and 1/8 of the genomes available, a 107-kernel sample will be required from each ear to save 99.9% of the mutants (22 kernels for 95%). In practice, screening a 100-kernel sample from 10,000 ears will save almost all mutants produced. This number (1×10^6) is feasible for seedling screens but not for older plant characters.

Recessive mutants may be identified using the minimum sample method. A one-kernel sample each from 10,000 selfed ears, when planted and selfed, will test 20,000 genomes and should express 10,000 mutants or roughly 10 recessive mutants for each locus in the genome, each of which will be a completely independent event. A repeated sample of the M1 would produce an additional large number of new mutants, only 1/8 of which should be duplicates of the previous sample. These would be at the cost of 10,000 M1 selfs and 10,000 single-sample M2s for

20,000 total selfed plants to produce 10,000 mutants for a mutant-producing efficiency of .50, which is comparable to pollen treatment.

A labor-saving procedure would be to densely plant the treated seed to reduce ear size. Allow to open pollinate, harvest and shell by machine, mix to randomize sampling, and take repeated single-seed equivalent samples to screen for the dominant mutants; or, use a single-kernel sample to grow, self and test for recessive mutants.

M.G. Neuffer

Designation of a dominant lax midrib mutant, *Lxm1*

Lxm1 (previously *Lxm*-1600*) leaves have a wide, flat, flexible midrib. The plants are small. Of special note is the ligule which extends down the midrib, leaving the proximity of the stem and forming an elongate "V". The apex of the V becomes a series of small yellowish wart-like enations.

Location data from T *wx* translocation tests indicate positive placement on chromosome 3; T *wx* translocation tests for other chromosomes indicated nonlinkage. The backcross data below show *Lxm1 Wx* linkage for T3-9c.

	T M Wx	*N Wx*	*M wx*	*N wx*	%co	χ^2
T3-9c (3L.09, 9L.12)	78 32	6	7	33	17	34.69

M.G. Neuffer

Expression of *zb4*: temperature effects

I have been using *zb4* to test linkage with other loci (*hcf3, hcf6*) on chromosome arm 1S. The penetrance of the trait is variable. I find that it depends on night temperatures. In progeny that should be segregating ¼ *zb4 zb4*, the highest frequency I have observed is 0.15. Often a second planting of an ear that expressed *zb4* did not display the *zb4* phenotype, a yellow green/green banding pattern. Expression tended to be strongest in greenhouse plantings during early fall, when heat had not been turned on in the greenhouse and nights were cool. To test the temperature dependence, I grew flats of material segregating *zb4 zb4* in 85F chambers and on germination, transferred some to a 55F refrigerator overnight (14 hours) with return to lights and 85F during the day. Material which remained in growth chamber (70F during 8hr dark) did not express *zb4*, whereas the night-refrigerated seedlings developed fine, yellow-green horizontal bands on leaves that were expanding at the time of cold treatment. The maximum number of bands per leaf equalled the number of nights (3) spent in the refrigerator. I have since been able to obtain expression of *zb4* using 16 hours light at temperatures of 84-86F and 8 hours darkness at 64F. Generally expression was not observed in seedlings until the fourth leaf had begun to emerge.

M. Polacco

Gaspé – Mo17 hybrids: life cycle

A Gaspé flint line obtained from Dennis Hourcade (Monsanto) is 12-18 inches tall and has a short life cycle (dry kernel-dry kernel) of 35-50 days. The shorter cycle is obtained at growth temperatures of 90-95F and the longer cycle when grown at 85F. Fertility of Gaspé is generally low, ear shoot bags block ear development and when grown in soil, the ears rot, perhaps due to their location at base of plant. Growth in soil-less medium at top of pots (vermiculite or perlite) prevents ear rot.

When Gaspé is crossed with Mo17 (maternal parent) the life cycle and stature of plants is intermediate to that of the 2 parents. A backcross of a Mo17/Gaspé (maternal) with Gaspé resulted in plants very similar to Gaspé with respect to lifespan and the number of leaves (7 or 8), but with much higher fertility and somewhat larger plants. When grown under field conditions and with ear shoot bags, approximately ⅓ of this material developed ears with very good seed set. These all had ears several inches above soil level. In the case of ears near the soil, all rotted in the field.

M. Polacco

Location of *Les1* on chromosome 2S

The dominant lesion mutant, *Les1*, has been located between *sk1* and *wt1* on the short arm of chromosome 2. Results from the testcross of (+ *Les1 wt1*)/(*sk1* + +) × *sk1* + *wt1* are as follows:

Reg.	Genotype	No.	Totals
0	+ *Les1 wt1*	70	
	sk1 + +	93	163
1	*sk1 Les1 wt1*	2	
	+ + +	3	5
2	*sk1* + *wt1*	1	
	+ *Les1* +	6	7
1,2	+ + *wt1*	0	
	sk1 Les1 +	0	0

sk1 Les1	0.029 ± 0.013
Les1 wt1	0.040 ± 0.015

These data place *Les1* approximately mid-way between *sk1* and *wt1*. *Les1* should be an excellent seedling marker for silkless plants by choosing the white-tip, non-lesion seedlings resulting from the above cross.

Dave Hoisington

Linkage data for *Les8* on chromosome 9S

Testcross data for the cross (+ *Les8* +)/(*c1* + *wx1*) × *c1* + *wx1* were as follows:

Reg.	Genotype	No.	Totals
0	+ *Les8* +	31	
	c1 + *wx1*	27	58
1	*c1 Les8* +	11	
	+ + *wx1*	4	15
2	+ *Les8 wx1*	3	
	c1 + +	1	4
1,2	*c1 Les8 wx1*	0	
	+ + +	2	2

c1 Les8	0.215 ± 0.046
Les8 wx1	0.076 ± 0.030
c1 wx1	0.241 ± 0.048

The data are consistent with the placement of *Les8* approximately 8 map units distal to *wx1*. Additional tests with other markers in that region (*w11, lo2*, and *l7*) will be necessary to place precisely *Les8* on the linkage map.

Dave Hoisington

dek16 and *dek23* are tightly linked on chromosome 2L

Among 97 ears from selfs of the cross + + × (+ *dek16*)/(*dek23* +), no ears were found which did not segregate 3:1 for a defective kernel mutant, although it

was impossible to distinguish between *dek16* and *dek23* segregating ears. These data indicate that while these two phenotypically similar mutants are non-allelic, they are very tightly linked.

Dave Hoisington

CORVALLIS, OREGON
Oregon State University

DNA rearrangement associated with Mutator-homologous sequences in non-Mutator somaclones

We have been examining the fate of DNA sequences homologous to the transposon family Robertson's Mutator during tissue culture and regeneration of non-Mutator maize lines. The inbred line W182BN and eleven somaclonal variants derived from it were given to us by Elizabeth Earle. Nuclear DNA from the inbred and somaclones were compared by restriction digest, Southern blotting and hybridization with Mutator probes. W182BN was found to have three types of *Mu*-homologous sequences, as we have described previously (V. Chandler et al., Genetics 114:1007, 1986; C. Rivin et al., MNL 61:53, 1987): 1) an endogenous element structurally like *Mu2*, 2) *Mu* termini unassociated with internal sequences, 3) 2 copies of internal sequences without associated termini. An example of the latter type of sequence, designated *MRS*-A for *Mu* Related Sequence, has recently been cloned and sequenced (Talbert and Chandler, submitted).

The somaclonal variants showed no fragment length differences when restriction sites that lie within the *Mu*-homologous sequences were examined. However, when the DNA was cut with enzymes that recognize sites outside the Mutator sequences, two types of differences were observed: In several somaclones there appear to be new termini-homologous bands, and in one of the somaclones (designated 2049) there is a fragment length polymorphism for one of the internal sequences. Segregation analysis of F2s from the 2049 × W182BN cross shows that this second type of change is not a transposition. We do not yet know about the terminal RFLPs, though it seems possible that these might act as independent transposons.

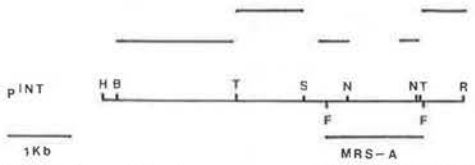


Figure 1. Mutator-related sequence and flanking DNA probes. A clone of *MRS*-A and flanking sequences (pINT) derived from W23 was given to us by Luther Talbert and Vicki Chandler. Subclones used for genomic mapping are shown above the map of pINT.

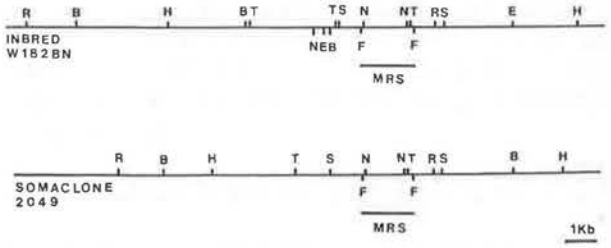


Figure 2. Genomic restriction map of a Mutator-related sequence and flanking DNA from the inbred line W182BN and its somaclonal variant 2049. R: *Eco*RI, B: *Bgl*II, H: *Hind*III, T: *Tth*111, N: *Nco*I, E: *Bst*EII, F: *Hinf*I, S: *Sst*I.

The rearrangement occurring in the internal sequence of somaclone 2049 was mapped using probes from *MRS*-A and its flanking sequences (Fig. 1). The genomic restriction maps (Fig. 2) show that the *Mu* related sequence and sequences flanking it on one side are unchanged in the somaclone. However, a major rearrangement has occurred in the DNA adjacent to the other side, as the inbred and somaclone restriction maps are different for at least 10Kb.

Chee Harn and Carol Rivin

DAEJON, SOUTH KOREA
Chungnam National University

MET maize - update

In order to improve the biological yields of modern maize hybrids, a different breeding approach has been attempted using land races with multiple tiller and ear habits (MNL 56:62; 58:85). Problems associated with the plants with multiple tiller and ear (MET) include the size of ear and kernel, ear height and lodging. Genetics of tillering and earing habits were also not clearly defined. Some well-established facts have accumulated concerning MET, and the aim of this article is to present information obtained.

Inheritance of tillers—An inbred line, IK, was developed from the MET population (Choe et al., SABRAO 19(2), 1987) and used as a female parent on six inbreds: A-type, Hi31, CI66, Hi26, A632 and B73. IK produces two to three tillers per plant under ordinary plant density and all the tillers bear a few harvestable ears. The male lines used were assumed to produce no tillers. Table 1 shows the plants segregating for tillers and less than one tiller in F2 populations. Assuming a 3 to 1 ratio for tillers to less than one tiller in F2 generations, all the families studied showed that the gene involved in tillering habits of MET is a single dominant gene. Chi-square values calculated for individual family, pooled and heterogeneity were all non-significant. The dominant effect of the gene was also confirmed in the F1 plants. However, it should be emphasized that the penetrance and expressivity for tillering was variable depending upon the genetic background of the parents. The variable penetrance and expressivity for the tiller phenotype may be explained by physiological aspects such as apical dominance of the main stem or heterosis revealed in the hybrid combination and by other genetic systems suppressing or enhancing the expression of the major dominant tiller gene. The variability in the penetrance and expressivity of tiller types didn't seem to be due to any obvious environmental effects.

Table 1. The Chi-square values assuming a 3:1 ratio for tiller to no-tiller plants in F2 generations.

Family	No. of plants/family: More than two tillers	No. of plants/family: Less than one tiller	X^2 values
IK x A-type	53	19	0.07 NS
IK x Hi31	24	4	1.71 NS
IK x CI66	18	10	1.71 NS
IK x Hi26	17	6	0.01 NS
IK x A632	36	15	0.53 NS
IK x B73	30	9	0.08 NS
Homogeneity			4.12
Pooled	178	63	0.17 NS
Heterogeneity			3.96 NS

Kernel size—The original MET materials and released lines all had small kernel size (less than 15 grams/100 kernels). However, from two different studies we found

that the kernel size is not related to the tillering habits of plants. The first evidence was obtained from the study conducted to improve the kernel size or weight by means of simple mass selection. In 1987, we found that kernels selected based on the weight of seed from our base populations, which are maintained by sibbing, did not show any discrepancies in the number of tillers. Namely, the kernels weighing over 25 grams/100 kernels also tillered as much as the kernels weighing less than 15 grams/100 kernels. More apparent evidence was obtained when we planted the F2 segregating populations with large kernels. F2 seeds from MET by U.S. lines were all large in kernel size and showed segregating plants for tillers. At the same time we found that the small kernels of the original MET materials were never recovered in the subsequent generations when they were crossed with large kernels of U.S. dent lines. But the tillering habits were retained regardless of the kernel size.

Ear height—The original MET materials all had high ear height. However, ear height of the F1 crosses between MET and a U.S. line with low ear height was very low (Table 2). The ear height of the MET type would be no problem for future breeding.

Table 2. Comparison of average ear height of IK and F1 plants between IK and a low eared U.S. dent line.

	No. of tillers /plant	Plant height* (cm)	Ear height* (cm)
IK	2.0 ± 1.1	195 ± 11.5	117 ± 10.5
(IK x LE)F_1	1.8 ± 0.8	206 ± 21.4	84 ± 14.4

* Heights of main stem and tillers were measured and averaged.

Table 3. Fresh and dry weight of MET and uniculm hybrids.

	Fresh weight, ton/Ha	Dry weight*, ton/Ha
MET hybrid**	120	12
FR29 x FR34***	80	9

* oven dried, ** measured at the silking stage and *** measured 10 days after pollination

Biological yields—Fresh and dry weight of F1 hybrids between the MET lines were compared with those of ordinary uniculm hybrids (Table 3). The fresh and dry weights of some of the F1 hybrids with multiple tillers and ears were much higher than those of common uniculm hybrids. However, the fresh weight as well as dry weight were very variable depending upon the time of harvesting.

Peroxidase activity—Quantitative and qualitative measurements of peroxidase activities in various parts of plants showed typical band patterns among the MET types, uniculm inbreds and their hybrids.

B. Choe, H. Lee, Y. Seo, B. Kim and J. Park

DEKALB, ILLINOIS
Northern Illinois University

Transposable elements, allelism tests and RFLP mapping

When an RFLP probe detects a single polymorphic band in different maize lines, the common assumption is that these bands are allelic. While this assumption is undoubtedly valid in most cases, the prevalence of transposable elements (TEs) in maize can complicate the issue, i.e. how can a low copy number TE present in different locations in different maize lines be distinguished from a fixed gene locus? As an example, *Bs1*, a retrovirus-like transposon, is present in 1-5 copies in most maize lines, and its hybridization patterns look similar to those of standard RFLP probes (Johns et al., EMBO J., 1985). Unfortunately, the standard tests for allelism, analysis of backcross and F2 self progeny, are remarkably insensitive to this phenomenon.

Let us call the hybridization band in one line "A", the band in another line "B", and the absence of a band "O". The F1 heterozygote between these lines can be represented as A/B if the bands are allelic, and A/O B/O if the bands are non-allelic and unlinked. On a Southern blot there are 4 possible conditions: A, B, AB, and O. It may be possible to distinguish AA from AO in some conditions, but this cannot be reasonably assumed.

In a backcross, the A/B heterozygote is crossed to the A line, resulting in 50% A progeny and 50% AB progeny. For unlinked TEs, the progeny will be 25% A/A B/O, 25% A/O B/O, 25% A/A O/O, 25% A/O O/O; this reduces to 50% AB, 50% A. Thus, a backcross is completely incapable of distinguishing allelic bands from unlinked TEs.

Selfing the F1 to produce an F2 gives a 1:2:1 ratio of A:AB:B if the bands are allelic, and a 9:3:3:1 ratio of AB:A:B:O if the bands are unlinked. A closer look at the latter ratio reveals some problems. The diagnostic class is the O, or null class: if null progeny are detected, then A and B cannot be allelic. However, the nulls are only 1/16 of the total progeny, and it would take an examination of 46 progeny to be 95% sure that getting zero null progeny is not a chance variation from 1/16. The situation is worse if the TEs are linked: the frequency of the null class varies inversely with the square of the linkage distance. If two TEs were 25% linked, it would take an examination of 191 F2 progeny to be 95% sure that zero null progeny was not due to chance variation.

There is another problem with null progeny: if no band appears in a particular lane of a blot, it is easy to decide that this is due to experimental error and simply ignore these lanes. If this is done, the 9:3:3:1 ratio expected from unlinked TEs degenerates into a 3:1:1 ratio. It would take an examination of 150 progeny to be 95% sure that a 3:1:1 ratio was not a chance variation of the 2:1:1 ratio expected from alleles. The situation is, of course, much worse with linked TEs.

So, if neither backcrosses nor F2 selfs will reliably distinguish TEs from alleles, what does work? The best allelism test would seem to be crossing the F1 heterozygote to a third line with a different hybridization band "C". For alleles, the resulting progeny are 50% AC and 50% BC. For unlinked TEs, the progeny are 25% C, 25% AC, 25% BC, 25% ABC. The C and ABC progeny, half of the total, are diagnostic for non-allelism. Also, there is no null class: a lane with only the C band is clearly distinguishable from a completely blank lane. It thus takes an examination of only 5 progeny for there to be a greater than 95% chance that at least 1 ABC or C will be seen, if unlinked TEs are involved. If 25% linkage exists between the TEs, examination of 11 progeny gives a greater than 95% chance of seeing at least one diagnostic offspring. This method is clearly much more powerful in distinguishing TEs from alleles in RFLP mapping than the standard backcross or F2 self methods.

One other RFLP method should be discussed: recombinant inbred (RI) lines. Completely homozygous RI lines yield the major advantage of an outcross to a third line:

there is a 1:1:1:1 segregation of AB:A:B:O progeny. The problem of null lanes being considered experimental error can be partially eliminated by testing more than one individual from a given line; however, there is no C band acting as an internal control. The major problem with RI lines is that they are not completely homozygous. Burr et al.'s (Genetics, 1988, in press) RI lines are estimated to be 7.5% heterozygous for all loci (they expected 3.125%, based on 5 generations of selfing). Also, their mapping method deliberately ignored null lanes. Thus, there is certainly room for a small number of TE probes in their collection, and an examination of these RI lines with a new probe will not necessarily distinguish between alleles and TEs in different locations (especially linked TEs). However, if a new probe detected only A and B types, and never AB or O types, among the RI lines, then one could be reasonably certain that A and B are allelic or at least tightly linked. The presence of heterozygous or null RI lines suggests the need for further allelism tests.

From the preceding discussion it can be seen that distinguishing low copy number TE from alleles by genetic tests is best done by outcrossing F1 heterozygotes to a third line; use of RI lines is also an acceptable method. Examination of F2 self progeny is unlikely to reliably distinguish between these alternatives, and backcrosses are useless. The importance of these observations depends on how prevalent low copy number TEs are. *Bs1* certainly falls into this category: a number of lines containing a single *Bs1* element exist, and only sophisticated genetic analysis or DNA sequence analysis would determine that *Bs1* is a TE and not a fixed gene. It may well be that other low copy number TEs exist; they may even be common.

Mitrick A. Johns

EUGENE, OREGON
University of Oregon

Genetic properties of four Mutator-induced alleles of *B-Peru*

A goal of this laboratory is to ascertain the molecular mechanisms controlling paramutation and the expression of the *B* gene, a regulatory locus involved in anthocyanin pigment synthesis in plant tissues. A necessary first step toward analyzing the *B* locus is to clone the genomic sequences. The *B* gene product is unknown and is likely to be present in small quantities; therefore, transposon tagging appears to be the best approach for cloning this gene. We have been using Robertson's Mutator as a transposon tag for this purpose. Several years ago Mutator stocks were obtained from D. Robertson and used to isolate 4 variegated mutations at *B-Peru* (V. Chandler and V. Walbot), an allele that specifies anthocyanin pigment in the aleurone and embryo. The mutations were isolated at frequencies of $1\text{-}3 \times 10^{-5}$, while in control experiments the spontaneous mutation frequency of *B-Peru* was determined to be $< 5 \times 10^{-6}$. All 4 alleles have been mapped to the *B* locus on chromosome 2S using 3-point testcrosses. In this report we describe the phenotypes and segregation properties of these *B-Peru* alleles. In a separate report in this issue we describe our molecular analyses of these insertion mutations.

The phenotypes of the progenitor *B-Peru* allele and the 4 Mutator-induced variegated alleles are described in the accompanying table. All of the independently isolated insertions affect both the kernel and plant tissues, suggesting that each insertion is in a region of the gene required for expression in all tissues. Three of the mutant alleles have colorless backgrounds, while 1 allele has pale pigment in both kernel and plant tissues. Each mutant allele has a characteristic pattern of somatic reversion that is observed in both kernel and plant tissues.

Allele	*Kernel Phenotype*	*Plant Phenotype*
B-Peru	purple aleurone & embryo	spotted irregular pigment on leaf sheaths and plant stalk; strong red pigment on tassel branches and glumes
b-Perumu5	colorless background; low frequency of tiny revertant sectors	green plant; tassel has low frequency of tiny revertant sectors
b-Perumu216	colorless background; medium frequency of tiny to medium sized revertant sectors	green plant; tassel has revertant sectors of variable size and frequency
b-Perumu218	colorless background; high frequency of tiny to large revertant sectors	green plant; tassel and plant with high frequency of revertant sectors of variable size
b-Perumu220	pale background; medium frequency of tiny revertant sectors	pale pigment in tassel and plant with small revertant sectors of variable frequency

Most characterized Mutator-induced mutations have been the result of a *Mu1* element insertion into the gene of interest. The typical reversion pattern of these alleles shows small and frequent somatic reversion events and rare germline reversion. The transmission of several *Mu1*-induced alleles through several generations has been reported. Typically the allele segregates as a single gene (when heterozygous, 1:1 segregation in outcrossed progeny and 3:1 in selfed progeny), with occasional plants transmitting more colorless kernels than expected to progeny (J.L. Bennetzen, V.L. Chandler, M. Freeling, D.S. Robertson, V. Sundaresan, and V. Walbot). The colorless kernels may still contain the mutant allele, but the element is no longer able to cause somatic reversion. DNA modification of *Mu1* elements has been correlated with this loss of somatic reversion for two mutant alleles (*bz2-mu1*, Chandler and Walbot; a *Mu1* insertion at *bz1*, Sundaresan and Freeling).

To determine the segregation properties of the 4 unstable *B-Peru* alleles, each has been outcrossed for several generations to a *b r-g* tester (W23/K55 hybrid) and the ratios of spotted, colorless and purple kernels determined. The genetic properties of each allele are summarized below.

b-Perumu5: This allele segregates as a single gene, and ears that transmit fewer spotted kernels than expected are rarely observed. No loss of somatic instability was observed after 4 generations of self-pollination and 2 generations of outcrossing to the *b r-g* tester. In the next 5 sequential outcrosses of *b-Perumu5*/*b* individuals to the *b r-g* tester, 9 in 10 ears showed the expected segregation: 50% spotted, 50% colorless ($.95 > P > .5$). One in 10 outcrossed ears had an increased number of colorless kernels, usually 60-70% instead of the expected 50% ($P < .01$). With more than 10^5 kernels examined no germline revertants have

been recovered from plants transmitting the typical pattern of infrequent, tiny somatic revertant sectors.

The *b-Perumu5* allele has a similar phenotype and transmission pattern to several other Mutator-induced alleles, although variants showing different patterns of somatic and germline reversion of *b-Perumu5* have been isolated. In progeny from both outcrosses and self-pollinations, kernels with more frequent and larger somatic revertant sectors are observed in approximately 1/300 kernels. Plants resulting from these kernels also produce progeny with larger somatic revertant sectors and produce germline revertants at a frequency of approximately 10^{-4}. In addition, we have identified 2 genetic stocks that contain a factor or factors that cause a dramatic alteration in this allele's phenotype: every kernel receiving the *b-Perumu5* allele and the factors contains frequent and large somatic revertant sectors, and germline revertants are isolated at frequencies between 10^{-1} and 10^{-3} (G. Patterson and V. Chandler). Bedinger et al. have reported another type of background effect on the *b-Perumu5* allele. When *b-Perumu5* is crossed by certain maize inbreds fewer spotted kernels are transmitted than expected (MNL, this issue). These results suggest that different backgrounds contain factors that can increase or decrease the activity of the element at *b-Perumu5*.

b-Perumu216: This allele has never segregated as expected for a single gene. In all selfed and outcrossed progeny fewer spotted kernels are observed than predicted for a single heterozygous gene. Three classes of segregation ratios are observed upon outcrossing a *b-Perumu216/b* plant to a *b r-g* tester. For example, in one generation a total of 32 ears were examined; 18 of the ears contained ¾ colorless, ¼ spotted kernels (.95>P>.2); 6 ears contained ⅝ colorless, ⅜ spotted kernels (.95>P>.3); and 8 ears contained more than 80% colorless kernels (P<.001 that the deviation from 3:1 or 1:1 is due to chance alone). Purple kernels are observed in each class and range from 0-2% of the kernels on each ear.

When the *b-Perumu216* allele is transmitted as female, approximately 1 in 25 plants contain large ear sectors of germline revertants, and 1 in 8 plants contain large ear sectors of colorless kernels. This demonstrates that reversion and inactivation can happen early in ear development. Small sectors of revertant tissue are frequently observed in the tassel, but rarely observed in the plant body. In about 1 in 200 plants larger revertant sectors of ⅛ to 1/32 of the tassel are observed as well as sectors on the plant body.

The *b-Perumu216* allele does not have the phenotype and segregation properties of the typical Mutator-induced mutation. The class segregating ¾ colorless, ¼ spotted may represent the segregation of an unlinked regulatory locus necessary for the somatic reversion of the element at *b-Perumu216*, and the class segregating ⅝ colorless, ⅜ spotted may represent the segregation of two unlinked regulatory loci. The class with greater than 80% colorless kernels may represent the inactivation of the element at *b-Perumu216* or the regulatory gene. Alternatively, the observed segregation ratios may not represent the segregation of unlinked regulator genes, but may represent a high frequency of inactivation of the element at *b-Perumu216*. Whatever the mechanism, these segregation patterns are reproducible in that they have been observed every generation for 5 sequential outcrosses.

b-Perumu218: This allele segregates as a single gene, with plants heterozygous for the allele producing 50% spotted and purple kernels upon outcrossing and 75% spotted and purple kernels upon selfing (.95>P>.2). Ears that have an increased number of colorless kernels (P<.01) are rarely observed (1 in 15 ears). However, the *b-Perumu218* allele is unstable, producing a large number of revertant kernels each generation. The number of revertants on each ear varies between 5 and 25% of the kernels, and when the *b-Perumu218* allele is transmitted as female large sectors of revertant kernels are seen in approximately 1 in 7 ears. No sectors of colorless kernels have been observed. Somatic revertant sectors are also frequently observed in the tassel and plant body. The large and frequent somatic sectors and the high frequency of germline revertants is not typical of most Mutator-induced mutations, but is similar to that observed for *b-Perumu5* in certain backgrounds.

b-Perumu220: The original *b-Perumu220/b* kernel had a pale pigmented background with tiny dark purple sectors. When planted and outcrossed to *b r-g* tester 4 kernel phenotypes were observed: pale purple with darker revertant sectors, colorless with revertant sectors, uniformly pigmented with no sectors, and colorless. The same 4 phenotypes were observed upon self-pollination. In the original crosses 2 of 4 outcrossed ears segregated as expected for a single gene, with 50% of the kernels spotted and purple and 50% colorless (.7>P>.5). However, the other 2 outcrossed ears segregated 65% and 70% colorless kernels (P<.05). The uniformly pigmented kernels varied in intensity, ranging from pale to dark purple, and their phenotypes were stable in subsequent generations, suggesting they were partial and full revertants, respectively. The spotted kernels with colorless or pigmented backgrounds have given rise to all 4 kernel classes in 2 subsequent generations of outcrossing. The uniformly pigmented kernels accounted for 1-3% of the ear, and sectors of revertant kernels are observed in 1 in 25 ears. Approximately ⅓ of the ears segregate 50% spotted and purple and 50% colorless kernels. The other ears contain larger numbers of colorless kernels, usually in the range of 55-65% of the total kernels. No ear sectors of colorless kernels have been observed. The coloration of plants arising from the variegated kernels is also highly variable, but the majority of plants have pale pigmentation and small, infrequent revertant sectors. The somatic reversion frequency and kernel phenotype is typical of many Mutator-induced mutations, but the high germline reversion frequency (10^{-2}) is not typical.

In summary, 3 of these *B-Peru* alleles have phenotypes or reversion frequencies different from most Mutator-induced mutations. Other groups have also reported Mutator-induced mutations with exceptional phenotypes. Robertson has reported a Mutator-induced waxy allele, *wx-mum2*, that has large somatic revertant sectors and a germline reversion frequency of 10^{-4} (MNL, this issue), and Freeling's laboratory has reported a high reversion frequency of an *Adh1* allele caused by a *Mu3* insertion. One possibility is that the differences in phenotypes between these mutants and most Mutator-induced mutations is because the *B-Peru* and waxy mutations are not caused by *Mu1* elements. Support for this idea comes from the molecular characterization of 3 of these *B-Peru* alleles

which demonstrated that no characterized *Mu* transposable elements are linked to the mutant alleles (Chandler and Turks, this issue).

Vicki L. Chandler, Susan Belcher
and Devon Turks

Three Mutator-induced alleles of *B-Peru* are caused by previously uncharacterized transposable elements

We have been using the transposable element Robertson's Mutator as an insertional mutagen and "transposon tag" to clone the *B* locus. The *B-Peru* allele was used as the target for our Mutator experiments since it causes pigment expression in the kernel. Four mutations that have a variegated phenotype have been isolated from Mutator stocks and their phenotypes and segregation properties are described in a separate report (V. Chandler, S. Belcher and D. Turks, this issue).

Our cloning efforts were initially focused on the *b-Perumu5* allele because it had a reversion pattern more characteristic of *Mu1*-induced mutations: small somatic revertant sectors and infrequent germline revertants. Our strategy has been to identify the particular *Mu* element linked to *b-Perumu5* using Southern blot analysis of DNA from plants segregating the mutant phenotype. We first used an internal *Mu1* probe that will recognize both *Mu1* and *Mu1.7* elements, because insertions of these elements are responsible for most of the molecularly characterized Mutator-induced mutations. There are only 2 restriction fragments homologous to this internal *Mu1* probe in the non-Mutator *b* tester stock. We determined that there were 10 to 20 *Mu1*-like elements and 10 to 20 *Mu1.7*-like elements in our *b-Perumu5* plants. We screened for the plants containing the lowest number of *Mu* elements and outcrossed these to a non-Mutator *b r-g* tester (W23/K55). After several generations of outcrosses, the total number of *Mu1* and *Mu1.7* elements had been diluted to 4 to 10 copies. In these lineages, it was possible to unambiguously identify the only *Mu1*-homologous restriction fragment that segregated with the *b-Perumu5* allele.

Using standard protocols, the *Mu1*-homologous restriction fragment that segregated with the *b-Perumu5* phenotype was isolated from a total genomic library. An 850bp *Hin*fI restriction fragment adjacent to the *Mu1* element was subcloned and used as a hybridization probe to examine the progenitor *B-Peru* stock and other *b-Perumu5* plants. This sequence recognized a single 5.4kbp *Bgl*II fragment in the progenitor *B-Peru* stock and a 6.8kbp *Bgl*II fragment in the *B-Perumu5* stock, suggesting that a 1.4kbp *Mu1* element had inserted into this region of the genome.

A combined molecular and genetic analysis was then done to determine if the sequences adjacent to the cloned *Mu1* element represented the *B-Peru* gene. RFLP mapping placed this sequence to within 4 map units of the *B* locus on the short arm of chromosome 2 (T. Helentjaris, personal communication). This confirmed our previous segregation analysis that suggested the cloned *Mu1* element was tightly linked to the *B* locus. If the *Mu1* element that inserted into this chromosomal region is responsible for the *b-Perumu5* phenotype, there should be a 100% correlation between the presence of the 6.8kbp *Bgl*II fragment (*Mu1* element insertion) and the spotted phenotype. We used Southern blots to examine DNA from 38 individuals that derived from the original *b-Perumu5/b* kernel. Thirty-five individuals fit the above criteria, but 3 exceptions that transmitted the spotted phenotype and no longer contained the *Mu1* element at this site were identified. Other RFLP markers were then used to demonstrate that these exceptional individuals had undergone recombination events separating the *Mu1* element insertion from the *B-Peru* locus. This demonstrated that the *Mu1* element we cloned was not responsible for the *b-Perumu5* phenotype.

These data suggested that we had cloned a sequence that maps close to *B*, but is not part of the *B* locus. Our data further demonstrated that neither a *Mu1.7* nor a *Mu1* element was responsible for the *b-Perumu5* phenotype, as we had cloned and characterized the only restriction fragment homologous to *Mu1* that mapped to chromosome 2S. This was initially surprising as most of the molecularly characterized Mutator-induced mutations have been caused by the insertion of a *Mu1* element. However, during the past two years, several exceptions have been reported in addition to our *b-Perumu5* allele.

Using probes specific for all characterized *Mu* elements we have investigated whether any of these elements were inserted into the *b* locus, generating the *b-Perumu5* allele. Karen Oishi and Mike Freeling provided a probe specific for *Mu3*, the element that inserted into *Adh1*, and Sue Wessler provided a probe specific for the *Mu* element that inserted into the *wx-mum5* mutation isolated by Don Robertson. We have also used probes specific for 4 other *Mu* elements, *Mu4, Mu5, Mu6,* and *Mu7*, that were cloned from maize stocks by cross-hybridization with *Mu* terminal probes (Talbert, Kelly, and Chandler, this issue). All 6 of these elements are homologous to *Mu1* termini, but share no internal homology with each other or with *Mu1* and *Mu1.7*. Each probe recognized 2 to 8 unique restriction fragments, but none of these fragments segregated with the *b-Perumu5* allele.

A similar analysis has been performed with plants segregating the *b-Perumu216* and *b-Perumu218* alleles. We have not identified any restriction fragments homologous to any cloned *Mu* element cosegregating with these two mutant alleles. After 4 successive outcrosses, the *b-Perumu220* allele still contains numerous *Mu1* and *Mu1.7* elements (40-80), so it has not been possible to determine if a *Mu1*-homologous fragment is cosegregating with this mutant allele. However, none of the restriction fragments homologous to the other 6 elements segregate with the *b-Perumu220* allele.

Our results demonstrate that 3 unstable mutations isolated from Mutator stocks are not caused by previously characterized *Mu* elements. All maize lines contain additional copies of *Mu* termini that are not part of characterized *Mu* elements, so it is likely that additional *Mu* elements remain to be discovered. One or more of these may be responsible for the *B-Peru* alleles we have isolated. Alternatively, another type of element unrelated to *Mu* elements by sequence similarity may be responsible for these mutations. Genetic tests and Southern blots suggest an *Ac* element is not responsible for the *B-Peru* alleles. Experiments are in progress to test for *Spm* activity.

We are trying to "trap" the element(s) transposing in these stocks by screening for mutations in genes for which

we have probes available. We have crossed *b-Perumu5* and *b-Perumu218* plants (that contain only a few *Mu1* and *Mu1.7* elements) to testers for *bz1*, *sh1*, and *a1*. Multiple, independent insertions into each gene have been isolated at frequencies ranging from 5×10^{-3} to 10^{-5}. This winter the newly isolated mutations will be characterized to determine what kind of insertion element they contain. If new elements are found they will be cloned, and we will determine whether any of these elements are responsible for the *B-Peru* mutations. If so, we will use these elements as probes to clone the *B-Peru* gene.

Vicki L. Chandler and Devon Turks

Characterization of a class of *Mu* elements present in normal lines

Normal stocks of maize contain sequences homologous to *Mu* transposable elements. One class of *Mu*-homologous sequence has *Mu* terminal inverted repeats unassociated with the *Mu1* internal sequence. This class is present in multiple copies in normal maize lines. Two examples, termed *Mu4* and *Mu5*, were cloned from the maize inbred line B37 based on homology to the *Mu1* termini and sequenced. A structural comparison of *Mu4* and *Mu5* with *Mu1* (Figure 1) illustrates the following points.

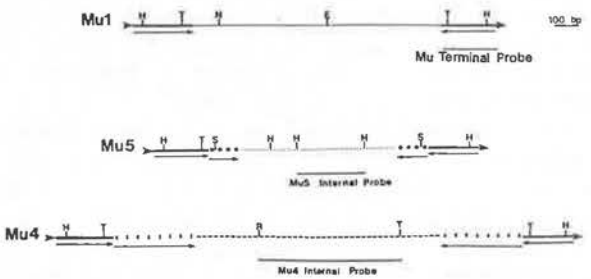


Figure 1. Comparison of *Mu4* and *Mu5* with the transposable element *Mu1*. The *Mu* terminal inverted repeats which are the only regions of homology are indicated by thickened bars. The arrowheads flanking the elements represent the 9 bp direct repeats. Restriction sites indicated are H:*Hin*fI, T:*Tth*111-I, N:*Nco*I, B:*Bst*EII, S:*Sal*I, R:*Eco*RI. There are additional *Hin*fI sites in *Mu4* which are not shown.

1. *Mu4* and *Mu5* are 2.0 and 1.3kb in length, respectively, and both have *Mu* terminal inverted repeats which are approximately 90% similar to those of *Mu1*.

2. *Mu4* and *Mu5* are flanked by short (9bp) direct repeats as are other *Mu* elements.

3. The terminal inverted repeats of *Mu4* and *Mu5* are longer than those of *Mu1*, extending internally from the *Mu1*-homologous termini by 280 and 130bp, respectively, as shown in Figure 1.

4. Except for the sequence similarity of the *Mu* termini, *Mu1*, *Mu4*, and *Mu5* have no sequences in common.

Our sequencing results suggest that *Mu4* and *Mu5* have the structures of transposable elements, and the apparent 9bp target site duplications suggest that they inserted into their current genomic locations in B37. In addition, Southern blot analysis using a 200bp probe made from the DNA immediately adjacent to the cloned *Mu4* element demonstrates that *Mu4* elements are not in the same genomic locations in all maize lines. The DNA flanking the cloned *Mu4* element hybridizes strongly to a single *Bgl*II restriction fragment in all maize lines we have examined, but only in B37 and B73 does the *Bgl*II restriction fragment also hybridize to the *Mu4* internal probe. Other lines do not have a *Mu4* element adjacent to the flanking probe. However, all lines examined have 2 to 4 *Mu4*-homologous sequences elsewhere in the genome. Similar experiments are in progress for the cloned *Mu5* element.

Southern blot analysis reveals multiple restriction fragments homologous to *Mu1* termini in all related *Zea* species we have examined, including *Zea mexicana*, *Zea luxurians* and *Zea diploperennis*. A few of these sequences are also homologous to internal probes for *Mu4* and *Mu5*. In contrast, *Tripsacum dactyloides* contains no sequences homologous to the *Mu1* termini or to the *Mu4* and *Mu5* internal probes shown in Figure 1. Our hybridization conditions would detect sequences that are at least 86% homologous to the *Mu1* termini. These results suggest that *Mu4* and *Mu5* elements have been in the genome during the evolution of the genus *Zea*. Experiments are in progress to determine if *Mu4* and *Mu5* may still contribute to genomic variation in either Mutator or non-Mutator lines of maize.

Mu elements in addition to *Mu1* and the closely related *Mu1.7* have been shown to transpose in Mutator stocks. These include the *Mu3* element (Chen et al., Genetics 116:469, 1987) and an element cloned by Sue Wessler from the *wx-mum5* allele isolated by Don Robertson. In addition, we have characterized two other elements, termed *Mu6* and *Mu7*, which were cloned from a Mutator stock based on homology to the *Mu1* termini. All of these elements share the *Mu* terminal inverted repeats, but have no other sequences in common. Internal probes specific for each element also hybridize to restriction fragments in non-Mutator lines. We estimate that the 8 characterized elements account for approximately 50-70% of the restriction fragments homologous to *Mu1* termini found in non-Mutator maize lines. Thus, additional *Mu* elements remain to be characterized.

L.E. Talbert, A.J. Kelly and V.L. Chandler

GAINESVILLE, FLORIDA
University of Florida and USDA-ARS

Mature pollen contains transcripts of the constitutive sucrose synthase (*Css*) gene

Two different enzymes condition the sucrose synthase activity that is detected in the maize plant (Chourey, MGG 184:372, 1981; Echt & Chourey, Plant Physiol. 79:530, 1985). Genes coding for these enzymic activities have been identified, cloned, sequenced and genetically mapped (Chourey & Nelson, Biochem. Genet. 14:1041, 1976; Geiser et al., EMBO J. 1:1455, 1982; Werr et al., EMBO J. 4:1373, 1985; Sheldon et al., MGG 190:421, 1983; Zack et al., Maydica 31:5, 1986; McCarty et al., PNAS 83:9099, 1986; Shaw and Hannah, unpublished). Whereas the activity coded for by the *Sh* locus is found almost exclusively in the endosperm, the activity coded for by the second gene is found in the endosperm, embryo, roots, shoots and cultured cells (see references above). To our knowledge, there is only 1 report which describes a maize tissue lacking sucrose synthase activity. W.H. Bryce and O.E. Nelson (Plant Physiol. 63:318, 1979) reported that pollen of maize lacked this enzymic activity whereas detectable levels of several other starch synthetic enzymes were found in this tissue.

To ask more directly whether either of the 2 structural genes for sucrose synthase is expressed in the maize pollen, Northern analyses of RNA from the pollen and from seeds were performed. As shown in the figure, *Css* mRNA having the same mobility as that of the seed is detected in the mature pollen of maize. Thus it would appear that the *Css* locus is expressed in this tissue as well as in most if not all tissues of the maize plant.

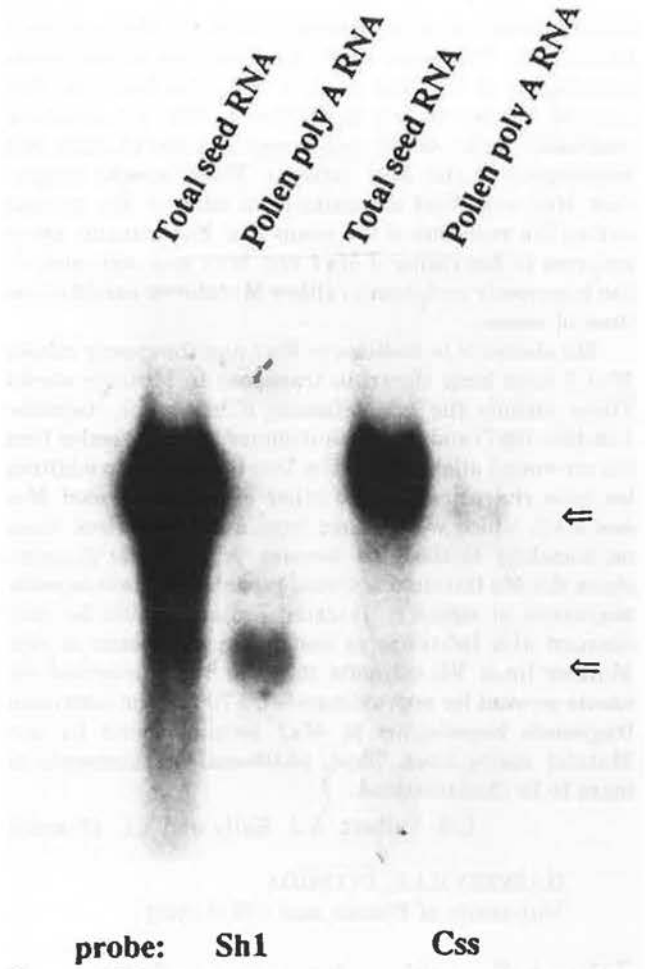


Figure. RNA was extracted from pollen and developing seeds using the methods of McCarty (MNL 60:61, 1986). Purification and oligo dT chromatography for the pollen preparation as well as subsequent Northern analysis followed methods described previously (McCarty et al., PNAS 83:9099, 1986). Developing seeds came from self-pollination of F1 plants of the genotype W64a × 182E. Pollen was collected from W22 plants which were homozygous for a *vp* mutant. Tracks contain approximately 7μg of RNA in every case. Poly A RNA was used for the pollen assays whereas total RNA was used for the seed samples. Gels were developed after 40 hours of exposure. Two intensifier screens were used.

Perhaps unexpectedly, the *Sh* probe also detects a transcript in the mature maize pollen. The transcript is significantly smaller than the *Sh* transcript of the endosperm. The origin and genetic control of this mRNA are under investigation.

Chourey (MNL 61:55, 1987) has correctly pointed out some of the confusion associated with the nomenclature of the genes encoding sucrose synthase enzymes of maize. The major problem arises from the fact that the gene shrunken, or *Sh*, which encodes the predominant endosperm sucrose synthase was named for its characteristic kernel phenotype and not for its biochemical function. Because of this, the naming of the gene coding for the second sucrose synthase has not been trivial.

We (McCarty et al., MNL 60:58, 1986) had initially used the abbreviation *Ss2* as the gene symbol for the second gene. However, three problems with this symbolism became apparent during the review of the paper describing this gene. The criticisms are the following: (1) *Ss2* is misleading in that there is not an *Ss1* or *Ss*. In order to have an *Ss*, *Sh* would have to be changed to *Ss* or we would have to have 2 symbols for 1 gene. Neither possibility is particularly appealing to us. (2) If in fact *Ss* is really *Sh* then, the argument goes, *Ss2* must really be *Sh2*. This, of course, is wrong. *Sh2* is on another chromosome and affects a step later in the starch biosynthetic path, ADP-glucose pyrophosphorylase. Although we initially thought that this was common knowledge, the mistake of equating *Ss2* with *Sh2* has been made on many occasions (public talks, question and answer sessions, poster sessions and even in our own lab meetings). Continued usage of *Ss2* and the compelling logic in the above argument will quickly lead to 1 gene symbol for 2 genes. (3) *Ss2* does not follow the recommendations of our group concerning gene symbols. One need only look to recommendation 1 (Burnham et al., MNL 49:3, 1975) to see that all new genes (after 1975) should be given a three letter symbol. This point clearly rules out *Ss2* if we are to follow our own rules. As an alternative, we have used *Css* for *constitutive sucrose synthase*. The symbol has a number of attractive features: (1) It gives a fair description of the expression of the gene. While we have not examined every cell in every tissue of a corn plant, the data to date point to the fact that *Css* is expressed in many if not all tissues of the plant. As we have pointed out above, *Css* transcripts can be found in a tissue which reportedly lacks sucrose synthase activity. Whether this reflects a form of control at the translational level or differences in sensitivity of the enzyme assay versus RNA assay is presently unknown; however, there is now no reason to think that the gene is not expressed in this tissue. The term constitutive should not be equated with non-inducible. An examination of the literature concerning the original *operator-constitutive* mutants of the *lac* operon of *E. coli* shows that these mutants were still inducible; the level of induction, however, is less than that of wild type. (2) It is difficult to confuse *Css* with *Sh2*. The symbol *Css*, like *Sh1*, differs substantially from the words sucrose synthase. (3) *Css* follows the rules of maize nomenclature.

It is our hope that the problems of gene symbolism surrounding the sucrose synthases can now be resolved.

L.C. Hannah and D.R. McCarty

The structure of the viviparous-1 locus

We recently succeeded in cloning the viviparous-1 locus by transposable element tagging using the Robertson's Mutator-induced *vp-mum1* allele isolated by Philip Stinard and Donald Robertson at Iowa State. In the course of confirming our clone, pVPM1B (Figure 1), we have done some preliminary molecular analysis of several *vp* mutant alleles. Figure 2 shows a Southern blot of total DNA restriction digests from normal and mutant plants of each stock probed with sequences flanking the *Mu* insertion in pVPM1B (see Fig. 1). In every case we detect differences between the mutant and wildtype alleles. In all cases, the

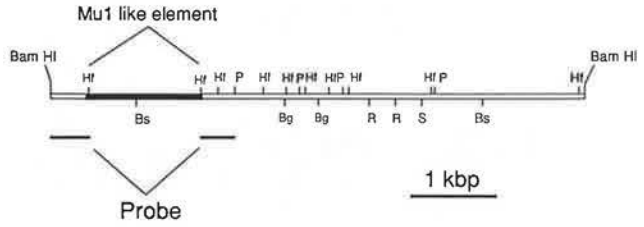


Figure 1. Restriction map of pVPM1B. Bs = *Bst*EII; Hf = *Hin*fI; P = *Pst*I; S = *Sst*I; Bg = *Bgl*II; R = *Eco*RI.

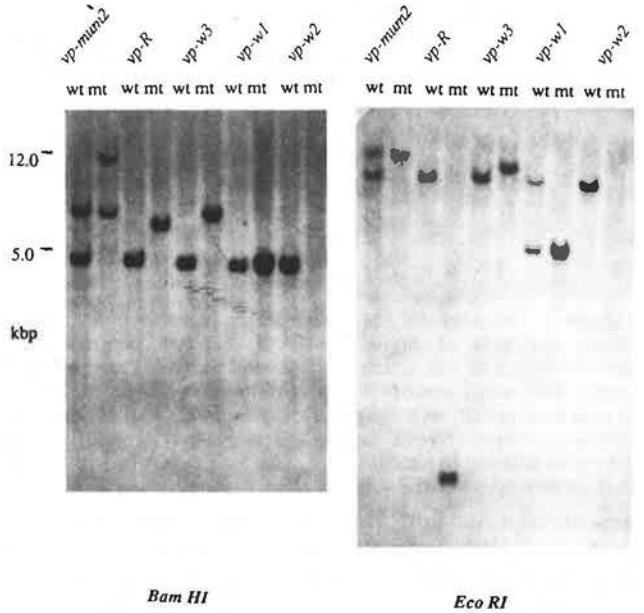


Figure 2. Southern blot analysis of *vp mutant stocks.*

wildtype alleles segregating in these stocks resemble those found in W22. At least two of these mutants, *vp-w1* and *vp-w2*, arose spontaneously in W22 inbred stocks. In addition, further restriction analysis (not shown) indicated the W22 allele as the probable progenitor of *vp-mum1* and *vp-w3*. Therefore, in these cases direct comparisons can be made. The rearrangements we see in these stocks, particularly the deletion in *vp-w2*, are convincing evidence that we have cloned *vp*. We can summarize the data as follows:

vp-mum2: This mutable allele should contain a Mutator insertion at *vp*. The normal plant in this case is a heterozygote. The apparent incomplete digestion of the mutant DNA by *Bam*HI is possibly due to DNA methylation associated with *Mu* element inactivation. (In other *mum2* plants we see complete digestion.) Note that we see different size shifts with *Eco*RI and *Bam*HI. Based on these and other polymorphisms detected by the righthand half of our clone we conclude that the wildtype (W22-like) allele segregating in this stock is not the progenitor of *vp-mum2*. However, using these polymorphisms we have identified the probable progenitor allele in the parental Mutator stock (obtained from Donald Robertson). Relative to that allele we see an approximate 1.4kbp *Bam*HI size polymorphism in *vp-mum2* consistent with a *Mu1*-size insertion.

vp-R: This is the standard mutant allele described by Robertson (Genetics 40:745). Although the progenitor is unknown, this mutant is clearly different from the wildtype W22 allele.

vp-w3: This apparently stable mutant arose in one of O. Nelson's *Ac Ds* stocks. In digests with at least 4 enzymes including the 2 shown we see a consistent 2kbp size shift in the mutant restriction fragment. Analysis of the parental stock (a gift of O. Nelson) revealed only the W22 wildtype allele. We conclude that this allele contains a 2kbp insertion, possibly a *Ds* element. We have confirmed that this stock contains active *Ac* by crossing it to a *sh2-m* (no *Ac*) stock. However, we see no evidence of *vp* mutability.

vp-w1: This mutant arose spontaneously in a homogeneous W22 background. One parent carried *R-st*, thus it may have been induced by the *I-R* transposon of *R-st* (J.L. Kermicle, personal communication). Interestingly, the rearrangement in this allele is detected in digests with *Eco*RI and *Sst*I (not shown), which cut to the right of the *Mu* insertion in pVPM1B (Fig. 1), but not with *Bam*HI (The normal plant is heterozygous). Therefore, the lesion must occur beyond the left border of pVPM1B, and the *vp* gene must extend beyond our clone. The data are consistent with either a large insertion containing several of the relevant restriction sites or a large deletion (>10kbp).

vp-w2: This allele also arose spontaneously in an *R-st* W22 stock. In this mutant the entire probe sequence has been deleted (at least 1kbp). We have confirmed this in digests with 4 different enzymes. However, sequences to the right of the 0.5kpb *Pst*I fragment in pVPM1B are detected in *vp-w2* DNA (not shown), therefore one breakpoint of the deletion occurs within the cloned region, roughly 2kbp from the site of the *Mu1* insertion in *vp-mum1*.

Figure 3 summarizes our conclusions from the Southern data. Curiously, all of the mutants for which we have firm data map toward the left end of pVPM1B. Nonetheless, probes from the right end hybridize to the largest *vp* transcript.

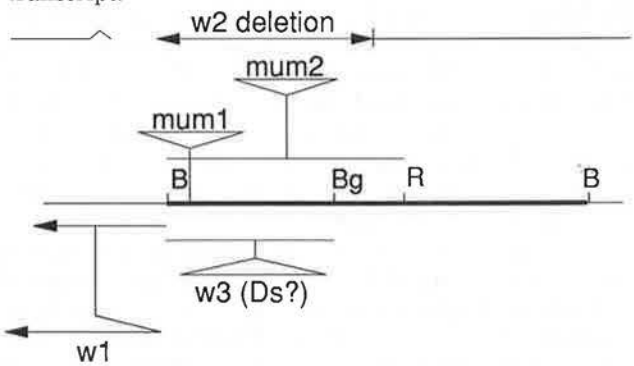


Figure 3. A physical map of *vp*. This schematic shows the approximate positions of 5 *vp* mutations relative to our clone. The drawing corresponds to the wildtype allele found in W22, which is apparently the progenitor of all the mutants shown with the exception of *vp-mum2*. B = *Bam*HI; Bg = *Bgl*II; R = *Eco*RI.

We are indebted to Hugo Dooner for sharing his extensive collection of *vp* mutants and to Stinard and Robertson for their *vp-mum1* and *vp-mum2* stocks.

Donald R. McCarty, Christian B. Carson
and S. Caprice Simonds

An activity stain for sucrose synthase

An activity stain was developed to detect sucrose synthase (SS) in starch gels. Sucrose synthase (EC 2.4.1.13)

catalyzes the reversible reaction of sucrose with UDP to produce UDP-glucose and fructose. The stain is based on a triple enzyme coupling mechanism. The production of fructose is coupled to hexokinase (HK) followed by phosphoglucoisomerase (PGI), and glucose-6-P dehydrogenase (G6PDH). The last enzyme is coupled to the tetrazolium stain.

Crude endosperm and embryo extracts from 25-day-old W22 kernels were obtained by grinding tissue with extraction buffer (Tris-HCl, 0.1 M, pH 7.5) in a 1:1 ratio. The homogenate was clarified by centrifugation at 16,000g for 30 min at 4 C. Immunoprecipitations were carried out according to Chourey (Mol. Gen. Genet. 184:372, 1981). An equal volume of antibody was added to the crude extract; PEG (8000 MW) was also added to a final concentration of 0.5%. The mixture was incubated for 4h at 4 C, and then centrifuged at 16,000g for 30 min at 4 C. Electrophoretic separation was performed in a discontinuous buffer system in 10% starch gels for 3.5h at 4 C at a constant voltage of 10.8V/cm gel. The gel buffer was histidine·HCl, 5mM, pH 7.0 (adjusted with NaOH); the electrode buffer was Tris 135mM/citric acid 43mM, pH 7.0. The staining solution contained sucrose 100mM, UDP 0.75mM, ATP 2.36mM, HK 1U/ml, PGI 0.5U/ml, G6PDH 0.3U/ml, $NADP^+$ 0.04mM, MTT 0.48mM, PMS 0.13mM, $MgCl_2$ 20mM, and Tris 0.1M, pH 7.5. It should be noted that ADP, an alternative for UDP as a substrate, is also present in this stain, due to the HK reaction. Gels were stained at room temperature, and SS bands appeared after about 1h.

Four criteria were used to determine the identity of the sucrose synthase bands in the gels: substrate specificity, tissue specificity, a deletion stock, and immunoprecipitation. The use of crude extracts in combination with a 3-enzyme coupling system leads also to the production of bands other than those representing SS activity. Modifications of the SS stain were used to account for all bands in the gel. Similar banding patterns were obtained when UDP was replaced with ADP; however, the 2 bands closest to the origin appeared at a much slower rate (Fig. 1). This would be expected for maize SS activity because the enzyme prefers UDP as a substrate. The stain can also detect invertase activity since this enzyme produces both fructose and glucose. Removal of PGI and UDP eliminated only the two bands closest to the origin, indicating that they corresponded to SS bands; invertase bands appeared only after prolonged incubation (12h). Finally, exclusion of all the coupling enzymes, ATP, and UDP yielded only the 4 most anodal bands. This is further evidence indicating that the bands closest to the origin represent zones of SS activity.

There are two loci which encode proteins with SS activity in the seed. While the product of the *Sh* locus is found only in the endosperm, the other isozyme is present in both endosperm and embryo. Accordingly, the *Sh-W22* endosperm extract (Fig. 1, lane a) had 2 bands corresponding to the 2 different enzymes, whereas the embryo extract (lane b) had only the most anodal of the 2 bands, SS-2. Furthermore, the endosperm extract of the *sh* deletion mutant, *sh bz-m4*, lacking most, if not all, of the coding region of the *Sh* locus, did not produce the SS-1 isozyme (lane c). Finally, endosperm extracts treated with extraction buffer or nonspecific antiserum (lanes d and e,

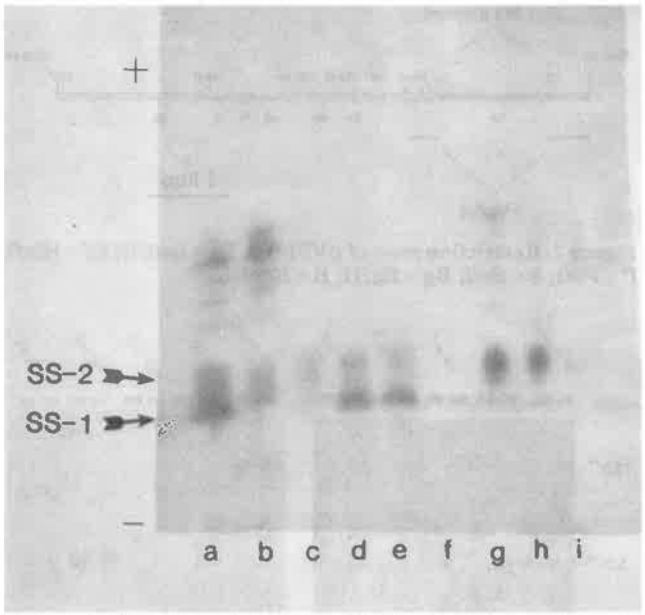


Figure 1. Gel stained for SS activity. Lanes a and b are crude extracts of *Sh-W22* endosperm and embryo, respectively. Lane c is crude endosperm extract from *sh-W22, bz-m4*. Lanes d-f are *Sh-W22* endosperm extracts incubated in extraction buffer, nonspecific antiserum, and *Sh* specific antibody, respectively. Lanes g-i are *Sh-W22* embryo extracts incubated in extraction buffer, nonspecific antiserum, and antibody, respectively.

respectively) had both isozymes, whereas treatment with antibody raised against the *Sh* protein (lane f) produced only the SS-2 band with markedly reduced activity. Embryo extracts incubated in extraction buffer (lane g) or nonspecific antiserum (lane h) showed the SS-2 band, however, incubation in the antibody (lane i) eliminated the band. Reduction in activity of the 4 most anodal bands in the immunoprecipitation treatments was due to dilution of the samples.

To our knowledge, this is the first report of an activity stain for sucrose synthase. Prior to this, it was only possible to measure overall SS activity in a sample. It will now be possible to examine and compare activities of the two isozymes.

B.L. Bournival, C.E. Vallejos, P.S. Chourey and L.C. Hannah

Recombinants lacking in detectable levels of both sucrose synthases are functionally normal

The two sucrose synthase isozymes, SS1 and SS2, encoded by the *Sh and Ss2* loci respectively, have been described previously (Chourey, MGG 184:372, 1981; Chourey & Still, Proc. Intl. Genetics Symp. p. 207, ed. Reddy & Coe, Oxford & IBH Publ., 1983; Echt & Chourey, Plant Phys. 79:530, 1985). All spontaneous *sh* mutants analyzed so far and a *sh* deletion mutant (*sh bz-m4*) show only the SS2 protein; the SS1 protein is completely lacking in the mutants. Western blot analyses show that both the *Sh* and *Ss2* genes are expressed in the wild type (*Sh*) genotype leading to either both homotetramers (S1S1S1S1 and S2S2S2S2) as in the developing endosperm cells, or a total of 5 isozymes (3 heterotetramers, S1S1S1S2, S1S1S2S2, and S1S2S2S2, and 2 homotetramers) as in the seedling cells (Chourey et al., MGG 203:251, 1986). The *Sh-r5* revertant described previously (Courage-Tebbe et al., Cell 34:383, 1983) has led to isolation of the first *Ss2-null*

allele (Chourey et al., 1986 CSH meeting, p.65 & manuscript submitted), a single protein band of SS1 electrophoretic mobility is seen in all tissues of this revertant. Because the *Ss2-null* (i.e. *ss2 ss2*) expression did not segregate independently from the homozygous *Sh-r5* phenotype, we suggested that both the SS encoding genes are on chromosome 9. Two independent molecular mapping studies have now mapped *Ss2* to 9L (McCarty et al., PNAS, 83:9099, 1986; Gupta et al., Plant Molec. Biol., 1987, in press).

The demonstrated loose linkage between *Sh* and *Ss2* genes (approx. 30 to 40 map units) led to the obvious question: can the double homozygotes lacking both SS1 and SS2 proteins be isolated? The data given below indicate that such recombinants can be isolated and surprisingly are functionally normal. Because the *Ss2-null* expression is not associated with any detectable plant/seed phenotype, individual *sh* segregants from the cross given below were examined by Southern and Western analysis. The usefulness of genomic Southern tests in such analysis is related to the fact that the *Ss2-null* allele in *Sh-r5* strain is unique (manuscript submitted). Briefly, the *Eco*RI genomic digests of the *ss2/ss2* (null) homozygotes yield 2 fragments (≈23kb and 8kb), the *Ss2/Ss2* homozygous allele in *sh bz-m4* stock shows a ≈16kb fragment and the *Ss2/ss2* heterozygote shows a combination of the 3 fragments.

We made the following cross: *Sh-r5/Sh-r5,ss2/ss2* × *sh/sh,(bz-m4),Ss2/Ss2*. The *sh* segregants on the F2 ears were selected, genomic DNA from a certain number of such individual *sh* seedlings was *Eco*RI restricted and examined for *Ss2* hybridizing fragments.

Number of individual F2 sh seedlings

Ear #	16 kb	16 + 23 + 8 kb	23 + 8 kb	Total
1	13	17	5	35
2	10	14	4	28
3	9	12	1	22
Total 3	32	43	10	85

Root extracts from some of the individual seedlings used for the above Southern tests were also examined by Western blot analysis for the SS protein. A total of 4 seedlings showing the 23 + 8kb fragments (the presumptive *ss2 ss2* genotype) from two separate ears showed no detectable levels of SS protein. This entirely unexpected observation leads us to suggest that the lack of detectable levels of SS1 and SS2 proteins is not associated with impaired functions relating to development, maturation or germination of the kernel. Similar immunoblot tests on root extracts from several individual seedlings of the other 2 classes (the presumptive *Ss2/Ss2* and *Ss2/ss2*) showed, as expected, a single SS2 protein band. Enzymatic analyses on these genotypes have not been done because routine tests have shown that Western blots are far more sensitive than the assay for enzyme activity, particularly in seedling extracts where the enzyme activity is highly labile.

In a second set of experiments, a F2 *sh/sh* plant from a sibbed progeny of the ear #1 was identified as having the 23 + 8kb *Ss2* hybridizing fragments (i.e. putative *ss2/ss2*, it was not typed for SS protein expression). It was used as a male parent in the following cross (lack of flowering synchrony prevented selfing): *Sh/Sh-r5,Ss2/ss2* × *sh/sh*, putative *ss2/ss2* (The female parent was also heterozygous for *Gl15/gl15* but is disregarded here). Four possible genotypes of the seedlings derived from this cross and the predicted SS protein pattern on Western blots are:

	SS bands
Sh/sh,Ss2/ss2	5
Sh/sh,ss2/ss2	1
Sh-r5/sh,Ss2/ss2	5
Sh-r5/sh,ss2/ss2	1

A total of 30 F1 individual seedlings have been typed so far by Western analysis. A segregation of 17 seedlings for 5 bands and 13 for 1 band pattern has led us to conclude that the male parent in the above cross was indeed of the *ss2/ss2* genotype. The important conclusion from this analysis is that plants lacking in detectable levels of both the sucrose synthases are not only nonlethal but are functional in completing the life cycle. Efforts are in progress to obtain a SS-null (*sh/sh, ss2/ss2*) stock so as to better understand the physiological role of sucrose synthase in the maize plant.

Prem S. Chourey

GRAND FORKS, NORTH DAKOTA
University of North Dakota

Protocol for fixation, embedding, sectioning, and staining of embryos

Below we describe our current protocol for the processing of maize kernels for paraffin sectioning of embryos. This protocol is a revised and more detailed version of that previously reported (J.K. Clark and W.F. Sheridan, J. Hered. 77:83, 1986).

Preparation of kernels and fixation: Immature and mature kernels are removed from freshly harvested ears and the sides of the kernels are trimmed with a razor blade in order to facilitate penetration of the fixative. The kernels are fixed in FAA [by volume, 60 parts 95% EtOH, 20 parts 20% formaldehyde, 5 parts glacial acetic acid, and 15 parts distilled water]. After 24 hours or longer of exposure to the fixative the vials containing the kernels are placed in a dessicator under vacuum, in order to remove any air remaining in the kernels.

Dehydration and embedding: Kernels are dehydrated by the tertiary butyl alcohol (TBA) procedure (D.A. Johansen, Plant Microtechnique, 1940). It is important to leave the kernels for an adequate time period for each step in the dehydration series; we use a 48-hour minimum time for each step until the kernels are moved into the oven, and then 8 to 24 hours for each step thereafter. The dehydration and embedding steps are as follows: one each of 20% EtOH and 30% EtOH, followed by 2 48-hour exposures to each of the following:

Grade 1 50% EtOH, 10% TBA, 40% distilled H_2O
Grade 2 50% EtOH, 20% TBA, 30% distilled H_2O
Grade 3 50% EtOH, 35% TBA, 15% distilled H_2O
Grade 4 50% EtOH, 50% TBA, saturated with Eosin Y
Grade 5 25% EtOH, 75% TBA, saturated with Eosin Y

We then place the vials on top (outside surface) of the embedding oven and provide 2 48-hour exposures to 100% TBA saturated with Eosin Y, followed by a 48-hour exposure to a mixture of equal parts of TBA and paraffin oil. The kernels are then placed in a small volume of fresh TBA:paraffin oil (1:1) and poured into fresh vials half filled with warm semi-hard paraffin wax. These vials are

placed in the 60C paraffin oven. After 8 hours (or overnight) the fluid is poured off and the kernels are exposed to 2 changes of paraffin wax of 8 to 12 hours each. This is followed by replacement of the wax with Paraplast plus for 8 hours, a change of Paraplast plus for 24 hours, and a final change of Paraplast plus for 48 hours. The kernels are then embedded in fresh Paraplast plus using Peel-away plastic models to cast the blocks. Casting is facilitated by use of a hot plate and slide warmer to keep the Paraplast plus molten and to allow arrangement of the kernels in the mold prior to placing on the surface of ice water (See Johansen for details). We orient the kernels so that the embryonic axis is facing downward toward the bottom of the mold.

Sectioning and Mounting: The blocks of Paraplast plus are trimmed so as to provide a 2-3mm margin of paraffin around the edges of the kernel, and then are attached with the aid of a heated dissecting needle to the end of a small oak block which has been previously soaked in melted paraffin. Sections are cut on a rotary microtome (see S.E. Sass, Stain Tech. 20:93, 1945), to obtain 15 micrometer thick serial sections. These ribbons are arranged on black construction paper in necktie boxes and scanned under a dissecting microscope. Sections lacking embryo material are discarded and the remaining sections are cut to convenient lengths and are mounted onto glass slides. A drop of Haupt's adhesive (A.W. Haupt, Stain Tech. 5:97, 1930) is spread on the surface of the slide using a fingertip and the surface is flooded with a 3% formalin solution, in order to float the ribbons. After gentle warming of the flooded slides so that wrinkles in the ribbons flatten out, they are drained and allowed to dry at least 24 hours.

Staining and coverslipping: The slides bearing the mounted sections are passed through 2 changes of xylene, one change of xylene:TBA (1:1) and 2 changes of 100% TBA, to remove the Paraplast plus and prepare the sections for staining in aqueous buffer. The slides are passed through 3 or more changes of distilled water (until the odor of TBA is no longer evident), and then one change of 0.025M citric acid-sodium phosphate buffer, pH 4.0. They are then placed in 0.005% Toluidine Blue O in the same buffer (pH 4.0) for 4 to 15 minutes. During staining the slides are periodically removed from the staining solution and examined under a dissecting microscope using transmitted light, to evaluate the staining intensity. When the sections are adequately stained they are rinsed for 1 minute in distilled water and air dried overnight. Overstained slides may be destained in a 1:1 mixture of TBA:Buffer followed by washing in the buffer before restaining. The air-dried slides are passed through 2 changes of xylene into clean xylene and coverslips are placed over 3 drops of Preserv-a-slide by gently lowering them from one end with a clean dissecting needle while holding the other end. The stained sections are photographed with Kodak Technical Pan 2451 which is developed with Technidol or Dektol.

We have had good success with this protocol in sectioning kernels aged from 0-80 days after pollination. For very young kernels we increase staining time. Older kernels frequently require a longer time in fixative, and an increase in the time allowed for the dehydration and embedding steps. Kernels may be stored in the FAA solution indefinitely. Note that TBA crystallizes at temperatures below 25C; therefore we add normal butyl alcohol (1 part to 99 parts TBA) to prevent this.

William F. Sheridan, Janice K. Clark, Guy Farish and Shelley M. Horne

HYDERABAD, INDIA
University of Hyderabad

c-m55437 - an autonomously mutating allele at the *C* locus

The *c-m55437* allele at the *C* locus has been isolated originally by Peterson (1963). This mutable allele is under the control of the *En* transposable element system. Test crosses of *c-m55437* (*c-m Sh Wx/c sh wx* × *c sh wx/c sh wx*) gave rise to an excess of colourless round kernels among the progeny (Table 1). This could be due to the segregation

Table 1: Test cross of spotted type. The cross was *c-m Sh Wx/c sh wx* × *c sh wx/c sh wx* (shrunken progeny not given).

Pedigree	Round Progeny		
	Spotted	Colourless	Coloured
87K 0331-1 × 0343	53	2	0
-2 × 0341	160	38	1
-3 × 0341	160	52	1
87K 0335-1 × 0341	97	13	2
-3 × 0341	124	29	11
-4 × 0342	115	28	6
-5 × 0341	160	27	7

of three or more *En* elements or the allele is autonomously mutating giving rise to a high frequency of colourless derivatives. In order to differentiate between these two alternatives, plants grown from colourless round sib kernels were crossed to the *En* tester, *c-m(r)/c-m(r)*, and an *En* line *c sh wx/c sh wx; En*. No spotted kernels were found among the progeny of either cross. These results confirm that the *c-m55437* allele is autonomously controlled by the *En* element.

Ch. Jayaram, P.A. Peterson and A.R. Reddy

pr-m860192 - a mutable allele at the *Pr* locus

An instance of instability at the *Pr* locus has been detected while analysing the *c-m55437* allele. The mutant phenotype exhibited red and purple spots on a colourless background on the same kernel. This spotting pattern is due to the mutability of an *En*-induced (autonomous) mutable *c-m55437* allele superimposed with the *pr* mutability. That the mutability is associated with the *Pr* locus was confirmed in crosses with *pr* tester. The progeny kernels exhibiting *pr* mutability showed purple sectors on a red background. This mutant has been designated *pr-m860192*. Preliminary data suggest that the mutability is induced either by an autonomous element or by more than one independently segregating copies of the same

Table 1: Test cross of sectored type. The cross *pr-m/pr* × *pr/pr*

Pedigree	Sectored	Purple	Red
87K 0376-1 × 0391	96	19	120
-3 × 0390	84	16	97
-4 × 0391	121	27	151
87K 0377-2 × 0390	76	13	105
-3 × 0394	115	23	152
-4 × 0391	83	42	88

element (Table 1). The *pr* mutant stocks also contained the *En* element. Further characterization of the allele is in progress.

A.R. Reddy, P.A. Peterson and Ch. Jayaram

Quantitative and qualitative analysis of amylolytic enzymes of etched mutant

Earlier we have reported (MNL, 1985) the effect of *et* mutation on chlorophyll pigmentation and chloroplast ultrastructure during greening of seedlings under light. We report here on the changes in total amylase in germinating kernels and young seedlings. The total amylase levels were estimated in et^+et^+ and *et et* kernels during germination and greening of seedlings (Table). The total amylase levels and the specific activity of amylase were observed to increase from the 3rd day to the 5th day after germination in et^+et^+ kernels, whereas in *et et* kernels the activity levels decreased during the same period. Further, the total amylase content was significantly higher in et^+et^+ kernels as compared to *et et* kernels at all tested stages. In the developing seedlings too, the total amylase levels were significantly higher in normal (et^+et^+) leaves than that of virescent leaves of *et et* genotype. Total amylase levels peaked at the 8th day followed by a decrease by the 10th day in leaves of both genotypes. Similarly, the specific activity of total amylase peaks at the 8th day in et^+et^+ seedlings. However, the mutant seedlings show continuous increase up to the 10th day. α and β amylase studies in kernels and seedlings of both genotypes are in progress. Electrophoretic profiles of total amylase did not show any qualitative differences between et^+et^+ and *et et* genotypes throughout the period of kernel germination.

Days after germination	Total amylase activity* (n.Kat/mg kernel/gm.leaf)	
	et^+et^+	*et et*
3	162	100
4	198	78
5	236	52
6	282	200
8	508	278
10	312	212

* Each value represents an average of at least 5 independent experiments.

H.G. Sangeetha and A.R. Reddy

IOWA CITY, IOWA
University of Iowa

Suppression of meiotic loss by distal regions of the B chromosome

B^A chromosomes are subject to meiotic loss in the female meiosis. The rate of meiotic loss increases greatly when specific regions of the B chromosome are deleted. For TB-9Sb, deletion of the centric heterochromatin produces very high rates of B^9 loss (Carlson, Crit. Rev. Pl. Sci. 3:201, 1986). The effect of deleting a distal (9^B) region on meiotic loss of the B^9 is reported here.

9^B-2150 is a deletion derivative of the standard 9^B. It lacks virtually all of the distal B chromatin on 9^B except for a small piece of heterochromatin adjoining the translocation breakpoint. Previous data indicated that recovery of *C* in crosses of 9(*c*) 9^B-2150 $B^9(C)$ × *cc* is quite low compared to that in standard TB-9Sb crosses. Consequently, a controlled test of B^9 loss was developed. Hyperploid standard TB-9Sb plants were crossed as female to a homozygous TB-9Sb-2150 stock: 9(*wx*) $9^B(Wx)$ $B^9(C)$ $B^9(C)$ × 9^B-2150(*Wx*) 9^B-2150(*Wx*) $B^9(C)$ $B^9(C)$. Among the progeny, *Wx Wx* plants were selected by pollen classification. These contain $9^B(Wx)$ 9^B-2150(*Wx*) $B^9(C)$ $B^9(C)$. Next, the *Wx Wx* plants were crossed as female to a *c wx* tester. Two types of progeny were produced in this cross: 9(*c wx*) $9^B(Wx)$ $B^9(C)$ and 9(*c wx*) 9^B-2150(*Wx*) $B^9(C)$. The progeny were classified for 9^B type by a test of nondisjunction. Each plant was crossed as male to a *bz* tester and the presence or absence of nondisjunction determined (9^B-2150 lacks a region that is required for B^9 nondisjunction.) The same plants were backcrossed as female to a *c wx* tester in order to determine rates of meiotic loss. The ears produced were classified for meiotic loss according to a method reported in the MNL in 1985 (p. 54). Results are given below:

Plant Number of Female Parent	Rate of Meiotic Loss
Standard 9^B	
7118- 5	15% (33-34/223)
7118-16	2- 3% (0-3/198)
7118-37	27-28% (49-51/181)
7118-54	11-12% (20-21/180)
7118-60	16% (28-29/179)
7118-64	18-19% (33-35/186)
7118-65	17-18% (37-39/213)
7118-66	2- 3% (4-5/182)
7118-67	19-20% (34-36/182)
7118-68	10-11% (23-25/227)
Deficient 9^B	
7118- 4	56-57% (94-96/168)
7118- 7	50% (107-108/215)
7118-10	60% (93/155)
7118-27	65-66% (148-149/226)
7118-36	51-52% (103-105/202)
7118-57	40-41% (75-77/186)
7118-71	51% (84/164)
7118-73	43% (71-72/167)
7118-74	61% (85-86/140)
7118-76	65-66% (89-90/137)

A range of values is given for most plants because the correct value for meiotic loss cannot be determined precisely. Nevertheless, the data show that deletion of a distal B segment on 9^B produces a large increase in B^9 loss. Suppression of meiotic loss by the standard 9^B must occur by a *trans* effect on the B^9. [Note: the calculation of meiotic loss was recently modified from the prior formula in terms of the treatment of a small crossover class, *c Wx*. The change is insignificant to the results and will be discussed elsewhere. The new formula is: *c wx* - *C Wx* - (0.0 to 0.5 *c Wx*) *divided by* total *wx*.]

W. Carlson

Male/female differences in crossing over: the effect of B chromosomes

Robertson (Genetics 107:117, 1984) reported a dramatic effect of B-A translocations on crossing over in the male meiosis. He showed that the hypoploid (A A^B) constitution gives a markedly higher rate of crossing over than the standard (A A) constitution for regions near the translocation breakpoint. The effect seems to be a general one, since a number of different translocations were tested.

Several explanations were suggested for the hypoploid effect. One possibility was a difference in recovery of gametic classes between male and female, based on the heteromorphic nature of A A^B bivalents. Another explanation was that heterochromatin from the B chromosome may enhance male crossing over on A^B chromosomes. It is also possible that the partial hemizygosity in A A^B plants affects crossing over.

The alternative explanations were tested using TB-9Sb. The 9^B chromosome contains most of the distal B heterochromatin. The ability of a standard 9^B to enhance crossing over was compared to the effect of a modified 9^B The latter 9^B (designation 9^B-2150) is a deletion derivative that lacks virtually all of the B heterochromatin (Fig.

2, Carlson & Curtis, Can. J. Genet. Cytol. 28:1034). A family was constructed which contained two types of plants: 9 9^B (standard) B^9 and 9 9^B (deletion) B^9. Five plants of each type were tested for crossing over in the *wx-v* interval, which is adjacent to the translocation breakpoint. In each plant, chromosome 9 carried *wx v* and 9^B had *Wx V*. Reciprocal testcrosses were made, with the results listed below.

Plant type	Plant Number	Crossing over in male (2 ears)	Crossing over in female (1 ear)
Standard 9^B	6825-22	10.5% (66/627)	7.3% (24/330)
	6825-29	9.1% (51/560)	4.9% (18/371)
	6825-30	16.8% (95/566)	8.7% (38/437)
	6825-32	10.2% (51/499)	5.1% (14/277)
	6825-34	6.3% (36/568)	6.4% (22/344)
	Average	10.6% (299/2820)	6.6% (116/1759)
Deletion 9^B(2150)	6825- 2	3.9% (26/669)	5.6% (11/191)
	6825- 3	2.7% (15/546)	3.7% (13/354)
	6825-17	1.6% (8/514)	2.9% (9/308)
	6825-27	3.5% (20/577)	5.7% (16/279)
	6825-28	2.8% (18/652)	6.8% (15/222)
	Average	2.9% (87/2958)	4.7% (64/1354)

The number of plants tested is small and needs to be expanded. Nevertheless, the data indicate that crossing over in the male meiosis is considerably higher in the presence of a standard 9^B compared to the heterochromatin deficient 9^B. The range of crossover rates in the male is 6.3-16.8% for standard 9^B vs. 1.6-3.9% for the deficient 9^B. The rates for crossing over in the female meiosis are much closer between the two groups and there is overlap in the ranges: 4.9-8.7% for standard 9^B vs. 2.9-6.8% for the deficient 9^B.

The findings suggest that Robertson's crossover effect is due in part to the presence of B heterochromatin on the A^B chromosome. The effect is not as strong as that found by Robertson, suggesting that other factors may also be involved (note that tests were conducted with balanced heterozygotes rather than hypoploids, and consequently any effect of hypoploidy on crossing over was not measured.) However, it is most interesting that three classes of maize heterochromatin (B, knob and centromeric) have now been implicated in effects on male vs. female crossover rates (Rhoades, Maize Breeding and Genetics, 1978).

W. Carlson

A technique for producing duplications of distal chromosomal segments

Various cytogenetic techniques have been employed for producing whole chromosome or partial chromosome duplications in maize. However, most of the methods are difficult to utilize, since the duplications produced are not heritably stable. For example, nondisjunction of B^A chromosomes can be used to construct hyperploid plants (A A^B B^A B^A). These plants are trisomic for a distal A chromsome segment. The condition, however, is not uniformly transmitted in crosses due (in part) to B^A nondisjunction.

A procedure for constructing stable or nearly stable duplications has been developed. This method combines the chromosomes of a standard (A-A) translocation and a B-A translocation in a unique manner. The basic idea is to utilize certain adjacent I gametes produced by A-A translocation heterozygotes. Adjacent I gametes are both duplicate and deficient. They are normally inviable due to their deficiency. However, the addition of an appropriate B^A chromosome will cover the deficiency and rescue the gamete along with its duplication.

The technique was tested by combining T8-9(4453) with the B^9 from TB-9Sb. Translocation breakpoints for T8-9(4453) are 8L 0.86 and 9S 0.68. The adjacent I gamete to be rescued is 8 9^8. This gamete lacks the distal 0.32 of 9S. In TB-9Sb, the breakpoint on 9S is 0.4 (Roman and Ullstrup, 1951). The B^9, therefore, carries the distal 0.6 of 9S and can rescue the 8 9^8 gametic class (it also produces a second duplicated region in the gamete due to overlap between the B^9 and 9^8).

The steps involved in producing the duplication gamete (8, 9^8, B^9) are as follows:

1. Cross hyperploid TB-9Sb as female to homozygous T8-9.
   9(*wx*) 9^B(Wx) B^9B^9 × 9^8(*Wx*) 9^8(*Wx*) 8^9 8^9
2. Germinate progeny and select *Wx wx* plants by pollen classification. These plants contain 8, 8^9, 9(*wx*) 9^8(*Wx*) B^9. They are heterozygous for T8-9 and contain a B^9 chromosome (the 9^B was selected against in this step. Its absence can be confirmed by a test of nondisjunction, since the 9^B is required for B^9 nondisjunction).
3. Cross the selected plants as female to a *wx wx* tester. Some of the progeny should receive 8, 9^8 (*Wx*) B^9 from the female.
4. Germinate progeny from (3) and self pollinate. Select ears that show a reduced (less than 75%) frequency of *Wx* kernels (low *Wx* transmission is due to pollen competition against duplication gametes).
5. From the selected ears, germinate seeds and select *Wx Wx* plants. These should be duplication homozygotes (8 8 9^8 9^8 B^9 B^9).
6. Self pollinate to establish a homozygous stock.

By following this protocol, two plants (6799-1 and 6801-1) were identified as putative homozygotes (step 5) and self-pollinated. The seeds produced were tested to confirm presence of the duplication. The first test involved germinating five kernels from each ear and checking chromosome numbers in the roots. Results are currently available for 6799-1. All five plants derived from ear 6799-1 contained 22 chromosomes. The extra two chromosomes were shown to be B^9's, as expected.

Next, outcrosses were made of the putative homozygotes. Two crosses to tester lines were selected for analysis: *bz bz wx wx* × 7011-2 and *bz bz wx wx* × 7012-6 (Family 7011 derives from plant 6799-1 while family 7012 comes from 6801-1). These crosses gave *Bz* progeny as expected, although an occasional *bz* kernel or variegated (*Bz/bz*) kernel was produced. Forty-six *Bz* progeny from the 7011-2 cross and 43 *Bz* progeny from the 7012-6 cross have been checked for chromosome constitution. All 89 had 21 chromosomes, as expected for crosses of a homozygous duplication. (The *bz* and variegated kernels have not yet been analyzed, but their appearance in these crosses suggests that B^9 instability may be present).

One final test was performed. Homozygotes derived from 6799-1 and from 6801-1 were crossed (as male) to a *wx* tester. Next, the *Wx wx* progeny were testcrossed as male to *wx wx*. Transmission of *Wx* through the pollen was determined. It should be less than 50%, due to pollen competition, if the duplication was present. Results for eight testcrosses are given below. In each cross, selection against *Wx* pollen is strong. The findings indicate that the homozygous duplication 8 8 9^8 9^8 B^9 B^9 has been constructed.

Type of Cross	Designation of crosses	Classification (Two ears each) Wx	wx	%Wx
1. wx wx X duplication heterozygote (from 6799-1)	7002 X 7013-2	89	620	12.6%
	7002 X 7013-3	54	506	9.7%
	7002 X 7013-5	60	585	9.3%
	7002 X 7013-6	44	559	7.3%
2. wx wx X duplication heterozygote (from 6801-1)	7002 X 7014-4	56	620	8.3%
	7002 X 7014-5	44	671	6.1%
	7002 X 7014-6	39	530	6.9%
	7002 X 7014-7	68	563	10.8%

On theoretical grounds, the duplication is expected to be relatively stable because a) each of the three chromosomes carries vital genes not supplied by the other chromosomes, and b) the B^9 is incapable of nondisjunction in the absence of 9^B. However, a potential source of instability is residual nondisjunction of the B^9. The occasional (<1%) appearance of *bz* kernels in crosses of *bz bz* × homozygous duplication indicates that some nondisjunction may occur. Also, the appearance of variegated kernels in these crosses may provide another source for changes in B^9 number. However, changes in B^9 dosage do not destroy the integrity of the duplication itself. A reversion to normal chromosomes can only occur by crossing over between the short, overlapping regions of 9^8 and B^9 to restore a normal 9. The cytological results presented earlier suggest that such crossing over is not common. The n+1 gamete is regularly transmitted by the homozygous duplication. Current tests, therefore, indicate a high level of stability of the duplication chromosome structure, even though B^9 dosage may vary occasionally.

W. Carlson and R. Roseman

Stability of "double A^B" duplications

A method for duplicating proximal chromosomal segments using A^B chromosomes was reported by Carlson and Curtis in 1986 (Can. J. Genet. Cytol. 28:1034). Two duplications were constructed, one for chromosome 9 and one for chromosome 3. In both cases, overlapping A^B chromosomes were utilized. For example, the 9^B from TB-9Sb and the 9^B from TB-9La were combined to produce plants containing 9^{BSb} 9^{BSb} 9^{BLa} 9^{BLa} in place of two normal 9's. Preliminary cytological data indicated that this type of duplication is relatively stable in inheritance.

A genetic test for stability of the chromosome 9 duplication is reported here. Stability depends on the formation of homomorphic bivalents in meiosis: 9^{BSb} 9^{BSb} and 9^{BLa} 9^{BLa}. If heteromorphic bivalents form or if a quadrivalent forms, crossing over between 9^{BSb} and 9^{BLa} could occur. Crossing over would produce a normal 9 and break down the duplication.

To test for formation of a crossover 9, duplication homozygotes carrying the *Wx* marker were crossed as female to a *wx* tester. If the crossover 9 was not produced in the female, all progeny should contain 9(*wx*) 9^{BSb} (*Wx*) 9^{BLa}(*Wx*). However, if crossing over occurred between 9^{BSb} and 9^{BLa}, some progeny should carry a normal 9(*Wx*). The constitution of progeny was checked by germinating forty-nine kernels and crossing the plants as male parents to a *wx* tester. One ear per male parent was classified for *Wx* vs. *wx* phenotypes. Forty-six of the forty-nine plants tested gave *Wx* frequencies below 50% (average 29% *Wx*. Range 15-42% *Wx*). Three of the plants gave *Wx* rates of 50% or more (52%, 59%, 66%). The frequency expected in the presence of a crossover 9(*Wx*) is at least 50%. Therefore, forty-six plants must have carried the (noncrossover) duplication chromosomes. Selection against the duplication by pollen competition gave the reduced *Wx* frequencies. The three plants that gave 50% or more *Wx* probably contained a crossover 9(*Wx*) (in addition, they may have contained an extra 9^{BSb} or 9^{BLa} or crossover 9, depending on chromosomal segregation patterns.) An alternative possibility to the presence of a crossover 9 in the aberrant crosses is the presence of extra chromosomes through meiotic nondisjunction of 9^{BSb} or 9^{BLa}. This would mean that plants which gave aberrant ratios contained either 9(*c wx*) 9^{BSb}(*Wx*) 9^{BSb}(*Wx*) 9^{BLa}(*Wx*) or 9 (*c wx*) 9^{BSb}(*Wx*) 9^{BLa}(*Wx*) 9^{BLa}(*Wx*). Both types of plants might transmit *Wx* at high frequency due to the extra chromosome carrying *Wx* (chromosome number = 22). However, *Wx* progeny from a 22 chromosome plant should usually contain 21 chromosomes. When *Wx* kernels on the three aberrant ears were sampled (ten per ear) all had 20 chromosomes. Consequently, crossing over rather than meiotic nondisjunction appears to be the cause of ears with a high *Wx* frequency. This finding makes utilization of the proximal "double A^B" duplications more difficult, since periodic re-testing of stocks will be needed to insure their constitution.

W. Carlson

IRKUTSK, USSR
Siberian Institute of Plant Physiology and Biochemistry
KRASNODAR, USSR
Krasnodar Research Institute of Agriculture

High level of RNA synthesis in mitochondria of spontaneous and induced corn mutants

We found that the rate of mitochondrial DNA transcription in the mitochondria isolated from dwarf mutant A344 *sin/sin* seedlings proved much higher than in A344 +/+ (MNL 61:61, 1987). The high level of mtRNA synthesis was revealed for the mitochondria of newly induced small kernel mutant Gb334 *meg/meg* (MNL 60:70, 1986) and spontaneous mutants W64A *wx/wx* and Sg25 *o2/o2* (Table). To characterize the rate of mtRNA synthesis in mutants the ratio of absolute rate of RNA synthesis in mitochondria of spontaneous mutants to that of relative controls carrying dominant alleles of the genes was used.

Mutant	Relative rate of RNA synthesis in mitochondria			
	5 min	10 min	15 min	20 min
A344 *sin/sin*	0.60	6.54	7.26	3.87
Gb334 *meg/meg*	2.00	2.58	3.94	2.50
W64A *wx/wx*	1.36	1.48	1.03	1.32
Sg25 *o2/o2*	1.31	1.47	1.06	1.86

The positive correlation between the value of phenotypic effect governed by the recessive allele and relative and absolute rates of mtRNA synthesis is typical for the given set of mutants.

Yu.M. Konstantinov*, A.S. Mashnenkov, G.N. Lutsenko* and V.A. Podsosonny*
*Siberian Inst. Plant Phys. Biochem., Irkutsk

ITHACA, NEW YORK
Cornell University

Evaluation of tissue culture-derived methomyl resistant *cms-T* lines

We have previously reported the recovery of many plants resistant to methomyl following in vitro selection using 6-8 month old callus derived from normally sensitve *cms-T* inbreds and hybrids (MNL 61: 59-60). Field tests were conducted this past summer using first (R1) and second (R2) generation progeny from regenerated plants (R0). Lannate (whose active ingredient is methomyl) was applied at 1.12 lbs/A, twice the rate recommended for sweet corn, before evaluation. Results indicate a maternal mode of inheritance for the methomyl resistance trait. All methomyl-resistant plants were also male-fertile and HmT toxin resistant. Segregation for resistance and sensitivity was seen among ear-to-row progeny from some self- and cross-pollinated R0: 4/21 rows from sensitive R0 and 3/69 rows from resistant R0. None of the R2 examined showed segregation in the field. The N- and T- cytoplasm checks were uniformly resistant and sensitive, respectively. Test crosses indicate that restoration of male-fertility was not due to a novel or newly activated dominant *cms-T* restorer gene.

Regenerated plants were also recovered from callus cultured for 14-16 months. Many (19/31 or 61%) methomyl-resistant R0 were obtained from non-selected control callus. Only 2% of the plants regenerated from callus 6-8 months in culture showed such spontaneous resistance. This result suggests that prolonged time in culture increases spontaneous resistance and concurrent reversion to male-fertility. Not all genotypes responded similarly, suggesting an effect of nuclear background on frequency of these traits.

Analysis of phosphorylation by mitochondria isolated from resistant R1 and R2 and N-cytoplasm checks, shows that resistant mitochondria are unaffected by the addition of 1mM methomyl. Mitochondria isolated from sensitive R1 and R2, and T-cytoplasm checks, show over a 70% decrease in their phosphorylation activity in the presence of 1mM methomyl. These results suggest that the tissue culture-derived resistance may arise from an alteration in the mitochondrion. Molecular studies of mitochondrial DNA from resistant and sensitive lines are currently in progress.

A.R. Kuehnle and E.D. Earle

Effects of HC toxin on growth and regeneration of susceptible and resistant callus

Helminthosporium carbonum race 1, a fungal pathogen of corn, produces a host-specific toxin (HC toxin) known to be a pathogenicity factor for the disease Helminthosporium leaf spot. The toxin has been purified and the structure is known (J. Walton and E.D. Earle, Biochem. Biophys. Res. Comm. 107:785. Major host resistance and insensitivity to the toxin are conditioned by a single dominant gene *Hm* (O.E. Nelson and A.J. Ullstrup, J. Hered. 55:195). Root-growth assays have shown that certain corn genotypes susceptible to the fungus are sensitive to low toxin concentrations (root growth is inhibited). Corn genotypes resistant to the fungus are also sensitive to the toxin, but at higher concentrations. We describe here studies which demonstrate a differential effect of HC toxin on growth and regeneration of susceptible and resistant callus and our attempts to recover novel resistant genotypes using an in vitro selection system.

Our initial attempts to establish regenerable callus of susceptible and resistant near-isogenic inbred lines, Pr and Pr-1, resp., were unsuccessful. Therefore, to obtain susceptible genotypes which were amenable to tissue culture, we used the following approach. The susceptible inbred Pr (*hm hm*), which does not produce regenerable callus, was crossed to the resistant inbred W182BN, which readily produces regenerable callus. The resulting F1 progeny were self-pollinated. Plants derived from those self-pollinations were grown in the field and inoculated with a spore suspension of *H. carbonum* race 1 to identify susceptible genotypes. A F2 population of that cross segregated 136 resistant to 39 susceptible plants, which was an acceptable fit to the expected 3 resistant:1 susceptible ratio if the *Hm* locus were segregating. In 1985 and 1986, immature embryos from susceptible and resistant plants were excised ten days after pollination and plated onto a maintenance medium containing 15 μM 3,6-dichloro-o-anisic acid (D.R. Duncan, M.E. Williams, B.E. Zehr and J.M. Widholm, Planta 165:322). Resistant and susceptible callus was subcultured at three-week intervals. Plants were regenerated by transferring callus onto a modified LS medium with no hormones and decreasing percentages of sucrose (10, 4 and 2 percent - two to four weeks on each).

The toxin preparation used in these studies was kindly provided by Dr. V. Macko at the Boyce Thompson Institute for Plant Research, Cornell University, Ithaca, NY. Toxin was incorporated into the medium at the appropriate concentrations just before the media were poured into plates.

To determine toxin concentrations inhibitory to callus growth, susceptible and resistant calli were placed on media containing concentrations of 0, 0.5, 1, 2, 5, 10, and 20 μg/ml toxin. The callus used in those experiments was not friable and had many organized areas. Initial and final (4 weeks later) fresh weights of the calli were recorded. The percent increase in growth for each treatment was calculated (Figure 1). Exposure to increasing toxin concentrations gave progressively greater growth inhibition, as well as amount of necrotic tissue (data not shown) of susceptible callus. Resistant callus was not inhibited at concentrations up to 20 μg/ml (Figure 1) but was inhibited at the concentration of 50 or 100 μg/ml (Figure 2).

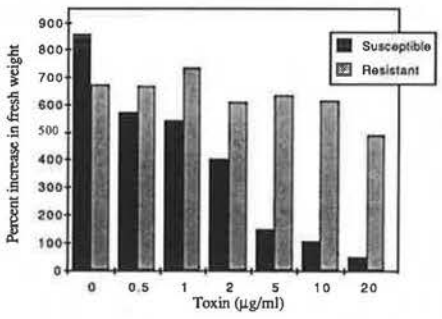


Figure 1. Percent increase in fresh weight of susceptible and resistant callus exposed to various concentrations of HC toxin for four weeks.

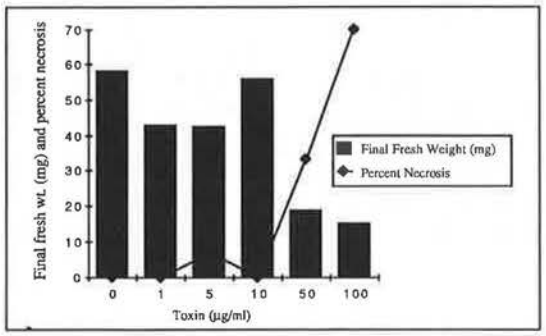


Figure 2. Final fresh weight and percent necrosis of resistant callus exposed to various concentrations of HC toxin for four weeks. Initial fresh weight of individual calli was 10 mg.

To study toxin effects on regeneration 16 susceptible and resistant calli were plated onto regeneration media with 0, 1, 2, 5, 10 and 20 µg/ml toxin (Table 1). At 5 µg/ml and higher, susceptible callus formed no large shoots, whereas resistant callus formed large shoots at all toxin levels.

Table 1. Organogenesis on Susceptible(S) and Resistant(R) Callus with Various Levels of HC Toxin.

Toxin[a] µg/ml	# calli w/ small shoots		# calli w/ roots		# calli w/ large shoots		total large shoots	
	S	R	S	R	S	R	S	R
0	14	13	16	15	14	5	20	17
1	10	9	10	16	10	7	14	14
2	11	14	10	13	5	2	10	6
5	8	11	1	13	0	7	0	10
10	5	11	1	11	0	3	0	9
20	1	8	0	9	0	3	0	9

[a] Sixteen calli were on regeneration medium (10 and 4% sucrose) containing the toxin concentration indicated for four weeks.

Those data were used as the basis for the following selection scheme. Objectives were to recover novel resistant genotypes and compare the efficacy of mutagens, age of callus and selection pressure for the recovery of resistant genotypes. A total of 5,676 calli (10 to 20 mg) were used. The mutagens sodium azide and ethyl methane sulfonate were applied to callus at predetermined sublethal concentrations prior to toxin exposure. Callus which had been in culture for 3 months (established 1985) and 15 months (established 1986) was subjected to toxin selection. Initial toxin concentrations of 2, 5, and 10 µg/ml were used. Those calli on 2 µg/ml were subjected to a stepwise toxin increase of 5 µg/ml at the first transfer. Some of the calli exposed to 5 µg/ml were subjected to 10 µg/ml at the first transfer. At each selection or transfer (approximately 3-week intervals) individual calli were scored for percent necrosis. All nonnecrotic areas were transferred. The percentages of calli (based on initial number of calli) which were eliminated in each selection regime for each of six selection cycles are shown in Figure 3. To date, we have not recovered any resistant callus in any selection regime. Mutagen application or varying callus age did not affect the recovery of resistant callus. Growth of all susceptible calli was inhibited by the toxin and all eventually became 100% necrotic. Callus that we knew to be resistant (W182BN) was exposed to similar toxin levels for the same amount of time. That callus readily grew and regenerated resistant plants at the fourth, fifth, and sixth selection cycles. Failure to recover resistant callus might be due to an inability to identify resistant cells within relatively

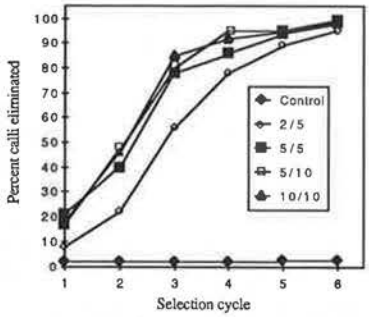


Figure 3. Percent calli eliminated in various selection regimes during six cycles of selection. Paired numbers in legend refer to toxin exposure (µg/ml) of the first selection cycle followed by toxin exposure of the subsequent selection cycles (2 to 6).

slow-growing callus cell populations. Moreover, susceptible cells surrounding a resistant cell might leak components which are lethal to the resistant cell. To address those possibilities, additional selection experiments using fast-growing cell suspensions are underway.

S.J. Wolf and E.D. Earle

Differential effects of HC toxin on susceptible and resistant protoplasts

In addition to studying the effects of HC toxin on growth and regeneration of susceptible and resistant callus, we looked at the effects of HC toxin on protoplasts. The objective was to study the effects of HC toxin on protoplasts derived from susceptible and resistant near-isogenic genotypes in an effort to provide information which might prove useful for understanding the mechanism of action of the toxin

Mesophyll protoplasts were isolated from leaves of 10 to 12 day-old susceptible (Pr × K61) and resistant (Pr1 × K61) corn plants by incubating leaf pieces in an enzyme solution containing 2% cellulysin, 0.5M sorbitol, and 10 mM $CaCl_2.2H_20$ for 3 to 4 hours. Isolated protoplasts were cultured in a minimal medium containing 0.5 M sorbitol + 10 mM $CaCl_2$ + 5 mM MES buffer at a density of 1.0×10^5 protoplasts/ml unless otherwise stated. The toxin used in these studies was the same preparation as that used for the callus work (see previous article).

The most striking differential effect of the toxin was in distribution of chloroplasts. Populations of susceptible protoplasts treated with low toxin concentrations had more protoplasts with chloroplasts well distributed throughout the cytoplasm than control (untreated susceptible and resistant) or resistant protoplasts treated with low toxin concentrations (Figure 1). That effect occurred after a

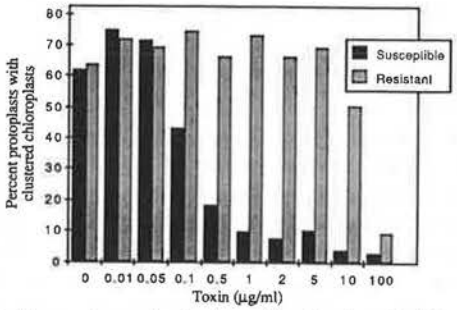


Figure 1. Percent protoplasts with clustered chloroplasts after exposure to various toxin concentrations. Protoplasts were cultured in the dark in minimal medium for 60 hours. Greatest standard deviation is 10.

minimum of 36 hours of toxin treatment. Control and resistant (low toxin levels) protoplast populations had chloroplasts localized and clustered within the cytoplasm. Like previously reported assays, resistant genotypes showed a similar toxin effect, but at higher toxin concentrations. A greater percentage of resistant protoplasts treated with high toxin concentrations had well-distributed chloroplasts. Protoplasts with well-distributed chloroplasts looked similar to protoplasts cultured in a complete nutrient medium. For that reason we compared the effects of HC toxin on susceptible and resistant protoplasts when cultured in a complete medium (D.S. Brar, S. Rambold, O.L. Gamborg, and F. Constabel, Z. Planzenphysiol. 96:269). In those experiments, a differential response for chloroplast distribution was not as pronounced as that which occurred in a minimal medium and was noted only after a much longer toxin exposure.

In addition to studying differences in chloroplast distribution, we looked at differences in viability by staining with fluorescein diacetate (FDA) or Evans Blue. Low toxin concentrations increased long-term viability of susceptible protoplasts whereas high toxin concentrations increased long-term viability of resistant protoplasts. Like differences in chloroplast distribution, viability differences were not immediate (Table 1). Similar differences in long-term viability were not noted when susceptible and resistant protoplasts were cultured in a complete nutrient medium. Additional studies are underway to determine components of the complete medium which influence the toxin effect.

Table 1. Viability rating (based on fluorescence) of populations of susceptible and resistant protoplasts cultured with various concentrations of toxin in minimal medium for 8, 11, 14 and 18 days.

	Susceptible				Resistant			
Toxin µg/ml	0	2	10	100	0	2	10	100
8 days	5*	5	5	5	5	5	5	5
11 days	5	5	4	2	5	5	5	4.5
14 days	3	5	4	1	3	3	4	4.5
18 days	2	5	4	1	2	2	2	4

*Rating (based on number and intensity)
0 = no protoplast fluorescence
3 = moderate protoplast fluorescence
5 = strong protoplast fluorescence

S.J. Wolf and E.D. Earle

JOHNSTON, IOWA
Pioneer Hi-bred International

An independent, EMS-induced dominant male sterile that maps similar to *Ms41*

We received remnant seed from an EMS seed-mutagenized W23 plant in 1978 from Dr. R.L. Phillips at the University of Minnesota. This seed was brought to my attention because plants had exhibited male sterility segregation, even after being crossed by normal W23. Subsequent crosses established that the male sterility was the result of a dominant, nuclear mutation at a previously undescribed locus. In 1983 sufficient data had been collected on this mutant to locate it to chromosome arm 4L using the waxy translocation series. We did not publish these results because we had wanted to complete an on-going developmental study of the microsporogenesis of this mutant and to present it with the genetic segregation data. In 1987, Dr. M.G. Neuffer from the University of Missouri presented mapping information on a dominant male sterile he had isolated from his EMS pollen mutagenesis work. He gave this dominant male sterile the designation *Ms41* and located it on 4L using the waxy translocation series. When we compared his data to our data, we discovered that our dominant male sterile, which we had designated *Ms*-7255*, seemed to map very similarly to his *Ms41*.

INTERCHGE	BRK PT		IPL	MsWx	N Wx	Mswx	N wx	INDEPEN. CHI SQ.	%CO	S.E.
1-9c	S.48	L.22	316	109	86	64	57	0.275	47	0.03
1-9(4995)	L.19	S.20	141	38	27	44	32	0.005	50	0.04
2-9b	S.18	L.22	248	64	82	53	49	1.591	54	0.03
2-9d	L.83	L.27	310	83	95	59	73	0.114	50	0.03
3-9c	L.09	L.12	171	41	43	44	43	0.053	51	0.04
4-9b	L.90	L.29	173	76	9	9	79	108.5**	10	0.02
4-9g	S.27	L.27	119	51	26	17	25	7.362**	36	0.04
5-9a	L.69	S.17	554	97	89	194	174	0.016	51	0.02
6-9a	S.79	L.40	335	68	81	105	81	3.874+	56	0.03
8-9(6673)	L.35	S.31	126	30	22	39	35	0.307	48	0.04
9-10b	S.13	S.40	204	57	88	35	24	6.783+	60	0.03

** Significant at the 1% level
+ See text below

These data show independence chi-square values that are highly significant for T4-9b and T4-9g. The value is extremely high for T4-9b, indicating a strong departure from independence and, therefore, indicating linkage. The value for T4-9g is not nearly as high. We do not know whether this represents reality or not because the breakpoint for T4-9g is across the centromere from *Ms*-7255* on the short arm of chromosome 4. There are 2 other interchanges that show significance. Although the significant values could indicate factors that impact expression of *Ms*-7255* are on these chromosomes, the barely significant values more likely represent problems in sterility classification. Much of the background of this mutant is W23. This inbred has the tendency for fertile plants to appear sterile in certain environments. Unfortunately, it was not possible to use the same source of *Ms*-7255* for each cross with the waxy translocation series. Some of the sources were better than others for stable expression of the male-sterile phenotype. We have repeated some of these crosses. Results will be available in 1988.

We do not know whether *Ms41* and *Ms*-7255* represent the same locus or not. Because *Ms41* can shed pollen in some environments, we may be able to make testcrosses with *Ms*-7255*. If this fails, we will rely either on RFLP's or conventional mapping to determine allelism of these 2 dominant male steriles. It is of interest to note that both of these dominant male steriles apparently resulted from EMS mutagenesis. In one case the EMS treatment was on kernels, and in the other, it was on pollen.

M.C. Albertsen and L.M. Sellner

Use of pollen mutagenesis to alter flowering initiation of tropical lines

One of the factors that has hindered introgression of tropical germplasm from its area of adaptation into the central corn belt has been sensitivity to the long-day conditions characteristic of the corn belt growing season. Most tropical germplasm will not flower until late September or October, if at all, under long-day conditions. Genes that condition this photoperiod sensitivity have been reported by various researchers to be recessive. There may be, however, many other factors involved in flowering that are dominant. If this hypothesis is correct, then one method that may be useful to alter flowering initiation is mutagenesis. In 1985, we began a project to move one of the tropical populations into the corn belt without first

introgressing corn belt germplasm to facilitate flowering. We based this project on pollen mutagenesis. Our approach was to mutagenize a photoperiod-sensitive population grown in a short-day environment, self the resulting M1 plants in a similar short-day environment, and grow the M2's (S1's) in a long-day environment to select for those plants that could flower and set seed.

Mutagenesis was conducted according to Neuffer's standard pollen mutagenesis protocol (M.G. Neuffer, Maize for Biological Research, p. 61, 1982). In addition to the standard 0.063% EMS in paraffin oil (Treatment 1), we also used 0.10% EMS (Treatment 2). Pure paraffin oil was used as the control treatment. Pollen from a tropical population was bulked, treated with EMS in paraffin oil, and used to pollinate 226 plants at Homestead, Florida. One hundred and six of these were pollinated by Treatment 1, while 120 ears were pollinated by Treatment 2. In addition, 100 ears were pollinated with pollen that had been exposed to paraffin oil only. At Homestead, 74 ears from Treatment 1 were harvested with M1 seed set ranging from 3-121 kernels per ear. Sixty-one ears from Treatment 2 were harvested with seed set ranging from 3-107 kernels per ear. The control treatment yielded 77 well-filled ears.

During summer 1985, M1 seed of the three treatments was planted at Homestead. One hundred fifty M2 ears were obtained for Treatment 1, 197 for Treatment 2, and 94 for the control. M2 progenies were grown at Johnston, Iowa. Self-pollinations were made on those plants that silked and shed pollen during the long-day conditions at Johnston during summer 1986. Following is the flowering distribution of the M2 families grown at Johnston in Growing Degree Units (GDU) = (degree F max + degree F min) divided by 2; 86F is upper max and 50F is lowest min.).

Treatment	1	2	Control
Less than 1599 GDU	3	1	0
1600 - 1699	7	4	1
1700 - 1799	12	23	2
1800 - 1899	44	35	17
more than 1900 GDU	10	23	8

Treatment 1 resulted in 66 families that flowered between 1550 and 1900 GDU's. Treatment 2 resulted in 63 families that flowered between 1550 and 1900 GDU's. The control treatment had 20 families that flowered less than 1900 GDU's. The earliest control family that flowered was 130 GDU's later than the earliest family from either Treatment 1 or Treatment 2. M3 seed was harvested from these plants and planted at Johnston in summer 1987. One hundred seven M3 (S2) families were selected based on agronomic appearance and early flowering. These are being topcrossed with 2 elite proprietary inbreds this winter at Homestead. Progenies will be yield tested during summer 1988 at 3 Pioneer research stations. A total of 3 locations for each station will be grown. Each location will have 2 replications of each topcross combination, resulting in 18 reps of data for each topcross/S2 combination. The best performing S2's will be advanced either as inbreds per se or will be used as a source of germplasm for introgression into elite inbred lines.

M.C. Albertsen and W. Salhuana

Chromosome arm location for *ms23*

I grew out progenies of AB-interchanges that had been crossed with the 3 unmapped genetic male steriles described by West and Albertsen (MNL 59:87) to determine chromosome arm location of each male-sterile locus. These progenies were from crossing either homozygous or heterozygous AB-interchanges as male onto known heterozygotes of each of the 3 genetic male steriles. I had decided to use known heterozygotes as female parents rather than homozygous recessives because of the number of crosses that had to be made with the AB-interchanges. Known heterozygotes for each of the male steriles were obtained by selecting male-sterile plants from the stocks originally used to describe *ms22, ms23,* and *ms24* and intercrossing with normal inbred B73. Given equal transmission of the *Ms* and *ms* alleles through the female, the critical mapping cross will result in half the hypoploids being fertile and half being sterile. Not all crosses with every AB-interchange were possible, and no male-sterile hypoploids were observed from crosses with *ms22* and *ms24*. With *ms23*, however, crosses with AB-interchanges involving the long arm of chromosome 3 yielded progenies in which the hypoploids segregated 1:1 for male sterility. One was TB-3La; the other was compound interchange TB-3La-2S6270. Data for these interchanges are shown below.

INTERCHGE	TOT # HYPOPLOIDS	# ms HYPOPLOIDS	TOT # PLTS
3La	13	6	120
3La-2S(6270)	42	23	113

The breakpoint for TB-3La is at 0.1 of the length of the long arm of chromosome 3. The other AB-interchange involving 3L is a compound interchange. These two interchanges provide us an approximation of the location of the *ms23* locus on 3L. The distal breakpoint for TB-3La-2S6270 is at 0.6, and the proximal breakpoint is at 0.1. Therefore, the *ms23* locus must be located within this chromosome segment. Crosses will be made this summer to specifically locate the *ms23* locus. We will continue efforts to locate *ms22* and *ms24*.

M.C. Albertsen

Identification of quantitative-trait loci for plant height and ear height using RFLPs

One of the major problems facing plant breeders is the complex genetic nature of most important agronomic traits. Examples of these quantitative traits include such whole-plant characters as stalk quality, disease resistance and grain yield. Maize breeders have been very successful at producing better inbreds using conventional genetic methods. Unfortunately, as more and more favorable alleles have been accumulated in their working germplasm, the effort required to carry all of them into the next inbred generation has increased. This is due to the fact that each new favorable allele often contributes only a few percent to the final whole-plant characteristic and therefore many plants must be screened and their phenotypes averaged to get reliable estimates of each genotype's worth. Recently developed methods using molecular markers offer a new approach to studying these complex traits. Instead of determining only the phenotype of the plant, it is now possible to determine the genotype as well. This means

that for the first time, maize breeders have a tool that should allow them to follow the individual genetic components of complex traits. Earlier studies (for example see Edwards et al., Genetics 116:113, 1987) have shown the utility of co-dominant molecular markers (isozymes) in identifying quantitative trait loci (QTLs) and following their transmission in sexual crosses. These studies indicated the power of the technique but were limited by the relatively small number of markers available. RFLPs, because of the almost unlimited number available in any given cross, have the potential to provide the breeder with unprecedented and detailed knowledge about the maize germplasm pool. As an initial step in our study on the practicality of using RFLPs in maize breeding, we have identified and mapped QTLs for plant height and ear height.

Seed of the F2 of B73/Mo17 was planted in 1986. Each plant was self-pollinated to produce F3 ears. A winter nursery was used to produce seed for field testing. Twenty-four kernels from each of 112 F3 ears were planted in an isolated crossing block interplanted with rows of Pioneer inbred V78. This resulted in 112 F3 topcrosses to inbred V78. In addition, a separate 24 kernels from each F3 ear were planted, 10 individuals from each row selfed and the seed pooled. This produced the F4 bulks which were tested per se.

The F3 topcrosses were evaluated in field performance tests in 1987. Each of the F3 topcrosses comprised an entry in a randomized complete block design. The experiment was grown with 2 replications per location at 4 locations in central Iowa with each replication of an entry planted in a 2 row plot. The F4 bulks were tested identically except there were 3 test locations instead of 4; 2 locations in central Iowa and 1 in central Indiana.

Data were collected on the progenies rather than the original, individual F2 plants because the heritability of these complex traits is very low when measured on a single plant basis. Each trait is probably governed by more than one gene and expression is affected by environmental conditions. Thus, by testing F3 topcrosses and F4 bulks in replicated trials, a more accurate measure of phenotype was obtained. These measures of phenotype of F3 and F4 progeny were considered accurate estimates of the phenotype of each of the F2 plants from which they were derived.

Individual probes were hybridized to DNAs prepared from each of the F2 plants. Each plant was scored for its allelic composition at the locus defined by the probe. A genetic linkage map was constructed from these data by establishing linkage between pairs of RFLP marker loci by estimating the recombination values using the maximum likelihood method. Genetic linkage groups were assigned to chromosome arms by 1) using as probes DNA sequences whose location was known and/or 2) using as markers isozyme loci whose location was known. Linkage groups whose chromosomal location is unknown are indicated as Xn. Sixty-six markers were mapped to 10 linkage groups that cover approximately 50% of the genome.

Plant height and ear height evaluated as topcrosses (TC) and F4-bulks (PS) showed significant variability among genotypes and no significant genotype by location interactions (Table 1). Both traits are highly heritable among the progeny from this cross: TC heritability = 0.68 or 0.63, PS heritability = 0.86 or 0.78 for plant height or ear height respectively.

Table 1. Analysis of variance of plant height and ear height evaluated as topcrosses and F4-bulks in 1987.

	Plant Height (inches)				Ear Height (inches)			
	TC[1]		PS[2]		TC		PS	
Source	df	SS	df	SS	df	SS	df	SS
Total	707	28105	645	99998	707	17334	645	37343
Location	2	11604	2	50593	2	2327	2	3026
Rep (Loc)	3	303	3	415	3	83	3	79
Genotype	117	6168*[3]	110	28999*	117	5070	110*	16862*
Genotype by Location	234	4001	210	7692	234	3765	210	7172
Error	351	6028	320	12299	351	6090	320	10204

1) topcross

2) F4 bulks tested per se

3) asterisk denotes significance at the 0.001 level

Averaged values (across reps) of plant height and ear height were used in a combined (across locations) single factor analysis of variance, which was calculated for each pairwise combination of plant height or ear height with marker locus. An F-test was used to determine if a significant (PR>F 0.05) amount of variability in plant height or ear height could be explained by the genotypic expression of each marker locus. Significant additive and dominance effects (PR>F 0.05) were estimated for QTLs that were associated with marker loci associated with plant height and ear height (Table 2).

Table 2. Estimates of additive and dominance effects for probes that explain significant phenotypic variation in plant height and ear height.

		PLANT HEIGHT (inches)					EAR HEIGHT (inches)					
		TOPCROSS			PER SE			TOPCROSS			PER SE	
	PROBE[1]	%V[2]	ADD[3]	DOM[4]	%V	ADD	DOM	%V	ADD	DOM	%V	ADD	DOM
CHROMOSOME 1													
58[5]	BNL12.06	1*[6]	0.3		3*		-1.3	2*	0.3		7*		-1.1
6	AMP1												
5	PIO20668	2*	0.9										
11	PIO20661	1*	0.8										
14	PIO2044				2*		-0.9				4*		0.9
7	PIO20575	2*		0.4	1*		0.8	3*		0.4	3*		0.6
4	PIO20644				1*		0.7				2*		0.6
4	PIO20674				1*						3*		0.6
2	PIO20682												
2	BNL5.59												
	PIO20654												
CHROMOSOME 2													
14	UMC034				4*	-2.9	-0.9	4*	-0.9	0.3	5*	-1.9	
12	PIO1012				3*	-2.6					5*	-2.0	
12	PIO205				1*	-2.1		2*	-0.7	0.3	3*	-1.8	
	PIO2017												
CHROMOSOME 3													
2	E8												
25	PIO206H	6*	-1.6		8*	-4.2		8*	-1.5	0.3	10*	-2.7	
11	PIO20576	5*	-1.6		3*	-2.4		7*	-1.4		4*	-1.6	
14	PIO20511	3*	-1.7		2*			4*	-1.3		4*		-0.6
40	BNL5.37	5*	-1.8		4*	-3.5		10*	-1.7	0.4	6*	-2.8	
13	PIO1080	1*	-1.0										
9	UMC1A10	2*	-1.0		2*	-2.0					5*	-1.8	
1	PIO20521	5*	-1.5		2*	-2.0		4*	-1.0		4*	-1.7	
13	PIO1533	3*	-1.3		1*	-1.7		4*	-0.9		3*	-1.5	
	BNL6.16	3*	-1.2		2*	-1.6	-0.8	3*	-0.7	-0.3	4*	-1.5	-0.5
CHROMOSOME 6													
6	PIO20599												
3	PIO20595												
14	MDH2												
22	PIO1014												
	PIO1016												
CHROMOSOME 9													
16	PIO105				1*		-0.7						
36	SHRUNKEN				1*	1.4							
3	WAXY	1*	0.7		5*	3.6		3*	0.8		10*	2.9	
8	PIO2052				3*	2.5					6*	2.1	
3	CSS				1*	1.8					2*	1.3	
6	PIO20554				3*	2.9					7*	2.4	
50	BNL8.17				1*	1.6					2*	1.3	
5	BNL14.28												
	PIO20562												
CHROMOSOME 10													
12	PIO1033												
4	GLU1	5*	1.7		2*	2.2		4*	0.8	0.4	4*	1.2	0.6
10	PIO20646	5*	1.8		2*	2.6		6*	1.1	0.4	3*	1.4	
	PIO65	3*	1.1					3*	0.8				
CHROMOSOME X1													
4	PIO20558	3*	-1.2		1*	-1.6		5*	-1.2		2*	-1.1	
5	PIO20509	5*	-1.7		2*	-2.4		7*	-1.5		5*	-1.8	
3	PIO20508B	4*	-1.5		2*	-2.5		9*	-1.6		5*	-2.0	
	PIO20508E	4*	-1.5		2*	-2.1		8*	-1.7		4*	-1.8	
CHROMOSOME X2													
4	PIO20746												
3	PIO20684												
3	PIO20708												
14	PIO1537												
40	PIO20569	1*	-0.7					3*	-1.0				
	PIO20581												

CHROMOSOME X3											
15	PIO20589										
9	PIO1518			1*		0.8					
4	PIO612						2*				
7	PIO1524										
1	PIO20622										
55	UMC043										
11	PIO20531										
14	PIO2026H	1*	-0.4				2*	0.7	-0.3		
39	PIO20566										
4	PIO20527										
14	PIO20528	1*	0.7								
	PIO67										
CHROMOSOME X4											
18	PIO20690	2*	-1.3								
4	PIO20537										
3	PIO20603										
16	PIO20689						2*	0.7			
	PIO20640			2*	2.1		2*	0.8	5*	2.0	

1) probes developed at Brookhaven National Laboratory (BNL, Burr et al., Genetics, in press), Pioneer Hi-Bred Int., Inc. (PIO) and University of Missouri-Columbia (UMC, Hoisington, pers. comm.)

2) per cent of phenotypic variation explained by RFLP loci

3) estimated additive effects (inches)

4) estimated dominance effects (inches)

5) separation of probes in cM; the Kosambi mapping function (Owen (1950) Advances in Genetics 3:117-157) was used to convert percent recombination into map distances

6) asterisk denotes significance at the 0.05 level

Thirty-one and 29 marker loci explained a significant amount of the variability in expression of plant height and ear height in F4 bulks (PS) while these traits were associated with 25 and 23 marker loci, respectively, in F3 topcrosses (TC).

Based on linkage information and QTL expression of additive and dominance effects, we have identified 7 to 10 QTLs for both traits when evaluated as either TC's or PS. In general, the QTLs we have identified affect both plant height and ear height, although there are exceptions. For example, the QTL on chromosome 2 affects TC expression of ear height, but does not affect the TC expression of plant height. Most QTLs identified for topcrosses were the same as those identified for F4-bulks. The most obvious exception was the QTL near PIO20554 on chromosome 9 that affected expression of both traits in PS tests, but neither trait when evaluated as topcrosses.

Our future work will first concentrate on filling in gaps in our RFLP linkage map. We also intend to expand our study of QTLs to include many of the agronomic traits that are of interest to maize breeders. Finally, we plan to evaluate the feasibility of using RFLP/QTL linkages to improve our ability to select for superior genotypes.

David Grant, Debi Blair, Wendy Behrendsen, Ruth Meier, Bill Beavis, Sue Bowen, Robin Tenborg, Joanie Martich, Rob Fincher, Stephen Smith, Howie Smith and Joe Keaschall

LLAVALLOL, ARGENTINA
Instituto Fitotécnico de Santa Catalina, Conicet

Path analysis on dry matter production and its components in flint type maize

Data from research previously reported (MNL 58:116-118, MNL 60:77), which include 1985 results, were analyzed using the path coefficient technique with the aim of investigating the relationships of dry matter production and its components. F1 hybrids of crosses between flint type inbreds differing in photosynthetic activity were used. According to their influence on dry matter (DM) production the traits classified as first order factors were straw DM and kernel DM; second order factors were cob DM, ear length, leaf area index (LAI) and plant height (related to straw DM) and ear length, ear diameter, ear

Table 1. Direct (D) and indirect effects of first order factors on yield.

Via:	ear width	ear length	100 kernel weight	cob weight	ear/ plant	Total r_{iA}
Effect due to:						
ear width	(D)0.236**	- 0.003	0.047	0.208	- 0.171	0.317**
ear length	- 0.008	(D)0.083ns	0.009	0.185	0.042	0.311**
100 kernel weight	0.165	0.012	(D)0.067ns	0.128	- 0.169	0.203*
cob weight	0.097	0.030	0.017	(D)0.508**	0.219	0.871**
ear/plant	- 0.091	0.008	- 0.025	0.249	(D)0.447**	0.588**

P^2_{yA} = 0.181 (squared residual path coefficient)

*,**: significant at the 0.05 and 0.01 level

r_{iA}: phenotypic correlation coefficient

Table 2. Direct (D) and indirect effects of first order factors on straw.

Via:	cob weight	ear length	LAI	plant height	Total r_{iB}
Effect due to:					
cob dry weight	(D)0.346**	0.095	0.065	0.007	0.513**
ear length	0.106	(D)0.308**	0.128	0.049	0.591**
LAI	0.067	0.116	(D)0.338**	0.064	0.585**
plant height	0.020	0.121	0.175	(D)0.123ns	0.439**

P^2_{xB} = 0.389

*, **: significant at the 0.05 and 0.01 level

r_{iB}: phenotypic correlation coefficient.

Table 3. Yield and straw effects on total dry matter.

	Via kernel DM	Via straw/plant	Total r_{iC}
Effect due to:			
Kernel DM	0.605**	0.248	0.853**
Straw DM	0.260	0.577**	0.837**

P^2_{xC} = 0.001

** significant at the 0.01 level

r_{iC}: phenotypic correlation coefficient.

per plant, cob weight and 100 kernel weight (related to yield). Direct and indirect effect estimates are shown in Tables 1, 2 and 3. Both first order factors show similar direct effects on total dry matter production. Second order factors explain 61% of variance in straw and 82% in grain yield. Among them, ear length, cob DM and LAI are the main traits influencing straw through direct effects; in a similar way, cob weight, ear/plant number and ear diameter are shown to be the main traits influencing grain yield. Results are in general agreement with those obtained in 1983 and 1984. Differences are attributable to a higher ear/plant number in 1985 which caused diminution of ear size traits.

Jaime R. Jatimliansky, María I. Urrutia and Miguel J. Arturi

LLAVALLOL, ARGENTINA
Instituto Fitotécnico de Santa Catalina,
Universidad Nacional de La Plata

Colchicine treatment induces quadrivalent formation, suggesting ancient polyploidy

Zea mays ssp. *mays* (2n = 20) is commonly considered as diploid with x = 10. Recently, Molina and Naranjo (Theor. Appl. Genet., 73:542, 1987) and Naranjo and Molina (MNL 61:62, 1987) presented new cytological evidence supporting x = 5 as the basic chromosome number of the genus.

Treatments of meiocytes prior to the onset of meiosis with dilute solutions of colchicine can induce intergenomic pairing, revealing cryptic genome homologies (Jackson & Murray, Theor. Appl. Genet. 64:219, 1983). This technique disrupts a bivalent promoting mechanism in *Alopecurus* and *Triticum*, and revealed the polyploid origin in "diploid" species of *Helianthus* (Jackson & Murray, ibid.; Murray et al., Kew Chrom. Confer. II:165, 1983).

In 10 plants of maize, colchicine solution (0.5×10^{-4}M) was applied prior to the onset of meiosis by injection of the solution in the node. This material showed one quadrivalent in about 8% of the cells in every plant examined.

In another experiment the stems of 5 plants were cut and placed in 0.5×10^{-4}M colchicine solution for 12h followed by 24h in water before fixation in 3:1 alcohol:acetic acid. The quadrivalent frequency was higher than in the injected material. A detailed study of 155 cells from one plant showed the following results:

Configurations			No. of Cells
II	IV	I	
10	—	—	42
8	1	—	39
6	2	—	24
4	3	—	17
2	4	—	15
–	5	—	17
9	–	2	1
			155

In both treatments the control material was fixed without pretreatment and showed only bivalent formation.

These results constitute new evidence to support the Molina and Naranjo hypothesis that the *Zea* species with 2n = 20 are of polyploid origin. Although the meiotic configurations that maize presents normally are those expected in a typical allotetraploid (AABB), the induced quadrivalent formation by colchicine suggests a higher affinity between the postulated genomes and the existence of a genetic mechanism similar to that of wheat, which promotes bivalent formation. Finally it underlines the agronomic potential of this technique, as pointed out by Jackson and Murray (ibid.), since the induction of intergenomal recombination can give rise to new and extraordinary recombinant genotypes.

Lidia Poggio, María del Carmen Molina
and Carlos A. Naranjo

A double spindle in the genus *Zea*

Molina and Naranjo (Theor. Appl. Genet. 73:542, 1987) and Naranjo and Molina (MNL 61:62, 1987) presented cytological evidences for a basic chromosome number x = 5 in *Zea*:

1. In 2n = 30 hybrids the most frequent meiotic configuration was 5 III + 5 II + 5 I, and in *Z. perennis* and hybrids with 2n = 40 it was 5 IV + 10 II.
2. In all species and hybrids with 2n = 20, secondary association was observed, and a maximum of 5 groups of 2 bivalents each was frequent at diplotene-diakinesis.
3. In 2n = 30 hybrids, there is a clear tendency for A, B and C genome separation through the respectively grouped trivalents, bivalents and univalents.

As well as the evidences mentioned it has recently been observed that a high frequency of cells showed formation of 2 spindles with 5 bivalents each in metaphase-anaphase I, in all taxa with 2n = 20. In several cells an asynchronous initiation of anaphase was observed. It is interesting to point out that when cut stems were placed in colchicine solution (Poggio et al., this MNL), the 2 spindles were clearly seen in cells without multivalent formation.

More studies are needed to clarify the origin of the double spindle and the position of different genomes in it. The double spindle is, probably, a relictual condition of diploidy.

María del Carmen Molina, Carlos A. Naranjo
and Lidia Poggio

Nuclear DNA content in *Zea*

Microdensitometry measurements revealed significant differences in 4C DNA content between and within taxa in the genus *Zea*. Rayburn et al. (Am. J. Bot. 72:1610, 1985) and Laurie and Bennett (Heredity 55:307, 1985) demonstrated that there are variations in DNA content among varieties of *Zea mays* ssp. *mays* and ssp. *mexicana*. Moreover, Rayburn et al. (ibid.) have shown that there is a significant positive correlation between C-band number, % of heterochromatin, and genome size (DNA content). They also show that DNA content decreases with increasing latitude. Their results indicate that intraspecific variation in DNA content in maize is largely caused by differences in the amount of heterochromatin and is significantly correlated with geographical distribution, suggesting that these characteristics have adaptative significance and are, therefore, of potential agricultural interest (Laurie and Bennett; Bennett, New Phytol. 106:171, 1987).

In the present work we have studied the 2C nuclear DNA content in 4 taxa of the genus *Zea* and in 3 interspecific hybrids by microdensitometry of Feulgen-stained root-tip telophase cells. The technique was done according to Poggio and Hunziker (J. Hered. 77:43, 1986). The optimum time of hydrolysis occurred after 30 min in 5N HCl at 20C. *Allium cepa* var Ailsa Craig was used as standard. The results are summarized in Table 1.

Rayburn et al., and Laurie and Bennett, have demonstrated that there is a highly significant variation in 2C DNA content in maize, ranging from 4.92pg in the Seneca 60 hybrid to 6.74pg for a Zapalote Chico accession. The 9063 line analyzed in this work has 2C DNA content values similar to those of the races of Mexican maize regarded as "primitive"; namely Chapalote (5.82pg) and Nan-Tel (5.96pg), reported by Laurie and Bennett. The 9063 line has a high value when compared with Seneca 60 (2C = 4.92pg) or Knobless Tama Flint (2C = 5.14pg) values published by Laurie and Bennett. The same authors found significant differences among races of annual teosintes (ssp. *mexicana*). The line of ssp. *mexicana* analyzed in this work shows a very high 2C DNA content (2C =

Table 1. 2C nuclear DNA content in the genus *Zea* and artificial hybrids.

TAXON	2n	Ploidy level*	2C ADN (pg) $\bar{X} \pm$ ES	DNA (pg) per basic genome*
Section Zea				
Z. mays ssp. mays (9063)	20	4x	5.966 ± 0.060	1.491
Z. mays ssp. mexicana (4024)	20	4x	6.783 ± 0.053	1.684
Z. mays ssp. mays (9063) X Z. mays ssp. mexicana (4024)	20	4x	6.543 ± 0.076	1.636
Section Luxuriantes				
Z. diploperennis	20	4x	6.503 ± 0.096	1.626
Z. perennis	40	8x	11.561 ± 0.166	1.445
Z. diploperennis X Z. perennis	30	6x	8.287 ± 0.121	1.381
Z. diploperennis X Z. perennis	40	8x	11.819 ± 0.249	1.478

* The basic chromosome number of the genus Zea is x=5 according to Molina and Naranjo (Theor. Appl. Genet. 73:542-550, 1987).

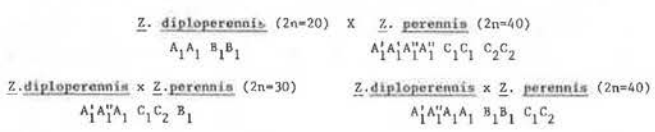
6.73pg) considering that the highest value reported by Laurie and Bennett was 2 C = 6.44pg for the line K65-1. The artificial hybrid between both lines studied here shows an intermediate value between them and differs significantly from both progenitors.

Variation in the amount of heterochromatin (number of C^+ bands) would appear to be an important cause of differences in DNA content, and significant differences in number of C^+ bands in metaphase and prophase were found between *Z. mays* ssp. *mays* (9063) and ssp. *mexicana* (4024). The 9063 line presents 12 C^+ bands distributed in 10 chromosomes. The 4024 line has a higher number of C^+ bands (17-18) and only 2 chromosomes are devoid of bands.

Preliminary studies in another line of ssp. *mexicana* (4025) show the presence of heterozygosity for terminal C^+ bands. This fact could account for differences in karyotypes, DNA content and morphological characters in hybrids with the same progenitors. These data indicate that the DNA content of ssp. *mays* (9063), compared with the higher DNA content of ssp. *mexicana* (4024) could be attributed to a smaller amount of heterochromatin. The hybrid presents, as is expected, an intermediate number of C^+ bands in metaphase and C^+ chromocenters in interphase (Poggio et al., in preparation).

Laurie and Bennett reported 2C = 5.28pg for *Z. diploperennis*. The line studied here shows 2C = 6.55pg (*Z. diploperennis*, Mexico Jalisco, Sierra de Mananthan Occidental, 2Km E of Las Joyas, Leg. Rafael Guzman & M.A. de Guzman - No. 1120, Nov. 1980). The same authors reported that *Z. perennis* had a DNA content close to twice that of *Z. diploperennis* (*Z. perennis*, Mexico, Jalisco, Ciudad Guzman. Leg. Dra Prywer, cultivated in "Instituto Fitotécnico de Santa Catalina" since 1962). In the present work *Z. diploperennis* shows less DNA content per basic genome than *Z. perennis* (Table 1). This difference is statistically significant. This fact is not surprising. Although in polyploids of recent origin the DNA content is directly proportional to the DNA content of diploid progenitors, there are cases in which this proportionality does not occur (Poggio and Hunziker, J. Hered. 77:43, 1986; Martinez and Ginzo, Can. J. Genet. Cytol. 27:766, 1985; Bennett, New Phytol. 106:171, 1987; Poggio et al., Darwiniana 27:25, 1986).

The hexaploid hybrid *Z. diploperennis* × *Z. perennis* has an intermediate value between the progenitors. The octoploid hybrid probably originated by the fertilization of an unreduced egg cell from *Z. diploperennis* by a normal male gamete from *Z. perennis*. The hypothetical genomic constitutions of these species and hybrids are, according to Molina and Naranjo (Theor. Appl. Genet. 73:542, 1987):



The expected result is that the octoploid hybrid will have more DNA content (about 6%) than the octoploid *Z. perennis*. In fact, the hybrid has more DNA than *Z. perennis* although the difference is statistically nonsignificant. The expected difference is small and perhaps of a magnitude difficult to detect by Feulgen cytophotometric method.

Finally, it is interesting to point out that the differences found in DNA content and C^+ band number in *Z. mays* ssp. *mays* and ssp. *mexicana* do not interfere with normal pairing since the hybrid shows 20 bivalents (II) in meiosis and normal fertility (Naranjo and Molina, MNL 61:62, 1987). The *Z. diploperennis* × *Z. perennis* hybrids (2n = 30 and 40) show the expected pairing according to the genomic constitution postulated by Molina and Naranjo.

Claudia M. Tito, Lida Poggio
and Carlos A. Naranjo

LLAVALLOL, ARGENTINA
Instituto Fitotécnico de Santa Catalina
BUENOS AIRES, ARGENTINA
Centro de Ecofisiología Vegetal

In vitro culture of immature embryos

In order to determine an adequate culture medium for obtaining the best growth of immature embryos, plants of sweet corn and Colorado Klein were grown in the greenhouse, out of the normal age for this species (for which embryos grew slowly). The embryos were excised 22 and 28 days post-pollination, at 1.2 to 2 and 2.2 to 4.0mm in length, respectively.

The culture media used contained mineral salts of N6 medium (Chu, Proc. Symp. Plant Tissue Cult., pp.43-50, Science Press, Peking, 1978), with the addition of (mgl^{-1}) glycine, 3.0; thiamine, 0.75; ascorbic acid, 20.0; nicotinic acid, 1.0; vitamin B_6, 0.2; adenine, 0.2; succinic acid, 25.0; pantothenic acid, 0.5; biotin, 0.003 and asparagine, 1500 (Haagen Smit et al., Science 101:243, 1945); agar, 0.8% and 4 different concentrations of sucrose, 2.5% 5.0% 10.0% 15.0%, resulting in 4 treatments labeled A, B, C and D respectively. The media were adjusted to pH 5.8.

The embryos were isolated and placed on the culture media with the rounded scutellar side exposed and the flat plumule radicle axis side in contact with the medium. The embryos were maintained at 28C with a 16/8h photoperiod for 60 days. Then the plants were transplanted to pots with a sterile mixture of soil-vermiculite (1:1) and grown in the greenhouse. The plants were finally transplanted to soil 108 days after sowing.

The immature embryos on all media showed similar behavior at the beginning, independently of their initial size, developing normally up to a determined length, after which all of them germinated (Table 1).

Table 1. Growth of embryos before germination.

Culture medium	Average of initial length of embryos (mm)	Average of embryos length before germination (mm)
A	1.65	2.20
	3.39	3.99
B	1.76	2.31
	3.38	3.99
C	1.75	2.17
	2.86	3.47
D	1.55	1.95
	3.24	3.82

The youngest embryos did not continue their growth on medium A. Almost all the embryos developed into plants on media B, C and D, with the greatest number developing on medium B. Concentrations of sucrose affected the survival of the youngest embryos more than the oldest ones (Table 2).

It seems likely that sucrose concentration increases osmotic value in the medium, which is necessary for

Table 2. Influence of culture medium and initial length of the embryo over the number of plants transplanted to pots.

Initial heigh of embryos (mm)	Average of transplantable plants Medium A	Medium B	Medium C	Medium D
1.2 to 2.0	33.3	93.7	66.7	78.9
2.2 to 4.0	75.0	100	70.0	88.9

Table 3. Influence of culture medium and initial length of the embryo over the number of plants that reached maturity.

Initial height of embryos (mm)	Average of plants which grew to maturity Medium A	Medium B	Medium C	Medium D
1.2 to 2.0	0	43.7	33.7	36.9
2.2 to 4.0	37.5	100	40	66.9

Table 4. Relationship between plant height and type of flowering.

Type of flowering	Height (mm)
non branching tassel	1,26
normal flowering	1,14
tassel seed	0,86
ear developing where normally there is a tassel	0,51

growth induction in proembryos of several species, probably to allow an effective flow of metabolites (Raghavan and Srivastava, 9. Embryo Culture. In Experimental Embryology of Vascular Plants, Johri, ed., p.195, 1982).

In a short period after transplantation, 46.8% of plants died. Plants from medium B showed the largest percentage of survival (100%), while none of the plants from the smallest embryos growing on medium A was successfully transplanted. On the same culture medium, the number of plants arising from the largest embryos always exceeded the number of plants arising from the smallest ones (Table 3).

Plants began to flower about 135 days after embryo sowing and 20 days later all of them had flowered; 81% of plants showing abnormalities such as an ear developing where normally there is a tassel, tassel seed, and non-branching tassel. Plants did not reach their normal height, showing differences related to the different types of flowering (Table 4). Notwithstanding growth abnormalities, cytological analysis revealed a somatic chromosome number of $2n = 20$ in every tested plant.

Dina García[1] and María del C. Molina
(1) Fellow, Com. Inv. Cient. Prov. Buenos Aires

LOMAS DE ZAMORA, ARGENTINA
Lomas de Zamora University
LLAVALLOL, ARGENTINA
Instituto Fitotécnico de Santa Catalina

Variation within teosinte: numerical analysis of agronomic traits

As part of a project whose purpose is the evaluation of wild genetic resources of maize, especially the study of possibilities offered by teosinte as a genetic diversity source, a series of agronomic traits in wild taxa of *Zea* were quantified. During the last few years, a great deal of information about teosintes has been given by several authors, including us (see MNL 59:61, 1985; MNL 60:79, 1986 and MNL 61:67, 1987). All this information is based in large part on the study of specific important traits.

All the available information has contributed to strengthening some ideas or putting into action some other ones about phylogenetic relationships among teosintes and between teosintes and maize. Nevertheless, the characterization of teosintes for non-specific features or those of apparent non-taxonomic significance, for example common agronomic traits, has not deserved the same attention. Consequently, the purpose of these investigations is to gather all the teosintes in clusters on the basis of agronomic traits, and then compare these groupings with those derived from the utilization of specific traits.

The wild taxa of the genus *Zea* (teosintes), *Z. perennis* (Zp), *Z. diploperennis* (Zd), *Z. luxurians* (Zl), *Z. mays* ssp. *parviglumis* var. *parviglumis* (Zmpp), *Z. mays* ssp. *parviglumis* var. *huehuetenangensis* (Zmph) and *Z. mays* ssp. *mexicana* (Zmmx) constitute the 6 operational taxonomic units (OTU's). These 6 OTU's are part of a complete randomized block design with 3 replicates and they were cultivated during the 86/87 growing season in our experimental field.

This experiment gave enough information to compare teosintes among themselves. The average values for each trait were used to make analysis by numerical techniques.

Twenty-two traits were measured in each one of the OTU's:

(A) *Evolutive cycle traits*: (1) days to tassel, (2) days to silking, (3) days to pollen; (B) *Prolificity traits*: (4) number of productive nodes per tiller, (5) number of female spikes in the uppermost node, (6) number of female spikes per tiller, (7) number of female spikes per plant, (8) number of fruit cases per spike; (C) *Plant traits*: (9) total number of tillers, (10) number of tillers with spikes, (11) plant height (cm), (12) number of leaves per tiller, (13) leaf width in the uppermost productive node (cm), (14) leaf width in the 10th node (cm), (15) leaf length in the uppermost productive node (cm), (16) leaf length in the 10th node (cm), (19) stalk diameter in the uppermost productive node (cm), (20) stalk diameter in the 10th node, (21) whole plant protein content at tillering stage (%), (22) whole plant protein content at stalk forming stage (%).

The average values obtained for each one of the traits were set into a basic data matrix (BDM) (see Table 1).

Table 1. Basic data matrix (BDM): agronomic traits.

	Characters										
OTU's	1	2	3	4	5	6	7	8	9	10	11
Zp	166.1	183.1	186.7	2.9	1.7	6.2	113.5	5.2	37.2	16.4	243.0
Zd	168.4	184.1	187.4	6.2	1.9	11.7	140.2	5.5	16.1	13.0	248.1
Zl	185.7	188.3	200.4	10.6	3.7	68.9	941.2	6.4	22.6	19.5	300.5
Zmpp	181.5	186.7	193.8	8.3	2.4	35.6	710.7	5.9	24.7	22.6	280.0
Zmph	199.2	197.4	203.4	7.9	1.6	19.6	134.8	5.8	12.1	10.0	294.0
Zmmx	140.9	158.7	152.5	8.6	6.8	101.6	281.4	8.4	3.1	3.1	305.0

	Characters										
OTU's	12	13	14	15	16	17	18	19	20	21	22
Zp	18.3	2.9	3.4	21.6	54.1	9.2	11.3	0.3	0.6	19.8	13.3
Zd	19.6	3.1	3.5	21.3	55.6	11.8	13.7	0.4	0.8	24.1	13.8
Zl	21.1	3.4	4.7	19.5	116.9	7.2	20.9	0.4	1.3	17.0	12.5
Zmpp	20.4	2.8	3.8	17.0	92.9	5.7	20.5	0.4	1.0	15.2	10.3
Zmph	22.2	3.5	4.5	21.8	110.7	7.0	20.9	0.4	1.3	20.7	17.6
Zmmx	18.0	5.7	7.3	44.1	85.7	13.4	22.4	0.9	2.0	20.0	NC

With these results, we built phenograms derived from correlation index matrix and mean taxonomic distance matrix (MTD), OTU × OTU, using the UPGMA method (see Figure 1).

Finally, the results obtained through these measurements and evaluation procedures point out that when the different taxa are clustered on the basis of these agronomic importance traits, they are partially congruent with the clusters derived from specific characters (see MNL 60:81, 1986). This fact is suggesting that possibly these chosen traits have taxonomic significance. It must be specially emphasized that one of the teosintes of the Sect. *Luxuriantes* (*Z. luxurians*) is more similar to the taxa belonging to the

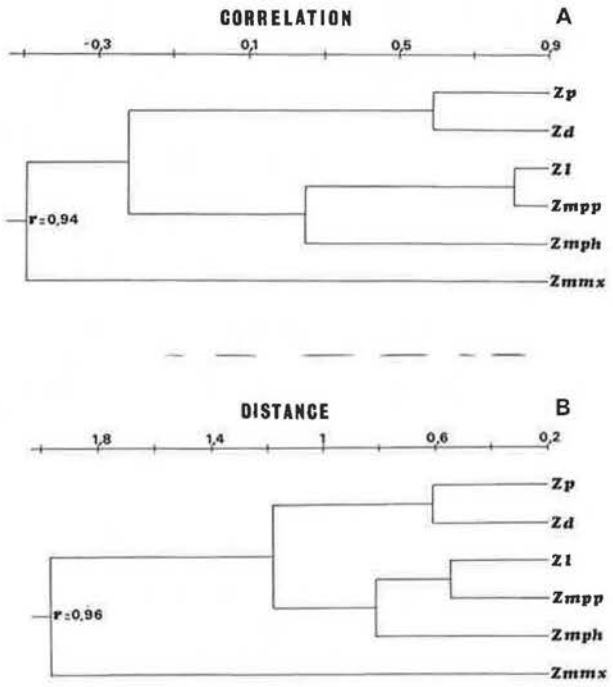


Figure 1. Phenograms of 6 OTU's resulting from: (A) UPGMA cluster analysis of the OTU × OTU correlation matrix; (B) UPGMA cluster analysis of the OTU × OTU distance matrix. r: cophenetic correlation coefficient.

other taxonomic section (Sect. *Zea*) than to the ones of its own when we analyze the studied traits. This fact agrees with the general impression that one generally has about Guatemala teosinte when it is compared with Zmpp and Zmph, and without considering those specific traits that make the difference among themselves.

One wonders if *Z. luxurians* could be the link between Sect. *Luxuriantes* and Sect. *Zea*, as those teosintes belonging to the first are the most primitive, but the ones included in Sect. *Zea* are the most evolved.

We finally think that as more information is collected, new questions would be brought out; then it is necessary to go on researching until it is possible to have a better understanding about phylogenetic relationships within teosinte.

Mónica B. Aulicino[1] and Jorge L. Magoja
[1]Fellow, Cons. Nac. Inv. Cient. Técni. (CONICET)

Diploperennial teosinte-maize hybrids: inheritance of evolutive cycle

At the latitude in which most cultivated maize is concentrated in Argentina (35° southern latitude), generally all the teosinte species express a strong photoperiod response, and they flower during autumn, which makes seed collection difficult. Consequently they show a long cycle. Diploperennial teosinte is a species which under these environmental conditions has a cycle 3 times longer than cultivated maize.

Evolutive cycle traits were measured in 4 different populations to obtain the necessary basic information for future projects in which diploperennial teosinte germplasm will be used for maize genetic improvement. The 4 populations studied are: diploperennial teosinte (P1); a sweet variety of maize Ever-Green (P2) and their F1 and F2 progenies derived from crossing teosinte with maize.

On the basis of individual plants, cycle traits measured are: 1) days to tassel (T), 2) days to silking (S), and 3) days to pollen (P). The results obtained are summarized in Table 1. The average values of the F1 are smaller than those of the mid-parent value (MP), being nearer to maize values. The F2 average values are higher than those of the F1 and the frequency distribution for them is wide enough to reach their parent extreme values (see Table 1 and Table 2).

Table 1. Number of plants (N), means and ranges for evolutive cycle traits in diploperennial teosinte (Zd), Ever-green maize (Eg) and F1 and F2 populations.

	N	Days to tassel Mean ± SD	Range	Days to silking Mean ± SD	Range	Days to pollen Mean ± SD	Range
P_1 (Zd)	60	167.1 ± 3.9	159-175	182.8 ± 2.3	178-187	185.4 ± 3.2	180-194
P_2 (Eg)	60	54.8 ± 2.4	50-60	62.7 ± 3.4	55-71	57.4 ± 2.1	53-63
F_1	100	79.5 ± 4.0	67-83	101.6 ± 5.3	93-113	94.6 ± 5.5	84-108
F_2	650	95.7 ± 18.4	66-153	116.7 ± 22.5	66-157	107.1 ± 19.6	75-157

Table 2. Differences between means for evolutive cycle traits of diploperennial teosinte(Zd), Ever-green maize (Eg), F1 and F2 populations and mid-parent values (MP).

	T	S	P
P_1 (Zd)	167.1^{a}(*)	182.8^{a}	185.4^{a}
P_2 (Eg)	54.8^{b}	62.7^{b}	57.4^{b}
F_1	79.5^{c}	101.6^{c}	94.9^{c}
F_2	95.7^{d}	116.7^{d}	107.1^{d}
MP	111.0^{e}	$122,8^{e}$	121.4^{e}

(*) Individual means within a column followed by different letters are significantly different at 1% level

These data previously shown in this paper also point out that a very low environmental influence exists over the expression of measured traits, by which we estimate that they have a high heritability. The different traits measured are closely entailed among themselves and their hereditary behaviour is similar.

Last, an interesting phenomenon to note is the degree of simultaneity in sexual maturation. Diploperennial teosinte, as the other teosintes, is a protogynous plant, but maize is generally protandrous. Opposite to other hybrids between maize and perennial teosinte, and maize-diploperennial teosinte hybrids (see MNL 58:130, 1984), the F1 progeny is protandrous as are the F2 individuals. We cannot generalize conclusions about this fact, but for this particular case protandrous trait of maize is a dominant trait.

From the results we have obtained it can be deduced that selection of short evolutive cycle individuals would be easy (previous investigations we have done demonstrate it). This fact offers a considerable advantage as you can obtain individuals derived from these interspecific crosses which have such a cycle duration that they may be grown in the field and mature under normal environmental conditions.

Víctor R. Corcuera[1] and Jorge L. Magoja
[1]Fellow, Com. Inv. Cient. (CIC), Prov. Buenos Aires

Diploperennial teosinte-maize hybrids: inheritance of prolificity

Generally, hybrids between maize and its nearest wild relatives are very heterotic. On the basis of our investiga-

Table 1. Number of plants (N), means and ranges for prolificity traits in diploperennial teosinte (Zd), Ever-green maize (Eg) and F1 and F2 populations.

	N	Number of productive nodes Mean ± SD	Range	Ears in the uppermost node Mean ± SD	Range	Ears per tiller Mean ± SD	Range
P_1(Zd)	60	6.2 ± 1.7	5-8	1.9 ± 0.6	1-3	11.8 ± 3.6	7-18
P_2(Eg)	60	1.1 ± 0.4	1-3	1.0 ± 0.0	-	1.1 ± 0.4	1-2
F_1	100	5.1 ± 1.0	3-7	6.2 ± 3.1	2-14	29.4 ±14.0	8-64
F_2	350	6.1 ± 2.5	2-14	5.3 ± 2.5	2-15	32.7 ±16.2	5-97

Table 2. Differences between means for prolificity traits of diploperennial teosinte (Zd), Ever-green maize (Eg), F1 and F2 populations and mid-parent values (MP).

	PN	EUN	ET
P_1 (Zd)	6.2^{a}(*)	1.9^{a}	11.8^{a}
P_2 (Eg)	1.1^{b}	1.0^{b}	1.1^{b}
F_1	5.1^{c}	6.2^{c}	29.4^{c}
F_2	6.1^{ac}	5.3^{d}	32.7^{c}
MP	3.7^{d}	1.5^{e}	6.5^{d}

(*) Individual means within a column followed by different letters are singificantly different at 1% level.

Table 3. Percent of heterosis for prolificity traits.

Character	% heterosis (*)
Number of productive nodes	39.7
Ears in the upper most node	318.9
Ears per tiller	357.2

(*) % heterosis = (F_1-MP/MP)x 100

tions on hybrids between maize and perennial teosinte (*Zea perennis*) (see MNL 56:104, 1982 and MNL 57:66, 1983) and between maize and diploperennial teosinte (*Z. diploperennis*) (see MNL 61:63 and 61:66, 1987) it is clearly evident that prolificity is the most heterotic trait.

Normally, maize is a non-prolific plant, which means that it is not capable of producing several ears per plant. On the other hand, the wild relatives of maize are prolific, in different intensity depending on the species considered. In spite of that, prolificity reaches its maximum expression in hybrids between maize and its wild relatives.

We have evaluated prolificity in progenies derived from crosses between diploperennial teosinte and maize, and their respective parents through 3 different traits (see Table 1): 1) Number of productive nodes per tiller (PN); 2) Number of ears in the uppermost node (EUN); 3) Number of ears per tiller (ET).

The results obtained for these kinds of hybrids are very similar to those previously found in perennial teosinte-maize hybrids (see MNL 57:66, 1983).

As can be easily seen in Table 2, these hybrids between diploperennial teosinte and maize generally not only exceed the mid-parent prolificity values, but the most prolific parent values too, in a highly significant way. In hybrids the prolificity frequency distribution is completely displaced to the right side, relative to their parental frequency distributions. The degree of heterosis expressed through these traits is really very high, especially when the number of ears per tiller (ET) is considered (see Table 3).

According to the results we have finally obtained, it can be stated that prolificity is transgressively inherited in a positive direction (heterosis). Unlike what happens in perennial teosinte-maize hybrids, in which the real prolificity probably can be disguised by sterility problems (lack of fecundation helps the development of a great number of ears), in this case diploperennial teosinte-maize hybrids are highly fertile and the prolificity they express cannot be attributed to sterility.

This particular means of inheritance for prolificity traits has a high practical value, since through the utilization of wild germplasm we could increase in a very significant form the prolificity degree in maize populations (see MNL 61:65 and 61:66, 1987). The results obtained also let us deduce that if the combination of maize germplasm with teosinte finally increases in a very significant way the prolificity degree, this trait in the different types of teosintes, depending on their expression level, perhaps can be taken as indicative of the introgression degree they assimilated from maize.

Victor R. Corcuera and Jorge L. Magoja

Diploperennial teosinte-maize hybrids: inheritance of tassel traits

Some traits of the male inflorescence (tassel) can have taxonomic importance, and have allowed clustering the different taxa of *Zea* on the basis of traits not traditionally used (Doebley and Iltis, Amer. J. Bot. 67:982, 1980). The study of interspecific hybrids between taxa belonging to the 2 different sections in which Doebley and Iltis have divided the genus *Zea* offers the possibility of obtaining information that contributes to supporting the force of the specific value of those traits.

We have previously studied (see MNL 58:122) the inheritance mechanism of 3 different tassel traits which have taxonomic value in perennial teosinte (*Zea perennis*) maize hybrids. This time we have repeated once more the study of those traits in hybrids between taxa belonging to the 2 different sections of the genus *Zea*, but replacing perennial teosinte with diploperennial teosinte (*Z. diploperennis*) as representative of the Sect. *Luxuriantes* and using maize as representative of Sect. *Zea*.

Four quantitative tassel traits were evaluated both in progenies derived from the crossing between diploperennial teosinte and maize, and their respective parents. The traits we studied are: 1) tassel branch number (TBN), 2) tassel branching axis length (TBAL), 3) tassel central spike length (TCEL) and 4) lateral tassel branch internode length (LTBIL) (see Table 1).

For the 3 traits of specific value (TCEL was not used by Doebley and Iltis) the parents differ significantly between themselves. On one side we have maize with high TBN and long TBAL and TBIL, on the other one there is diploperennial teosinte with low TBN and short TBAL and TBIL.

In tassel branch number (TBN) the results obtained let us deduce that a low number (teosinte) is dominant over a high number (maize). This phenomenon can also be seen for TBAL when traits are closely entailed. The average values of both traits for F1 and F2 progenies are significantly lower than the mid-parent value (see Table 2).

Table 1. Number of plants (N), means and ranges for tassel traits in diploperennial teosinte (Zd), Ever-green maize (Eg) and F1 and F2 populations.

	N	TBN Mean ± SD	Range	TBAL(cm) Mean ± SD	Range	TCEL(cm) Mean ± SD	Range	LTBIL(mm) Mean ± SD	Range
P_1(Zd)	60	9.3 ± 3.3	5-18	3.2 ± 0.9	1.5-5.7	16.0 ± 1.3	13.7-17.5	4.2 ± 0.4	4-5
P_2 (Eg)	60	31.8 ± 7.5	17-51	12.8 ± 2.8	8-17.7	22.7 ± 4.4	9.5-30.5	6.0 ± 1.0	4-8
F_1	100	11.6 ± 6.3	2-22	5.3 ± 2.5	1-11	16.6 ± 5.2	11-26	5.0 ± 0.8	3-6
F_2	400	10.3 ± 6.7	2-54	5.1 ± 2.5	1-29.5	20.8 ± 5.3	1-23.2	4.6 ± 0.9	3-7

Table 2. Differences between means for tassel traits of diploperennial teosinte (Zd), Ever-green maize (Eg), F1 and F2 populations and mid parent values (MP).

	TBN	TBAL (cm)	TCEL (cm)	LTBIL (mm)
P_1 (Zd)	9.3^{a}(*)	3.2^{a}	16.0^{a}	4.2^{a}
P_2 (Eg)	31.8^{b}	12.8^{b}	22.7^{b}	6.0^{b}
F_1	11.6^{a}	5.3^{c}	16.6^{a}	5.0^{c}
F_2	10.3^{a}	5.1^{c}	20.8^{c}	4.6^{d}
MP	20.6^{c}	8.0^{d}	19.4^{c}	5.1^{c}

(*) Individual means within a column followed by different letters are significantly different at 1% level.

The way in which these traits (TBN and TBAL) are inherited is different from that previously reported for perennial teosinte-maize hybrids (see MNL 58:122). By the opposite, the TBIL trait is inherited in a similar way in both kinds of hybrids: the F1 and F2 average values are intermediate when they are compared to their respective parent average values, and the F2 frequency distribution nearly reaches the parents' extreme values, by which one can deduce, as was previously remarked (see MNL 58:122), that this trait is quantitatively inherited and possibly that the action of additive genes is predominating.

The fact that TBN and TBAL are inherited in a very different way, depending on the type of teosinte used in the cross, though all these species belong to the same section of the genus *Zea*, and the great variability shown by maize for these traits, offers an infinite number of different inheritance possibilities derived from a great deal of possible genetic combinations.

These results coinciding with our previous publications (see MNL 58:122), let us suppose that from the 3 different traits studied, only one has specific value (TBIL). Nevertheless, it is necessary to analyze a greater number of hybrid combinations between representatives of both sections of the genus *Zea* to obtain strong definitive conclusions. As there is a great diversity in maize, we think that probably the better thing would be analyzing hybrids between the different teosintes to study the specific traits.

Víctor R. Corcuera and Jorge L. Magoja

Races of maize from Salta (Argentina): numerical analysis of storage protein data

Different grain samples representative of the 21 native races of maize (*Zea mays* ssp. *mays*) from Salta, Argentina, were used to study the storage proteins through different evaluations. The material was kindly supplied by Prof. Melchiore, who has also studied and classified it on the basis of traditional morphological traits in the Laboratory of Botanics, Faculty of Agronomy, Buenos Aires University.

The races studied are: Pisinga púrpura (PP), Colorado (C), Chaucha (CH), Amarillo de ocho (AO), Chiriguano (CHO), Orgullo cuarentón (OC), Criollo amarillo (CA), Amarillo ancho (AA), Capia blanco (CB), Capia tabanito (CT), Capia púrpura (CP), Capia rosado (CR), Chulpi (CHI), Socorro (S), Perla (P), Blanco ocho rayas (BOR), Ocho rayas (OR), Altiplano (A), Pinto (PO), Culli (CI) and Negro (N). These races constitute the 2 operational taxonomic units (OTU's).

Some morphological traits of these kernels were evaluated: 1) type (amylacea, amylaesaccharata, indurata, orysae), 2) pericarp (colourless, light coloured and dark coloured), 3) aleurone layer color (coloured or colourless), 4) amyloproteic parenchyma color (colourless, coloured), 5) kernel shape (rostrated, cuneiform, round or extended), 6) hard endosperm ratio, 7) kernel weight, 8) kernel length, 9) kernel width and 10) kernel thickness. The biochemical traits evaluated are: 11) protein content, 12) saline soluble protein content, 13) zein content, 14) glutelin content.

Polyacrylamide gel electrophoresis of zein (PAGE) let us obtain traits 15 to 51 using the criterion of presence-absence of bands (the electrophoretical patterns can be seen in Figure 1). Finally through SDS polyacrylamide gel electrophoresis of soluble proteins, we obtained traits 52 to 64 using the previous criterion (the electrophoretical patterns can be seen in Figure 2).

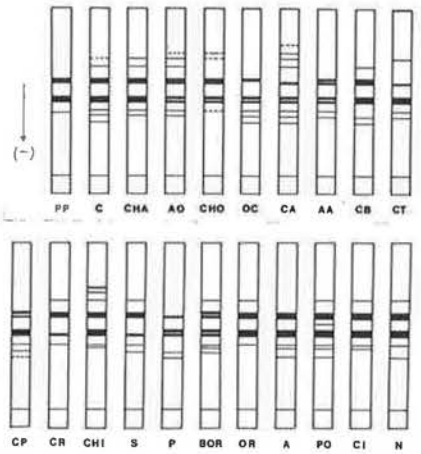


Figure 1. Polyacrylamide electrophoretic patterns of zein of races of maize from Salta.

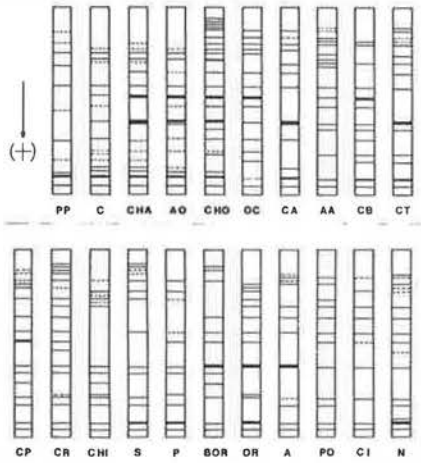


Figure 2. SDS-polyacrylamide electrophoretic patterns of soluble proteins of races of maize from Salta.

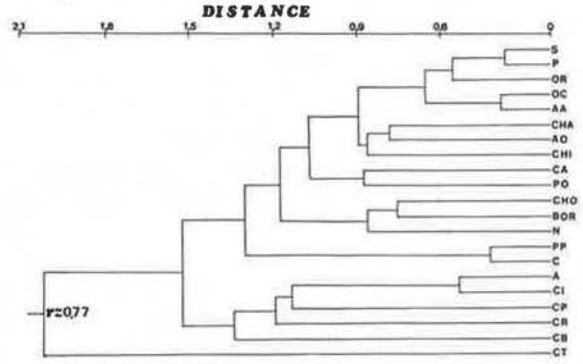


Figure 3. Phenogram of 21 OTU's resulting from UPGMA cluster analysis of the OTU × OTU distance matrix, based on characters 1 to 14. r: cophenetic correlation coefficient.

We have made a basic data matrix (BDM). Using traits 1 to 14 a mean taxonomic distance matrix (MTD) was built. Then, with this MTD we built the phenogram shown in Figure 3, by the unweighted pair group using arithmetic averages (UPGMA). On the basis of those traits derived from the electrophoretical studies, an association matrix (OTU × OTU) was built, using the simple matching coefficient (SMC), from which the resulting phenograms (see Figures 4 and 5) were built by the UPGMA method.

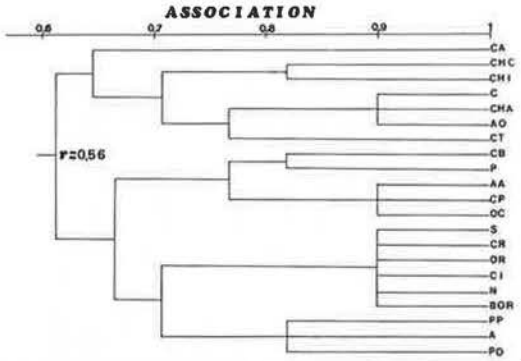


Figure 4. Phenogram of 21 OTU's resulting from UPGMA cluster analysis of the OTU × OTU association matrix, based on zein electrophoretic patterns. r: cophenetic correlation coefficient.

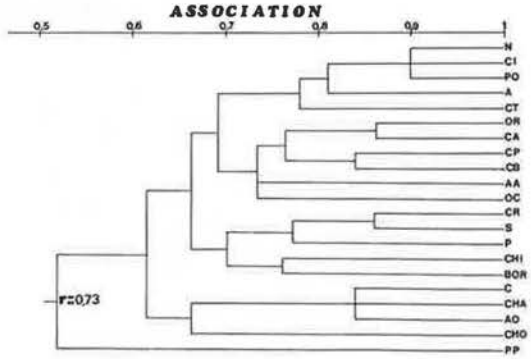


Figure 5. Phenogram of 21 OTU's resulting from UPGMA cluster analysis of the OTU × OTU association matrix, based on SDS-PAGE soluble proteins. r: cophenetic correlation coefficient.

According to what we have previously analyzed, it can be deduced that those races studied can be clustered into 2 or 3 very well-defined racial complexes, in which there is a smaller distance among the different races. The different groupings show a partial congruence though statistically significant. Otherwise, the results obtained on the basis of protein storage traits are also partially congruent with those measured on the basis of traditional morphological traits (Melchiore, personal communication).

Finally these results point out that biochemical traits can be appropriate to make a clustering analysis and in addition they are demonstrative of the considerable variation among the different races. This fact is in accord with what we have previously communicated in similar studies and documents the enormous variability present in maize.

Angel A. Nivio and Jorge L. Magoja

Effect of perennial teosinte introgression in maize on kernel protein content

It is very well known that the wild relatives of maize have a kernel protein content 2 or 3 times greater than maize (see MNL 59:61, 1985). As was previously published (MNL 59:69, 1985), the F2 and F3 progenies derived from crossing perennial teosinte (*Zea perennis*) and maize there is a high protein content, which is dominant, and is given by the wild parent of the cross (*Z. perennis*). This fact led us to believe that possibly the teosintes, or perennial teosinte in this particular case, could be used to improve the kernel protein content in maize.

A perennial teosinte introgressed population of maize was studied to find out the effect played by perennial teosinte germplasm on protein content (see MNL 61:66, 1987). It is important to point out that the introgressed population is indistinguishable from conventional ones as all the plants essentially resemble the phenotype of cultivated maize, the only difference being the high prolificity and variability attributed to the effects of wild germplasm introgression.

Table 1. Whole kernel protein content of a perennial teosinte introgressed population of maize.

Year	Protein (%) Mean ± SD	Range
1986	13.5 ± 1.3	11.4-15.1
1987	12.4 ± 1.6	8.8-15.4

During two years running, a plant sample was taken at random from the introgressed population, and then the chosen individuals were evaluated through different morphological traits and by their kernel protein content. Table 1 summarizes the results obtained for kernel protein content. As can be seen, the average values obtained are not too high, though they are greater than control average values. When we correlated protein content with 12 plant and ear traits, statistically significant associations were not detected: for example, prolific vs. nonprolific plants or high yield vs. low yield plants do not differ significantly between themselves in their kernel protein content. This fact let us budget that plant selection by kernel protein content perhaps does not significantly affect agronomically important traits.

As the population studied does not show a great difference in mean kernel protein content, relative to the controls used (11%), the relatively high variability existing for this trait in the introgressed population could eventually permit a selection cycle on high protein content. The most outstanding individuals have up to 50% higher protein content than commercial maize, and this fact can be attributed to the effects of wild germplasm introgression.

While mean kernel protein content of the introgressed population is lower than that found in progenies derived from interspecific hybrids (see MNL 59:69, 1985) it is useful to remember that those plants were morphologically very similar to teosinte, and had a lot of little spikes each with small kernels. On the other hand the actually studied material is essentially maize, and consequently the increasing of protein content in a great deal of individuals belonging to that population is considered significant, showing the positive effect of wild germplasm introgression on the expression of the studied traits.

Liliana A. Perini[1] and Jorge L. Magoja

[1]Fellow, Com. Inv. Cient. (CIC), Prov. Buenos Aires.

Variation of free amino acid content during germination of maize-Balsas teosinte hybrids

With the aim of studying the mobilization of storage proteins during seed germination, we have evaluated free amino acid content in hybrids between a maize inbred line (OU) and Balsas teosinte (*Zea mays* ssp. *parviglumis* var. *parviglumis*) (Zmpp). F1 hybrid seeds resulting from the OU × Zmpp cross and their respective parent seeds were arranged to germinate in Petri dishes at a constant temperature of 28 C and darkness. Ten mature grains, previously sterilized, were placed into each dish. Each 24 hours, and during six hours running, samples of 10 grains each were taken from each dish (2 repetitions) for each representative of this experiment.

Table 1. Free amino acid content (FAA) during germination in OU maize inbred Balsas teosinte (Zmpp) and its hybrids derived from OU × Zmpp cross.

Days germination	Parent or cross	FAA (uMLeu) per seed or seedling	per 100 mg of seed	per 100 mg of protein
0	OU	2.4	1.3	11.8
	Zmpp	0.5	1.5	15.5
	OU x Zmpp	2.9	1.5	13.6
1	OU	3.5	1.9	17.3
	Zmpp	0.8	2.1	21.6
	OU x Zmpp	4.2	2.2	20.0
2	OU	5.6	3.0	27.3
	Zmpp	1.2	3.2	33.0
	OU x Zmpp	6.7	3.5	31.8
3	OU	16.9	9.1	82.7
	Zmpp	3.5	9.5	97.9
	OU x Zmpp	18.4	9.7	88.2
4	OU	22.5	12.1	110.0
	Zmpp	3.7	10.3	106.2
	OU x Zmpp	20.2	10.6	96.4
5	OU	29.8	16.0	145.5
	Zmpp	4.7	13.0	134.0
	OU x Zmpp	41.1	21.6	199.1
6	OU	30.0	16.1	146.4
	Zmpp	5.5	15.2	156.7
	OU. x Zmpp	41.8	22.0	200.0

Figure 1. Variation of free amino acid content (FAA) expressed in μMLeu/100 mg protein, during germination of maize inbred (OU), Balsas teosinte (Zmpp) and OU × Zmpp hybrid seeds.

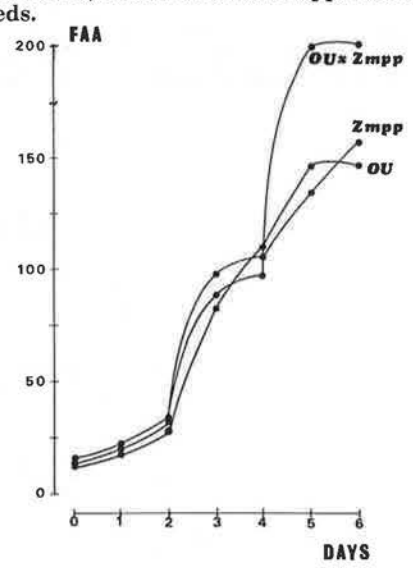
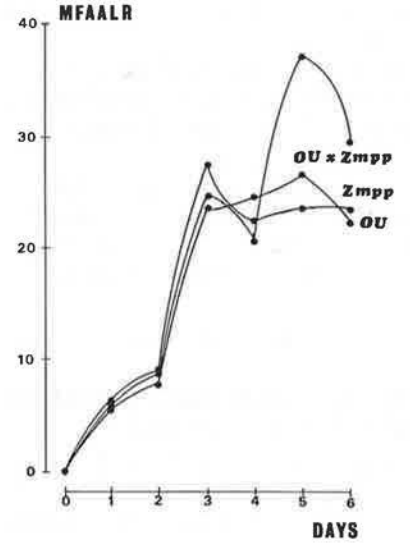


Figure 2. Variation of mean free amino acid liberation rate (MFAALR) expressed in μMLeu/100 mg protein day, during germination of maize inbred (OU), Balsas teosinte (Zmpp) and OU × Zmpp hybrid seeds.



The different samples were dried in the oven at a temperature of 65 C until they reached a constant weight, and then protein and water-soluble free amino acid (FAA) content was evaluated, expressing them in equivalent leucine micro mols (μm Leu) per 100mg of seeds. On the basis of seed dry weight and protein content, FAA was expressed per seed and per 100mg of protein (see Table 1). In mature grains which were not placed in Petri dishes to germinate, we have taken the measurements for the zero day (day 0), to be able to calculate the mean free amino acid liberation rate (MFAALR), which was expressed in leucine micromols/100mg of protein per day. The experimental results obtained are shown in Table 1 and we give a graphical representation of the free amino acid content variation and their liberation rate in Figures 1 and 2.

The mature grains of the hybrids and their respective parents do not significantly differ in their initial free amino acid and protein contents. The liberation of free amino acids during the germination process does not show any significant difference until 4 days after the initial incubation. From the fifth day, hybrids show a free amino acid content which is significantly greater than that of their parents. At the end of the experiment, free amino acid content was increased 10 or 15 times if we compared to the initial one. These produce a greater liberation of free amino acids in hybrids, as a direct consequence of a higher liberation rate (see Figure 2).

After 6 days incubation, hybrids have almost a 50% higher free amino acid content than their parents. The results obtained point out a greater proteolytic activity in hybrids, probably associated with a greater seed vigour, expressed through faster growing and mobilization of grain storage.

The results obtained through this simple experiment document once more the possibilities offered by maize wild relatives to increase the biological efficiency of the cultivated species (maize).

Ida G. Palacios and Jorge L. Magoja

Heterosis for dry matter and protein production per plant in diploperennial teosinte-maize hybrids

As we have previously reported (MNL 61:63, 1987), the heterosis produced in interspecific hybrids between diploperennial teosinte (*Zea diploperennis*) and a sweet variety of maize (Ever-green) can be detected in an early stage through the quantitation of the same traits during the earlier developmental stages of the younger plants. The results previously obtained and some other results obtained subsequently have let us make a most complete study through which the greater biologic efficiency of these hybrids can be demonstrated.

As was previously pointed out, this experiment let us obtain information about dry matter and protein production per plant in these hybrids and their respective parents. The different measurements were taken from seeding throughout the first 30 days of growing. The data obtained were used to calculate dry weight per plant (DW/PL) and protein per plant (P/PL). The increase in dry weight per plant (DW/PL) and protein per plant (P/PL) was evaluated on the basis of initial dry weight and protein in seeds respectively. The mean growth rate (MGR) expressed in mg of dry weight/plant day and the mean protein production rate (MPPR) expressed in mg of protein/plant day, were evaluated on the basis of the initial values for both

Figure 1. Variation of dry weight per plant (DW/PL) expressed in mg in Ever-green maize (Eg), diploperennial teosinte (Zd) and its hybrids (Zd × Eg).

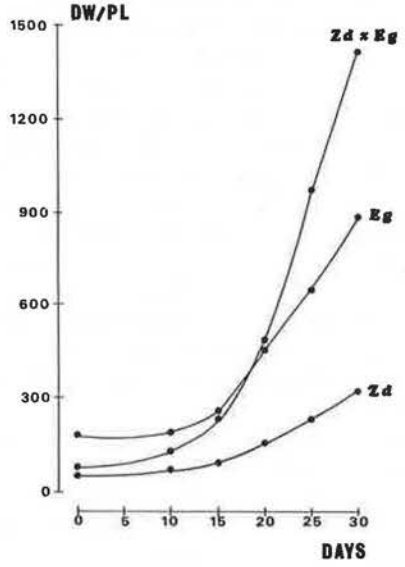


Figure 2. Variation of protein content per plant (P/PL) expressed in mg in Ever-green maize (Eg), diploperennial teosinte (Zd) and its hybrids (Zd × Eg).

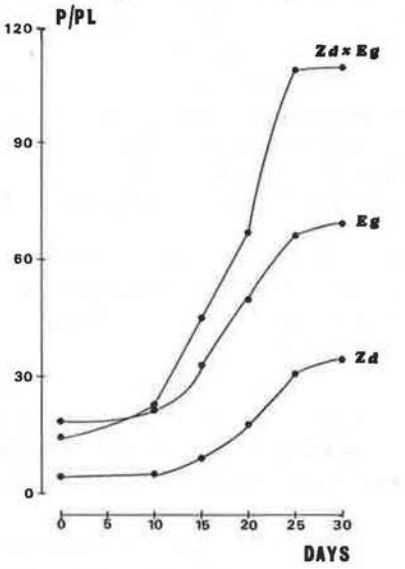


Table 1. Heterosis of diploperennial teosinte maize hybrids for the expression of the following traits: dry weight per plant (DW/PL), protein per plant (P/PL), dry weight increase per plant (DWI/PL) and protein increase per plant (PI/PL).

	% heterosis respect	
Character	Mid parent	Better parent
DW/PL (mg)	136.1	60.2
P/PL (mg)	110.2	57.6
DWI/PL (mg)	176.4	89.1
PI/PL (mg)	134.6	88.4

traits. The results obtained are illustrated in Figures 1 to 4.

As can be seen, hybrids are remarkably better than their parents when we analyze dry weight and protein production per plant. Thirty days after sowing, the hybrids had produced almost 2 times more dry weight and protein content than the better parent (maize). The greater efficiency of these hybrids to express the measured traits is mainly a direct consequence of both a high mean growth

Figure 3. Variation of mean growth rate (MGR) expressed in mg dry weight/plant · day in Ever-green maize (Eg), diploperennial teosinte (Zd) and its hybrids (Zd × Eg).

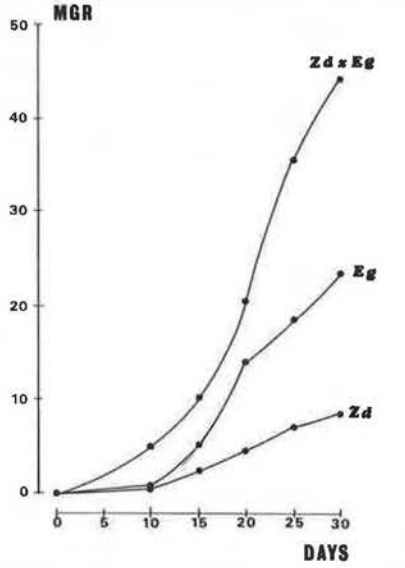
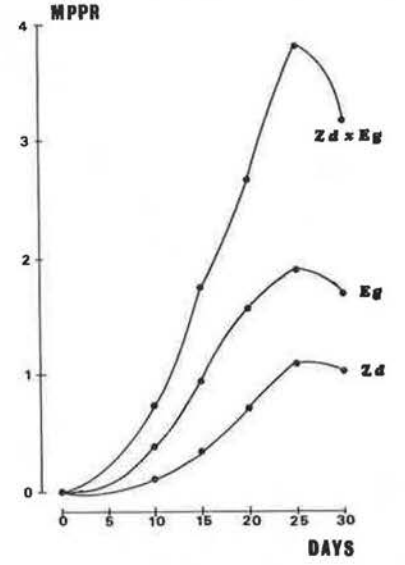


Figure 4. Variation of mean protein production rate (MPPR) expressed in mg protein/plant · day in Ever-green maize (Eg), diploperennial teosinte (Zd) and its hybrids (Zd × Eg).



rate and protein production rate during the period we have considered.

The heterosis expressed by those considered traits (see Table 1) is not only high when it is calculated in relation in the mid-parent, but also when it is evaluated in relation to the better parent (maize).

Finally we can say that though maize can be considered as one of the most efficient vegetable species the results we have obtained show that this particularity could probably be increased through the utilization of wild germplasm.

Ida G. Palacios and Jorge M. Magoja

Effect of four defective kernel mutants on seed free amino acid and protein content

Some mutants which condition defective kernels in maize, as we have previously studied (see MNL 56:108, 1982; 57:71, 1983 and 58:120, 1984), produce multiple

morphological, physiological and biochemical modifications in seeds. Especially, the study of biochemical changes conditioned or associated with defective mutations gives opportunity to understand the nature of the mutation and the possible changes promoted in most of cases, in which the character is lethal.

With the purpose of studying if the degree of defectiveness is associated with other biochemical characters, we have enlarged our investigations to another 3 spontaneous mutants which condition defective kernels. Four spontaneous mutants (*de*-7670; de*-7510; de*-7547 de*-7601*) produced in different inbred lines and conditioned by recessive genes of single inheritance were used in this analysis. Over a total of 58 segregant ears for each one of the mutations, the weight of defective and normal kernels was evaluated, determining the "defectiveness degree" through the calculation of the weight proportion of defectives relative to their normal equivalent (see Table 1).

Defective and normal kernel samples were taken at random from each ear. For each sample protein and free water-soluble amino acid content were determined. Free amino acids were expressed in equivalent leucine micro mols (μMLeu) per each 100mg of grain or protein (see Table 1).

Protein content did not differ significantly between normal and defective kernels. In contrast, highly significant differences are detected for free amino acid content in 3 of the mutants used (*de*-7601; de*-7670; de*-7510*), which notably affect endosperm and embryo.

Mutant *de*-7547* does not affect the viability of the embryo or condition significant differences in free amino acid content.

In Table 2, results were expressed on the basis of the individual kernel. As weight decreases it can be noted that defective kernels generally have a smaller protein and free amino acid content than their normal equivalent. In the case of *de*-7601* and *de*-7510*, this does not occur in free amino acid content. As a consequence of the high level of free amino acid content despite weight reduction, these defective kernels have almost the same free amino acid content per grain as their normal equivalent.

In Table 3 some of the possible associations that can be studied among the evaluated traits are shown. From these results, it can be deduced that the biggest kernels have a greater whole protein and free amino acid content, and relative level of free amino acid content is inversely associated with kernel weight. This fact points out that the most defective kernels have a greater percentage of free amino acids.

Three of the mutants (*de*-7601; de*-7670; de*-7510*) produce a strong weight reduction in kernels: defective kernels have from 1/10 up to 1/4 of the weight of their normal equivalents. These mutants are lethal as defective kernels are not capable of germinating under normal conditions, but if they do, they generate plants which die in an early stage of their development.

Defective *de*-7547* does not condition such a strong weight reduction in kernels (near 50% of the weight of their normal equivalents), nor the normal viability of kernels, producing plants which come up to a reproductive stage.

The lethal mutants studied condition from 2 to 6 times more free amino acid content in defective kernels relative to the normal ones, while the viable defective does not show any difference.

The results we show in this paper point out that at least for this little sample of mutant genes that condition defective kernels the "defectiveness degree" is significantly related to free amino acid content. This phenomenon can be attributed to the next: mutant genes, we believe, produce an early blockage in the synthesis of storage proteins or a repression of determined kinds of protein, which determine that part of those amino acids translocated to kernel do not form any part of the protein fraction. Some of these non-synthesized proteins could be essential for normal embryo development. This fact would possibly explain the lethality of some mutant genes.

Ida G. Palacios and Jorge L. Magoja

Table 1. Mean values of kernel weight and biochemical traits for defective kernel mutant segregating ears.

	Phenotype	Kernel weight (mg)	Weight of defectives (% of normals)	Protein (%)	Free amino acids content uMLeu/100 mg kernel	uMLeu/100 mg protein
de*-7670	+	168.1 ± 42.6	—	8.5 ± 0.1	2.2 ± 0.1	25.9 ± 0.4
	de	15.1 ± 6.9	9.0 ± 1.8	9.9 ± 0.8	4.1 ± 1.2	41.7 ± 9.2
de*-7510	+	188.3 ± 30.1	—	10.0 ± 1.1	1.7 ± 0.1	17.0 ± 0.3
	de	33.3 ± 8.8	17.7 ± 2.0	9.3 ± 0.7	9.2 ± 2.1	98.9 ± 12.5
de*-7547	+	256.2 ± 31.0	—	8.8 ± 0.4	1.6 ± 0.9	18.3 ± 10.2
	de	115.2 ± 20.8	45.0 ± 6.2	10.1 ± 1.1	1.9 ± 0.5	18.2 ± 3.9
de*-7601	+	214.7 ± 41.5	—	11.5 ± 0.9	2.0 ± 0.5	17.1 ± 4.5
	de	59.1 ± 17.8	28.3 ± 9.4	11.4 ± 0.5	5.4 ± 2.9	48.0 ± 26.9

Table 2. Mean values of protein and free amino acids expressed per kernel for segregating ears of defective mutants.

	Phenotype	Protein/kernel (mg)	Free amino acids/kernel (uMLeu)
de*-7670	+	14.3	3.7
	de	1.5	0.6
de*-7510	+	18.8	3.2
	de	3.1	3.1
de*-7547	+	22.5	4.1
	de	11.6	2.2
de*-7601	+	24.7	4.3
	de	6.7	3.2

Table 3. Correlation coefficients between some traits of defective kernel segregating ears.
***: significant at 5% level; **: significant at 1% level**

	r
Kernel weight vs. protein/kernel (a)	0.98**
Kernel weight vs. uMLeu/kernel (a)	0.74*
Protein/kernel vs. uMLeu/kernel (a)	0.75*
uMLeu/100 mg protein vs. kernel weight (b)	-0.62**

(a): calculated for the four mutants; (b): calculated for total ears studied.

Perennial teosinte introgressed population of maize: variation within S1 derived lines

As part of a project whose objective is increasing heterosis and genetic variability in maize, a perennial teosinte (*Z. perennis*) introgressed population of maize was obtained, in a first stage. This population was obtained using selected progenies derived from hybrids between perennial teosinte and maize, as donors of wild germplasm, and a wide diversity of maize stocks as recurrent parents (see MNL 60:82). Finally, as a result of that controlled introgression, the resulting population shows a wide variability, and unusual individuals combining exceptional characteristics can be found in it, as they combine precocity, prolificity and a high yield potential (see MNL 61:66).

During the 1985/86 growing season, we started the second stage, in search of the objectives previously pointed out, and then we initiated a first cycle of recurrent selection. A great number of plants of the population were self-pollinated. The progenies of these plants, or S1 derived lines, were grown during the next growing season (86/87). Each was crossed by two testers to measure their

combining ability (c.a.), and then, among themselves, the best S1 derived lines were crossed completing the recurrent selection (cycle).

Actually, the F1's derived from cross-breedings are being evaluated through yield comparative designs, while the preliminary results for different characters of agronomic importance have let us evaluate the variability range among S1 lines.

As shown in Table 1, the quantitation of some characters let us deduce that in most cases a wide variation is detected.

Table 1. Relevant traits of S1 lines derived from a maize population introgressed with perennial teosinte.

Character	Mean ± SD	Range
Days to tassel	62.1 ± 4.6	48-69
Days to pollen	67.6 ± 4.2	57-79
Days to silking	70.1 ± 4.2	60-85
Protoandrous (days)	2.5 ± 0.2	1-5
Plant height (cm)	180.0 ± 19.1	130-255
Ear insertion height (cm)	78.7 ± 17.9	45-150
Number of tillers	1.2 ± 0.3	1-3.8
Number of ears per plant	2.1 ± 0.4	1.1-3.2
Ear lenght (cm)	12.6 ± 1.7	8-17.1
Ear diameter (cm)	3.4 ± 0.4	2-4.4
Ear weight (g)	64.6 ± 23.0	20-133.3
Number of kernel rows	12.5 ± 1.5	8-18
Weight of 50 kernels (g)	11.5 ± 2.8	5-21.2

One of the most significant traits, and one which can be attributed to the primary effect of introgression, is the high prolificity present in most of the S1 derived lines. Some lines have, in all the cultivated plants, up to 3 perfectly developed ears per tiller. The S1 lines have on average a smaller number of ears and tillers per plant than the original population (see MNL 61:66), and the ears weigh less.

The enormous variability produced when combining wild germplasm with germplasm of the cultivated species (*Z. mays* ssp. *mays*) is not adequately reflected through those few quantitative traits measured. Qualitative variation is greater, and can be appreciated when you observe ears and plants.

As it is true that in some cases inbreeding depression has conditioned a reduction in the amplitude of variation, in other traits this amplitude is kept without significant differences when compared with the original population.

The results obtained point out that the increase of the genetic base and germplasm produced by introgression can effectively contribute to the development of new genetic resources, from which new lines able to produce better hybrid combinations can probably be obtained.

Gabriela Pischedda[1] and Jorge L. Magoja
[1]Est. Exper. Reg. Agropec. Pergamino, INTA

Diploperennial teosinte introgressed population of maize: variation within S1 derived lines

At the same time that perennial teosinte is being used in wild germplasm introgression projects in maize, we have also obtained a diploperennial teosinte (*Zea diploperennis*) introgressed population of maize. As was previously pointed out (MNL 60:82 and MNL 61:65), this population was originated from the recombination of different maize stocks with diploperennial teosinte germplasm given by interspecific hybrids. A recurrent selection cycle was started from the introgressed population, as was done when we used perennial teosinte (see preceding), with the only purpose of making the developed individuals capable for practical purposes.

Table 1. Relevant traits of S1 lines derived from a maize population introgressed with diploperennial teosinte.

Character	Mean ± SD	Range
Days to tassel	68.9 ± 3.2	63-77
Days to pollen	79.0 ± 3.6	68-86
Days to silking	75.4 ± 3.8	66-83
Protoandrous (days)	-3.6 ± 0.2	-2-(-5)
Plant height (cm)	185.9 ± 17.1	145-215
Ear insertion height (cm)	91.2 ± 15.8	55-120
Number of tillers	2.1 ± 0.6	1-4.4
Number of ears per plant	2.7 ± 0.5	2-4
Ear lenght (cm)	12.1 ± 1.8	8.5-15.7
Ear diameter (cm)	2.7 ± 0.4	1.9-3.5
Ear weight (g)	44.9 ± 20.8	25-100
Number of kernel rows	10.0 ± 1.7	8-14
Weight of 50 kernels (g)	8.6 ± 2.1	5-14.2

A preliminary evaluation of S1 derived lines from self-pollinated plants of the original population is summarized in Table 1. There is considerable variation in most of the traits we have measured, but this is smaller than that shown by the original population (see MNL 61:65). When these lines are compared with those derived from the perennial teosinte introgressed population, it can be deduced that they have more tillers with a higher number of ears per plant, though these ears are smaller in size.

Generally a smaller variation is noted in this case than in the previous one, though it can be considered that there is a wide germplasm and genetic base produced by the introgression of foreign germplasm in maize. As in the case of S1 derived lines from the perennial teosinte introgressed population, those derived from a diploperennial teosinte introgressed population show a lower number of tillers and ears per plant than the original population from which they arise.

The results we have obtained do not let us deduce that perennial teosinte germplasm introgression in maize is more successful than diploperennial teosinte germplasm introgression. But we must say that this comparison is not possible, as we have not used the same maize stocks as recurrent parent. Especially as in the case of introgression with diploperennial teosinte germplasm, the genetic base of the maize stock used was really very narrow.

The results obtained let us deduce that the utilization of germplasm belonging to two of the most phylogenetically distant taxa of *Zea*, from the cultivated species (maize), produces a great variability, probably similar to the variability found by primitive man from whom modern maize is derived. If this is true now, then man once more has the possibility of leading the evolution of maize to more productive forms (another plant architecture?) through less empirical methods.

Gabriela Pischedda and Jorge L. Magoja

Seedling abnormalities in Argentine breeding populations and advanced generations of their crosses

Trial plots of selfed parents, selfed F1, and F2 (F1 sibbed) from a diallel between local populations grown in summer 1986 showed an unusual frequency of chlorophyll abnormalities, mostly albinos. In spite of the fact that the experiment was not carried out for this purpose, the data on abnormality frequency and the population backgrounds would enable us to discuss the probable causes.

Five populations were used in the experiment. Punzó INTA is a commercial variety, obtained by stratified mass selection in 3:1 A synthetic in EERA Pergamino INTA (Sanguinetti, personal communication; for 3:1 A synthetic, see Illia et al., IDIA suppl. 32:32, 1976). BSR is the Syn-3

from the second cycle of recurrent selection for combining ability with the unreleased single cross ER × EX in 3 gene pools of the IFSC (Aguilar Riega, unpublished report). The remainder were unimproved stocks of the IFSC maize breeding programme.

The 5 populations and 2 others not included in the study were crossed in summer 1983 by one of us (F.J.B.) with bulked pollen, and the advanced generations of each cross were obtained in 1985. Composites of seeds from each generation of each cross were used for 2 yield trials in Llavallol and Chacabuco, Buenos Aires, in 1986. Abnormality frequencies only were taken in Llavallol. Data expressed in percent were analyzed using arc sin transformation, modified for zero percentage. Means of each entry were compared with suitable values for zero percentage (Table 1).

Table 1. Chlorophyll abnormal percentages in five populations and their crosses in 1986-87.

	Punzó	BSR	BHDC	BG	BCb
Punzó	1.89*	4.13*	3.29*	0.00	0.00
BSR	1.85*	8.39*	0.00	2.71*	0.00
BHDC	0.00	0.00	0.00	1.98*	0.91
BG	0.00	0.00	0.00	0.00	0.46
BCb	0.00	0.00	0.89	0.49	0.00

Percent mean of abnormalities observed in selfed parents (diagonal), selfed F_1 (above) and F_2 (below).

* Significantly different from zero at 0.10 level.

Table 2. Chlorophyll abnormality percentages in five populations and their crosses in 1987-88.

	Punzó	BSR	BHDC	BG	BCb
Punzó	0.48	6.49*	3.53	0.00	0.00
BSR	1.04*	4.74*	1.92*	0.98	0.00
BHDC	0.00	0.00	0.48	1.43	2.82*
BG	0.00	0.00	0.00	0.00	0.00
BCb	0.00	0.00	0.00	0.00	0.00

Percent mean of abnormalities observed in selfed parents (diagonal), selfed F_1 (above) and F_2 (below).

* Significantly different from zero at 0.10 level.

Rough estimates of heterozygous individual frequencies in BSR and Punzó were 30.14 and 7.4%, respectively. In the other populations selfed, abnormalities were not detected probably due to lower frequency that required a larger sample.

BSR × Punzó selfed, showed an intermediate abnormality frequency between those observed in the selfed parents. No abnormalities were observed in 4 crosses with BSR or Punzó, probably due to the low frequencies supposed in the other parents. All crosses between parents without evidence of abnormalities (BG × BHDC, BG × BCb, BHDC × BCb) exhibited these abnormalities. This could be explained by fixed alleles in different chlorophyll loci in each parent, and repulsion phase linkage in F1. Therefore the frequency of recessive homozygotes for any locus in selfed F1 was increased so it was easier to detect the abnormalities.

Seed stocks were increased in summer 1986, and the experiment was repeated in summer 1987, using mixed samples of seed of both years. Data of abnormality frequencies were also taken in Llavallol as during the previous year, and the analysis was performed as above (Table 2).

Abnormality percentages in BSR and Punzó were lower than in 1986; but we observed albinos in BHDC selfed and BHDC × BSR selfed. From F2, only BSR × Punzó showed abnormalities, although BHDC × BCb showed them in an additional trial with F2 and backcrosses. In this trial we also observed seedling abnormalities in (Punzó × BHDC) × Punzó and (BSR × Punzó) × BSR.

Selection in Punzó and BSR would have increased the frequency of heterozygous chromosome segments, marked by chlorophyllic abnormalities. Presence of different fixed alleles in the rest of the populations could be related to the divergence among them, and random fluctuations (genetic drift) due to assortative mating or effective population number during maintenance.

Francisco J. Babinec and Laura A. Pflüger
[1]Fellow of CONICET

LONDON, ONTARIO, CANADA
University of Western Ontario

Embryo maturation proteins

Maize embryo maturation proteins comprise 9 to 11 polypeptide species when separated by 2D IEF-SDS PAGE, resolving over a range in pH of 5.0-8.0 and in molecular weight of 10,000-100,000. Individual species (A-K) have approximate pHs and molecular weights, respectively: A (7.7, 66,000); B (7.2, 66,000); C (6.8, 52,000); D (5.5, 39,000); E (6.4, 29,000); F (6.6/6.5, 81,000); G (6.3, 34,000); H (7.7, 73,000); J (7.4, 73,000), and K (7.2, 73,000). Although the precise age at which synthesis is initiated varies among the different polypeptides, synthesis of all species occurs within a 5 to 10 day period beginning 20-25 days after pollination (DAP) under field conditions. Maturation protein synthesis has been observed as early as 26 DAP in embryos from greenhouse-grown plants.

Following their initial detection, the maturation proteins rapidly become the most prominently synthesized species of embryo polypeptides, and high levels of synthesis relative to other polypeptides are maintained until maturity at 40-50 DAP. In studies involving 3 inbred cultivars (Oh43, M14, W23) and hybrids derived from their reciprocal crosses, developing embryos of all genotypes exhibited a similar pattern of maturation protein synthesis. Isoelectric variation between cultivar was detected for only 1 polypeptide, whose assignment to the maturation protein group is tentative.

Although a number of polypeptides are common to both embryo and seedling tissues, the maturation proteins are embryo specific. They are not observed on fluorograms from radicles, mesocotyls or plumules of 5 day old seedlings or from germinating embryos between 4 and 6 hours of imbibition. Maturation proteins appear to be present in tissues of both the scutellum and embryonic axis since they are observed in excised scutella and are not noticeably reduced in embryonic axes from which most of the scutellum has been removed.

With the exception of 1 species (E), maturation proteins cannot be identified among the products obtained from the in vitro translation of either total cellular RNA or intact polysomes when separated under conditions similar to those employed for in situ extracts. These results suggest that translational or post-translational controls may be involved in maturation protein expression. However, the in vitro translation of total cellular RNA in the presence of a dog pancreas microsomal membrane post-translational modification system does not result in the expression of additional maturation proteins.

J.G. Boothe* and D.B. Walden
*Current address: Plant Research Institute, Saskatoon, Sask.

In vitro studies with genic male steriles

Chemical reversion of genic male steriles to produce homozygous male sterile seed has been proposed by several researchers (e.g., Conn. Agric. Exp. Stn. Bull. 550, 1951; MNL 50:116, 1976; MNL 59:87, 1985). Since genic male sterility in maize, as in other species (Can. J. Bot. 51:2473, 1973), might be the result of genetic block in the synthesis or metabolism of endogenous plant growth regulators (MNL 50:116, 1976) or other molecules, treatments with appropriate chemicals might revert the genic male steriles to fertility. The present study was initiated to test the possibility of chemical reversion of genic male steriles of maize using the in vitro tassel culture system described previously (MNL 59:72, 1985; MNL 60:89, 1986). Two genic male steriles of maize, *ms14* in Oh43 background and *ms24* (UWO stocks, obtained originally from Earl Patterson) were selected for this study because these mutants cause the male gametophyte to degenerate at late microsporogenesis. We assume that these mutants provide a better chance for reversion than do other genic male steriles which fail earlier in development (Can. J. Genet. Cytol. 23:195, 1981; MNL 59:87, 1985).

Immature tassels of these male steriles were cultured in vitro as described previously (MNL 59:72, 1985). While *ms14* tassels grew well and produced approximately 100-150 normal spikelets per tassel, the tassels of *ms24* developed poorly and produced only 30-80 normal spikelets per tassel. However, the microspore development and breakdown in the cultured tassels of both male steriles was similar to that grown in vivo. Microspores from cultured *ms14* tassels degenerated at the late vacuolate microspore stage. Microspores from cultured *ms24* tassels underwent normal development through meiosis and the first mitotic division. After the first binucleate pollen stage, the generative nucleus degenerated followed by degeneration of the vegetative nucleus. At maturity, the spikelets on cultured *ms24* tassels extruded anthers and occasionally engorged pollen was produced. These results are similar to that of in vivo studies here or reported elsewhere (Can. J. Genet. Cytol. 23:195, 1981; MNL 59:87, 1985). Thus, immature cultured tassels of these genic male steriles (*ms14* and *ms24*) express male sterility without the influence of the rest of the plant. Similarly, the expression of the male sterile-mutant characteristic was observed in the cultured tassels of 2 other genic male steriles (*ms2* and *ms10*).

The effects of 12 different plant growth regulators (PGRs) at various concentrations were studied for the potential reversion of male sterility in vitro (Table 1). None of these treatments reverted either of the 2 genic male steriles to produce normal pollen. In the case of *ms24*, while a few engorged pollen grains formed in some instances, the frequency of such pollen was not increased with any treatment. One might conclude therefore that at least in these genic male steriles of maize, the metabolic blocks which lead to pollen breakdown are not overcome by the application of specific molecules at the concentrations and in the forms applied. Despite the negative results obtained in this study with PGRs, the possibility of reversion of genic male steriles with other chemicals can not be excluded and therefore, further research is warranted.

Table 1. PGRs used to study their effect on reversion and development of male steriles.

Abbr.	PGR	Conc.	ms14	ms24
Z	zeatin (4-hyroxy-3-methyl-trans-2-butenylaminopurine)	10^{-9} - 10^{-6} M	-	*
ZR	zeatin riboside	10^{-9} - 10^{-6} M	-	*
BAP	6-benzylaminopurine	10^{-9} - 10^{-6} M	-	*
K	kinetin (6-furfurylaminopurine)	10^{-9} - 10^{-6} M	*	*
GA3	gibberellic acid	10^{-9} - 10^{-6} M	*	*
CCC	2-chloroethyl trimethylammonium chloride	10^{-9} - 10^{-6} M	*	*
AMO	AMO-1618 (2-isopropyl-4-dimethyl amino-5-methylphenyl-1-piperidine carboxycate methyl chloride)	10^{-9} - 10^{-6} M	*	*
PHO	phospon-D (2,4 dichloro benzyl	10^{-9} - 10^{-6} M	*	*
IAA	3-indole acetic acid	10^{-8} - 10^{-5} M	*	*
TIBA	2,3,5-tri-iodeobenzoic acid	10^{-8} - 10^{-5} M	*	*
E	ethephon (2-chloroethyl phosphonic acid)	10^{-8} - 10^{-5} M	*	*
ABA	abscisic acid	10^{-8} - 10^{-5} M	*	*

D.R. Pareddy[1], R.I. Greyson and D.B. Walden
[1]Present address: United AgriSeeds, Inc., Champaign, IL

Variation in leaf polypeptide synthesis for several cultivars

We reported (MNL 61:72, 1987) a study to monitor variation in leaf polypeptide synthesis for various inbred and hybrid maize cultivars grown under different field management practices. The extraction, electrophoretic separation of polypeptides and fluorography of samples from 1985, 1986 and 1987 provide a large data set which has been analyzed using multivariate analysis techniques (Comput. Biol. Med. 17(1):29, 1987). These methods involve an eigenanalysis which defines and outlines trends

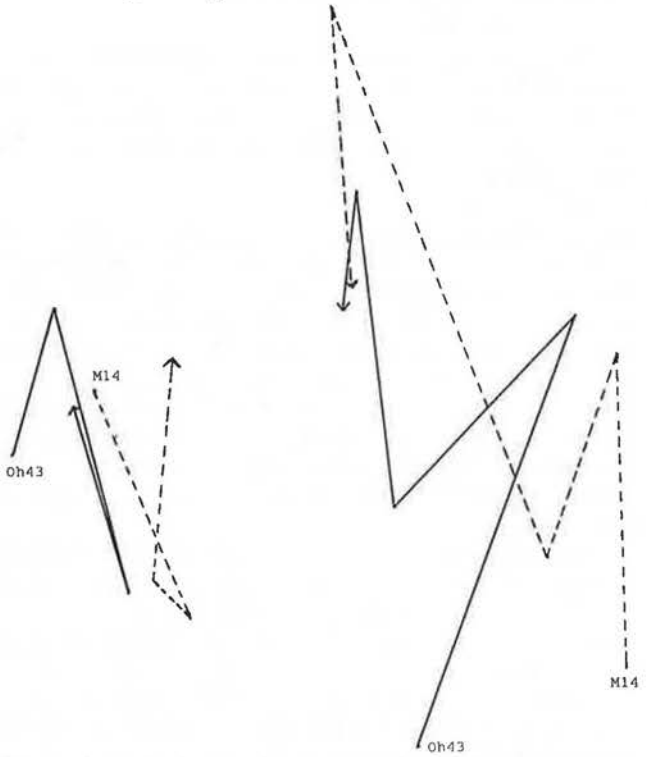


Figure 1. Trajectories drawn using the first two principal coordinates for the genotypes Oh43 and M14 grown in the control field plot. The trajectories drawn for the 1985 samples are grouped to the right while those for the 1986 samples are grouped to the left.

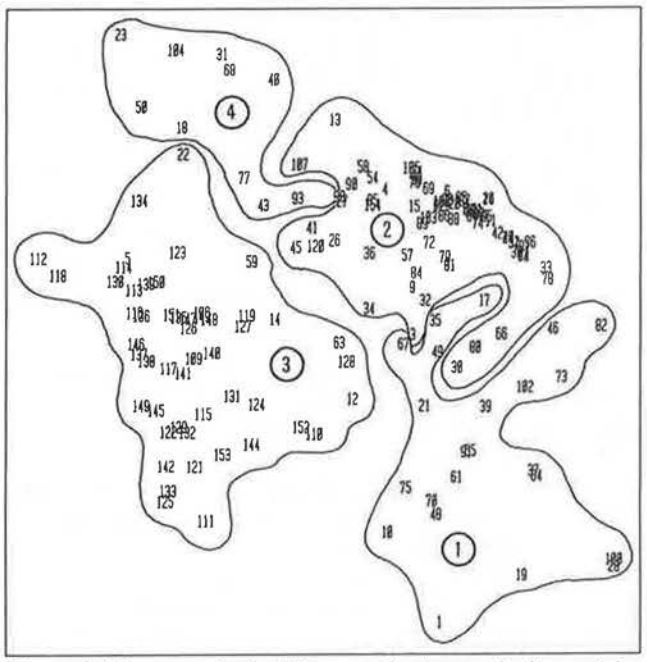


Figure 2. Diagram derived from a cluster analysis containing the genotypes Oh43, M14, Oh43/M14, and M14/Oh43 sampled for both the 1985 and 1986 growing seasons. The four major cluster groups represent the following developmental ages: group 1 - 25 days after planting, group 2 - 43 and 60 days after planting, group 3 - 35, 52, 76, and 110 days after planting, group 4 - 139 days after planting. Groups 1, 2 and 4 belonged to the 1985 data and were separated from the 1986 data, group 3.

in variation in any or all of the data subsets, e.g. cultivars, field plots, etc. Principal coordinate analysis summarizes variation found among the original data set variables and provides values which can be presented graphically as trajectory plots. Finally, a sum of squares clustering method was used to examine the grouping of individuals.

The study examined phenotypic (protein) variation among the various maize cultivars in an attempt to identify: 1) proteins that might be unique to cultivars; 2) proteins that might have a role in heterosis; and/or 3) proteins that might be of developmental significance. Approximately 5% of the polypeptides synthesized in the leaf tissue of different cultivars were found to be variable in expression and unique to specific cultivars and/or specific management plots. Principal coordinate analyses suggested that not necessarily the same polypeptides were varying in expression from 1 field season to the next since the trajectories for any 1 cultivar were separated by years (Figure 1). Polypeptide synthesis patterns throughout development show both similarities and differences between inbred and hybrid cultivars. Multivariate analyses led to the suggestion that groupings in the cluster analyses occur as a function of development (age) (Figure 2).

A subset of polypeptides was found to be expressed uniquely in certain inbred and hybrid cultivars. Heterosis was not fully described in terms of variant expression of polypeptides when inbred and hybrid genotypes were compared.

T.G. Crowe and D.B. Walden

Oncogene-related sequences transcribed in maize

We reported (MNL 60:91, 1986; 61:72, 1987) on the presence of oncogene-related sequences in maize. In those reports, ^{32}-P labelled probes (v-myb, v-myc, v-src, v-Ki-ras and v-Ha-ras) detected homologous sequences in the maize genome through Southern blot analysis. Under similar hybridization conditions (MNL 61:72, 1987), no fos-related sequences were detected.

The myb-related sequence in maize has been characterized by an alternative method. Paz-Ares et al. (EMBO J. 6:3553, 1987), by transposon tagging and subsequent sequence analysis, have revealed that the *c1* gene product in maize is homologous to *Drosophila* and human c-myb gene products.

Using hybridization conditions and techniques reported previously (MNL 61:72, 1987), preliminary results indicate that maize has sequences homologous to v-abl but not to v-sis. V-sis is the viral homologue of animal platelet-derived growth factor; consequently, it is not surprising that there are no maize sequences homologous to v-sis. V-abl, like v-src, encodes a highly conserved tyrosine kinase domain. It is possible that this domain is the only encoded domain that is common between the animal src genes and the maize src-related sequence. If such were the case, v-abl should bind to the src-related sequence in maize. Southern hybridizations indicate that v-abl does not bind to the src-related sequence in maize. Thus, it appears that the homology between v-src and the related maize sequence encompasses a greater region than the encoded tyrosine kinase domain. However, proof for the extent of homology between the putative maize src gene and animal src genes must await sequence analysis.

We have initiated RNA blot analysis of poly(A)$^+$ selected RNA from 5-day-old plumule tissue. Total RNA

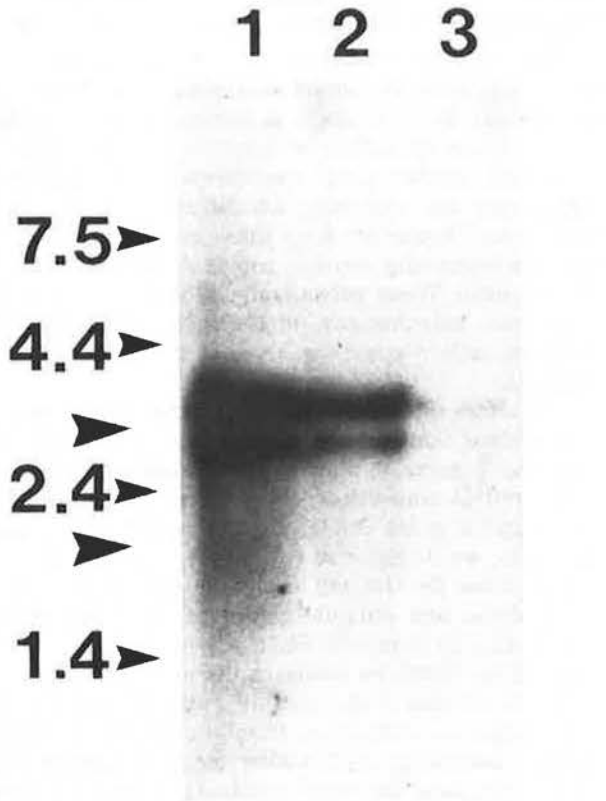


Figure 1. RNA blot of maize poly(A)$^+$ selected RNA hybridized with Ha-ras. Lanes 1-3 have 15, 7.5, and 2μg of mRNA respectively. The size in kilobases and the relative positions of RNA ladder fragments (BRL) are indicated. Larger arrowheads indicate the relative positions of the maize 26s and 17s ribosomal subunits.

was isolated by the guanidine isothiocyanate method (Methods in Molecular Biology, eds. Davis et al., 1986) and selected twice for poly(A)$^+$ mRNA using mAP paper (Amersham). The poly(A)$^+$ mRNA was electrophoretically separated on glyoxal/DMSO denaturing agarose gels (1.4%) and blotted to Zeta-Probe membrane (BioRad) using 20X SSC as the transfer buffer. The prehybridization buffer consisted of 30% deionized formamide, 5.5X SSPE, 1% SDS, 0.5% "Blotto" (Carnation low fat milk powder), and 500μg/ml of sheared and denatured salmon sperm DNA; incubation temperature was 56 C. The hybridization buffer consisted of the prehybridization buffer plus 20% dextran sulphate and 50ng of radio-labelled oncogene probe; the incubation temperature was 56 C. Washes were done at 60 C in 1X SSPE and 2% SDS.

Figure 1 shows initial Northern blot analysis of poly(A)+ selected maize mRNA probed with Ha-ras.

The Ha-ras detected 2 transcripts (3.2 and 2.8Kb) in young rapidly growing plumule tissue. Ha-ras has been found, in animals, to be expressed in dividing cells during early development. These results provide further evidence that the oncogene-related sequences in maize are genes which may be active during periods of rapid growth.

Work is underway to determine if other maize oncogene-related sequences are transcribed in rapidly growing plumule tissue.

R.B. Zabulionis, D.B. Walden, J.D. Procunier*
and B.G. Atkinson*
*Department of Zoology

Ubiquitin mRNAs synthesized by control and heat-shocked radicles and plumules of seedlings

Ubiquitin is a 76-amino-acid protein found in all eukaryotic cells. Its amino acid sequence is highly conserved and, in most cases, is derived from a multigene family. The genes coding for ubiquitin appear to consist of a variable number of repeated ubiquitin coding elements which are not separated by introns or other spacer sequences. Transcripts from these genes consist of large mRNAs containing multiple copies of information coding for ubiquitin. These 'polycistronic-like' mRNAs are translated into 'polyubiquitin' proteinaceous products which must be split precisely to produce the final functional ubiquitin.

Although the general structure of the ubiquitin genes is conserved, there are species differences in the number of genes per genome, in the number of repeats and in the size of the mRNA transcripts. As a prelude to characterizing the ubiquitin genes and the regulation of their expression in maize, we determined (by use of an avian ubiquitin cDNA probe) the size and level of ubiquitin transcripts in the radicles and plumules of control and heat-shocked (25 - >42.5 C) 5-day-old Oh43 maize seedlings. Results from these Northern analyses disclosed striking differences in the size of the ubiquitin mRNAs constitutively synthesized in each organ. Plumules produced ubiquitin mRNAs consisting of 1200-2000 nucleotides while primary radicles from the same seedlings produced ubiquitin mRNAs which range from 700-1500 nucleotides in length. Although the Northern analyses did not detect any changes in the organ-specific size of the ubiquitin mRNAs from heat-shocked seedlings, quantitation from dot-blots reveals that heat shock causes a marked increase in radicle (4-fold) and plumule (2-fold) ubiquitin mRNA levels. These results suggest (1) that maize ubiquitin is, as it is in animal cells, a heat shock protein, and (2) that the ubiquitin synthesized by the radicles and plumules of control or heat-shocked 5-day-old maize seedlings are either the products of different genes being expressed in each organ or are products of the same gene which undergo organ-specific processing.

Ling Liu*, D.B. Walden and B.G. Atkinson*
*Department of Zoology

Quantitation and intracellular distribution of the 70 and 18kd hsp mRNAs in seedlings

Exposure of maize seedlings to a rapid elevation in temperature (heat shock, hs; 25-42.5 C) results in the synthesis of heat shock polypeptides (hsp's) with Mr's of 108, 89, 84, 73 (hereafter referred to as 70) and 18kd. These hsp's are detected in vivo and are prominent among the in vitro translation products of polyribosomal poly(A)$^+$ RNAs from heat-shocked plumules.

Northern hybridization analyses suggest that the hs-enhanced synthesis of the 70kd hsp results from an increase in the accumulation of 70kd RNA transcripts on both free-cytoplasmic and membrane/cytoskeletal-associated ribosomes. In contrast, RNA blot analyses indicate that synthesis of the more prominent 18kd hsp variants results from the new transcription of 18kd RNA transcripts and the subsequent accumulation of these transcripts on free-cytoplasmic and membrane/cytoskeletal-associated ribosomes. In each case, this response occurs after a 5 to 10 minute exposure to hs and is maximal after 1-2h of hs.

Continued exposure to hs or a return of maize seedlings to 25 C (after a 1h hs) results in a decline in both the 70kd and 18kd hs mRNAs associated with these fractions. The kinetics of hs-induction and recovery are reflected in both the in vivo and in vitro (cell-free translation of polyribosomal poly(A)$^+$ RNAs) protein synthesis profiles. Although hybridization studies (using a maize hsp 70 and a maize hsp 18 cDNA) indicate discrete variances in both the time and degree of accumulation of 70kd and 18kd hsp mRNAs between the free-cytoplasmic and membrane/cytoskeletal-associated ribosomal fractions, these variances are not significant. However, the induction and recovery of the 70kd and 18kd hsp mRNAs are non-coordinate.

N.C. Hogan*, D.B. Walden and B.G. Atkinson*
*Department of Zoology

The response of seedlings to a variety of stress-inducing agents

Exposure of maize seedlings to heat-shock induces the synthesis of a set of heat shock polypeptides (MNL 6:111, 1982). Treatment of maize seedlings with a variety of other stressors induces synthesis of a set of polypeptides with Mr's similar to the heat-shock polypeptides (MNL 60:92, 1986; MNL 61:69, 1987). The 18kd polypeptides induced by cadmium chloride treatment and by treatment with the insecticide lannate (methomyl-$C_6H_{10}N_2OS_2$) are recognized by antibodies raised against 18kd heat-shock polypeptides (MNL 61:69, 1987). The similarity in Mr's of the polypeptide sets and the antigenic similarity of the 18kd polypeptides induced by different stressors led to the suggestion that heat-shock polypeptide synthesis is a general response to stress in maize.

Here we report on a comparison of 2D IEF-SDS PAGE profiles of newly synthesized polypeptides from maize seedlings treated with the following stress-inducing agents:

Treatment	Conditions for HSP-like polypeptide induction		Relative amount of radioactivity in HSP-like polypeptides
	Radicles	Plumules	
Control	-	-	-
Heat Shock	42°C	42°C	++++
Cadmium chloride	10^{-2}M	10^{-3}M	+++
Zinc Sulphate	10^{-2}M	10^{-1}M	++
Copper sulphate	ND	10^{-2}M	+
Lead nitrate	ND	10^{-2}M	+
Methomyl (lannate)	0.052M	0.13M	+

(ND - not done)

cadmium chloride, zinc sulphate, copper sulphate, lead nitrate and lannate (methomyl).

Plumules or radicles of intact 5-day-old maize seedlings (Oh43) were immersed in treatment solution for 3 hours. ^{35}S-methionine was added during the last 2 hours of treatment. Extracted proteins were separated by 2 D IEF-SDS-PAGE and fluorograms were compared. The pI's of the polypeptides induced by the different stressors were indistinguishable from those of the heat-shock polypeptides (HSPs). The relative amount of radioactivity in the HSP-like polypeptides varied among different stressors. The concentration of each agent found to induce synthesis of HSP-like polypeptides are given in the table, along with the relative amount of radioactivity in the HSP-like polypeptides in comparison with the amount found following heat shock.

This study is being repeated using the genotype Oh51A. Preliminary results show that the relative amount of radioactivity incorporated into HSP-like polypeptides is significantly higher in Oh51A than in Oh43 following treatment with the insecticide methomyl (lannate).

Carol A.B. Rees and David B. Walden

MADISON, WISCONSIN
Agrigenetics Advanced Science Co.

Agrigenetics maize RFLP linkage map

The Agrigenetics RFLP map is derived from linkage data observed in an A619Ht × Mangelsdorf's tester F2 population consisting of 87 individuals. The Mangelsdorf's tester stock was obtained from the University of Minnesota and A619Ht from Jacques Seed Co. (Prescott, WI). DNA was extracted from greenhouse-grown F2 plants which were scored for the following Mangelsdorf's tester markers: *bm2, lg1, su1, y1, j1, wx1, and g1*. This tester stock did not contain a *gl1* marker on chromosome 7 (D. Weber, pers. commun.) and the *a1* and *pr1* markers were not useful in this cross. We also used three isoenzyme markers (*Pgd2, Enp1, and Mdh1*), several cloned genes for well-known genetic markers (Table 1) and 37 of the Brookhaven National Laboratory markers (courtesy of B.Burr). In the early stages of map development, monosomic stocks (for all chromosomes except 1 and 5) obtained from David Weber were used to confirm the location of some linkage groups.

No differences in marker linear order were detected among 75 probes used to analyze a separate F2 population (B68Ht × B73Htrhm). The r92b, r111b, r118b, r167b, c333b, *Got2* and *Rp1* loci were mapped in other populations. *Rp1* was mapped following field inoculation and scoring of 45 F2 individuals that were segregating for *Rp1*.

Genetic linkage was detected using the method of expected phenotypic categories as described by Mather (The Measurement of Linkage in Heredity, Methuen and Co., London, 1938). Orthogonal coefficients for the 9-cell

Table 1. Identified clones obtained from other researchers.

Identified clones obtained from other researchers.

Cloned gene	Plasmid	Source
P1	P-VB.4	T. Peterson
Adh1	pZML793	E. Dennis
A1	pALC2-2	S. Schwarz-Sommer
Adh2	pZML841	E. Dennis
Sh1	p17.6	L.C. Hannah
Bz1	pD3MS9	O. Nelson
Wx1	pSalC	S. Wessler
Ss2	p21.2	L.C. Hannah
ubi	H76	P. Quail, A. Christensen
β zein	PZG15RX	B. Larkins
γ zein	γZM5	A. Esen

classification were used to determine lack of independent segregation, to examine deviation from Mendelian expectation for each marker, and to test the possibility of genetic linkage. This method reduces the number of inaccurate or physically nonexistent "mathematical" linkages which result from causes other than linkage. The method of maximum likelihood (Allard, Hilgardia 24:235, 1956) was used to calculate recombination frequencies. We have also used a method described by Burr et al. (Theor. Appl. Genet., in press) in which linear order is determined by seeking to minimize crossovers among all pairwise probe comparisons.

Most of our DNA markers are cDNA clones made from two different RNA populations. The first clones, prepared from etiolated coleoptile total poly(A) mRNA are indicated by the prefix "c". These clones were constructed by G-C homopolymer tailing into the *Pst*I site of the vector pSP64 (Promega). A high percentage (about 20%) of these cDNA clones proved unusable due to high background hybridization. Subsequent analysis suggested that excessive G-C tracts were a major cause of the non-specific hybridization. This conclusion was strongly supported when a second seedling root polysomal poly(A) cDNA library (prefix "r") was prepared by A-T tailing and less than 1% of the clones were rejected for reasons of high background. Overall, 38% of the cDNA clones were rejected for reasons of weak signals, high background or complexity. More than 80% of the useful clones were polymorphic with *Eco*RI or *Eco*R5

Table 2. Chromosome locations of duplicate molecular markers.

Chromosomes	Molecular markers
1,4	p83
1,5	c259 c362 r103 r235 r238 r278
1,9	r92 r153 r294
2,2	r85
2,7	c265 r267 c333 r111
2,10	r113 p168 r167
3,8	c514 c568 r50 r116 r144 r184 r274 r209
3,10	r43
4,5	c563 ubi r248
6,9	r118
9,10	c255

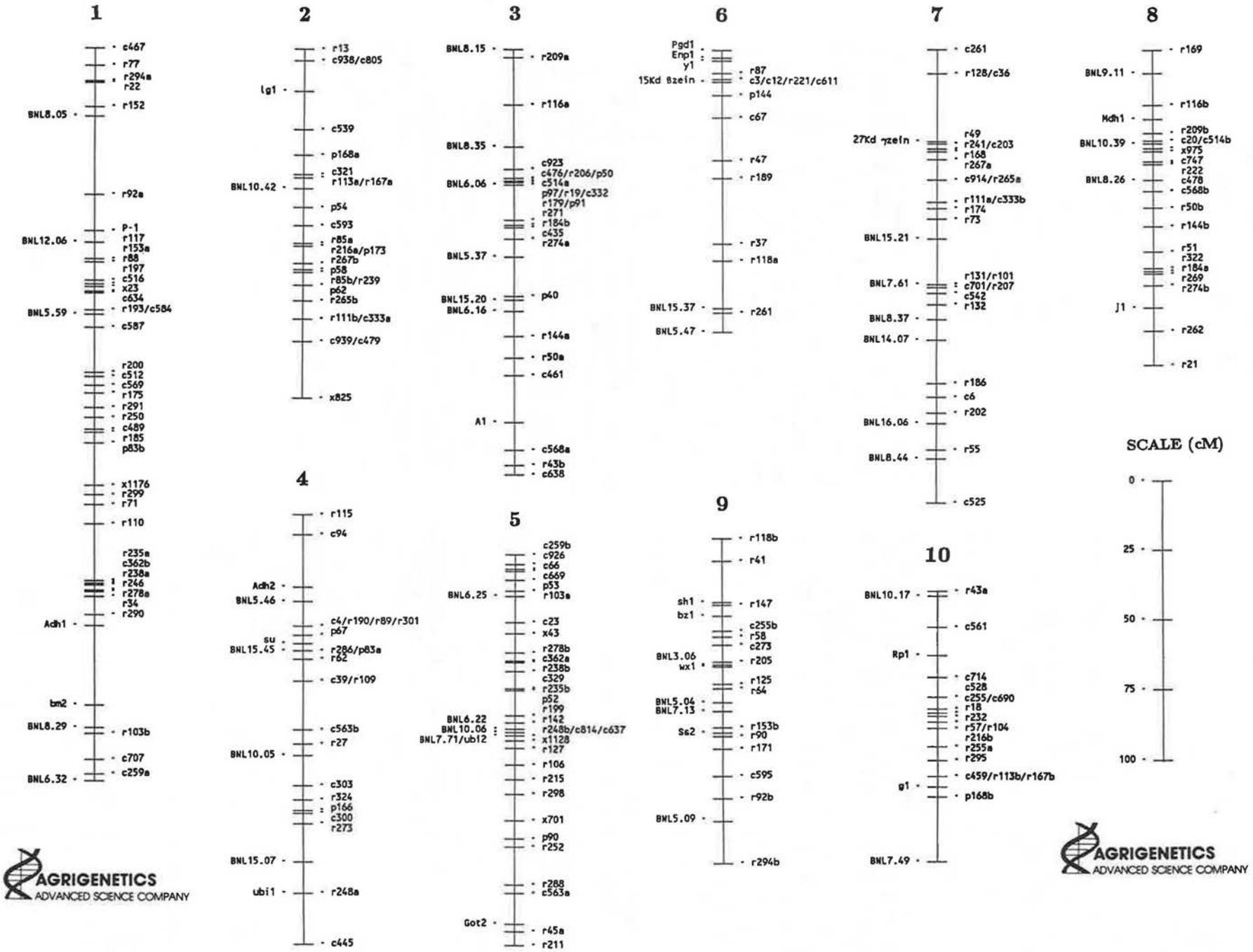


Figure 1. Established genetic markers and clones obtained from other researchers are shown on the left side of each chromosome. Codes are: BNL = Brookhaven National Laboratory; r = root cDNA clones; c = coleoptile; x = *Xho*I genomic clones; p = *Pst*I genomic clones; a or b = loci recognized by duplicated sequences. Independent clones between which no recombination was observed are shown on the same line.

digests, making the use of additional enzymes unnecessary. Single locus clones were preferentially selected, since they facilitate practical utilization of the map. However, our map does include a number of two-locus markers (Table 2) which are designated "a" and "b" on the map (Figure 1).

Our map includes some clones derived by exploiting the apparent undermethylation of single copy sequences (Burr et al., Theor. Appl. Genet., in press). An initial attempt using 1-2 Kbp *Xho*I fragments cloned into a vector *Sal*I site (prefix "x") gave only a 10-fold enrichment for single copy sequences. Subsequent libraries using 0.5-3 Kbp *Pst*I fragments (prefix "p") were much more efficient and about 70% of these clones were mappable.

Several new genes of known identity have also been positioned on the map (Table 1, Figure 1). A 15 Kd β zein gene obtained from Brian Larkins has been mapped to chromosome 6. A 27 Kd γ zein obtained from Asim Esen has been mapped to chromosome 7. A ubiquitin sequence (H76) obtained from Al Christensen and Peter Quail hybridizes to two loci (*ubi1* and *ubi2* on chromosomes 4 and 5 respectively). Clone r248 also proved to be a ubiquitin sequence by cross-hybridization.

M. Murray, J. Cramer, Y. Ma, D. West,
J. Romero-Severson, J. Pitas, S. DeMars,
L. Vilbrandt, J. Kirshman,
R. McLeester, J. Schilz and J. Lotzer

MEADVILLE, PENNSYLVANIA
Allegheny College
LONDON, ONTARIO, CANADA
University of Western Ontario

Effects of inhibitors and uncouplers in the synthesis of 52kD heat shock protein by mitochondria

Corn seedling mitochondria respond to a 10-degree increase in incubation temperature by in organello synthesis of a 52kD protein (Nebiolo and White, Plant Phys. 79:1129, 1985). Mitochondrial translation of this protein is insensitive to cycloheximide and erythromycin and sensitive to chloramphenicol (MNL 61:73, 1987). Sodium arsenite, an inducer of the heat shock response in intact seedlings, induces synthesis of the 52kD protein at concentrations greater than 50μM (Nebiolo and Walden, J. Cell Biol. 79:258a, 1986). In our hands, seedling mitochondria purified on both sucrose and Percoll gradients are free of bacterial contamination, measured by plate counts of mitochondrial pellets and minimal protein synthesis in the presence of acetate as carbon source, and synthesize the 52kD protein only when "stressed" by temperature or the mitochondrial uncoupler arsenite.

To extend our investigation of this mitochondrial response to stress, we treated purified seedling mitochondria isolated under sterile conditions with various inhibitors and uncouplers of mitochondrial function. We measured 35S-methionine incorporation under optimum conditions for detecting only mitochondrial protein synthesis: succinate/ADP as energy source, erythromycin and cycloheximide to inhibit plastid and 80S ribosome translation respectively, and vigorous shaking to maintain sufficient oxygen concentrations for efficient electron transport.

The general protonophoric uncoupler 2,4-dinitrophenol inhibits 35S-methionine incorporation by mitochondria by 65-70% at concentrations of 10μM but has no apparent effect at 50 and 100μM. No change was observed in protein profiles; the 52kD protein was not induced at any concentration. The uncoupler valinomycin, which acts as a mobile ion carrier of K^+, also had no effect on incorporation or on protein profiles.

The mitochondrial inhibitor rotenone had no effect on mitochondrial translation at treatments of 10, 25 and 100μM rotenone. Similar results were found using 10-100μM KCN, a potent mitochondrial electron transport inhibitor.

Methomyl, a carbamate insecticide, uncouples corn seedling mitochondria of T male sterile cytoplasm at concentrations as low as 4mM (Klein and Koeppe, Plant Phys. 77:912, 1985). Lannate, a commercial product with methomyl as the active ingredient, induces heat shock protein synthesis in corn seedlings of both T and N cytoplasm at concentrations as low as 20mM (MNL 61:69, 1987). We treated purified mitochondria from T and N seedlings with various concentrations of lannate and methanol, the solvent in which methomyl is dissolved to produce lannate.

At concentrations of 13 and 26mM lannate inhibits 35S-methionine incorporation by N mitochondria by 35% while showing no significant effect on T mitochondria at the same concentrations. At lower concentrations (1.3 and 2.6mM lannate) T mitochondria incorporation is inhibited while N mitochondria are not affected. This may imply 2 different mechanisms of action of lannate on mitochondria, one toxic effect to which N mitochondria are more susceptible and a second effect at lower concentrations, approximately those which uncouple the mitochondria, which affect T cytoplasm differentially.

Methanol at concentrations found in lannate treatments slightly inhibits incorporation of both N and T mitochondria.

We are currently investigating alterations of in organello translation products by lannate and methanol in both T and N seedling mitochondria, specifically looking for induction of the 52kD protein produced in response to heat and arsenite "stress".

David B. Walden and Christine M. Nebiolo

MILAN, ITALY
University of Milan
Istituto Biosintesi Vegetali, C.N.R.

Further results on *dek1* embryo-lethal mutant

The embryo-lethal mutant *dek1* shows pleiotropism, since, in addition to the block of embryogenesis at the proembryo stage, the endosperm is white, being devoid of carotenoids and anthocyanins, while the aleurone layer is absent. With the aim of better characterizing this mutant, two nuclear parameters, DNA replication and DNA content, were analyzed and the data obtained submitted for publication.

The capacity of mutant embryonic cells to actively synthesize DNA, in spite of the arrest in differentiation, was tested by determining the ^{3}H-thymidine incorporation in 14 DAP kernels. Normal and mutant sib kernels were cultured for 48 h in sterile MS liquid medium supplemented with 30μCi/ml ^{3}H-thymidine (specific activity 51μCi/mmol) or alternatively injected with 5μCi ^{3}H-thymidine

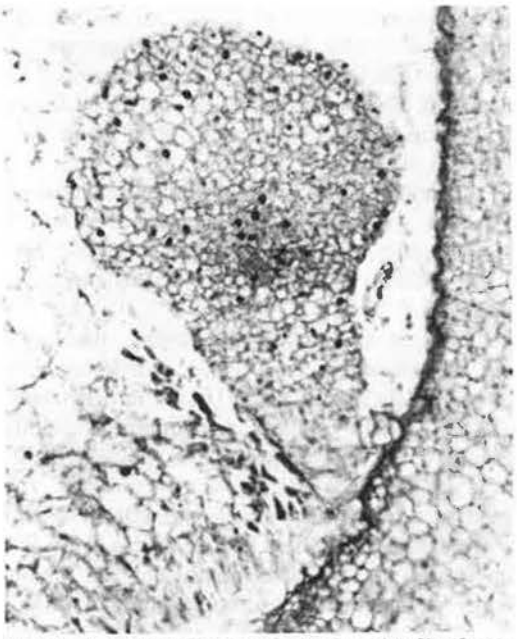


Figure 1. Autoradiograph of a longitudinal section of a 14 DAP mutant embryo after incubation in MS medium supplemented with ^{3}H-thymidine (30μCi/ml) for 48 h. Labelled nuclei appear as black dots over cells. ×135

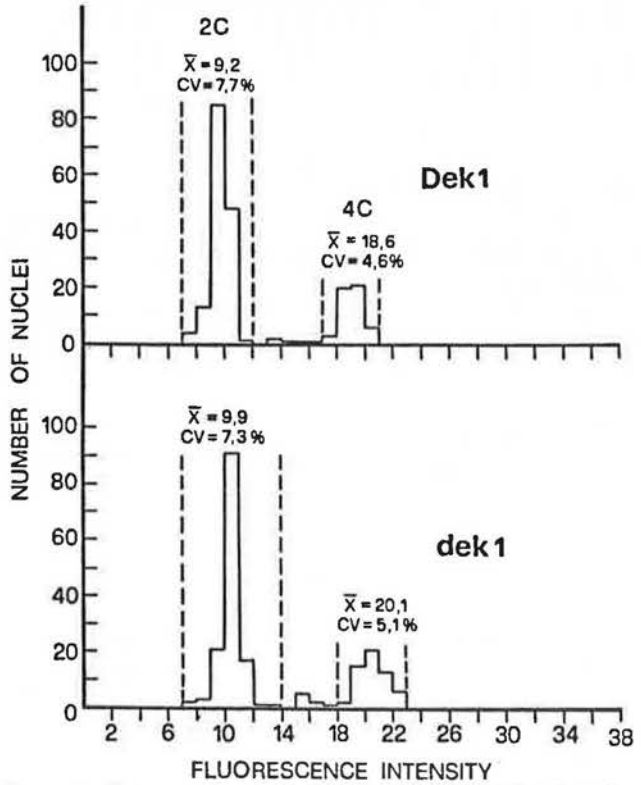


Figure 2. Fluorescence distribution of DAPI-stained nuclei isolated from normal *Dek1* (n = 206) and mutant *dek1* (n = 202) 16 DAP sib embryos.

and then cultured in MS medium for 48 h. Both procedures adopted gave positive results. Autoradiographic silver grains were preferentially located over nuclei of the central differentiated area in the mutant embryo, even though incorporation is also observed in nuclei located in the peripheral region (Fig. 1). Cell division capacity therefore is not affected by the mutation, as is also shown by the possibility of inducing callus from mutant embryos.

On the other hand, the increase in nucleolus and nucleus size observed both in embryo and in endosperm mutant cells (MNL 61:73, 1987) raised the question of whether larger nuclei might be the result of a higher DNA content and larger nucleoli of a consequently larger number of transcribed rRNA genes. DNA quantification by means of cytofluorimetry was therefore performed on nuclei isolated from normal and mutant embryonic cells at 16 DAP. The overlapping of the two fluorescence distributions of DAPI-stained nuclei (Fig. 2) leads one to exclude an increase in DNA content in mutant cells. Further autoradiographic experiments with ^{3}H-thymidine and ^{3}H-uridine incorporation are in progress.

Silvana Faccio Dolfini, Francesca Sparvoli
and Hélène Libert

Possible amplification of a *Zp* gene on the long arm of chromosome 10

It has been previously reported that a gene encoding for a particular zein polypeptide with a M.W. of 22Kd and focusing in the standard IEF analysis in position 2 (*Zp22/2*) was located on chromosome 10 closely linked to *O7* (Binelli et al., Plant Sci. Lett. 33:259, 1984). Pursuing our studies at the *R* locus, we decided to use *Zp22/2* (for brevity, *Zp*, capitals standing for the presence of the band and lower case for the absence) as a marker in our stocks. From the backcross *R-st Zp/r zp* × *R-st zp* (where the *R-st Zp* chromosome is derived from a trisomic *R-st R-nj r* while the *R-st zp* chromosome is in W22 background) we observed the following phenotypes:

dark st *Zp*:111	(endosperm genotype	*R-st*	*Zp/R-st*	*Zp/R-st*	*zp*)
light st *zp*:103	(endosperm genotype	*r*	*zp/r*	*zp/R-st*	*zp*)
dark st *zp*: 0	(endosperm genotype	*R-st*	*zp/R-st*	*zp/R-st*	*zp*)
light st *Zp*: 6	(endosperm genotype	*r*	*Zp/r*	*Zp/R-st*	*zp*)

These results are unexpected for two reasons: the number of recombinants is much lower than that expected on the basis of a close linkage between *Zp* and *O7*. In fact, assuming a distance of 26 cM between *R* and *O7*, the recombinants between *R* and *Zp* should be about 57. In addition, only one class of crossovers is found, namely the light st and *Zp*, while the reciprocal class, dark st and *zp*, is absent.

Two recombinant seeds (presumed genotype *r Zp/R-st zp*) were further analyzed. The plants were crossed to *r zp* and the progenies classified for *R* and *Zp* phenotypes. Results were as follows: st *zp* 77; *r Zp* 80; st *Zp* 3; *r zp* 0. Again, the frequency of recombination between *R* and *Zp* appears much lower than expected and only the recombinant class showing the presence of the *Zp* band is found. These results can be explained if in the *R-st Zp* chromosome (extracted from a trisomic stock provided by the Co-op) an amplified region of the chromosomal segment bearing *Zp* is present. The gene can be tandemly duplicated and mispairing with the homologous chromosome can occur. If this is the case, we expect two consequences: reduction of crossing over frequency between the two homologues due to structural heterozygosity; crossover events within the amplified region leading to strands with different copy number of *Zp*. As a consequence, st *zp* phenotypes cannot be recognized since the *zp* alleles are masked by the presence in the same strand of additional *Zp* copies.

Mariangela Menghetti, Carlo Soave[1]
and Giuseppe Gavazzi
[1]Istituto Biologia Agraria, Univ. Basilicata, Potenza

Structural DNA polymorphism of different *Sn* geographic alleles

The *Sn* locus conditions anthocyanin accumulation in specific plant and seed tissues. The *Sn* accessions we have been studying map on the long arm of chromosome 10 about 2 cM *R* distal and they differ in their rate of accumulation and in the quantity of pigment. Since they were originally present in different populations, they will be referred to as geographic alleles. Their origin and phenotypes are reported in Table 1. Their characterization

Table 1. Description of the *Sn* accessions analyzed. The signs + and − indicate presence and absence of pigmentation while (+) refers to weak pigmentation (sct:scutellum; al:aleurone; sn:scutellar node; msc:mesocotyl; plant:leaf basis and midrib, pericarp).

Symbol	Origin	tissue specificity				msc pigmentation(1)
		sct	al	msc	plant	(A530/msc)
Sn:bol1	bolivian population	−	−	+	+	1.01
Sn:bol2	bolivian population	−	−	+	+	1.27
Sn:bol3	bolivian population	−	−	+	+	6.43
Sn:Co-op	Co-op (Inv9a)	−	−	+	+	1.22
Sn:Stadler	R-ch:Stadler	−	−	+	+	0.58
Sn:hopi (2)	r-ch:hopi	+	(+)	(+)	+	0.38

(1) Anthocyanin content as determined after 96 hours of white light irradiation

(2) Sn presence inferred, since no evidence of recombinational separation of Sn from R has been so far obtained

at the molecular level could provide information useful in understanding the basis of the observed differences in expression. As reported in this MNL, *Sn:bol3* cross-hybridizes to a fragment (pR-nj:1) cloned from *R-nj*. Genomic DNA from plants homozygous for *r Sn:bol1*, *r Sn:bol2*, *r Sn:bol3*, *r Sn:Coop*, *R Sn:Stadler* and *r Sn:Hopi* were digested with *SstI* and analyzed by Southern blot. The pR-nj:1 probe detects *Sst*I fragments of different size in all genotypes tested. The *Sn:bol1* hybridization pattern is the same as that of *Sn:bol3*, thus showing a complex gene structure, while the *Sn:bol2* pattern appears less complex. The other three alleles show a high structural polymorphism indicating that they differ both from *Sn:Bol3* as well as from each other. These preliminary results indicate that the *Sn* alleles tested are related in terms of DNA homology because they all cross-hybridize but appear unrelated in terms of structural DNA polymorphism thus proving to be distinct alleles.

C. Tonelli, G. Consonni, S. Dellaporta[1]
and G. Gavazzi
[1]Yale University

Response of chlorophyll mutants to cytokinins

In plants cytokinins stimulate plastid differentiation and related processes like chlorophyll and carotenoid biosynthesis. It might well be that some of the "chlorophyll" mutants affected in their pigment content are due to reduction in endogenous cytokinin content. If so they should respond to exogenous hormone subministration by greening. Accordingly the response of several chlorophyll mutants was tested by applying a few drops of N6(benzyl) adenine (BA). Out of 25 mutants screened five (*lw1, l15, lc4, lc5, l11*) seemed to give some positive response. They were thus further analyzed. BA treatment in this case was done in two ways: 1) by germinating seeds in flasks containing 1μM BA on a rotary shaker for 48 hrs and 2) by applying a 20 μl drop of 100 μM BA on the scutellar node when the shoot was about 1 cm long. The effect of the treatment was measured by establishing the pigment content of the first two leaves of young seedlings (third leaf not yet expanded). The only mutant showing a significant increase in pigments following cytokinin treatment is *l11*. In the mutant treated with BA the PEPcarboxylase activity is restored to the level of the untreated control, thus suggesting that BA treatment leads to a restoration of photosynthetic functions. Correlated to this repair of BA on pigment levels and enzyme activity is the effect of this cytokinin on the mesophyll plastid development as determined from ultrastructural studies.

C. Tonnelli, C. Salvi, G. Gavazzi,
G. Marziani, G. Rossi and C. Longo

Is DNA modification involved in *Sn* instability?

We are trying to understand the basis of the instability of *Sn:bol3*, one of the *Sn* geographic alleles (MNL, 1988). In fact the progeny of individuals selected for strong red scutellar node and mesocotyl (referred to as *Sn-s*) consist of seedlings with parental phenotype as well as seedlings with significantly lower levels of pigmentation (referred to as *Sn-w*) and arising with a high frequency (from 20 up to 60% of the total progeny). *Sn-s* selections keep showing instability in succeeding generations while *Sn-w* selections never revert to the original phenotype.

Southern analysis of genomic DNA, digested with *Sst*I and *Hin*dIII and probed with the 600 bp *R-nj* fragment (as reported in MNL 1988), shows the same hybridization pattern in *Sn-s* and *Sn-w*, thus suggesting that no large DNA rearrangements are involved in this event.

Therefore we postulated that *Sn:bol3* instability could be correlated to DNA modification. Accordingly we treated *Sn-w* derivatives with the deoxycytidine analog 5-aza-2′-deoxycytidine (Aza) in order to see if the analog is capable of inducing a phenotypic reversion of *Sn-w* to *Sn-s*. Homozygous *Sn-s* and *Sn-w* seeds were germinated in darkness in the presence of the analog. Five days after germination they were exposed to light for 4 hours and the anthocyanin content of the scutellar node was then determined. The results obtained (Table 1) show striking differences in the level of pigmentation. The *Sn-w* derivatives treated with Aza show a dramatic increase in their pigment content, about 70 fold increase, when compared to the untreated ones. The effect of the analog is evident also in homozygous *Sn-s* seedlings, where a 4-5 fold

Table 1. Effect of 5-aza-2′ deoxycytidine (aza) on anthocyanin accumulation in scutellar node of *Sn-s* and *Sn-w* strains.

	Treatment	A_{530}/g. Fw	A_{530}/scutellar node
Sn-s	H_2O	8.15	0.407
	aza	33.10	1.930
Sn-w	H_2O	0.35	0.008
	aza	22.70	0.665

- Anthocyanin content expressed as A_{530} per gram of fresh weight (g.Fw) and per organ.
- Each treatment consists of 15-20 seeds.

increase in pigment content is observed. Furthermore a comparison between the values obtained in the treated *Sn-w* versus untreated *Sn-s* tissues show that *Sn-w* derivatives not only revert to the original phenotype but show a higher pigment content. These results thus suggest an involvement of DNA modification in *Sn* instability. Preliminary results of Southern blot analysis of genomic DNA from treated and untreated plants, digested with methyl-sensitive enzymes, seem to confirm this view.

C. Tonelli, G. Consonni, G. Gavazzi and A. Viotti
[1]Instituto Biosintesi Vegetali, C.N.R.

Sn is similar to *R* at the DNA level

Both *R* and *Sn* control the distribution of anthocyanins in plant and seed tissues. However, they differ in regard to their map position (*Sn* lying 2cM distal to *R*) as well as in their tissue-specific expression. In fact while some tissues such as anthers and roots represent a common domain of expression of both *R* and *Sn*, others like scutellar node, mesocotyl, leaf base, midrib, seed glumes and pericarp are exclusive of *Sn* expression.

Several *Sn* geographic alleles have been studied. One of them, symbolized *Sn:bol3*, differs from the others in its potential for pigment accumulation in the seedling tissues (at least five times greater than in others) and in its light independent pigmentation of seed integuments. *Sn:bol3* is unstable giving rise to a series of derivatives with a lower level of pigmentation.

The molecular cloning of the *Sn* gene is of particular interest in order to answer specific questions concerning its structure and the regulation of its expression. Since the product of the gene is unknown, an indirect approach to cloning *Sn* is necessary. Therefore the approach we adopted is based on the idea of a molecular similarity between *Sn* and *R*, cloned by S. Dellaporta. A 600 bp *Hin*cII - *BGl*II fragment (pR-nj:1) isolated from the *R-nj* allele cross-hybridizes to *Sn:bol3*. *Sst*I-digested DNA from plants homozygous for *r Sn:bol3* was analyzed by Southern blot. pR-nj:1 DNA hybridizes to three *Sst*I fragments of *Sn:bol3*. This pattern is similar to the hybridization pattern observed with *R-r* in which molecular analysis discloses the presence of three components. If a similar structure exists for *Sn* it is possible that these two genes are related in the sense that both derived from a common progenitor containing a triplicate structure. This finding should open the way to an analysis of the basis of the tissue specificity of *Sn*, its response to light, and the molecular events leading to *Sn:bol3* instability.

C. Tonelli, G. Consonni, G. Gavazzi and S. Dellaporta[1]
[1]Yale University

Ubiquity of *Mu* sequences in *Zea* taxa and other cereal species

The presence of sequences homologous to the internal fragment of the *Mu* transposable element in B chromosomes of Black Mexican lines has been already reported (MNL 61:77, 1987). In situ hybridization experiments with the entire *Mu-1* element confirm the localization of these sequences only in the two heterochromatic blocks adjacent to the proximal euchromatic region of the long arm of the supernumerary chromosome.

A parallel investigation by Southern blot analysis of genomic DNA from the Black Mexican lines with or without B chromosomes (never visited or having lost them) of different year harvests has been carried out. The DNAs, digested with several restriction enzymes (*Hin*fI, *Eco*RI, *Hin*dIII, *Xba*I) and probed with the internal fragment, denote the occurrence of three (*Mu-L, Mu-1, Mu-S1*, according to Walbot, MNL 59:96, 1985) elements of the *Mu* family and equal patterns of hybridizing bands, also independent of the year harvested, in the three Black Mexican lines. However, comparison of the *Eco*RI digestions probed with the entire element show among several hybridizing fragments the absence of a 4.2 Kb band in the Black Mexican line never visited by the B chromosome. The overall results could support the idea that B chromosomes may derive from a modified heterochromatinized A chromosome.

Southern blot investigation has been extended to other distantly or closely related cereals (respectively wheat, rye, barley, triticale or teosintes, sorghum, coix) that show the presence of partially homologous sequences to both the internal or entire *Mu-1* element. This rather surprising result could have interesting application in gene tagging provided that such sequences have maintained transposable activity.

C. Patrosso, L. Bernard, N.E. Pogna[1] and A. Viotti
[1]Instituto Sperimentale Cerealicoltura, S. Angelo Lodigiano, Italy

HSPs : more data on genetic variability

Last year we reported evidence of HSP variability in a limited sample of maize genotypes. The screening has been continued on a larger scale. In vivo-synthesized proteins (control and heat-shock) from 35 unrelated inbred lines and some F1's were analyzed by 1D SDS PAGE in a linear 5-20% gradient gel. The results allow maize HSPs to be divided into two major groups.

The first group includes HSPs expressed by the vast majority of the lines examined, namely HSPs 240, 94, 84, 82, 72, 70, 27, 22, and 18kD in molecular weight. This set, which includes exclusively high (over 70kD) and low (under 30kD) polypeptides, corresponds to HSPs generally reported in the literature, except for HSP 240kD which, to our knowledge, was never detected before. Genetic variability in this group is limited. HSPs 240, 72 and 18kD were invariant and mainly quantitative differences were detected for the others. Nearly 70% of the inbreds analyzed showed this "standard" pattern with only minor variations (at most 1 or 2 additional bands).

To the second group belong HSPs expressed only in a small number of genotypes. With the exception of HSPs 93 and 91kD (detected in 1 and 2 inbreds respectively), all HSPs of this group are in the medium (60-40kD) and low (34-17kD) molecular weight range. HSPs 52kD (9 inbreds), 44kD (12 inbreds) and 40kD (11 inbreds) were the most frequently found in this survey. Four inbreds showed a prominent 17kD HSP in addition to the standard 18kD. Analysis of F1's for this and other HSPs showed an additivity pattern.

Few lines exhibited a large variation in HSP pattern -4 to 6 different bands compared to standard lines. However, until now precise data are available with regard to in vivo thermotolerance of the inbreds considered. Analysis of this important aspect is in progress.

Carla Frova and Giorgio Binelli

Selection of viable male gametophytic mutants

A better understanding of the genetic control of the male gametophytic phase can be efficiently obtained on the basis of gametophytic mutants for pollen development within the anthers and for pollen function, i.e. pollen germination and tube growth.

We adopted an indirect approach to select viable mutants affecting the male gametophytic generation in maize, based on the observation that components of pollen development and function show positive correlation with endosperm development (Mulcahy, Science 171:1155, 1971, Ottaviano et al. T.A.G.63:249, 1982) and that some alleles determining defective endosperm in maize have been found to be expressed in the male gametophyte (Jones, 1928). Thus we selected endosperm defective mutants (*de*) and tested them for gametophytic gene expression studying the distortion from the expected Mendelian segregation.

Heterozygous plants were selfed and the resulting F2 ears scored for *de* and normal phenotypes. In order to study the effect of *de* alleles on pollen tube growth rate the F2 ears were divided into 3 sectors of equal length to obtain the proportion of *de* kernels in the top, middle and base sectors. Heterogeneity between sectors was interpreted as due to differential tube growth rate between pollen carrying the *de* allele and pollen carrying the normal allele. Thirty-four endosperm defective viable mutants, introgressed in B37 genetic background, that complementation tests revealed affected different genes controlling endosperm development, were analyzed.

The study of the segregation in F2 revealed 4 classes of *de* mutants: 1) mutants in which the mutation does not affect either gametophytic development or function; 2) mutants in which the effect on the gametophyte regards pollen development processes; 3) mutants showing effects on both pollen development and function; and 4) mutants where only pollen tube growth rate is affected. Some of the mutants included in class 2) and 3) showed also reduced pollen size and increased pollen sterility.

Positive and negative interactions between pollen and style were detected by means of mixed pollination (pollen produced by *de/de* plants and pollen from an inbred line used as a standard and carrying genes for colored aleurone) on *de/de* and *de/+* plants. Positive interactions were interpreted as metabolic complementation between defective pollen and normal styles.

We are currently involved in the characterization of some of those mutants at the biochemical level.

M. Enrico Pé and Ercole Ottaviano

Suspension cultures from embryogenic callus

In the aim of developing cell suspension cultures maintaining morphogenic competence, with a potential use in obtaining protoplasts capable of plant regeneration (K.K. Kamo et al., Planta 172:245, 1987), we are studying the conditions to grow liquid cultures from callus derived from genetic stocks with good embryogenic potential (A188, W64A, Mo17 × B73, A188 × B73, W64A × A188).

Seeds were a kind gift from the Istituto Sperimentale per la Cerealicoltura, Bergamo, and the Dipartimento di Genetica e di Biologia dei Microorganismi, University of Milano. Plant-regenerating callus cultures were initiated both from immature embryos (1mm long) and from the nodal region of mature embryos separated from ripe seeds, on agar-containing MSE medium (MS salts + 25mM proline + 20g/l sucrose + 1mg/l 2,4-D), modified from Armstrong and Green (Planta 164:207, 1985). After subculturing every 2 weeks on the same medium for 2 months, the faster growing and more friable calli were transferred (1g fresh weight/30ml) to liquid MSE medium and incubated at 27 C on a rotary shaker. The medium was changed every week; cell release was evaluated after 1 month.

No appreciable release of cells was observed from morphogenic calli derived from mature embryos, which presented a compact structure which was not affected by different additions of 2,4-D to the culture medium; calli from immature embryos from all the genotypes examined, on the contrary, easily released cells which could be cultivated. The influence of 2,4-D on cell release was examined in detail on the two inbred lines: while for A188 increasing concentrations of the auxin (1, 1.5, 3mg/l) appear to increase cell release to liquid medium (from 0.9 to 1.5ml packed cells per 1g callus), for W64A the effect is reduced (the stimulation was from 0.8 to 1ml packed cells/culture).

Growth curves of cells in suspension cultures were determined on 15ml cultures, containing at the beginning 1ml packed cells: the growth was not very enhanced (2 fold increase after 1 week) and the plateau was reached after 2 weeks. Experiments are in progress to optimize conditions for cell release and suspension growth; the concentration of 2,4-D seems not to affect either the cell growth or the secretion of a thick white substance, which has been described to be secreted by maize liquid cultures (Kamo et al., 1987; J.A. Miernyk, J. Plant Physiol. 129:19, 1987).

Suspension cultures from embryogenic callus of all the genotypes examined are composed of small, round-shaped cells, which tend to aggregate in small clusters; these features are the same as those described by Kamo and Hodges (Plant Sci. 45:111, 1986) for embryogenic maize suspensions, and are characteristically different from the aspect of non-regenerating suspensions from root callus (single big elongated cells) obtained from the same genotypes.

Silvana Castelli and Lucia A. Manzocchi

Pl effect on young seedlings

Stocks segregating for *Pl*, collected from different sources and selfed for several generations, yield plants differing in their anther pigmentation and easily classifiable into 2 groups, strong and weak red anther colour. We presume this effect is due to *Pl*. From each stock we derived two sublines breeding true for strong and weak red anther colour respectively. Seedlings of the former develop an intense red pigmentation in their roots even in darkness, while those of the latter turn red only after light exposure. Their pigment content, however, remains much lower than that of their sibling sublines. One *Pl* source (originally from Dr. Brink) differs from the others in conditioning a very weak root pigmentation while maintaining the intensive anther coloration, suggesting the existence of *Pl* alleles that differ in their tissue specificity of expression. An example of the effect of different *Pl* sources on root pigmentation is given in the table below where results referring to a weak and strong *Pl* source are given.

Pl Source	*Pl* Constitution	Root pigment A 530/g f.wt.
Brink	*Pl/Pl*	2.3
Brink	*pl/pl*	0.7
Co-op (l*-4120)	*Pl/Pl*	58.9
Co-op (l*-4120)	*pl/pl*	2.7

Two effects should be noted: 1) within each *Pl* source the *Pl* subline develops more pigment than the *pl/pl* subline; 2) the intensifying *Pl* over *pl* effect is more than 20 fold for *Pl* (Co-op) but only 3.3 for *Pl* (Brink).

By crossing two *Pl* stocks differing in their colour marker constitution, i.e. *A1 a2 C1 C2 R-g Pl* (from Dr. Coe) with *A1 A2 c1 C2 r-r Pl* (from Dr. Phillips, in A188 background) we obtained, upon 2 successive rounds of selfing, colourless seeds, *r-r/r-r Pl/Pl A2/a2* in genotype. Their progeny, obtained by selfing, yield seedlings with red and nonred roots in a 3:1 rato. The one-quarter of seedlings devoid of anthocyanin (*r-r/r-r a2/a2 Pl/Pl*) exhibit an unusual yellow pigmentation in their roots. Chromatographic results on hydrolyzed extracts of such roots indicate that the major component present in the extracts is quercetin. This compound is not found in coleoptiles of the same seedlings. Since *a2* is blocking one step of the conversion of dihydroflavonol to anthocyanins, it might well be that conversion of dihydroflavonols to flavonols is enhanced by the presence of *Pl*, leading to an accumulation of flavonols in roots, an event generally not observed.

G. Gavazzi, M.L. Racchi and G. Todesco

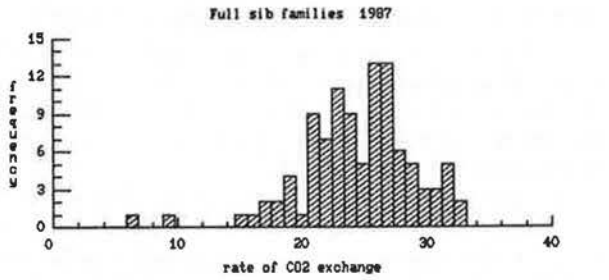
A new allele of *vp5* isolated in the progeny of regenerated plants

In last year's News Letter, we presented the first observations on nonparental phenotypes in selfed progeny of regenerated plants. In particular we reported the isolation of a viviparous mutant with white seedling. This mutant proved to be allelic to *vp5* on chromosome 1. Evidence of allelism was obtained by crossing the selfed progeny with the progeny of plants segregating for *vp5*. Out of 7 such crosses 2 ears were in fact segregating for the viviparous phenotype thus confirming the allelism of this mutant with *vp5*.

M.L. Racchi, M. Pontoglio and G. Todesco

Measurement of CO_2 assimilation in full sib families

Rate of photosynthesis seems to be a significiant component of yield at least in restrictive environmental conditions, and it could be used as an additional parameter of selection. More data, however, are needed. We have analyzed the variability of this physiological trait in a set of 104 full sib families extracted from a synthetic population developed for second cropping in North Italian climate. Measurements were made at flowering time on single leaves of 6 plants per plot with 2 replications of a completely randomized block design. The portable system of ADC Ltd. (Hoddesdon-England) (consisting of an IRGA, a leaf cuvette, an air supply unit and a data logger) was used. Photosynthesis rate was estimated as CO2 exchange rate (μ mol m^{-2} s^{-1}) in an open system. Data range from 5.93 to 32.77 with an average value of 24.498. As regards the genetic component of the trait the estimate of intraclass correlation coefficient was 0.43. The frequency distribution of the family mean values is reported in the Figure.



These preliminary data seem encouraging to start a two way intrapopulation selection procedure in order to assess the correlated response to selection of the main morphological and physiological components of yield in environmentally restrictive conditions.

A. Camussi, F. Stefanini and M. Bozza

Preliminary analysis of a housekeeping gene

The 250bp cDNA clone (PCM4), previously isolated from a cDNA library of polyA-RNA of maize endosperm polysomes, shown to be localized by in situ hybridization on the long arm of chromosome 5 and represented in endosperm polyA-RNA by 4 RNA species ranging from 1.5 to 4.5Kb in length, has been further characterized. In cDNA libraries from various maize tissues, M4 related clones represent about 1-3% of the total recombinant phages and their abundance in the endosperm cDNA library is comparable to that of zein sequences.

The M4 sequence does not show significant homology either to maize storage protein genes or to other published maize sequences. Southern blot analysis showed that the genomic region containing the M4 sequence is highly conserved into 2 unrelated maize lines, W64A and A69Y. The resulting hybridization pattern of the genomic DNA digested with several restriction enzymes (*Eco*RI, *Xba*I, *Bam*HI, *Hin*dIII, *Eco*RV) is exactly the same in these two maize lines. Other organisms have been analyzed for the presence of M4 related sequences; in particular, some related plants (teosinte, oat, barley, wheat), some dicots (petunia, bean, tobacco), and some unrelated organisms like yeast, rat and man. Their DNAs have been digested with *Eco*RI and *Hin*dIII and hybridized with the M4 fragment. The *Eco*RI digestion gives rise to a single hybridization band (about 8Kb in the monocots and about 3.5Kb in the dicots) while *Hin*dIII digestion results in 2 bands (of similar molecular weight in monocots, different weight in dicots) as a *Hin*dIII site is contained in the probe. No hybridization signals were detectable in yeast, man and rat. Copy number evaluation suggests the presence of 1-2 copies per maize haploid genome.

Northern blot analysis of either total or polyA-RNA from different maize tissues reveals the existence in all the RNA investigated of the 4 M4 transcripts previously shown in endosperm RNA. The relative abundance of each of the 4 RNA molecules is different in the various tissues in which one or two of the transcripts are barely detectable. A similar transcript pattern is also present in petunia and in bean tissues. The transcripts are identical in size in the two dicots but different from the maize M4-related RNAs. Studies are in progress to elucidate the tissue-specific regulation of the 4 transcripts, and genomic clones containing the M4 sequence are under characterization.

F. Quattrocchio, F. Sparvoli and A. Viotti

NEW HAVEN, CONNECTICUT
Yale University
STORRS, CONNECTICUT
University of Connecticut

How far does *Ac* jump on average? "About one foot" (34 cm)!

In order to understand the mechanism of *Ac* transposition, it is important to know the pattern of transposition. Greenblatt (Genetics 58:585, 1968) selected kernels from either twinned or untwinned red multikernel sectors derived from medium variegated ears (genotype: *P-VV/P-WR*). The red sectors are the result of a somatic transposition of *Ac* from *P-VV*. Greenblatt reported that from a total of 168 red sectors the average recombination distance of *tr-Ac* from the *P* locus is 34 CM (centi-morgan). In the course of isolation of *Ac* induced mutants at *P*, we collected *tr-Ac* from the *P* locus using a different selective screen, thus allowing a comparison of the effect of seed selection methods on the measured recombination results.

From crosses of *P-VV/P-WR, r/r* × *P-WR/P-WR, r-m3/r-m3* (*r-m3* is a *Ds* suppressed *R-sc* allele, isolated by Dr. Kermicle and here used as an indicator of *Ac* activity), we selected kernels on these medium variegated ears with the phenotype of red pericarp and variegated colored aleurone. The genotype of these kernels will be either *P-RR tr-Ac/P-WR* or *P-WR tr-Ac/P-WR* if the pericarp and the pollen mother cell share the same cell lineage. Otherwise, in cases where the pericarp and the female gametes are discordant, medium variegated will be expected along with 7 percent light variegated. The frequency of *tr-Ac* red vs. *tr-Ac P-WR* provides a direct measure of *tr-Ac P* recombination frequency and is comparable to red sector mapping results. Plants from all kernels were self pollinated (mating of course does not affect pericarp phenotype in this generation).

The seed selections yielded the following data:

312 ears with red pericarp
161 ears with colorless pericarp
1 ear with light variegated pericarp
17 ears with medium variegated pericarp

All ears showed *R-sc* mutability of the *r-m3* allele and hence had *tr-Ac* activity. The recombination frequency is 34% (161/(312+161). These results are indistinguishable from those of Greenblatt (1968), confirming an average length of transposition of *Ac* at 34 CM. While the two selection methods yield the same results, the use of the *r-m3* method is much the more efficient in terms of transpositions recordable per ear as well as the work required for confirmation of progeny genotypes.

Among the realized 312 red ears more than 10 percent (46) exhibited somatic instability of the red pericarp somatic lineages. We find this a high rate based on prior observations made by Greenblatt (unpublished) of this phenotypic class. Referring to the reports of Orton and Brink (Genetics 53:7, 1966), many of these unstable red cases illustrate the subsequent return of the *tr-Ac* back to the *P* locus. The somatic loss of pericarp pigment might also be due to *P* inactivation by other transposable element systems. This latter possibility raises the issue of the interactive behavior of different transposable elements and/or they may signal the reactiveness of different element systems to a common cellular stimulation of transposition.

Jychian Chen, Stephen Dellaporta
and Irwin M. Greenblatt

PEORIA, ILLINOIS
USDA, ARS, Northern Regional Research Center

Linkages among zein genes as determined by IEF

Seventy different zein polypeptides have been characterized in 18 maize inbreds by serial analysis with isoelectric focusing in agarose (IEF) followed by SDS-PAGE (Wilson, Plant Physiol. 82:196, 1986). Each zein polypeptide is identified by a letter and number related to its relative molecular mass and by a number (between 10 and 60) related to its isoelectric point. A-zein is often called 22 kDa zein, though its true mass is near 27 kDa. B-zein is called 19 kDa zein but is really about 24 kDa. D-zein is called 10 kDa, though its true mass has not been determined. A 1 to 10 scale gives finer discrimination to the A and B zeins. Band identifications were made by running inbreds and individual zeins on gels adjacent to well-characterized "standard" inbred lines.

Eleven different crosses were made among inbreds that had been characterized by serial analysis. Some lines contained *su1* as a marker for chromosome 4 and *y8* or *wx* T7-9(4363) for chromosome 7. Backcrosses by one or both parents or the F2s (for the translocation crosses only) were made, and seeds were analyzed for occurrence of zeins. As many as 120 seeds of each class were assayed, so that linkages could be determined (N.T.J. Bailey, Introduction to the Mathematical Theory of Genetic Linkage, 1961).

The results obtained from the cross W64 × A619 are presented here, along with a summary of results from all crosses. Fig. 1 shows the zein patterns obtained with agarose IEF. N is the inbred N28, a standard used to check the pattern. The zeins which differ between the two parental inbreds are identified on the right of the gel. Six backcross seeds pollinated by P1 (W64A) are on the left, pollinated by P2 (A619) are on the right of the gel. Note that bands 10 and 22 are present or absent (0 or 2 doses) in BCP1 while they occur in 1 or 3 doses in BCP2 on the right. They appear to be linked. Bands 32 and 33.5 occur or are absent together in 9 seeds, while 3 seeds show apparent crossing-over. It was possible to score 11 zeins for this cross. The results from 240 seeds are given in Table 1.

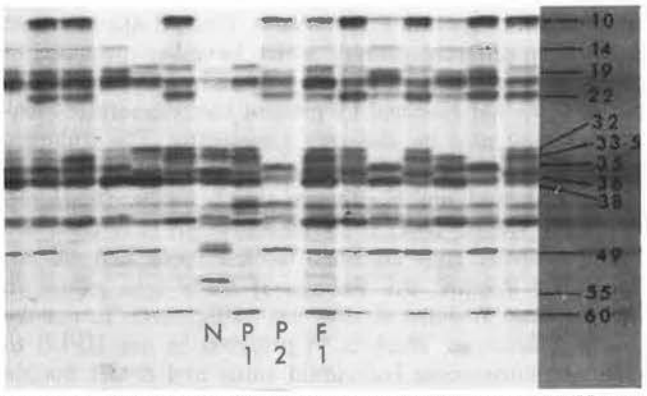


Figure 1. IEF of zeins from the cross W64 × A619. N is N28, a standard inbred, P1 is W64A, P2 is A619, and F1 is P1 × P2. Six BC-1 seeds are on the left, six BC-2 seeds on the right. The numbers are the IEF identifications for each band.

A. Linkages on chromosome 4:

	% Recombination						
ZEINS:	14	19	35	36	32	33.5	49
A2/60/W64A	7	7	7	7	7	30	33
A3/14/A619		0	0	0	0	23	26
A3/19/A619			0	0	0	23	26
B9/35/A619				0	0	23	26
B6/36/W64A					0	23	26
B8/32/W64A						23	26
A3/33.5/W64A							4
B9/49/A619							

Suggested order for 15 zeins and a marker on chromosome 4:
A2/60, B7/17.5, A3/14, A3/19, B9/35, B6/36, B8/32*, A1/37, A3/33.5, A3/33, A2/28, B6/49*, B8/54.5, su-1, A1/30.5, A2/44.5.
(*-two different recombination values were obtained for these zeins in different crosses.)

B. Linkages on chromosome 7:

	% Recombination	
ZEINS:	10	38
B9/22/A619	0	0
B9/10/A619		0
B9/38/W64A		

Suggested order for 4 zeins plus markers on chromosome 7:
7-9 waxy trans(4363), D/55, B9/22, B9/10, B8/38, y8.

Table 1. Zein gene linkages. Recombination frequencies calculated for backcrosses of W64A × A619, with suggested order for zein genes determined from this and 10 other crosses. Zeins are identified by SDS-PAGE mobility, IEF band number, and inbred origin in first column, by IEF number only on the upper row.

Assignments to chromosomes 4 and 7 were made with the crosses involving the markers. Chromosome 4 contains both sizes of the major zeins, A and B, and they occur in two regions of the chromosome separated by about 23 cross-over units. Five zein genes are so tightly linked that no confirmed crossovers were detected. Genes for three B-zeins and one D-zein were found on chromosome 7, with the B-zeins being closely linked.

Zeins 32 and 49 gave different recombination values for different crosses, suggesting that the bands represent genetically different zeins in different inbreds. Two inbreds were found to contain both A1/33.5 and A3/33.5 zeins, which could not be distinguished by IEF alone. Large-scale scoring by SDS-PAGE is not practical yet, but examination of a few F3 seeds revealed that A1/33.5 and A3/33.5 can be separated by crossing-over. Band D/55 was linked to *wx* T7-9(4363) in two crosses. Several bands scored as being D/55 were lifted from the IEF gel and run on SDS-PAGE to confirm their identity. Some inbreds were found to contain a B5/55 zein. Thus, it appears that zeins from different inbreds which have been mapped to the same position on IEF gels may have different genes. Some zeins not resolved by present electrophoretic techniques may also be different genetically. The linkages reported here differ in several instances from those reported by Ottoboni and Steffensen (MNL 59:99, 1985, and Biochem. Genet. 25:123, 1987). Although they used the same methods, they analyzed too few seeds and did not take into account the linkage of some zein genes on chromosome 7. Joint studies are being made to resolve these differences. Work is in progress to use HPLC to further characterize individual zeins and detect double zeins in IEF bands.

C.M. Wilson, T.C. Nelson and G.F. Sprague

PHILADELPHIA, PENNSYLVANIA
University of Pennsylvania

3S linkage data: *E8*

Previously we reported (Sorrentino and Poethig, MNL 59:86, 1985) that *E8* is located approximately 30cM distal to *d* on the short arm of chromosome 3. We have since mapped this locus more precisely using a null allele (*E8-0*) fortuitously present in our *d g2* stock, an extremely slow allele (*E8-S*) obtained from M.M. Goodman, and more common alleles of this locus which have similar faster

A. *+ + E8-S / cl d E8-F × cl d E8-F; Clm-4*

Families	Parental		*cl--d*		*d--E8*		Double	
	+ + S	*cl d F*	*+ d F*	*cl + S*	*+ + F*	*cl d S*	*cl d S*	*cl + F*
N43-2,3,12	54	58	10	7	23	25	1	1
Totals	112		17		48		2	

% Recombination (N=179) **10.6 ± 2.3** **27.9 ± 3.3**

B. *+ E8-S +/ d E8-F g2 × d E8-0 g2*

Families	Parental		*d--E8*		*E8--g2*		Double	
	+ S +	*d F g2*	*d S +*	*+ F g2*	*+ S g2*	*d F +*	*d S g2*	*+ F +*
B655-5	39	38	10	31	8	6	2	4
Totals	77		41		14		6	

% Recombination (N=138) **34.1 ± 4.0** **14.5 ± 3.2**

C. *E8-S +/ E8-0 g2 × E8-0 g2*

Families	Parental		*E8--g2*	
	E8-S +	*E8-0 g2*	*E8-S g2*	*E8-0 +*
B655-4	51	52	12	5
Totals	103		17	

% Recombination (N=120) **14.2 ± 3.2**

mobilities in starch gels (*E8-F*). The results of 3 sets of test crosses (Table) provide the following map distances, *cl* — 10.6±2.3(20/179) — *d* — 30.6±2.6(97/317) — *E8* — 14.3 ±2.2(37/258) — *g2*. These results indicate that *g2* is the terminal marker on 3S, and extend the current map by about 14cM.

N.G. Pasteris and R.S. Poethig

3S linkage data: *Cg*

Cg was originally mapped 5cM proximal to *d* on the basis of a 3-point test cross involving *lg2* and *d* (Galinat, MNL 26:51, 1952). Because *lg2* is on 3L, about 50cM from *d*, it seemed worthwhile to obtain additional linkage data for *Cg* using more closely linked markers. The table lists

A. *Lg3 d +/ + + Cg × + + d*

Family	Parental		*Lg3--d*		*d--Cg*		Double	
	Lg d +	*+ + Cg*	*Lg3 + Cg*	*+ d +*	*Lg3 d Cg*	*+ + +*	*Lg3 + +*	*+ d Cg*
C768	119	114	28	19	11	13	3	0
Totals	233		47		24		3	

% Recombination (N=307) **16.3 ± 2.1** **8.8 ± 1.6**

B. *+ + × Lg3 +/ + Cg*

Families	Parental		*Lg3--Cg*	
	+ Cg	*Lg3 +*	*Lg3 Cg*	*+ +*
C764	38	43	6	8
C765	38	28	13	12
C766	40	29	7	14
C767	40	36	12	12
Subtotals	156	136	38	46
Totals	292		84	

% Recombination (N=376) **22.3 ± 2.1**

the results of testcrosses between *Cg*, *Lg3* and *d*. These results confirm the tight linkage between *Cg* and *d*, but indicate that *Cg* is distal rather than proximal to *d*.

R.S. Poethig

Identifying genes that interact with *Tp1* and *Tp2*

The phenotype of *Tp1* and *Tp2* suggests that these loci play an important role in regulating the transition from juvenile to adult growth in corn. In order to define loci that interact with *Tp1* and *Tp2* I have taken 2 different approaches. The first involves testing the effect of hyperploidy for various chromosome arms on the expression of these mutations. This approach is based on the assumption that the effect of such gross variation in chromosome dose can be traced to one or a few genes within the hyperploid region. Having defined chromosomal regions that interact with *Tp1* and *Tp2*, the loci responsible for this effect can then be identified by efficient mutagenesis schemes. The second approach involves making "educated guesses" (based on the phenotypes of existing mutations) to search for mutations that interact synergistically with *Tp1* and *Tp2*. Preliminary results indicate that both approaches have considerable merit.

Tp1/+ and *Tp2*/+ stocks homozygous for various recessive endosperm markers were crossed by appropriate B-A translocations in order to generate diploid, hypoploid and hyperploid progeny. Seeds with mutant endosperms were assumed to have hyperploid embryos. Those with wild type endosperms were assumed to be either diploid or hypoploid; the latter class should be relatively infrequent because of the low rate of B-A non-disjunction in a W23 background, and because B-A deficient sperm preferentially fertilize the central cell. Plants that were smaller than normal were assumed to be hypoploid escapes and were excluded from this analysis.

Of the arms that have been tested so far (1L, 3L, 4L, 5S, 5L, 6L, 9S, 10L), only 3 had obvious effects on the expression of *Tp1* and/or *Tp2*. Hyperploidy for 1L suppresses tiller production by *Tp1* plants, but enhances the effect of this mutation on tassel morphology. Hyperploidy for 3L and hyperploidy for 4L enhances the effects of *Tp1* and *Tp2* on both vegetative and reproductive morphology. Although other arms also had an effect on the morphology of *Tp1* and *Tp2*, these effects were considered non-specific because wild type hyperploid siblings were similarly affected.

The fact that hyperploidy for 1L and 4L significantly influences the expression of *Tp1* and *Tp2* is particularly interesting because both arms possess mutations that interact synergistically with *Tp1* and *Tp2*. Plants homozygous for teosinted branched (*tb*), a recessive mutation on 1L that enhances tillering and transforms the ear into a tassel, and heterozygous for *Tp1* or *Tp2* have dramatically more tillers than singly mutant plants; even tillers bear tillers in double mutants. The effect of this combination on reproductive development is unclear because in some families all primary branches terminate in a tassel, while in others, primary branches terminate in ears. However, as a rule, secondary branches are female.

It should be noted that the effect of *tb* on tiller formation in double mutants is the exact opposite of that produced by hyperploidy for 1L: i.e., increasing *tb*+ suppresses tillering while a recessive (presumably loss-of-function) mutation of this locus enhances tillering. This observation is consistent with the hypothesis that the *Tp1* and *Tp2* mutations enhance tiller formation (at least in part) by suppressing the expression of *tb*+. However, *tb* is clearly not the only locus involved in this process because grassy tillers (*gt*) also interacts synergistically with *Tp1* and *Tp2*, although not to the same extent as *tb*.

The mutation of interest on 4L is *Tu*, a dominant mutation that increases glume size in heterozygous condition and has more extreme effects in homozygous condition. Since *Tp1* and *Tp2* also increase the size of glumes, and often transform them into leaf-like structures, it was of interest to determine how these loci interacted with *Tu*. *Tu*/+; *Tp1*/+ (or *Tp2*/+) plants have highly modified tassels and ears in which all vegetative structures are considerably larger than those produced by any of these mutations individually. One possible explanation for this result is that *Tp1* and *Tp2* stimulate the expression of *Tu*+, and *Tu* is a hypermorphic (over-expressing) mutation that enhances this stimulatory effect. A problem with this explanation is that dosage analysis of *Tu* indicates that this mutation is neomorphic rather than hypermorphic (MNL 61:85). If *Tu* is a constitutively active mutation, this explanation may have some validity.

R.S. Poethig

3S linkage data: *ys3* and *ra2*

Listed here are the results of testcrosses involving *ys3*, *ra2* and a number of markers on 3S. These data, and previous results for the distance between *Lg3* and *Rg* and from *d* to *ra2* (MNL 60:109, 1986), provide the following map distances: *ys3* — 1.5± 0.7(4/259) — *Rg* — 1.8±0.5(11/623) — *Lg3* — 16.5±1.8(71/429) — *ra2* — 4.9±0.7(48/971) — *d*. If one takes the single *ys3 Rg Lg3* double recombinant seriously,

A. *ys3 + Lg3 + +/ + Rg + ra2 d* x *ys3 ra2 d*

	Families C799	C780	Subtotal	Total	% Recombination	
a. Parentals						
ys3 + Lg3 + +	73	30	103			
+ Rg + ra2 d	69	27	96			
				199		
b. Recombinants						
+ + Lg3 + +	2	0	2			
ys3 Rg + ra2 d	1	0	1			
				3	*ys3-Rg*	1.5 ± 0.7
+ Rg Lg3 + +	3	0	3			
ys3 + + ra2 d	2	0	2			
				5	*Rg--Lg3*	2.3 ± 0.9
+ Rg + + +	16	10	26			
ys3 + Lg3 ra2 d	13	5	18			
				44	*Lg3--ra2*	17.0 ± 2.3
ys3 + Lg3 + d	1	3	4			
+ Rg + ra2 +	2	1	3			
				7	*ra2--d*	2.7 ± 1.0
ys3 Rg Lg3 + +	1	0	1			
+ + + ra2 d	0	0	0			
				1		
Total progeny:				259		

B. *+ +/ ra2 d* x *ra2 d*

	Families C755	C756	Subtotal	Total	% Recombination
a. Parentals					
+ +	64	82	146		
ra2 d	64	64	128		
				274	
b. Recombinants					
ra2 +	2	6	8		
+ d	6	3	9		
				17	5.8 ± 1.4
Total progeny				291	

then these results suggest that there is no interference between recombination events in the *ys3 Rg* and *Rg Lg3* intervals. This is significant because there is usually no interference across the centromere. Since *ys3* is located on 3L, this lack of interference may mean that *Lg3* is located on 3S.

R.S. Poethig

PIRACICABA, SÃO PAULO, BRAZIL
ESALQ, Universidade de São Paulo

Preliminary results on the segregation of knobs (C-bands) in inbred lines derived from a flint variety

During the development of work with the purpose of selecting families of isogenic lines with slight differences in knob composition within families, a C-banding procedure was employed (Aguiar-Perecin and Vosa, Heredity 54:37, 1985). The experience has proved useful for the analysis of a large number of plants in a population with knob positions well defined. Despite the data being incomplete, it is interesting to present them. Table 1 shows the knob constitution of 2 groups of S3 progenies, JD-14 and JD-06, each one derived, from one plant of the commercial flint variety Jac-Duro (Sementes Agroceres). Data on each progeny were obtained through the examination of C-banded metaphases in root tips of seedlings from individual ears. All the materials showed homozygous bands in the long arm of chromosomes 6, 7 and 8 (Fig. 1a, b). The analysis of pachytene showed that the band in chromosome 6 corresponds to KL2 and KL3. It has not been determined yet if the band on chromosome 8 corresponds to KL1 or KL2. So, we are using the provisional designation of "8L" for this position. Bands corresponding to K2L and K3L were also found in the two groups of lines, segregating in most of them (Fig. 1a). Only JD-14 lines displayed knobs at 7S and 9S (Fig. 1b). It can also be observed in Table 1 that the materials with more than 15 plants analyzed show the 1:2:1 expected Mendelian ratio. Deviations seem to be due to sample problems, at the present stage of the investigation. On the other hand, it is interesting that a preliminary survey of S4 and S5 ears shows that segregation is still occurring in at least one locus in most materials investigated. This is particularly consistent for S4 ears derived from JD-14-4, which, as can be seen in Table 1, have knobs segregating at 2L, 3L, 7S and 9S. Among 10 ears analyzed segregation was found in at least 2 knob positions. More progenies are under analysis and aspects like independent segregation of knobbed chromosomes will be checked in selected crosses and their F2 generations. But the possibility that in fact we have an excess of heterozygotes that we succeeded in selfing should not be discarded. This is an interesting hypothesis to be investigated, for our system could be simulating mechanisms of artificial as well as natural selection, when selection pressure for heterozygosity may occur (references in Jain, Ann. Rev. Ecol. Syst. 7:469, 1976). On the other hand, the inbreds JD-14-1 with segregation only at 3L and 9S, and JD-14-2, only at 9S, have already produced completely homozygous S6 progenies (see Table 1 of the communication on flowering time).

The lines obtained are being used in some experiments to study knob effects on development, involving flowering time screening and regeneration capacity in tissue culture experiments.

Margarida L.R. de Aguiar-Perecin
and Juliana U. Decico

Figure 1. Karyotypes of a) plant of the progeny JD-06-13, homozygous for bands at 2L, 3L, 6L (KL2, KL3), 7L and 8L (correspondence with KL1 or KL2 not determined yet); b) plant of the progeny JD-14-1 homozygous for bands at 6L, 7S, 7L, 8L and 9S.

Table 1. Position of C-bands corresponding to knobs and frequencies of homozygotes and heterozygotes for each position in inbred lines (S3) derived from a flint maize variety.

MATERIALS	C-Band positions on chromosomes																				
	2L			3L			6L KL_2, KL_3			7S			7L			8L			9S		
	++	+0	00	++	+0	00	++	+0	00	++	+0	00	++	+0	00	++	+0	00	++	+0	00
JD-14-1			21		4	15 2	21			21			21			21			5	12	9
JD-14-2			15	15			15					15	15			15			2	7	6
*JD-14-4	1	5	2	4	4		10			8	2		10			10			2	6	2
JD-14-5			12	1	7	4	12			12			12			12			6	6	
JD-14-6	3	9	4	16			16			7	6	3	16			16			2	8	6
JD-06-6	3	11	4	7	9	2	18					18	18			18					18
JD-06-13	3	2	2	7			7					7	7			7					7

++ Homozygous for band

00 Homozygous for absence of bands

*½ of a S_2 line (analysis for 2L and 3L positions incomplete in two plants)

Flowering time in inbred lines differing in knob composition

During a series of plantings to produce inbred stocks, some of which were used in experiments to standardize a C-banding method (Aguiar-Perecin, Caryologia 38:23, 1985), some routine observations on flowering time were made. Highly inbred stocks of Zapalote Chico, a high-knob Mexican race, were always observed to be very precocious in comparison with inbred stocks of Ceremonial and Entrelaçado, low-knob Brazilian races. This precocity was also observed in comparison with inbred lines derived from the flint variety JD, previously mentioned, with a difference of around 20 days in an experiment in 1987. In fact, the Zapalote Chico race was described as very precocious by Wellhausen et al. (Races of Maize in Mexico, Secr. Agric. Ganad. Mexico, Rockefeller Found., 1951). Due to the racial and certainly genotypic differences among the materials mentioned, it would be difficult from these simple findings to argue for high knob numbers causing precocity, in the conditions of these plantings in the fields of ESALQ/USP, Piracicaba, which were carried out always in summer, with flowering during January and February.

In this context, and regarding some reports in the literature, it would be interesting to investigate correlations between flowering time, the duration of mitotic cycle and knob composition. Cell cycle time has been shown to be species specific, depending mainly on the basic nuclear DNA content (Van't Hof, Exp. Cell Res. 39:48, 1965; Bennett, Proc. R. Soc. London B. 181:109, 1972). On the other hand, it has been shown in some species of Compositae that the amount of heterochromatin per genome is related to the reduction of the cell cycle times in annual plants with large DNA and heterochromatin contents, but that develop faster than the perennials (Nagl, Nature 249:53, 1974). This reduction was also found in the heterochromatin-rich *Allium carinatum* (Bösen and Nagl, Cell Biology Intern. Reports, Vol. 2:565, 1978). Further, Vosa, (University of Oxford, U.K., personal communication) also observed a species of *Allium* from Turkey, with extremely high heterochromatin content, to be very precocious. Price and Bachmann (Plant Syst. Evol. 126:323, 1976) reported that some annual species of Compositae with high DNA contents and longer mitotic cycle time yet displayed a rapid developmental rate.

Correlations between higher DNA content, lengthening of cell cycle and late flowering have been observed when B chromosomes are present in some species of cereals (Jones and Rees, Chromosoma 24:158, 1968; Rao et al., Experientia 35:1154, 1979). In a previous experiment, it was found that an increasing number of B's tends to delay the male flowering rather than the female, in inbred lines of Zapalote Chico (Ruas, Aguiar-Perecin and Miranda-Filho, in preparation).

In order to analyze the effects of knobs on development, lines of the JD variety have been selfed, as mentioned in the communication on knob segregation. In 1984, it was observed that one S2 line and respective S3 progenies (family JD-14) differed by a mean of 10 and 5 days respectively from another group of S3 lines (JD-06). The 2 groups and derivatives (S4, S5 and S6) have maintained this difference (5-10 days) in subsequent years. Only male flowering has been recorded in randomized block experiments. The cytological analysis showed that late materials (JD-06 lines) do not display knobs at 7S and 9S. Here again data appeared to show late material having knobs in fewer positions. As the cytological analysis showed differences in the frequencies of heterozygotes and homozygotes in the various stocks, the flowering time of S4, S5 and S6 progenies have been recorded in order to detect groups with conspicuous differences. These groups in fact appeared more consistently during the experiment of January to February, 1987. The estimates of flowering time between 3 families of S6 progenies derived from JD-14-1 showed that one of them, 300-14-1-3-3, was more precocious. In another group of 3 families derived from JD-14-2, one of them, 301-14-2-1-1, showed delay in flowering (mean of 5 days). The histograms of frequencies of days of flowering of these 2 lines are quite distinct. Table 1 shows the knob constitution of the 2 families but data referring to the 6 families are still incomplete. These results are, at present, the only ones that seem to be in agreement with the report of Chughtai and Steffensen (MNL 61:98, 1987), in the sense of the appearance of more precocious progenies with fewer knobs.

Table 1. C-bands corresponding to knob positions in two families of S6 progenies derived from a flint maize variety.

	C-band positions							
Materials	2L	3L	6L(L_2,L_3)	7S	7L	8L	9S	No. of plants analized
300-14-1-3-3	00	00	++	++	++	++	00	10 (2 progenies)
301-14-2-1-1	00	++	++	00	++	++	++	25 (5 progenies)

++ Homozygous for band

00 Homozygous for absence of band

The expectation at the present state of this investigation is that the comparison of subgroups within JD-14 families will provide information on the effects of different knob contents and positions, or interactions of knob-genotypes on plant development, evaluated through the flowering time. Selected crosses and their F2 will also be analyzed. Studies on genes affecting flowering have been controversial in estimating the number of genes (Bonaparte, Can. J. Genet. Cytol. 19:251, 1977). The importance of knob constitution in plant adaptation has been emphasized in the literature, as in the survey of knobs in maize races made by McClintock et al. (Chromosome Constitution of Races of Maize in the Americas, Colegio de Post Graduados, Chapingo, México, 1981). Further, the demonstration made by Viotti et al (TAG 70:234, 1985) that knob sequences may be found interspersed in the genome is also an additional point to be envisaged in this discussion.

Margarida L.R. de Aguiar Perecin

RALEIGH, NORTH CAROLINA
North Carolina State University and USDA-ARS

Knob inheritance in *Zea diploperennis* X maize

In previous reports (MNL 60:103, 1986; MNL 61:78-79, 1987) transposition of knobs from terminal to internal chromosomal positions in *diploperennis*-Chapalote hybrids and knob amplification in *diploperennis*-annual teosinte hybrids were described. Another unusual phenomenon in the inheritance of knobs has been observed in the F1 and F2 progeny of crosses between *diploperennis* and Palomero Toluqueno and is reported here.

The Palomero Toluqueno parent plant was grown from seed provided by M.M. Goodman (Mex 6, INIA, R-6, 70-71)

and had 3 internal knobs. The *diploperennis* parent was grown from seed provided by H. Iltis (Upper las Joyas, January 1979, Iltis et al. #1250) and it had 7 terminal knobs. When F1 sporocytes from the cross between these 2 parents were examined, no knobs were cytologically visible. Evidently correlated with the disappearance of knobs was the appearance of "nuclear bodies" that look like micronucleoli. Walters' (Chromosoma 17:78, 1965) study of nuclear bodies in maize revealed they contain RNA, protein, and phospholipids but no DNA. They appear in the earliest stages of meiosis and are believed to arise from the chromosomes.

In F2 sporocytes derived from selfing the knobless F1 progeny, knobs had reappeared. One internal knob and 2 terminal knobs were observed in the F2. In the F1, 1 to 6 nuclear bodies were present in every cell, but in the F2, nuclear bodies were observed in only 55% of the cells and no more than 2 were observed in any cell. These data imply that there is an inverse correlation between the cytological appearance of knobs and nuclear bodies.

It is significant that all knobs appear lost cytologically in the F1 but reappear in the F2. If knobs are transmitted by Mendelian inheritance, they could be lost in the F1, but they would not reappear in the F2. These findings suggest that knob DNA sequences may exist in alternating heterochromatic and euchromatic states. If they have this capacity to change states, it is conceivable that in the euchromatic state they might be transcribed, and thus, under certain conditions such as the genomic shock of interspecific hybridization, play a regulatory role in genomic expression.

Mary W. Eubanks

Linkage relationship between isozyme and morphological markers on chromosome 1L.

Table 1 shows data from 233 testcross progeny segregating for 2 morphological and 3 isozyme markers on chromosome 1L. These data were generated as part of an undergraduate research apprenticeship sponsored by RJR-Nabisco for D.M. Agnell.

Table 1. Testcross data.

Cross: brl, Ampl-4, Mdh4-12, bz2, Pgml-9 / + , Ampl-6, Mdh4-16.7,+ , Pgml-5 x brl, Ampl-4, Mdh4-12, bz2, Pgml-9

		SCO regions*				DCO regions*	
	Parental Types	1	2	3	4	2,4	All others
n = 233	74	5	12	1	23	2	0
	68	2	16	3	24	3	0
TOTALS	142	7	28	4	47	5	0
Recombination %		3.0	14.2	1.7	22.3		
(SE)		(1.1)	(2.3)	(0.8)	(2.7)		

*Regions 1, 2, 3, and 4 correspond to the segments brl - Ampl, Ampl - Mdh4, Mdh4 - bz2, and bz2 - Pgml, respectively. Only one class of double crossovers was observed. There were no triple crossovers.

D.M. Agnell, P.H. Sisco and C.W. Stuber

ST. PAUL, MINNESOTA
University of Minnesota

Plastid DNA in developing endosperm

We have initiated a study to characterize the amyloplast genome and to determine whether plastid genes are expressed specifically during amyloplast biogenesis in the endosperm. Five overlapping cosmid clones (from D.F. Lonsdale) representing the complete plastid genome were used in Southern hybridization analyses of total cellular DNA from 16-day-old endosperm of inbred A188. Sequences homologous to plastid DNA were present in the total DNA of endosperm tissue. When endosperm DNA was digested with *Bam*HI, *Pst*I or *Xho*I, the hybridization patterns for individual cosmid probes were identical to those of total DNA from green leaf tissue. Indistinguishable patterns also were obtained when one of the cosmids was hybridized to leaf and endosperm DNA digested with a 4-base-pair cutter, *Msp*I. We conclude that the amyloplast genome in the early stages of grain filling is very similar and most likely identical to the genome in differentiated chloroplasts.

The amount of DNA per nucleus in endosperm cells of A188 has consistently been shown to increase about 30-fold between day 14 and day 20 post-pollination (R.V. Kowles and R.L. Phillips, Proc. Natl. Acad. Sci. 82:7010, 1985). To determine whether the plastid genome copy number also increases during this period, the ratio of two plastid genes (Cytochrome f and P700) to a representative nuclear gene (rRNA gene) was measured. Total cell DNA was labeled with ^{32}P-CTP by random primer labeling, then hybridized to a predetermined excess of a cloned plastid gene or cloned nuclear rDNA bound to a nitrocellulose filter, and the amount of radioactivity bound was quantitated by scintillation spectroscopy. Under these conditions the amount of hybridization to the specific clone was assumed to be proportional to the amount of that sequence in the total DNA sample.

We found that plastid DNA also increases during endosperm development. For example, the ratio of the plastid cytochrome f gene to nuclear rDNA increased about 2-fold from day 10 to day 16 post-pollination and then dropped to about a 35% increase by day 30. Even though the amount of nuclear DNA per nucleus was not measured in these samples, it is apparent that the amount of plastid DNA keeps pace with or even exceeds the increase in nuclear DNA during endosperm development.

Andrew J. McCullough and Burle G. Gengenbach

Unique mtDNA restriction fragment in an S cytoplasm fertile revertant line retaining the S1 and S2 plasmids

Presence of S1 and S2 plasmids is characteristic of mitochondrial DNA of male sterile S cytoplasm maize. Male fertile revertants of S cytoplasm in most nuclear backgrounds typically have lost the S1 and S2 molecules; however, fertile revertants in the inbred Wf9 nuclear background still retain the S1 and S2 plasmids (L.J. Escote et al., MNL 59:100, 1985; MNL 60:127-128, 1986; T. Ishige and B. Gengenbach, MNL 59:98-99, 1985; T. Ishige et al., MNL 60:126, 1986). In addition to the G′, J′ and R′ fertile revertants of Wf9 we previously reported for S group materials from Japan, we also have obtained a similar S′ fertile revertant line we observed as a spontaneously fertile plant among standard Wf9S plants grown at St. Paul in 1984. We report here our initial observations of mtDNA from this S′ mutant.

S′ mtDNA contains the S1 and S2 plasmids apparently unaltered (data not shown). Restriction digests of S′ mtDNA with *Eco*RI revealed the presence of a ca. 6.5Kb

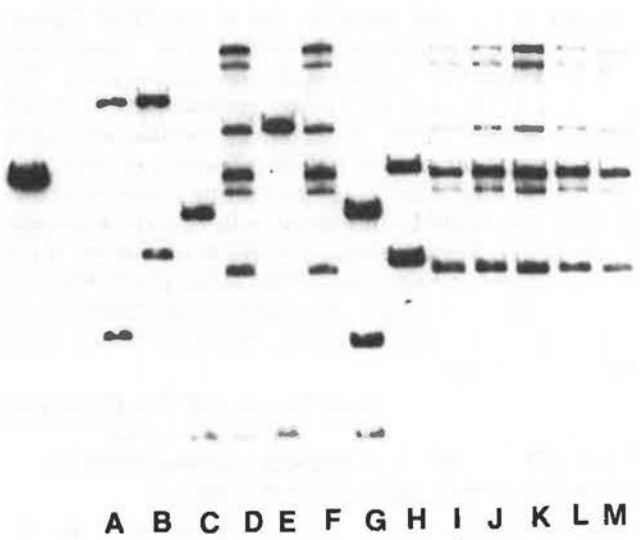


Figure 1. Hybridization of the 6.5Kb *Eco*RI fragment to mtDNA from: A. W182BN (N); B. Wf9 (N); C. Wf9 (R′); D. Putative progenitor of Wf9 (R′); E. Wf9 (J′); F. Putative progenitor of Wf9 (J′); G. Wf9 (G′); H. Wf9 (S′); I. Wf9*rf3rf3* (S) progenitor of Wf9 (S′); J. Wf9*Rf3Rf3* (S); K. A188*rf3rf3* (S); L. Wf9*rf3rf3* (S) from Illinois (S. Gabay-Laughnan); M. Wf9*rf3rf3* (S) from Japan (T. Ishige). Cloned DNA used as a probe is shown in the unmarked left lane. MtDNAs were digested with *Eco*RI and electrophoresed on an 0.7% agarose gel.

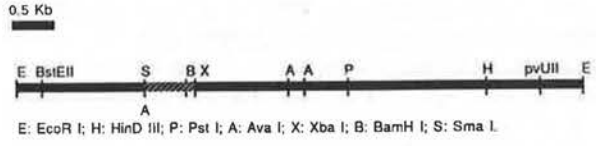


Figure 2. Restriction site map of the 6.5Kb fragment obtained from the S′ fertile revertant. Homology to the *atp9* gene was determined by probing with a 1.2Kb *Xho*I-*Xba*I fragment that contains the coding region of the gene.

fragment not visible in digests of the progenitor Wf9S*rf3rf3* line or in several other sources of S cytoplasm materials either with or without the *Rf3* allele. This 6.5Kb fragment was cloned into pUC119 and initially used as a hybridization probe to Southern blots of mtDNA (Fig. 1). Hybridization patterns for the intact 6.5Kb probe (unmarked left lane) were complex, but apparently identical, for 7 sterile S cytoplasm sources including Wf9S*rf3rf3* from Minnesota (lane I), from Illinois (lane L), or from Japan (lane M); Wf9S*Rf3Rf3* (lane J); A188S*rf3rf3* (lane K); and putative progenitors (lanes D and F, respectively) for the R′ and J′ fertile mutants. The hybridization pattern for the S′ mutant (lane H) was simpler with homology indicated to 2 mutant-specific fragments of ca. 6.5Kb and 4.4Kb. The R′ (lane C), J′ (lane E) and G′ (lane G) mutants had differential hybridization patterns with strong homology either to 1 (R′ and J′) or 2 (G′) mutant-specific fragments. N cytoplasm lines with W182BN (lane A) and Wf9 (lane B) nuclear genotypes exhibited hybridization patterns different from each other and from the S cytoplasm fertile mutant and sterile lines.

The cloned 6.5Kb fragment was hybridized to blots of clones containing the specific mitochondrial genes (*atp-alpha, atp6, atp9, cob, coxI, coxII, coxIII,* large rRNA, small rRNA) or other sequences (5Kb repeat of N cytoplasm, 1.94Kb plasmid, S1 and S2 subclones). Only the clone for *atp9* showed significant homology to the 6.5 *Eco*RI cloned fragment from S′. The *atp9* clone (a 2.2Kb *Xba*I fragment obtained from C.S. Levings) has a predicted protein coding sequence of 222bp, but the primary transcript in N cytoplasm is ca. 1.95Kb (R.E. Dewey et al., Proc. Natl. Acad. Sci. 82:1015, 1985). Figure 2 shows a restriction map of the 6.5Kb *Eco*RI fragment. A 1.2Kb *Xho*I-*Xba*I fragment from the 2.2Kb *atp9* clone when used as a probe showed the strongest hybridization to a ca. 600bp *Sma*I-*Xba*I fragment from the cloned 6.5Kb *Eco*RI fragment. The 2.2Kb *Xba*I clone of *atp9* contains a *Bam*HI site ca. 120bp from the terminal *Xba*I site which may also be present within the 600bp *Sma*I-*Xba*I fragment. Additional subcloning and transcriptional analyses are in progress to determine whether there are transcriptional differences related to *atp9* in the fertile mutants and S sterile cytoplasm lines or whether the structural differences have no effect on gene expression.

Yunxia Wang and Burle Gengenbach

SALT LAKE CITY, UTAH
NPI

Deletions of loci detected by RFLPs

We originally observed that very few loci exhibited "null alleles" during our efforts to prepare a linkage map based upon loci detected by RFLPs. A null allele at a RFLP locus is characterized by the complete absence of a detectable fragment after Southern hybridization with a cloned maize sequence. As our studies were principally geared towards mapping loci detected by random cloned sequences in a small set of segregating populations, this observation could not be taken as an accurate measure of the occurrence of deleted sequences within commonly used germplasm. More recently, by expansion of the number of inbreds examined (sometimes greater than 150 inbreds specifically from Corn Belt germplasm), we have now identified 5 loci, out of the approximately 400 loci we use, which occasionally detect deletions of the target sequences.

Four of the 5 clones identified loci which are duplicated on more than one chromosome, with only one of the pair of duplicate loci in each case exhibiting a null allele. A possible terminal rearrangement was observed in a Mangelsdorf tester line on the long arm of chromosome 2 at our most distal marker locus, #32. This same clone detects a duplicate locus on the long arm of 1, marker #82, for which we have never observed a null allele. The locus on chromosome 2 does not fit the pattern we have more commonly observed before, where almost all of the duplicated loci on the long arm of 2 have corresponding sequences found on the long arm of chromosome 7. Perhaps the presence of the locus #32 sequence on the terminus of chromosome 2 represents a recent translocation from locus #82 on 1, which is then only found in some maize inbreds. Another possible terminal rearrangement, this time on the short arm of 2, marker #417, was observed in B73 and some of its derivatives. This sequence is also found to be duplicated on chromosome 10 as marker #484. A third terminal rearrangement is found on the long arm of chromosome 5 at marker locus #363. Its corresponding duplicate locus, #292, is located on the long arm of chromosome 4 where we have never observed a null allele.

Possible internal rearrangements of chromosomal segments have also been identified in 2 other cases. Locus #483 exhibits a null allele and has been tentatively located near the centromere on chromosome 2. The sequence which detects this locus is also duplicated as marker locus #391 on chromosome 7, which we previously thought to represent a unique sequence due to the prevalence of the null allele at locus #483 in most of our "mapping" populations. The fifth clone recognizes a single, unique chromosomal location, which is undetectable in some maize inbreds. Marker #432 on the long arm of 3 appears to be the only unduplicated sequence of this group which exhibits a null allele. This deletion is evident in a majority of the Lancaster-derived germplasm examined by us but present in most other maize pedigrees.

Three of the 4 rearrangements reported herein occur on chromosome 2, which has been shown previously to display two different cytological forms (Neuffer, Jones, and Zuber, Mutants of Maize, p. 4, 1968). It is interesting to speculate that the possible terminal differences observed cytologically might also be reflected in the rearrangements of RFLP loci we report here. We do not yet know how well our results, obtained with various inbreds, correlate with these cytological observations; however, extension of this type of analysis should prove revealing as to how these duplications/deletions were generated during the evolution of the structure of the maize genome.

V. Turner, Scott Wright, J. Suzuki and T. Helentjaris

RFLP mapping of cloned genes and their related sequences

One of the many applications of restriction fragment length polymorphisms (RFLP) in plant genetics is determination of the chromosomal location of cloned gene sequences. In last year's news letter (Wright et al., MNL 61:89) we reported the mapping of 21 such cloned genes or related sequences in maize. Since that time, we have received and utilized another 9 genes in our RFLP studies, some of which had their genomic origins determined previously through conventional linkage analysis. As most of them hybridized to numerous locations in the genome, this resulted in the addition of 22 loci to our RFLP linkage map, bringing the total number of loci on our current map to 370 markers (Helentjaris et al., Genetics, in press). The following is in addition to this previous list of cloned genes, the approximate chromosomal location of them or related sequences, and the individuals who provided them.

Identity	Locus	Chromosome	Provider
ADP glucose-pyrophosphorylase (rice)[a]	309-Adp1	1L	J. Preiss
	310-Adp2	2L	
	314-Adp3	4[b]	
	318-Adp4	8L	
	319-Adp5	10S	
Anthocyaninless[a] (a1)	467-A1	3L	N. Federoff
	482-A1-2	1L	
	468-A1-3	2L	
	469-A1-4	5L	
	470-A1-5	7S	
Chlorophyll AB binding protein[a]	477-Cab1	3L	L. Bogorad
	478-Cab2,3	4L	
	479-Cab4	8L	
Colored aleurone and plant (R)	308-R	10L	S. Dellaporta
Opaque endosperm (o2)	480-o2	7	B. Schmidt/ B Burr
Oxygen evolving complex protein	472-Oec1	4[b]	L. Bogorad
	473-Oec2	2S	
	474-Oec3	5S	
	475-Oec4	7L	
	476-Oec5	8[b]	
Structural protein-L2	481-L2	1 or 5[b]	A. Huang
Structural protein-L3	471-L3	2L	
Superoxide dismutase-2[a]	463-Sod2-2	9[b]	R. Cannon/ S. Scandalios

"a" = Indicates duplicated loci; that is, individual clones detect more than one fragment, which have been mapped to different chromosomal locations

"b" = Locus has been assigned to chromosome, however, linkage data are not yet available

Anyone who is currently generating clones of known identity and would be interested in having their genomic location investigated, please feel free to contact us. If you are interested in using any of the above cited clones for your own research, please contact the original providers as we do not distribute these clones, only those generated in our own programs.

Scott Wright and Tim Helentjaris

Does RFLP analysis of ancient Anasazi samples suggest that they utilized hybrid maize?

While hiking in S. Utah, I was intrigued by the abundance of corn cobs lying around ancient Anasazi Indian ruins that were abandoned when the culture dissipated in the 12th-13th century A.D. While very dry, these cobs were still otherwise in very good condition. Realizing that the first step in our RFLP analysis is a DNA preparative protocol which utilizes lyophilization of the material, I wondered whether we could prepare DNA from these samples that would then be analyzable through our normal procedures.

I obtained 8 cob samples which had been tentatively dated to the 13th century from Winston Hurst, Curator at the Edge of the Cedars State Park in Utah. I tried several procedures to prepare DNA from the samples and found that one, with modifications, was most effective (C. Lichtenstein and J. Draper, In: DNA Cloning Volume II, D.M. Glover ed., IRL Press, Oxford, pp.67-120). I was able to repeatedly obtain DNA from the same set of 5 of the original 8 samples. Examination of the DNA on an agarose gel revealed that there was high molecular weight DNA present in the samples with only moderate degradation when compared to modern samples.

The 5 sample DNAs were digested with *Hin*dIII, electrophoresed on an agarose gel, and Southern-blotted by established procedures. These blots were then hybridized with several different clones from our repertory of informative maize clones. The clones were selected on the basis of: 1) they hybridized to only a single locus, 2) they were known to be very informative in a selection of Corn Belt germplasm composed of 150 inbreds, and 3) the loci detected by this group of clones were distributed on several different chromosomal segments.

The first positive result was that a defined hybridization signal could be detected in all 5 samples. It was weaker than that seen in the modern samples, most likely due to the moderate degradation in the ancient samples. The second aspect of interest here was that all of the Anasazi samples revealed an identical hybridization pattern, unique to each clone tested; i.e. there was no variation amongst these samples revealed by any of the clones. This is somewhat surprising in that all of these 5 samples originated from different ruins, although all in the same county and from about the same historical period. Obviously with the discrimination we usually see with this procedure, we can conclude that the people at these sites were using similar, if not identical, cultivars.

The third point is even more intriguing, if not difficult to interpret. Not only were the hybridization patterns identical among the 5 samples, but a number of the clones revealed more than one hybridizing fragment with them. There are a number of possible explanations for this result. For instance on average, 1 in 4 enzymes should exhibit a site within the genomic segment revealed by any particular clone and this would then result in 2 fragments. I do not believe this to be the case here as these clones had been tested against a large number of inbreds with *Hind*III and there were few or no cases of alleles revealed as 2 fragments. More importantly, I tested some of the clones against one of these ancient samples with multiple enzymes and in each case, 2 fragments were revealed, arguing that 2 distinct genomic segments were being detected. A second explanation for this result might be that there were extra copies of these sequences on chromosomes besides the normal complement of 20, perhaps on addition chromosomes from Teosinte or Tripsacum or even B chromosomes. It is difficult to believe that the former could result in genetically stable lines that would be maintained. The latter, B chromosomal sequences, is more difficult to rule out. However, I have checked some of these clones against modern B-A translocation stocks and see no evidence that any of these sequences are also found on the B chromsomal segments. The only other explanation that I have come up with is that the 2 fragments represent a heterozygous situation at single loci. This is supported by the fact that the intensity of the 2 fragments is usually equal and that they both often line up with single fragments detected in modern inbreds. For this situation to always be true in each of several samples as we have found, we cannot accept chance as the basis but would have to conclude that these identically heterozygous isolates were purposefully created, i.e. they were produced as part of a hybridization program. While surprising, the prospect that ancient Indians were aware of and utilized hybrid maize is not completely out of the realm of possibility; it was relayed to me that modern Indians in the region are known to maintain inbreds in a homozygous state by isolation, as part of their religious tenets. I am concerned that I am overlooking other possibilities or that there are other means to test this thesis. I would truly welcome ideas concerning whether this is indeed possible, what other data exists that would support or refute such an idea, and how I might further test this proposal.

Tim Helentjaris

SRINAGAR, KASHMIR, INDIA
Regional Research Laboratory, CSIR

Genetic variability and character association in maize grown in Jammu Province

Maize is an important cereal crop of Jammu and Kashmir State in India. It is cultivated over an area of 2.85lakh hectares, which is more than the area under any other cereal crop grown in the state, including paddy. Only about 10% of this area (0.29lakh ha) is under improved hybrids and/or composites. The rest of the area is still under local adapted varieties. These locals may not be as productive as the hybrids but they possess some useful traits which have helped them to survive long periods of domestication. They could, therefore, be useful material in the development of any superior hybrids suited to this region.

Table 1. 25 local varieties from Jammu Province.

S.No.	Variety	S.No.	Variety	S.No.	Variety
1	Udampur local	2	Udampur local 1	3	Ram Nagar Local
4	Ram Nagar 2	5	Billawar 2	6	Billawar 7
7	Mangnath 25	8	Mangnath 51-1	9	Mangnath 54
10	Mangnath 64	11	Jhallas 15	12	Jhallas 16
13	Jhallas 17	14	Jhallas 18	15	Jhallas 24
16	Jhallas 35	17	Jhallas 49	18	Qureai Manila 243
19	Bagnoti 559	20	Gandi 110	21	Batyar 43-1
22	Reasi 25	23	Reasi Local 1	24	Patha 392
25	Hami 250				

Table 2. F values, mean, range, phenotypic and genotypic coefficient of variability and heritability for seven characters in maize.

CHARACTERS	'F' values	Mean ± SE	Range	PCV	GCV	Heritability (%)
Number of leaves/plant	9.63**	14.82 ± 0.57	12.30-18.00	8.92	8.05	81.42
Leaf index $(cm)^2$	8.55**	54.95 ± 4.61	42.60-75.00	18.37	16.34	79.11
Tassel length from flag leaf (cm)	4.3**	40.90 ± 2.51	34.20-47.60	10.08	7.99	62.90
Number of ears/plant	3.7**	1.94 ± 0.28	1.40-2.80	9.79	5.67	57.89
Number of tassel branches/plant	10.8**	24.05 ± 2.68	12.80-41.40	26.64	29.21	83.16
Plant height (cm)	16.34**	169.10 ± 7.01	129.90-198.40	12.22	11.49	88.46
Days to 75% silking	5.44**	118.44 ± 2.98	107.00-123.00	4.52	3.75	68.95

Table 3. Phenotypic (P) and Genotypic (G) correlation coefficients of various characters with days to 75% silking in maize.

CHARACTER	Number of leaves/plant	Leaf index $(cm)^2$	Number of ears/plant	Tassel length from flag leaf (cm)	Number of tassel branches/plant	Plant height (cm)
CORRELATION (r)						
PHENOTYPIC (P)	0.5010*	0.3051	0.3585	0.1429	0.0445	0.2267
GENOTYPIC (G)	0.7468**	0.3815	-0.2372	0.2097	-0.0468	0.3115

*Significant at 5%
**Significant at 1%

The present study was undertaken to assess the genetic potential of 25 local varieties of maize (Table 1) from the Jammu province of Jammu and Kashmir State. The design for the experiment was a randomized block design with 4 replications at Srinagar. Each entry was grown in one-row plots 12m long, with row spacing 75cm. Plant-to-plant distance was maintained at 25cm and observations were recorded on 10 random plants in each plot. Only those characters were studied which were significant (Table 2). The genotypic and phenotypic coefficients of variability were estimated according to the formula given by Burton (Proc. 6th Int. Grassland Cong. 1:277, 1952) and heritability in the broad sense according to Hanson et al. (Agron. J. 48:268, 1956).

The analysis of variance revealed highly significant differences among varieties for all the characters, suggesting that there was a high degree of genetic diversity in the material for various characters. A wide range of variation was observed for most of the characters. Plant height, leaf index and number of tassel branches per plant showed wide variation, while the number of ears per plant and number of leaves per plant showed a narrow range of variation. In general the phenotypic coefficient of variation was higher than the genotypic coefficient of variation, indicating larger influence of environment on the expres-

sion of the characters. However, the number of leaves per plant, plant height and days to 75% silking showed very little difference in genotypic and phenotypic coefficient of variability, indicating that these characters were less influenced by the environment.

Plant height, number of tassel branches per plant and number of leaves per plant showed high heritability (88.46, 83.16 and 81.42% respectively), indicating the importance of these characters in varietal improvement through selection.

Data on the correlation coefficient (Table 3) indicated that the number of leaves per plant had a significant positive correlation with the days to 75% silking at both the genotypic and phenotypic level (r = 0.7468 and 0.5010), indicating a lot of scope for improvement in this character.

P.N. Jotshi, B.K. Bhat and M.K. Bhan

STANFORD, CALIFORNIA
Carnegie Institution of Washington

The transmittance of sunlight through husk tissue under field conditions

Anthocyanin accumulation in maize and sorghum has been exploited in several studies as a sensitive and convenient measure of a light-controlled biological process. Physiological studies (reviewed by A.L. Mancinelli in Bot. Rev. 51:107, 1985) show that anthocyanin accumulation in these plants is an example of a high irradiance response (HIR). HIR's are characterized by:(1) continuous irradiation required to produce the response; (2) strong fluence-rate dependency; (3) response does not show reciprocity.

In maize particular allelic combinations of genes exhibit a characteristic pattern of light dependence for anthocyanin accumulation. Some of the most familiar examples include the interaction between *B* and *pl*, which gives rise to the "sun-red" effect described by Emerson in 1921 (Cornell Univ. Agri. Exp. Stn. Mem. 39). Chen and Coe (Biochem. Genet. 13:333, 1977) have described an allele (*c-p*) of the *C1* gene which exhibits light-dependent anthocyanin accumulation in the aleurone during germination. A more recently described example is the deep purple pigmentation in the pericarp of kernels with a genetic constitution of *Sn* plus *Pl* (G.A. Gavazzi et al., Maydica 30:309, 1985). In order to produce anthocyanin in the pericarp of plants of genotype *Sn* plus *pl*, the developing kernels must be exposed to light. The light requirement for these phenomena is usually demonstrated by cutting back the overlying tissue and exposing the underlying "dark" tissue to direct sunlight or its equivalent. In approximately 24 hours the previously green (colorless, if aleurone or pericarp) tissue shows a dramatic increase in anthocyanin pigments.

As a first step to determining the precise light requirements necessary to elicit anthocyanin accumulation we measured the light characteristics that the plants are exposed to during much of their growing season. The spectrum of sunlight transmitted through either a single layer (Figure 1) of green or purple husk tissue or through the entire intact cob covering (Figure 2) was measured with a LI-COR 1800 portable spectroradiometer in 2nm increments. Measurements were made in August and September at Stanford, CA between 1200 and 1500 hours

Figure 1

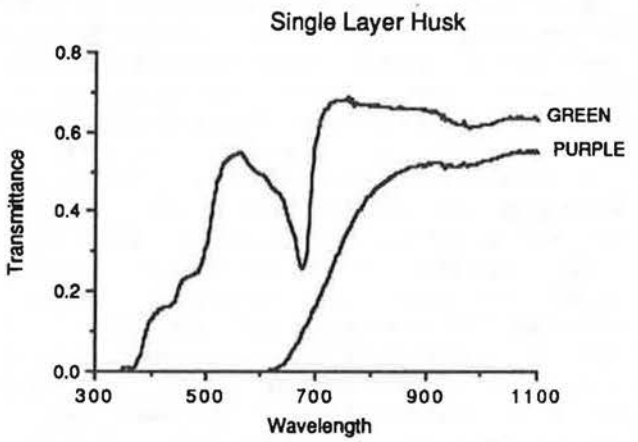
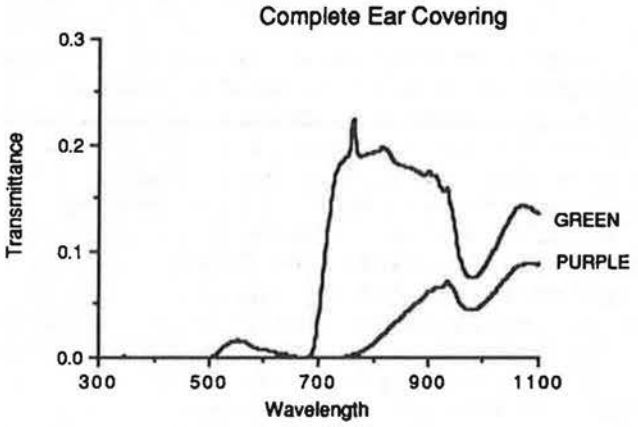


Figure 2



on bright cloudless days over the wavelength range from 300 to 100nm. Transmittance was calculated as the ratio of the spectral energy distribution of each tissue to that of the incident sunlight.

A single green husk or leaf sheath (Figure 1) transmits about 20% of the incident sunlight in the blue range (420-480nm), ~35% in the red range (@660nm) and ~70% in the far-red range (@730nm). In a *B pl* plant this is the light "seen" by the next underlying layer, i.e. the light transmitted by the newly exposed green husk. In approximately 24-48 hours, this outer husk turns purple and then transmits the purple husk pattern. Anthocyanin pigments in the husk drastically alter the amount and spectral quality of the transmitted light. Virtually no blue light is transmitted and the R:FR ratio is 0.21 vs. 0.5 transmitted by a single green husk.

Developing kernels are exposed to virtually no physiologically relevant light below 700nm (Figure 2). An intact green ear covering does transmit light in the far-red range at levels about ¼ that through a single layer of green husk. The R:FR ratio in an intact green ear covering is 0.14 compared to 0.005 for the complete purple covering. We have found that a single layer of green husk is sufficient to prevent pigmentation of the pericarp in *Sn pl* plants. This would indicate that the blue portion of sunlight must be the effector wavelength.

Loverine P. Taylor

STANTON, MINNESOTA
The New Northrup King

RFLP analysis of percentage recurrent parent

Backcrossing is used extensively to either transfer a specific gene from one genotype to another or to improve an inbred line for some quantitative trait (Hallauer and Miranda, Quantitative Genetics in Maize Breeding, ISU Press, Ames, IA, 1981). If the parent used for backcrossing is homozygous, the percentage recurrent parent after N generations of backcrossing is $1-(1/2)^{N+1} \times 100$. Six backcross generations are thus required to obtain greater than 99% recurrent parent. Analysis of backcross progeny with Restriction Fragment Length Polymorphism (RFLP) markers should allow one to compare the theoretical amount of inbreeding with actual levels of inbreeding observed.

Two lines, designated A and B, have been backcrossed 5 and 6 times, respectively, with a source of rust resistance (*Rp1-d*), A632. RFLP markers were utilized to quantitate the differences between the converted lines A-*Rp1-d* and B-*Rp1-d*, the original lines A and B, and the *Rp1-d* source, A632. Figure 1 shows the location of the 40 markers polymorphic for the lines tested and the chromosomal location of bands from the recurrent parent not found in the converted line. The chromosomal locations of the 10 recurrent parent (line B) alleles not incorporated into B-*Rp1-d* are spread over 5 chromosomes. The 2 unincorporated recurrent parent (line A) alleles are located on 2 chromosomes. A summary of allelic differences between the converted lines, the original lines and the *Rp1-d* source is shown in Table 1. The percentage recurrent parent observed for B-*Rp1-d* is significantly lower than expected.

Figure 1. Location of RFLP markers utilized to compare lines A, B, A-*Rp1-d*, and B-*Rp1-d*

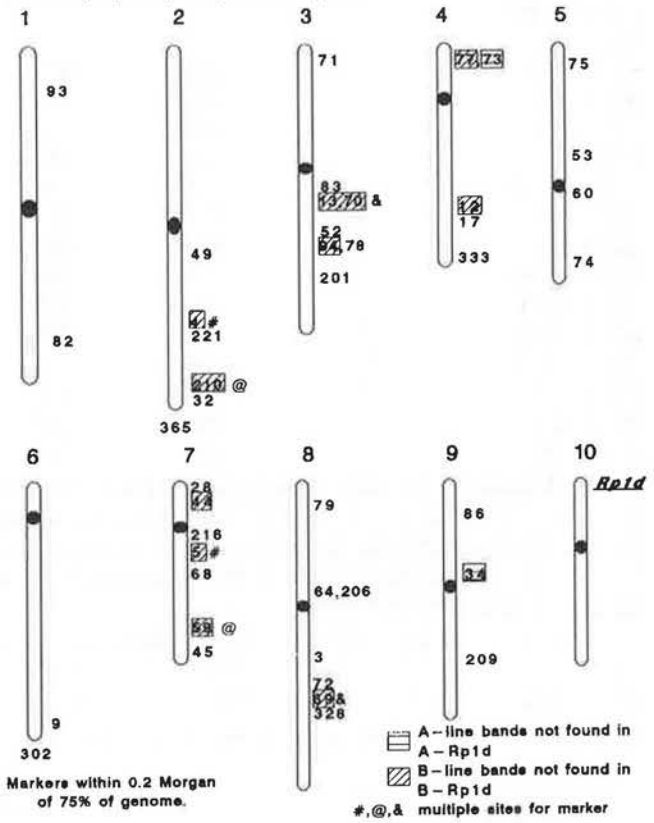


Table 1. Percentage recurrent parent for converted lines A-*Rp1-d* and B-R*p1-d*

line specific loci / total polymorphic loci	observed	expected
20 line A loci / 22 A–Rp1d loci	95% line A	99.2%
20 line B loci / 30 B–Rp1d loci	67% line B	98.4%
Percentage Nonrecurrent Parent for Converted Lines A–Rp1d and B–Rp1d		
2 A632 loci / 21 A–Rp1d loci	10% A632	0.8%
2 A632 loci / 21 B–Rp1d loci	10% A632	1.6%

Markers used are within 0.2 Morgan of 75.4% of genome. A polymorphism may not always distinguish all three lines from one another. The observed values do not total 100%.

The deviation of the B-*Rp1-d* results from expectations might indicate that selection was inefficient for recovery of the desired recurrent parent plant characteristics or that the non-recovered chromsomal regions have little effect on overall B-line plant structure. Although lines A and B are highly inbred, quantitative genetic theory states that a lower original parental level of homozygosity would lead to a lower percentage of recurrent parent. The B-line would simply be less homozygous than the A-line. The general availability of molecular markers should allow further testing of quantitative genetic theories at the molecular level.

Diana Beckman and Edward Weck

URBANA, ILLINOIS
University of Illinois

Glossy linkages

gl9 et: Earlier studies with TB translocations indicated that *gl9* was on the long arm of chromsome 3. F2 data from the cross + *et*/*gl9* + confirm this:

+ +	+ *gl9*	*et* +	*et gl9*
599	187	183	9

lg2 was also involved in this cross. The distance between *lg2* and *et* is too great to provide critical information but the data are consistent with the assumption that *gl9* is distal to *et*.

gl14 wt: Information had been available that both *gl14* and *wt* were on chromsome 2 but linkage data were limited. Backcross data were obtained as follows:

+ +	+ gl	wt +	wt gl
351	34	95	278

The data indicate about 17% crossing over. There is a discrepancy in size between the two cross-over classes. *gl14* is sometimes difficult to classify and in this case there is a deficiency of the combined *gl* class.

G.F. Sprague

Table. F2 breeding behavior in crosses of amt-types (colorless) with various ACR stocks (r-indicates recovery and nr nonrecovery) of the mottled phenotype among the segregants.

	aruq uq	amt-2	amt-3	amt-4	amt-6	amt-7	amt-8	amt-11	amt-12	amt-13	amt-15	amt-4	amt-18	amt-19
ACR(M14)	r	r			nr	r		nr	nr	nr	r		nr	nr
ACR(K55)	r	r	nr		nr	r	nr	nr	nr	nr		r	nr	nr
ACR(w23)	nr	nr	nr		nr	nr		nr	r	nr			nr	nr
ACR,uq	nr	nr	nr	nr		r		nr	nr	nr	r	r	nr	nr
A(m-1)CR	r	nr	nr				r		nr	nr	r		nr	
A(m-19)CR			r		r				r			r	r	

Mottled

A number of aleurone pattern factors (mottled) have been isolated from advanced generations of virus-exposed cultures. When these have been found in differing lines of descent they have been given temporary designations, *amt-1*, *amt-2*, etc. The *mt* designates it as of the mottled phenotype class with the number indicating the sequence of discovery. *amt-1* has been redesignated *a-ruq*, *Uq* by Peterson and Friedemann (Maydica 28:213, 1983). The remaining cases have been less extensively studied. Although of apparently independent occurrence the possibility of genetic similarity remains.

One approach to resolve this question involves response to a series of colored aleurone testers. Each of the mottled types exhibits mutations to both self-colored and colorless individuals. These can be stabilized upon further inbreeding. The colorless mutants were used in the testcrosses. The dominant mutations have been given temporary designations such as *A(m1) CR*; *m* following the *A* indicating the mutation to self color and the number identifying the mottled stock of origin. If a colored stock carries an appropriate regulator then colored × colorless crosses will exhibit some mottled seeds among the F2 segregants.

A small sample of data from such crosses is presented in the table. All of the crosses have not been obtained but the data available suggest that each "mottled" may have its own specific regulator.

G.F. Sprague

Dotted

A series of Dotted phenotypes have been recovered from advanced generations of virus exposed cultures. One, *Dt6*, is on chromosome 4. No linkage has been established for the remainder and they are being carried under temporary designations, *Dt(a)*, *Dt(b)* etc. Each is inherited as a simple dominant and each is of separate origin as judged by line of descent.

Unexpected behavior was noted in the F2's of a series of *dt/dt* crosses. Each *dt* stock was derived from a *Dt:dt* segregation and the *dt* parent had been selfed for at least one additional generation to ensure the correctness of the classification. Among the crosses all F1 seed were colorless and non-dotted. The F2 behavior is illustrated in the table. Three different patterns were observed. In the first group no dotted segregants were observed in F2. Most of the crosses involving *a-ml(Spm)* and *a-m4(Ac)* were in this group.

In the second group the F2 segregations approximated a 1:1 segregation with an excess of the non-dotted class, possibly through failure to recognize kernels with only one or two small dots.

In the third group the dotted class approximates a 1:3 ratio with, in most cases, a deficiency of the dotted class.

F3 progeny tests of the dotted class have been obtained for a number of the crosses tested. Among a large number of F3 progenies none bred true for the dotted phenotype. The range in breeding behavior roughly paralleled that exhibited by the F2's. In a few cases the more extreme dotted F2 kernels gave segregations approximating 3 *Dt:dt*. It is too early to speculate on the origin of *Dt* from the *dtxdt* crosses but some types of interaction between receptor or regulatory loci must be involved. Tests to establish the identity of some of the recovered *Dt* phenotypes are underway.

Pedigree	Phenotype	F2 Phenotypes Dotted	Colorless
a-m1 × *dt(b)*	colorless		all
a-m4 × *dt4*	"		"
a-m4 × *dt6*	"		"
a-m4 × *dt(b)*	"		"
dt2 × *dt6*	"		"
am1 × *dt3*	"	269	282
dt3 × *dt(a)*	"	456	523
dt6 × *dt(a)*	"	386	445
dt(b) × *dt(e)*	"	264	325
a-m4 × *dt2*	"	81	388
dt1 × *dt2*	"	158	393
dt2 × *dt(a)*	"	108	400
dt2 × *dt(b)*	"	131	392
dt3 × *dt(b)*	"	135	391
dt3 × *dt(b)*	"	129	333
dt3 × *dt(e)*	"	50	360
dt4 × *dt(b)*	"	32	133
dt4 × *dt(a)*	"	123	424
dt4 × *dt(e)*	"	147	326

The breeding behavior just described contrasts sharply with *Ac* or *Spm* behavior as illustrated by the following results. The cross of *a-m1/a1 dt2 wx* exhibiting dotting in F1 indicates the presence of *Spm* in the *dt2* parent. F2 and backcross data were as follows:

	Dt Wx	*Dt wx*	*dt Wx*	*dt wx*	*Recombination*
F2	552	53	217	170	24
BC	1107	286	480	1106	26

This appears to be a new location for *Dt* and the symbol *Dt7* is suggested.

G.F. Sprague

Amino-terminal sequence analysis of the embryo-specific *Prot* proteins

The protein products of the *Prot* gene are specific to the maize embryo where they accumulate to high levels during seed development (D. Schwartz, MGG 174:233). Characterization of these proteins with respect to solubility in saline solutions indicates that they belong to the globulin class of seed proteins (A.L. Kriz and D. Schwartz, Plant Physiol. 82:1069). Our previous studies on synthesis of the *Prot* proteins have shown that the polypeptide of higher molecular mass, PROT′, is processed post-translationally to form the "mature" gene product, PROT. The conversion of PROT′ to PROT is dependent on the product of the unlinked gene *Mep*, and embryos homozygous for the recessive *mep* allele accumulate PROT′ rather than PROT. The action of *Mep* is significant in that this product appears to be a protease exclusively specific for PROT′ and, to our knowledge, this is the only protease specific for a seed protein for which a defective allele is available. Characterization of *Mep* activity would therefore be a significant contribution to the knowledge of protein processing in the plant seed. To determine if *Mep* processing occurs in the amino-terminal portion of PROT′, we determined the N-terminal amino acid sequence (20 residues) of both PROT′ and PROT. As shown below, these sequences are not homologous and we thus infer that the *Mep* product proteolytically cleaves PROT′ to form PROT. Note that the sequence of PROT′ does not extend far enough to determine overlap with PROT, so it is not possible at this time to determine the site of *Mep* processing. We are in the process of screening an embryo cDNA library with *Prot*-specific oligonucleotides in order to identify clones corresponding to *Prot*, and eventual DNA sequence analysis of these clones will allow us to determine the precise site of *Mep* cleavage.

Sequences of polypeptides specified by the *Prot-I* allele

PROT′: NH2-XXX XXX Phe Glu Phe Leu Val Leu Met Arg Gln Gly Val Arg Met Tyr Lys Gln XXX Pro/Asn . . .

PROT: NH2-XXX Ser Pro Glu Glu Ser Ser Glu Asp Asp Asp Tyr Gln Phe Lys Val Lys Gln Gln Pro . . .

Alan L. Kriz

Effect of the nuclear background on the plasmid-like mitochondrial DNA molecules S1, S2, R1 and R2

The presence of 2 low molecular weight plasmid-like double-stranded DNA molecules, S1 and S2, in undigested mitochondrial DNA (mtDNA) preparations is characteristic of male-sterile maize cytoplasm of the S-type (*cms-S*). In some fertile Latin American races of maize carrying RU cytoplasm, plasmid-like mtDNA molecules, designated R1 and R2, are present. R2 is nearly indistinguishable from S2; both are about 5.4kb in length and differ in a single *BglI* site that is missing from S2. R1, at 7.4kb in length, is approximately 1kb longer than S1 and contains about 2.6kb that are unique to R1 (R.R. Sederoff and C.S. Levings III, Gene Flux in Plants, pp. 91-109, 1985).

The relative amounts of the S1 and S2 episomes in *cms-S* strains are determined by the nuclear background. In most inbred lines carrying *cms-S*, S1 and S2 occur in equimolar amounts. Several inbred line backgrounds in which this is not the case have been identified. M825 and the related R825 exhibit a reduced amount of S2 compared with S1; in 38-11 the reverse is true. It was previously demonstrated that when an inbred line whose mtDNA exhibits equimolar amounts of S1 and S2 is recurrently crossed with M825 as the male parent there is a decrease in the amount of S2 as compared with S1 (J.R. Laughnan et al., Stadler Symp. 13:93, 1981). The result is the same when the inbred line 38-11 is recurrently crossed with M825; the "reduced S1" pattern shifts to the "reduced S2" pattern characteristic of M825. Not only are S1 and S2 under nuclear control, they respond to the nuclear background independently of one another. It also should be noted that while cytoplasmic reversion to fertility of *cms-S* in the nuclear backgrounds M825, 38-11, H95 and W182BN results in the loss of the S1 and S2 episomes, revertants in the WF9 inbred background retain S1 and S2 in the same relative amounts as do their sterile progenitor strains. Therefore a number of fertile and sterile episome-containing strains are available for studies of the effects of changes in the nuclear background.

Agarose gel electrophoresis studies of ethidium bromide-stained undigested mtDNA from various episome-containing strains converted to different nuclear backgrounds are now essentially complete (Table 1). Each strain shown in the table was crossed a minimum of 6 times to the inbred line background indicated. It has been demonstrated that 5 crosses are sufficient to shift the 38-11 pattern to the pattern characteristic of M825.

The *cms-S* versions of a number of strains with equimolar amounts of S1 and S2 were recurrently backcrossed to both M825 and 38-11. Following conversion to the M825 nuclear background the "equimolar" pattern of S1 and S2 shifts to the "reduced S2" pattern and following conversion to 38-11 the pattern shifts to the "reduced S1" pattern. The "reduced S2" pattern characteristic of M825 is reversible since conversion to an "equimolar" nuclear background results in equimolar amounts of S1 and S2. The backcrossing of M825 to 38-11 has been carried out but the mtDNA has not been analyzed. We predict that it would exhibit the "reduced S1" pattern based on the other results presented herein. The "reduced S1" pattern of *cms-S* 38-11 strains becomes equimolar following conversion to "equimolar" strains; following conversion to M825 the pattern is that of "reduced S2".

The S1 and S2 episomes present in *cms-S* WF9 cytoplasmic revertants retain their ability to respond to the nuclear background. Revertants converted to M825 exhibit the "reduced S2" pattern while the same rever-

Table 1. Effect of conversion to different nuclear backgrounds on plasmid-like DNA molecules.

Relative amounts of plasmid-like DNA molecules in the progenitor (Cytoplasm Type)	Pattern of Progenitor	Pattern following conversion to: Equimolar Inbred Lines	M825	38-11
Equimolar S1 and S2 (S-Sterile)	— —	NA	— ····	···· —
Reduced S2 (S-Sterile M825)	— ····	— —	NA	not tested
Reduced S1 (S-Sterile 38-11)	···· —	— —	— ····	NA
Equimolar S1 and S2 (S-Fertile Revertant WF9)	— —	NA	— ····	···· —
Equimolar R1 and R2 (RU Fertile Accessions)	— —	— —	— —	— —

NA = not applicable

tants converted to 38-11 exhibit the "reduced S1" pattern. Whatever mtDNA changes occurred upon reversion to fertility, the S1 and S2 copy numbers are still under nuclear control. Reversion to fertility in WF9 does not alter the responses of S1 and S2 to the nuclear background.

Several RU cytoplasm accessions containing the R1 and R2 episomes were crossed by "equimolar" strains, by M825 and by 38-11. As expected, no effect of conversion to the "equimolar" strains was observed. However, conversion to 38-11 also exhibited no change in the relative amounts of R1 and R2. Since S1 responds to the 38-11 nucleus and R1 does not, this could mean that the target site for action of the 38-11 nuclear gene(s) in the S1 episome is in that region of S1 that contains no homology to R1. However, this region is present in S2 (and R2) and since S1 and S2 respond differentially to the nuclear background this is not a satisfactory explanation. This implicates a site or sites in the main mitochondrial genome as the target for the nuclear gene control. This conclusion is supported by the fact that R2 does not respond to the M825 nucleus as does S2; there is no change in the relative amount of R2 in the M825-converted RU cytoplasm. The 2 episomes, R2 and S2, are almost identical and if the target for nuclear control were present in S2 it would be expected to be present in R2 as well; results indicate that this is not the case. Restriction enzyme profiles show that the main mitochondrial genomes of *cms-S* and RU are very different, RU being similar to normal cytoplasms (A.K. Weissinger et al., PNAS 79:1-5, 1982). These results support the hypothesis that the target(s) for nuclear gene control of the maize episomes S1 and S2 resides in the main mitochondrial genome, not in the episomes themselves.

Heidi Feiler, Carol Leja, Susan Gabay-Laughnan and J.R. Laughnan

Reversion of *cms-S* to fertility in the presence of a nuclear restorer gene

Studies have been carried out to identify inbred line backgrounds in which the S1 and S2 mitochondrial episomes characteristic of S-type male-sterile cytoplasm (*cms-S*) occur in different relative amounts. Only a few nuclear backgrounds are known in which the molar amounts of the S1 and S2 molecules differ (J.R. Laughnan et al., Stadler Symp. 13:93, 1981). In all other inbred line backgrounds examined, S1 and S2 occur in equimolar amounts. A number of the more modern inbred lines not previously available with *cms-S* have been converted to S-type cytoplasm to determine if they condition S1:S2 differences. None was identified. In the course of these studies a fertile strain of the inbred line H95 (restores *cms-S*) which pedigree records indicated should carry *cms-S* was identified as lacking the linear S1 and S2 molecules entirely. This result could be explained by a seed mix with the normal (N) or C cytoplasm versions of H95 (T cytoplasm H95 is sterile) or by reversion to fertility of *cms-S*. In 2 of the 3 nuclear backgrounds in which cytoplasmic reversion to fertility has so far been characterized, the reversion event results in the loss of free S1 and S2 molecules. No *cms-S* strains have been observed to lose S1 and S2 and remain sterile. Because the H95 inbred line restores fertility to *cms-S*, molecular analysis of the mitochondrial DNA (mtDNA) was required to differentiate between seed mix and cytoplasmic reversion.

DNA was isolated from mitochondria of N and C cytoplasm sources of H95 as well as from *cms-S* W23 and from two H95 fertile S-cytoplasm strains derived by conversion of *cms-S* W23 to H95, one which maintains S1 and S2 as linear episomes (designated H95S+) and the strain in question which lacks S1 and S2 (designated H95S−). Ethidium bromide stained restriction endonuclease profiles were obtained after digestion of the mtDNA with enzymes *Hin*dIII, *Pst*I and *Sal*I. The restriction fragment patterns clearly demonstrated that the H95S− mtDNA was indeed of the S type and no seed mix had occurred. These gels were blotted onto Biodyne nylon membranes and probed with the following DNA fragments: (1) an S1 fragment which contains some homology to S2, (2) an S2 specific fragment, (3) an S1 specific fragment and (4) the cytochrome oxidase I (COXI) gene plus flanking regions. The S1 and S2 probes substantiate the fact that *cms-S* W23 and H95S+ maintain free copies of S1 and S2. They also indicate that integrated copies of S2 and a lesser number of integrated copies of S1 are present. In contrast the mitochondrial genome of the H95S− strain lacks S2 integrated copies as well as both the S1 and S2 free episomes; S1 integrated copies are retained. The COXI probe detects differences between H95S− and its progenitor strain H95S+. In the WF9, 38-11 and M825 nuclear backgrounds, cytoplasmic reversion to fertility also involves reorganization in the COXI region as well as in the S1 and/or S2 regions of the mitochondrial genome.

These data tell us that there has been reorganization of the *cms-S* mtDNA associated with loss of the S1 and S2 episomes. Loss of this type has been observed previously only in cytoplasmic reversion to fertility. However, it can not be determined by mtDNA studies alone whether reversion occurred in this H95 case. Genetic studies have been conducted on the H95S− strain in order to make this determination. Since the H95 inbred line carries a restorer-of-fertility (*Rf*) gene the H95S+ and H95S− strains are both fertile. Cytoplasmic reversion to fertility must be diagnosed by backcrossing the H95S− strain to maintainers of *cms-S*. This has been done and the H95S− strain retains complete fertility after 2 backcrosses by maintainer strains. This indicates that the mtDNA changes observed in H95S− were associated with reversion to fertility. This is the first reported case of cytoplasmic reversion to fertility occurring in the presence of restorer genes.

Heidi Feiler, Gracia Zabala,
Susan Gabay-Laughnan and J.R. Laughnan

VICTORIA, BRITISH COLUMBIA, CANADA
University of Victoria

Papago Flour Corn as a source of *a3*

Some years ago we grew some Papago Flour Corn plants from seed obtained from the Maize Genetics Cooperation Stock Center. Although the plants were shivering from the time they emerged, they did eventually produce some pollen which I used to make some crosses to some of my late stocks. Some years later I used some plants derived from these crosses in studies with the recessive anthocyanin factor *a3*. It appeared that the plants already carried *a3*, and, as far as I could tell, the Papago Flour Corn was the only possible source of the *a3* allele. This year I grew some more Papago Flour Corn plants to

confirm their plant phenotype. The plant pigment distribution is typical of that expressed in *r-r b a3* plants. I am unable to tell from the literature where the *a3* factor came from that was originally described by Lindstrom (MNL 8:10, 1934), but Papago Flour Corn is certainly one possible source of the allele.

E.D. Styles

Pigments in the cob pith

In 1927 Demerec (J. Hered.18:421) described a factor (*Md*) that determines pigment in cells surrounding the vascular bundles that themselves surround the 'pit' (pith). The pigment controlled by *Md* is independent of cob and pericarp pigment, and, according to Demerec, is not changed in the presence of homozygous *a a*. We have several stocks segregating for this type of pigment distribution, but we also have stocks that are pigmented in the pith itself, the most common of which are those carrying a 'strong' *P-RR* allele, which consistently have pigment in the cob pith as well as in the rest of the cob and pericarp. Such *P* alleles also determine tassel pith pigment, and plants can be characterized by breaking the tassel to expose the pith. *P-WR* and *P-WW* plants do not have this pith pigment, but another *P* allele which determines a 'grainy' pericarp and cob pigment consistently has strong pith pigment.

As well as stocks with *P*-determined pith pigments, we have stocks segregating for a factor determining strong anthocyanin pigment in the pith. *Pl* is required (we have not eliminated the possibility that a *Pl* variant might be responsible, although we think not), and also required is an *R-g* allele that 'responds' to *a3* by producing plant anthocyanins (i.e., Stadler's Group D or Group 4 *R* alleles). Expression is best when the plant is heterozygous or homozygous *a3*, but we have at least one *'R-g'* allele, that in *A3* stocks responds to the presence of *'Pth'* by producing not only pith pigment, but red anthers and pericarp pigment also. No other *R* or *B* alleles allow the pith pigment to be expressed, not even *R-ch'*, *r-ch*, or *B* which together with *Pl*, can produce strong purple pigments in other cob tissues.

E.D. Styles

'*R*-Mosaic'

We have reported previously (MNL 56:160) that certain *R-g* alleles, in the presence of *a3*, demonstrate a range of unstable plant color phenotypes including a diffuse purple and various grades of red and green variegation. Such variegations can be expressed in the pericarp as well as in leaves, tassels, culms, etc., but the pericarp patterns are not that obvious against the dark aleurone pigment. Incorporating *c* into these stocks produces a colorless aleurone but still allows full expression of the pericarp pigment. The patterns of sectoring in the pericarp mimic the sometimes uniform, sometimes irregular patterns that are typical of *P*-mosaic. Only the pigment differs.

E.D. Styles

WALTHAM, MASSACHUSETTS
University of Massachusetts

Palomero Toluqueno and certain Andean maize carry the short rachillae and reduced cupule traits probably descended from an independent domestication of teosinte

The string cob trait that became the basis of the slender, eight rowed sweet corn inbred MA-W401 and its hybrid, Candystick, was initially extracted from Confite Morocho of Peru (W.C. Galinat, Mass. Agric. Exp. Sta. Bul. 577, 1969.). It is characterized by a very slender cob resulting from three factors: reduced pith, reduced cupules and short rachillae. In most backgrounds the thick so-called normal cob segregates out in only 1/16 of the F2 as if two incompletely dominant genes controlled string cob. In teosinte the short rachillae trait is combined with well-developed cupules as if these were independent traits.

Allelism tests made by hybridizing W401 with White Cloud Popcorn (a white rice variety related to Palomero Toluqueno) have continued to breed true for the short rachillae. Selection for kernel row numbers above 20 increased the pith area but the cobs remained slender (semi-string cob) due to their short rachillae. The kernels of these high-row selections became long and thin, of the Shoe Peg type, and the cupule became narrow and greatly reduced, sometimes to the noncupulate condition.

It is suggested that the domestication of teosinte occurred at least twice and by different pathways. It seems that Chalco teosinte may have been domesticated by a combination of cupule reduction and kernel elongation that led to such diverse modern derivatives as Palomero Toluqueno, Confite Morocho and Gourd Seed Dent. Most maize may be predominantly from another independent domestication, apparently involving the tunicate locus and Guerrero teosinte. In this case the glumes become soft and the rachilla elongate in a way that elevates the grain almost beyond the chaff.

Selection by man for recessive alleles for a thick cob at the string cob loci increased the vascular supply that was necessary for the development of a more productive ear. The long rachillae plus the thickened pith area combine to make the enormous cob of modern maize.

Walton C. Galinat

Epimutations and morphogenesis

Phytomers and phases represent different levels of epigenetic control over the process of morphogenesis. As a result the form of particular organs and their repetitious sequence is programmed to function at high fitness in the overall design of the whole individual. The phenotype that is manifest during morphogenesis may change by epimutation just as somatic mutations produce chimeras. But epimutations are usually repaired during or just before gamete formation so that fertilization can reset the clock and repeat the whole sequence of morphogenesis in contrast to the standard mutations that can become fixed within the progeny (review by R. Holliday, Science 238:163, 1987).

In its original expression, A. Gray (Structural Botany, Amer. Book Co., New York, 1879) and W.E. Worsdell (The Principles of Plant Teratology, Ray Soc., London, 1915-16) considered the plant to be formed by a succession of

individual units (phytomers or phytons) each consisting of an internode with a leaf at its upper end and a root or potential root(s) at its base. This concept was modified by Galinat (Bot. Mus. Leafl., Harvard Univ., pp.1-19, 1959) with the vegetative phytomer formed by the leaf, its axillary bud and prophyll all at the base of the internode and then extended to the floral axis with homologous transformations in all lateral components. Occasional slippage, stuttering or other imperfections resulting from a non-rigid coupling between components of the phytomer and arrangement within the phytomer do not negate the role of the phytomer as a guideline, if not a framework, for directing morphogenesis.

Control at the level of the phase is higher than that of the phytomer. It is most obvious in the vegetative and floral phases, both of which are subdivided into juvenile and adult stages and in the case of maize and teosinte are further differentiated into separate male and female inflorescences. Nevertheless, underlying control at the level of the phytomer is a basic pattern of construction with the manifestation of its floral phase components being markedly different from their vegetative homologues.

The epimutations of morphogenesis occur by a methylation or demethylation of various genes according to the phytomer and phase programs. In reviewing this process, Holliday (1987) states "the control of gene expression in higher organisms is related to the methylation of cytosine in DNA, and the pattern of methylation is inherited."

A study of inherited defects in the normal methylation process that have been either induced by mutagens or are just of spontaneous origin should be helpful in understanding the epigenetic control of phase change and phytomer manifestation. Such mutations include the *Cg* (corn-grass) and *Tp* (teopod) loci that have difficulty in shutting the vegetative phase off after the floral phase is turned on as well as the contrasting loci of *is* (interrupted spikelets; after starting to use *ir* for interrupted rachis, I found it was being used by molecular geneticists to mean inverted repeat) and *Bif* (one of Neuffer's EMS induced mutants on chromosome 8S). The *is* mutant produces a barren condition in sectors at the base of the rachis while the *Bif* mutant does a similar thing at the apex of the rachis.

Walton C. Galinat

Recognition of phytomer homologies by the *B-b* allele

The *B-b* allele ordinarily puts a red or purple bar on the glume cushion at the base of the male glumes. The glume cushion is homologous to the leaf subtending an axillary bud in the phytomer. In teopod (*Tp*) and corngrass (*Cg*) the glume cushion may elaborate into a spathe subtending the spikelets, both male and female. In the presence of the *B-b* allele, the entire spathe may become red and the basal glume area has no glume cushion or bar color. It takes a smart gene to know what it is supposed to do and where to perform. There is a strong background effect on the performance of *B-b* and so it does not always live up to the above expectations.

Walton C. Galinat

Stabilizing the links connecting teosinte and maize

As a result of attempts to reconstruct stable stocks of the links that must have once been intermediate steps in the evolutionary emergence of maize from teosinte, I have come to some conclusions about how to proceed. The key trait genes have various types of behavior in each other's background. They may be silent with zero penetrance, especially when heterozygous, or they may have unstable expression, even along the length of a single ear or spike.

Judging from observations of the vascular anatomy at various positions along a rachis, it appears that the background effect involves a congruous balance between the vascular supply system and the energy requirements of its customers, the spikelets. The elaborate vascular system of the modern cob is both excessive and incongruous with a stable expression of the solitary female spikelets borne in two ranks that characterize teosinte. On the other hand, the simple vascular supply of the female spike in teosinte is inadequate to support a stable expression of the more energy-demanding traits of paired female spikelets and/or many-ranked spike of maize.

By trial and error it was discovered that the ideal background for a stable expression of the isolated key traits was a tiny, eight rowed popcorn inbred, MA1001, that we had bred in attempting to reconstruct the Tehuacan so-called "wild corn". The connecting links on this reconstructed background should be useful experimental material for genetic, molecular, and developmental studies designed to study the origin of maize.

Walton C. Galinat

Breaking the cycle of bad plants - bad seed - bad plants with a winter crop in temperate Chile

The summer of 1986 was the coolest and wettest in Massachusetts that I have experienced there in 32 years. My corn grew poorly, flowered late and produced poor, somewhat immature seed that was similar to that which I usually obtained from a winter crop planted in early November in the Homestead area of southern Florida. Had my winter crop been planted in Florida with this bad seed, it would have produced bad plants bearing bad seeds. But my winter crop of 1986-87 was grown in the Corn Belt of Chile in Rancagua about 100 miles south of Santiago. Although the poor quality of seed planted gave a poor stand of weak plants that flowered later than expected, under the Rancagua conditions, it produced good quality seed. When this good seed was planted and grown during this past favorable summer in Massachusetts, again normal good quality seed resulted. The sick cycle had been broken.

The consequences of this sequence of events might be falsely interpreted by some as resulting from epimutations to defective seed such as may occur in somaclonal variation from the weak plants regenerated from a scutellar clone that are subsequently repaired or reprogrammed in germline cells—a suspicion that cannot be ignored.

Walton C. Galinat

Sexual transmutation and the origin of maize

Of significance to understanding the assemblage and affiliation of secondary sex genes is an enhancer gene for single spikelets that has been discovered in crosses with Cuzco flour corn. This modifier gene raises the level of single spikelet penetrance by extending its phenotype to the male spikelets in the maize tassel. Is one to conclude that the maize tassel came from the teosinte ear? Not

unless one believes in the transmutations of Iltis. If the teosinte ear is not the precursor of the maize ear as claimed by Iltis, how then is it possible to genetically transfer the phenotype of single female spikelets from teosinte to both the maize ear and maize tassel? Also of importance is the fact that teosinte tassels that are feminized by any of the known tassel-seed genes or by environmental conditions have solitary female spikelets enclosed in indurate cupulate fruit cases rather than Tehuacan type ears as expected by Iltis. No transmutation of the Iltis type has ever been found to segregate from maize-teosinte hybrids. Only specific genes controlling the key traits separating teosinte and maize are found in their segregations.

Walton C. Galinat

WEST LAFAYETTE, INDIANA
Purdue University

Transformation of *Adh* null pollen to *Adh+* by "macroinjection"

The lack of an effective transformation system to introduce foreign and/or engineered DNA into developing, reproductively competent maize cells continues to be one of the major limitations in plant molecular genetics. One approach to the delivery of exogenous DNA into maize involves the direct transformation of gametes. This technology could circumvent the expense, time, and genome stress associated with the transformation of cells in culture and subsequent plant regeneration. The rapid cell division and high level of cell-cell communication in the developing maize tassel provides a possible window of opportunity for the introduction of DNA into male gametocytes. Upon cloning the *Adh1* locus in 1982, we decided to macroinject this gene into the developing spikelets of *Adh1* null maize plants. Although the approach was a canonical "long shot", the ease and rapidity with which the experiment could be performed and the results assayed by pollen staining, and the potential value of a pollen transformation system, overcame our unease regarding the rather foggy biological underpinnings of our approach. Much to our surprise, the first experiments performed suggested transformation of *Adh1* null pollen to *Adh+*. This original result led to intermittent experiments between 1982 and 1985, first at the International Plant Research Institute in San Carlos, California and later at Eli Lilly and Company in Greenfield, Indiana, designed to test this approach.

A number of preliminary experiments were performed to investigate the optimal mechanics of the macroinjection procedure. Seed from a homozygous *Adh1* null maize line (kindly provided by Dr. Drew Schwartz, Indiana University) were individually planted in ten gallon pots in the greenhouse and grown under high intensity lighting. Generally about 35 to 45 days after planting, depending on the growth conditions, a window was cut into the maize stalk just above the base of the developing tassel. The immature tassel was then gently pulled through the window and supported on a solid base for subsequent manipulations. A few young spikelets at various points along the tassel were then removed and microscopically examined for their stage in pollen development. We performed the majority of our macroinjections at a stage where the gametocytes of the central (and, therefore, furthest advanced) spikelets were in pachytene. Hence, all other spikelets injected would contain gametocytes that were at or just prior to pachytene and would, perhaps, be more likely to meiotically recombine any injected DNA into recipient chromosomes. We also found that handling and macroinjection of spikelets significantly younger than this led to death of the pollen-producing cells. We injected approximately 0.5 microliters of a solution of 0.1M glucose, 50mM Tris-HCl, 5mM $MgCl_2$ containing 0.25 micrograms/microliter DNA. The injections were into the approximate center of individual spikelets, thus bathing the developing anthers. A hand-drawn needle with an inner diameter of approximately 15 microns and an outer diameter of approximately 150 microns was found to be optimal for the injection process. Larger outer diameters tended to kill the immature spikelet and smaller inner diameters did not allow efficient delivery of the DNA solution. All of the reasonably developed spikelets on a tassel were individually injected, any spikelets not injected were excised or marked with a felt tipped pen as uninjected controls. The young tassel was then gently replaced within the central plant stalk and the previously excised window taped back into place. Within 15 to 19 days, depending on growth conditions, the somewhat distorted tassel shed pollen. Collected pollen samples were first weighed (estimating 3000 pollen/milligram) and then stained for ADHI activity by standard procedures (Freeling, Genetics 83:701, 1976).

In 14 preliminary experiments, in which the individuals performing the injections and pollen scoring were unaware of the nature of the injected solutions, 1 of the 8 controls (half no DNA and half *Adh−* plasmid DNAs) and 4 of the 6 *Adh1* injections yielded *Adh+* pollen. Since even a low level of contamination by pollen from an *Adh1+* plant would totally invalidate our approach, we found that we could not have any mature *Adh1+* plants in the same greenhouse with our injected plants and that the individual performing the pollen staining could not have recently visited a greenhouse or field that contained shedding *Adh1+* plants. Also, samples of *Adh1+* pollen commonly used as controls for the staining procedures could also be a major source of contamination if taken through the staining procedure by the same person or in the same room as the pollen from the injected plants. Despite all these problems, we were able to derive some provocative results when all known sources of pollen contamination were eliminated. The Table presents data derived from 3 such experiments.

In the properly controlled experiments performed, we found 19 out of 34 *Adh1* injections gave an apparent transformation frequency of .005% or higher. Only 3 of 20 control injections of *Adh1-* DNA or no DNA gave a similar high frequency of apparent transformants. We assume that these 3 false positives were due to mislabelling of a tube containing *Adh1+* DNA as a control, from contamination by putatively transformed pollen shed in the greenhouse or from some unknown source. In no case did the controls yield more than 4 putative *Adh+* pollen grains in a single staining. The highest frequency of putative transformants with an *Adh1* injection observed in any experiments was 0.1%.

Since we made no extensive attempt to recover putatively transformed pollen through a fertilization event, we have no absolute evidence to indicate that recovery of

Material Transformed*	ADH+/Total Pollen* Pollen Collection Date** 7-11	7-12	7-13	7-14	7-15
No DNA	-	-	-	-	1/430
No DNA	-	-	-	-	1/256
Adh1	-	-.	0/50	3/52	0/158
Adh1	-	-	2/220	0/38	1/8
Adh1	-	1/10	2/38	0/20	-
Adh1	96/434	13/150	2/100	-	-

	2-18	2-19	2-20	2-21	2-22	2-23
No DNA	-	0/16	-	-	-	-
No DNA	-	-	-	-	0/18	1/141
Adh1-	0/28	2/80	0/54	-	-	-
Adh1	-	0/40	3/60	-	-	-
Adh1	-	-	-	149/532	79/150	0/33

	4-5	4-6	4-7	4-8	4-9	4-10
No DNA	1/190	0/64	-	-	-	-
No DNA	-	0/26	-	-	-	-
Adh1	14/34	1/42	1/8	1/4	-	-
Adh1	1/112	0/150	0/30	-	-	-
Adh1	-	0/68	0/70	0/24	0/40	1/20
Adh1	-	0/128	-	0/40	0/10	-
Adh1	-	-	-	0/4	0/24	0/26
Adh1	-	-	-	0/92	0/60	0/18
Adh1	-	-	-	0/44	0/4	-
Adh1	-	-	-	0/3	-	0/20

* Data are presented as the number of pollen staining for ADH activity divided by the number (in thousands) of pollen grains scored.
** Pollen was collected from the same tassel on subsequent days and separately stained for ADH activity.
* Each line represents a single DNA sample injected into a single plant.

ADHI+ pollen from macroinjected *Adh1* null plants was not due to some unidentified source of pollen contamination. The tendency of a putatively transformed plant to continue to produce ADH+ pollen over 2 or more days, while those around it remained ADH−, supports a biological interpretation. Moreover, many of the putatively transformed pollen stained pink rather than fully purple, indicating a low content of ADH enzymatic activity. All *Adh1+* pollen controls and contaminants consistently stained fully purple. A low level of ADHI enzymatic activity in a pollen grain could result from a transient, non-integrating transformation by *Adh1* DNA or from an inefficient expression of transformed *Adh1* DNA due to a structural alteration in or position effect on the integrated gene.

We eventually terminated these experiments for a number of reasons. Variations in the time of injection, the injection buffer, and the presence of various replicative or integrative components in the transformation vector did not seem to improve the transformation efficiency. In addition, we did not become significantly more successful in achieving high apparent transformation frequencies (>1/2000) with practice. More importantly, the requirement to delegate an entire greenhouse and various personnel exclusively to this project became prohibitively expensive. Had we been able to perfect a technique for reproducibly storing pollen in a viable form, we would have been able to split the pollen collected into stored and stained aliquots. Any injected plants yielding a high level of ADH+ pollen could then be used in a pollination whenever the appropriate silks were available. The chief reason for the cessation of these experiments, however, was our decision to await the construction of recombinant vectors that could drastically simplify the interpretation and recovery of putative pollen transformants. These vectors, containing plant regulatory sequences that can drive gene expression in maize pollen (for instance, the promoter from maize *Adh1*) linked to selectable or scoreable markers not naturally encoded by maize, are now available.

J.L. Bennetzen, C. Lin,
S. McCormick and B.J. Staskawicz

WOOSTER, OHIO
The College of Wooster

Cloned maize genomic sequences recovered by homology to meiosis-specific cDNA clones from *Lilium* or their maize small heat shock gene cognate

A previous report (see MNL 60:71, 1986) described the recovery of a maize genomic DNA clone through its sequence homology to expressed meiotic prophase repeat (EMPR) cDNA clones from *Lilium*, a clone encoding a product with regions of strong amino acid similarities to small heat shock protein of soybean. Further studies have shown that this clone reanneals with high fidelity to an mRNA induced at least 50-fold in heat-shocked maize somatic tissue. A larger genomic clone containing the entire structural gene plus flanking regions has now been recovered. Collaborative sequencing studies (Dietrich, Bouchard, DeSilva, and Sinibaldi, J. Cell Biol. 105:245a, 1987) have now been performed on an extensive segment of this clone containing the gene and associated regulatory regions, and it appears by several criteria to be a genuine maize small *hsp* gene.

Maize cognate genomic clones have been recovered through their reassociation homology to either lily EMPR clones or the maize small *hsp* clone. They appear to fall into two classes: those containing inserts which reassociate with both the lily meiotic and maize small *hsp* gene probes, and those containing inserts reassociating only with the maize small *hsp* gene probe. Plasmid subclones for examples of each have been produced and are being characterized. Interestingly, RNAs complementary to these examples of both classes of cognate sequences are present in heat shocked and normal maize somatic tissues at comparable levels. Additional studies are underway to further examine the relationships and expression patterns of this complex group of related sequences.

Robert A. Bouchard

WOOSTER, OHIO
The College of Wooster
LA JOLLA, CALIFORNIA
The Salk Institute
LONDON, U.K.
Queen Mary College, the University of London

Abundance of oligo-dC/oligo-dG runs in the genomes of maize and other eukaryotes

The existence of "specious positive" genomic sequences, clones for which are frequently recovered by annealing of oligo-dC/oligo-dG tails in cDNA plasmids to genomic sequences (see MNL 60:72, 1986), has been further investigated. Quantification of the frequency of such sequences in maize DNA was performed by plating a maize phage-λ genomic DNA library at several dilutions and challenging for positive plaques with ^{32}P end-labelled synthetic oligo-dC probe. The result is that 2% of all clones from such a

library (average insert size 15,000 ntp) contain at least one oligo-dC/oligo-dG run. Such clones are readily detectable after brief radioautographic exposures and appear as strong positives on an overnight radioautogram. Of twelve specious positive clones recovered as phage which were examined on Southern transfers, one contained two positive fragments, suggesting the possibility that there may be some clustering of such regions in the genome. It thus appears that oligo-dC/oligo-dG runs are a widely distributed component of the maize genome.

We felt it would be of interest to determine whether this situation was peculiar to maize. Accordingly, we have examined the occurrence of such regions in other selected eukaryote genomes by comparing the relative intensity of hybridization to the synthetic probe of their DNAs with that seen for maize DNA. While the exact relative quantification is still preliminary, it is evident that such sequences are a widespread feature of higher eukaryote genomes, although their abundance varies considerably. Among monocotyledonous plants, wheat (*Triticum aestivum*), the closest relative to maize examined, has about three times the maize abundance per unit weight of DNA of oligo-dC probe positive sequences. Of other selected monocots, *Allium cepa* has about one-third the oligo-dC probe positive sequence abundance of maize, while *Yucca schidigera* and *Asparagus officinalis* have 4-5 times the abundance of maize. Abundancy differences may occur over short evolutionary distances, since in the genus *Lilium* the species *L. speciosum* shows four times the abundance of oligo-dC probe positive sequences as *L. henryi* or *L. longiflorum*. Oligo-dC probe positive sequences are also a feature of some dicotyledonous plant genomes, occurring in *Nicotiana tabacum* and *Hypochaeris radicata* with about twice the abundance seen in maize. In addition, they are present at significant levels in some animal genomes, such as *D. melanogaster* (at comparable concentration to maize) and salmon sperm DNA (*Salmo* ssp.) (at *very high* abundance; at least 25 times that seen in maize). Oligo-dC probe positive sequences are however relatively rare in some eukaryotic DNAs, such as those of *Phaseolus vulgaris* and *Bos taurus*, both of which show less than one-tenth the maize abundance; and they are entirely absent from the DNA of the prokaryotes *E. coli* and phage λ.

The biological significance of these sequences, if any, remains to be elucidated. Two sets of observations do suggest that their presence could have some impact on the genome. The presence of such short oligo-dC/oligo-dG runs has been shown to induce altered conformation upon neighboring regions when the DNA is placed under torsional stress (Kowhi-Shigematsu and Kowhi, Cell 43:199, 1985). Such runs also may positively influence the level of action of meiotic recombinase enzymes on DNAs containing them (Hotta & Bouchard, unpublished). In any event, as we have previously noted, the existence of such sites presents an important practical problem for workers using heterologous plasmid probes containing inserts with oligo-dC/oligo-dG tails (as many cDNA plasmids do) for the purpose of recovering maize genomic clones containing genes with sequence homology to those inserts. This caveat from maize should now be generalized to a number of other organisms, a number of which also have some degree of genetic interest.

Robert A. Bouchard, Michael McKeown
and John S. Parker

* 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
r refers to numbered references in Recent Maize Publications section

CHROMOSOME 1
Cat2 cDNA clone, RM, BS, transcripts --Bethards &, r52; Scandalios, r484; Skadsen &, r514
Reduction in endosperm due to paternal deficiency enhanced by maternal duplication: TB-1Sb by TB-1Sb, TB-1La, & TB-1OL19 but not by TB-4Sa, TB-5La, or TB-1OL32;
TB-1La by TB-1Sb, TB-1La, TB-4Sa, & TB-7Lb; TB-4Sa & TB-5La not by TB-1Sb; TB-1OL18 by TB-1Sb; TB-1OL19 by TB-1La --Birchler &, r54
Adh1-3F1124: Mu3 insertion in 5' region, organ-specific expression --Chen &, r88
P1 clone, from P1-vv via Ac; RM; cotwins carry transposed Ac elements on the same genomic fragment --Chen &, r89
Knob distribution in inbreds and varieties --Chughtai &, r96
cfr1: coupling factor reduction, affects chloroplast ATP synthase; uncovered by TB-1Sb --Echt &, r155
QTLs near P1, Mdh4, Adh1, Phi1; notably grain weight, ear characters;distorted ratios --Edwards &, r157, Stuber &, r545; Wendel &, r604
Adh1: 5' SI nuclease hypersensitive sites, dimethyl sulfate footprinting --Ferl &, r173, r174, r175; Paul &, r406
ms28 uncovered by TB-1Sb --Golubovskaya, r204
Adh1 promoter, modifications, chimeric constructs --Howard &, r233; Walker &, r593
bz2-mu1, bz2-mu2: clone, Mu1.4 inserts, RM, transcripts --McLaughlin &, r352
hcf3, hcf6, hcf31 uncovered by TB-1Sb; hcf2, hcf4, hcf12, hcf13, hcf41 uncovered by TB-1La; hcf44, hcf50 uncovered by TB-1Lc --Miles &, r365
Bz2 clone, from bz2-m via Ds; RM --Theres &, r558
P1-OVOV contains Ac in inverted order vs. P-VV --Peterson, 62:42
P1 clones from P1-vv, P1-rr, cDNA, transcripts --Lechelt &, 62:47
trisomel, duplex & simplex ratios --Doyle, 62:49
hcf3-Mu --Cook &, 62:50
QTLs for ear height vs. RFLPs --Grant &, 62:71
RFLP map --Grant &, 62:71, Murray &, 62:89
Ac transpositions average 34 cM from P1-VV --Chen &, 62:97
br1 3 Amp1 14 Mdh4 2 bz2 22 Pgm1 --Agnelli &, 62:102
NPI309-Adp1, NPI482-A1-2 on 1L; NPI481-L2 on 1 or 5 --Wright &, 62:104
w*-495A allelic to w*-571C, 8% to wx1 T1-9(8389)(1L.74), 26% to wx1 T1-9(035-10)(1L.89);
bz2 24 zb7 5 gs1 26 bm2; Les7 shows no linkage with 1L markers; dek22 shows no linkage with wx1 T1-9(4995) --Sisco, 62:124

CHROMOSOME 2
Knob distribution in inbreds and varieties --Chughtai &, r96
QTLs near Dia1, B1; notably grain weight, ear number; distorted ratios --Edwards &, r157, Stuber &, r545; Wendel &, r604
hcf1, hcf15 uncovered by TB-1Sb-2L4464 --Miles &, r365
Krn2 (tr2), 1tel linkages --Miranda &, 62:39
Pxl: wx1 T2-9d (2L.83) linkage; v24 16%; pgspt*-579B 5% -Whalen &, 62:34
trisome2, telo2Sa, duplex & simplex ratios --Doyle, 62:49
Les1: sk1 3%; wt1 4%. dek16 & dek23 tightly linked --Hoisington, 62:53
b-Perumu5, b-Perumu216, b-Perumu218, b-Perumu220 --Chandler &, 62:56,58
QTLs for ear height vs. RFLPs --Grant &, 62:71
RFLP map --Grant &, 62:71, Murray &, 62:89
NPI32-null, NPI310-Adp2, NPI468-A1-3, NPI471-L3 on 2L; NPI473-Oec2, NPI417-null on 2S; NPI483-null on 2 --Turner &, 62:103, Wright &, 62:104
gl14 17 wt1 --Sprague, 62:107

CHROMOSOME 3
Duplication of segment, 3S.5 to 3L.1, with 3B chromosomes from TB-3Sb and TB-3La --Carlson &, r81
Knob distribution in inbreds and varieties --Chughtai &, r96
QTLs near E8 (Est8), Hex1, Tpi4, Pgd2, Mdh3; notably ear weight; distorted ratios --Edwards &, r157, Stuber &, r545; Wendel &, r604
a1-m2-7991A1, a1-m2-7995, a1-m2-7977B, a1-m2-8004, a1-m2-8010A, a1-m2-8011,
a1-m2-8167B, a1-m2-8417, a1-m2-8745 derivatives, RM; Spm-w-8011, dSpm-7995, dSpm-7977B, dSpm-8004 RM, BS --Masson &, r342
hcf46 uncovered by TB-3La --Miles &, r365
A1: BS, inserts; transcript, translation, enzymatic activity --Reddy &, r433; Schwarz-Sommer &, r492
a1-m877527, Cy-regulated; A1-m(r)-Cuna, En-regulated --Peterson, 62:3
a1-sh2-Del, Def3 Mutator induced --Stinard &, 62:14,15
brn1: ra2 27%, g2 16%, d1 22% --Stinard, 62:17
trisome3, duplex & simplex ratios --Doyle, 62:49
Lxm1 (was Lxm*-1600), lax midrib; wx1 T3-9c 17% --Neuffer, 62:53
ms23 uncovered by TB-3La & TB-3La-256270 --Albertsen, 62:71
QTLs for plant height & ear height vs. RFLPs --Grant &, 62:71
RFLP map --Grant &, 62:71, Murray &, 62:89
g2 14 E8 31 d1 11 cl1; Cg1 9 d1 16 Lg3; d1 5 ra2 16 Lg3 2 Rg1 2 ys3 --Pasteris &, Poethig, 62:98,99
NPI432-null, NPI467-A1, NPI477-Cab1 on 3L --Turner &, 82:103, Wright &, 82:104
gl9 26 et1 --Sprague, 62:107

CHROMOSOME 4
Adh2, DNaseI hypersensitivity in chromatin at -40 to -70bp from start site, -150 to -180, and -250 to -285;
changes in induced chromatin or purified genomic DNA --Ashraf &, r19
Reduction in endosperm due to paternal deficiency enhanced by maternal duplication: TB-4Sa not by TB-1Sb, TB-3La, TB-5La, TB-1OL19 or TB-1OL32;
TB-1La, TB-7La, TB-1OL18, & TB-1OL19 but not TB-1Sb or TB-1OL32 by TB-4Sa --Birchler &, r54
Knob distribution in inbreds and varieties --Chughtai &, r96
hcf23 uncovered by TB-4Sa --Miles &, r365
Zein polymorphisms: T4-10f, T4-9(5974), T4-9g, and T3-4(5156) linkages --Ottoboni &, r396
Zein gene subfamilies --Rubenstein &, r461
QTLs near Aco1 --Stuber &, r545
c2-m85-2, c2-m85-3 --Peterson, 62:3
c2-m3 element Mpi1 --Weydemann, 62:48
trisome4, telo4Sa, duplex & simplex ratios --Doyle, 62:49
Ms*-7255: wx1 T4-9g, wx1 T4-9b linkage --Albertsen &, 62:70
RFLP map --Grant &, 62:71, Murray &, 62:89
Ubiquitin RFLP locus, AGR1002-ubi1 --Murray &, 62:89
Zein linkages with su1 --Wilson &, 62:97
NPI478-Cab2,3 on 4L; NPI314-Adp3, NPI473-Oec1 on 4 --Wright &, 62:104

CHROMOSOME 5
Knob distribution in inbreds and varieties --Chughtai &, r96
hcf18, hcf21, hcf38, hcf43 uncovered by TB-5La --Miles &, r365
QTLs near Pgm2, Mdh5, Amp3, Got2; notably grain weight, ear characters --Stuber &, r545
a2-m668291, Cy-controlled --Peterson, 62:3
Ael-5180, dominant glassy; linkage data; ael-Mu1, ael-Mu2, ael-Mu3, ael-Mu4, ael-Mu5, ael-Mu6, ael-Mu7 --Stinard &, 62:11
Dap1: wx1 T5-9(4790) 39%, wx1 T5-9a 3% --Stinard, 62:16
a2-Mus1, a2-Mus2, a2-Mus3 Mutator-induced --Stinard &, 62:18
trisome5, duplex & simplex ratios --Doyle, 62:49
vp1-w1, vp1-w2, vp1-w3, vp1-mum1, vp1-mum2: clone, RM --McCarty &, 62:61
prl-m860192 En-mediated --Reddy &, 62:64
RFLP map --Grant &, 62:71, Murray &, 62:89
Ubiquitin RFLP locus, AGR1002-ubi2 --Murray &, 62:89
Clone PCM4 localized in situ; transcripts --Quattrocchio &, 62:96
NPI363-null, NPI469-A1-4 on 5L; NPI474-Oec3 on 5S; NPI481-L2 on 1 or 5 --Turner &, 62:103, Wright &, 62:104

CHROMOSOME 6
Knob distribution in inbreds and varieties --Chughtai &, r96
QTLs near Pgd1, Enp1, P11, Hex2, Idh2, Mdh2; notably kernel characters; distorted ratios --Edwards &, r157, Stuber &, r545; Wendel &, r604
afd1 uncovered by TB-6Lc --Golubovskaya, r204
hcf5, hcf26, hcf323 uncovered by TB-6Sa; hcf34, hcf36, hcf48, hcf408 uncovered by TB-6Lc --Miles &, r365
y1-Mum Mutator induced --Buckner &, 62:18
trisome6, telo6La, telo6Lb, telo6Lc, duplex & simplex ratios --Doyle, 62:49
Beta zein RFLP locus, AGR1003-zeinB --Murray &, 62:89

CHROMOSOME 7
Reduction in endosperm due to paternal deficiency enhanced by maternal duplication: TB-1La by TB-7Lb --Birchler &, r54
Sod2 (superoxide dismutase 2; cytosolic) clone, RM, BS --Cannon &, r77, r78; Scandalios, r484
Knob distribution in inbreds and varieties --Chughtai &, r96
hcf*-Mu5 uncovered by TB-7Lb --Miles &, r365
O2-Rev alleles, from o2-m(r), gives rise to O2-Ver alleles (reversion-prone; chromosome instability) --Motto &, r377
Zein gene subfamilies --Rubenstein &, r461
o2-m20, o2-m21, o2-m22: Spm inserts, clone, RM, transcripts --Schmidt &, r485
Zein polypeptides Zp20/1, Zp20/2, Zp20/3 gene cluster, restriction fragment --Marotta &, 62:29
o2-m5: clone --Salamini &, 62:29
trisome7, telo 7La, duplex & simplex ratios --Doyle, 62:49
hcf104, Mu-induced --Cook &, 62:50
RFLP map --Grant &, 62:71, Murray &, 62:89
Gamma zein RFLP locus, AGR1004-zeinG --Murray &, 62:89
Zein linkages with y8 & wx1 T7-9(4363) --Wilson &, 62:97
NPI470-A1-5 on 7S; NPI475-Oec4 on 7L; NPI480-o2 on 7 --Wright &, 62:104

CHROMOSOME 8
Knob distribution in inbreds and varieties --Chughtai &, r96
QTLs near Idh1, Mdh1; notably grain weight, ear characters; distorted ratios --Edwards &, r157, Stuber &, r545; Wendel &, r604
b-32 protein cDNA clone --DiFonzo &, 62:29
trisome8, duplex & simplex ratios --Doyle, 62:49
hcf102 (was hcf*-1113-3), Mu-induced, uncovered by TB-8Lc --Cook &, 62:50
RFLP map --Grant &, 62:71, Murray &, 62:89
NPI318-Adp4, NPI479-Cab4 on 8L; NPI476-Oec5 on 8 --Wright &, 62:104

CHROMOSOME 9
TB-9S(Saraiva) half-translocation: 9S-near tip, B-euchromatin --Ahnert &, r3; Saraiva &, r473,r474
Duplication of segment, 9S.4 to 9L.4, with 9B chromosomes from TB-9Sb-2150 and TB-9La --Carlson &, r81
Mr: Mutator of R1-m; Sh1 17 Mr 22 Wx1 --Chang &, r86
Knob distribution in inbreds and varieties --Chughtai &, r96
c1-m5 clone via Spm tagging; RM & transcripts of alleles c1-m1, c1-m2, c1-m858, C1-I, C1-S --Cone &, r104
bz1-m13CS9 dSpm insert contains splicing site for excision from transcript --Kim &, r275
hcf42 uncovered by TB-9Lc --Miles &, r365
C1 clones, one genomic & 2 cDNA: BS, deduced protein --Paz-Ares &, r408
Deletions of Yg2, Mu-generated, progressively class 1 (male transmission 2:1 to 10:1), class 2 (male transmission nil,
TB-9Sb hypoploid viable), class 3 (hypoploid inviable, class 4 (female transmission reduced) --Robertson &, r445
TB-9S(Saraiva) half-translocation deficient for Yg2, not Bz1 --Saraiva, r473, r474
Wx1: in situ hybridization, pachytene localization at 32.25% of chromosome 9 (i.e., very near the centromere) from the short arm tip,
in heavy chromomere region of 9S --Shen &, r504
Sh1: transcripts, anaerobic induction (=ANP87); Css2 transcripts --Springer &, r525
QTLs near Acp1 --Stuber &, r545
bz1-mu2: Mu1.7 insert, RM --Taylor &, r557
Sh1 DNaseI hypersensitive sites 5' --Wurtzel &, r632
bz1-m877803, En-related --Peterson, 62:3
yg2-Mum, wd1-Mu, Bf1-Mu(del) --Robertson &, 62:24, 25
sh1-m6258 Ds inserts --Pahl &, 62:46
trisome9, duplex & simplex ratios --Doyle, 62:49
hcf113, Mu-induced, uncovered by TB-9Sb --Cook &, 62:50
Les8: c1 22%; wx1 8%; c1 wx1 24% --Hoisington, 62:53
Ss2-null (Css2-null) --Chourey, 62:62
c1-m5543? En-mediated --Reddy &, 62:64
QTLs for ear height vs. RFLPs --Grant &, 62:71
RFLP map --Grant &, 62:71, Murray &, 62:89
NPI463-Sod2-2 on 9 --Wright &, 62:104
Dt? 25 wx1 --Sprague, 62:108

CHROMOSOME 10
Reduction in endosperm due to paternal deficiency enhanced by maternal duplication: TB-1Sb by TB-10L19 but not by TB-10L32; TB-10L18 by TB-1Sb & TB-4Sa but not by TB-3Sb, TB-3La or TB-9Sd;
TB-10L19 by TB-1La & TB-4Sa but not by TB-4Lb, TB-5La, TB-9Sd or TB-10L32 --Birchler &, r54
R1-nj components, probable gene order G1 Sc Nj K --Kumar &, r292
hcf47, hcf316 uncovered by TB-10Sc; hcf28 uncovered by TB-10La --Miles &, r365
Zein polymorphisms, T4-10f linkage --Ottoboni &, r396
Zein gene subfamilies --Rubenstein &, r461
QTLs near Glu1; notably kernel characters --Stuber &, r545
K10-II deficiencies --Rhoades &, 62:33
trisome10, telo 10La, duplex & simplex ratios --Doyle, 62:49
RFLP map --Grant &, 62:71, Murray &, 62:89
QTLs for ear height vs. RFLPs --Grant &, 62:71
Zein Zp22/2 recombination with R1-st; possible amplification --Menghetti &, 62:92
Sn1 clone, by R1 homology --Tonelli &, 62:94
NPI319-Adp5 on 10S, NPI308-R1 on 10L --Wright &, 62:104

B CHROMOSOME
9B-2150, deletion of most distal B chromatin, derivative from TB-9Sb: meiotic loss --Carlson, 62:65

UNPLACED
Imidazolinone resistance: dominant, single gene --Anderson, r12
CgL1: anthracnose leaf blight [Colletotrichum graminicola (Ces.) Wils.] resistance --Badu-Apraku &, r23
Yg*-1459 (yellow-green); pro1-1058; dek1-792 (clf*-792); dcr*-1084 (defective crown); brn*-1314A (small, brown kernel); Fbr*-1602 (few-branched tassel); Tlr*-1590 (tillered); Hsf*-1603;
Rld*-1441, Rld*-1990 (rolled leaf); Lxm*-1600 (lax midrib); Rs*-1606 (rough sheath); noe*-2205 (no ear primordia); tye*-2171 (tiny ear) --Bird &, r55
ms*-aa: aborted anthesis, membranous lodicules --Cornu &, r108
Fasciated ear, recessive --Hake &, 62:2
Twin ears: penetrance, expressivity --Hallauer, 62:2
ba*-861059B --Pan &, 62:4
aml-su*-2162, su-sh*-3191, su-sh*-3328, su-sh*-5079, su-sh*-5081 Mutator-induced --Stinard &, 62:14
Multiple ears and tillers, dominant --Choe &, 62:54

TRANSPOSABLE ELEMENTS (see also specific loci affected)
Mu1 RM, methylation & inactivation --Bennetzen, r47
Ac transpositions from P1-vv occur in the same genomic fragment in cotwins --Chen &, r89
Ac methylation --Chomet &, r92; Schwartz &, r489
Mu elements in Italian varieties, 5-15 copies; stable --Del Guidice &, r134
Ac transcripts --Kunze &, r293
Derivatives Spm-w-8011, dSpm-7995, dSpm-7977B, dSpm-8004 RM, BS --Masson &, r342
Ds2 transcripts, splicing --Simon &, r512
Mu1.7: RM, BS --Taylor &, r557
Ds (4.3 or 1.5 kb) in Wx1 transcripts is mostly spliced out at sites in and near the inserted element --Wessler &, r608
Mu1, Mu1.7 elements in extrachromosomal, closed circular form --Sundaresan &, r549
Ac deletions, excision recognition and transposase ORF --Coupland &, 62:46
Mu4, Mu5, Mu6, Mu7: RM --Talbert &, 62:59
Ac transpositions average 34 cM from P1-VV --Chen &, 62:97

NUCLEAR cDNA/GENOMIC CLONES/PROBES
Glutelin-2 and zein-2 cDNA clones: RM, BS, flanking regions, transcripts --Boronat &, r61; Prat &, r424
Zein 27kD cDNA, tandem duplication, RM for region --Das &, r129
Zein cDNA clones, 19kD: BS transcripts, translation, flanking regions --Galili &, r185, Kriz &, r288
Histone H3, H4 genomic clones; H3 60-80 copies, H4 100-120 copies; RM, BS, transcripts --Gigot &, r199
PEP carboxylase cDNA clones: 4 genes (1 specific for C_4 enzyme; a 5th gene is likely), RMs, transcripts --Grula &, r211
Cytoplasmic aldolase cDNAs, translation --Kelley &, r259
RuBPCase small subunit (rbcs) cDNA: BS, flanking regions --Lebrun &, r308, Matsuoka &, r343
Zeins: DNaseI footprinting, protein binding site --Maier &, r335
Chlorophyll a/b binding protein (LHCP) cDNA: BS, flanking regions --Matsuoka &, r344
Zein cDNA clones & subfamilies: 100 genes, 4 subfamilies; BSs --Rubenstein &, r461
PEP carboxylase, pyruvate orthophosphate dikinase, NADP-malic enzyme, RuBPCase small subunit, LHCPII, oxygen-evolving polypeptides (OE33, OE23, OE16) cDNA clones, transcripts, translations --Sheen &, r500, r501, r502, r503
Pollen-specific cDNAs --Stinson &, r539
Lipid body protein L3 cDNA: BS, deduced protein --Vance &, r577
MRS-A, Mu-related sequence (without associated termini), RM --Harn &, 62:54

CHLOROPLAST
rRNA operon, promoter; tRNAvalGAC; RM --Delp &, r135
Zea species, subspecies, Tripsacum: comparative restriction fragments; deletions, insertions --Doebley &, r147, r149
Ribosomal protein genes rps4, rps7, rps12: BS, transcripts, S1 nuclease site analysis --Giese &, r198; Russell &, r464
Template active region, 9kbp, possible origin of replication --Gold &, r203
Ribosomal protein genes rpl23, rpl2, rps19, rpl22, rps3, rpl16, rpl22: order, BS --Larrinua &, r305; McLaughlin &, r353, r354, r355
psbB, psbF, petB, petD: RM, BS, transcripts --Rock &, r448
atpA, atpH BS; S1 nuclease-protected sites 5' --Rodermel &, r450
rps14, trnR(UCU), trnfM region: BS, transcripts --Rodermel &, r451; Srinivasa &, r526
tRNAproUGG of mitochondrion: sequence present but transcripts absent in chloroplast --Runeberg-Roos &, r463
tRNAserGCU: BS --Russell &, r465

MITOCHONDRION
cms-T, 2.3kb linear plasmid RM and terminal BS; homologies to S1 & S2, to ctDNA, and nuclear DNA --Bedinger &, r43
cms-T: urf13-T and fertile mutants; RM, transcripts, and polypeptides --Dewey &, r142, r143; Eckenrode &, r156; Fauron &, r168; Rottmann &, r460; Walker &, r594; Wise &, r625, r626, r627
cms-S, cms-CA, cms-D, cms-LBN: fertile revertants from tissue culture; loss of S1 & S2 plasmids --Earle &, r153
NCS2 transcripts vs. normal cms-T --Feiler &, r171
Ribosomal RNA genes, cytochrome oxidase subunit II gene of Zea diploperennis: RM, BS, flanking regions --Gwynn &, r214
cms-C, cms-BB, cms-ES, cms-PR, cms-RB: cosmid clones, restriction patterns --Pring &, r427
tRNAproUGG: map location, transcripts --Runeberg-Roos &, r463
atpA of N, cms-S, cms-T: arrangements, stoichiometry --Small &, r515
ORF25 in N, cms-C, cms-S, cms-T: BS, predicted protein --Stamper &, r535
cms-S: S2 plasmid URF, 130kDa polypeptide --Zabala &, r639
atp9, cms-S revertants --Wang &, 62:102

RESISTANCE/TOLERANCE/QUANTITATIVE INHERITANCE ANALYSIS
Stem borer (Chilo partellus Swinhoe) R in cultivars R to Diatraea grandiosella Dyar but not in cultivars R to Ostrinia nubilalis (Hubner) --Ampofo &, r11
Imidazolinone R selection in tissue culture (tolerant line XA17); dominant, single gene --Anderson, r12
Anthracnose stalk rot (ASR) and leaf blight (ALB) [Colletotrichum graminicola (Ces.) Wils.]: ALB R in inbred LB58 vs. A632 dominant, single gene (CgL); in RD6502 vs. A632 two complementary dominants.
ASR R in LB31 vs. B37 & others dominant, single gene, in RD6502 vs. B37 quantitative, BSH 18.8% --Badu-Apraku &, r23, r24, r25
Modifiers of o2 endosperm texture from two sources monogenic, one dominant, one recessive; other sources more complex --Belousov, r45
Sorghum downy mildew [Peronosclerospora sorghi (Weston & Uppal) C.G. Shaw] R/S among 6 lines, additive + non-additive, maternal effects --Borges, r59
Ear rot (Gibberella zeae) R/S diallel --Chiang &, r91
Kernel depth mass selection, gca --Cross &, r117
Differences in 2D-PAGE proteins, qualitatively and quantitatively, vs. gca in diallel --Damerval &, r122
Saline tolerance in Arizona 8601 germplasm --Day, r132
Tripsacum dactyloides monoecious vs. gynomonoecious, recessive major gene --DeWald &, r140
QTLs vs. marker loci (allozyme and flavonoid) --Edwards &, r157, Stuber &, r 545
Saline tolerance in Protador & Across 8024; differences in Na concentration in tissues --Hajibagheri &, r215
Maize-streak virus, Puccinia polysora, Helminthosporium maydis, Curvularia leaf spot, Fusarium moniliforme stalk rot, Striga hermonthica (witchweed) R germplasm --Kim &, r276
Isozyme diversity (10 loci) in populations and derived lines, selection changes --Kling 7, r279
Heritability for yield estimates 53-80% among 121 selection experiments of 7 types --Lamkey &, r296
Isozyme differences vs. hybrid performance among inbreds --Lamkey &, r297; Tsaftaris &, r568
Al tolerance in CMS30 & CMS36, gca, sca --Lopes &, r327
Maize dwarf mosaic virus R diallel --Milinko &, r366
Anther culture response, gca, sca --Petolino &, r416
Tassel culture response partially dominant --Rhodes &, r441
Glycine betaine levels in inbreds --Rhodes &, r442
Maize chlorotic dwarf virus R, gca, sca --Rosenkranz &, r459
Heat tolerance in pollen and tassel, gca --Schoper &, r487
Somaclonal variation --Scowcroft &, r494; Zehr &, r640
Maize weevil (Sitophilus zeamais) R, feeding deterrence, phenolics --Serratos &, r495
Isozyme and protein characterization & diversity in corn belt dent varieties, relationships --Smith &, r516; Smith, r517
Gray leaf spot (Cercospora zeae-maydis Tehon & Daniels) R/S mainly additive, readily transferred --Thompson &, r 560
Aflatoxin contamination, R vs. S in two subpopulations --Widstrom &, r617
Regenerability of 37 inbreds vs. 3 media --Wilkinson &, r618
Heat-shock vs. cold-shock proteins vs. cold tolerance --Yacoob &, r633, r634
QTLs (plant height, ear height) vs. RFLPs --Grant &, 62:71
RFLPs vs. percentage recurrent parent --Beckman &, 62:107

--Assembled unrestricted by Prof Ligate

MAIZE GENETICS COOPERATION STOCK CENTER

During the past calendar year, 2270 seed samples were provided in response to 184 requests. Both figures represent a volume of service activity somewhat greater than that of the previous year. Stock requests continue to be received from a diverse clientele representing research, teaching and commercial interests. Increased requests for certain categories of stocks clearly reflect current cooperative efforts in various approaches to chromosome mapping.

During the past growing season, plantings were made of numerous types of stocks, with special emphasis on increasing seed supplies of stocks in low supply or high demand. In addition, seed increases were made of the entire collections of chromosome inversions, B-A translocations, tetraploids, primary trisomics and wx-marked translocations. Increases were also made of multiple gene stocks and selected chromosome testers. The summer plantings consisted of about 5 acres of nurseries, of which about one-fourth were devoted to observations relating to confirmation of pedigrees, allelism testing, location of genes to chromosome and chromosome mapping. In addition, pedigree information on seedling traits was developed from some 2,000 sand bench cultures during the winter.

The Maize Stock Center functions at two distinct levels. Its basic responsibility is to preserve and perpetuate genetic stocks for posterity. At a second level, it has the potential and the mission to enhance and expand current research productivity by supplying verified, vigorous stocks suitable for immediate use as research tools. To this end, there are continuing efforts to develop improved procedures and genetic marking for monitoring, confirming and perpetuating certain types of genetic stocks. Examples of such adjuncts are already very valuable in the perpetuation and use of certain of the primary trisomics and B-A translocations. We would welcome further suggestions any of you may have regarding more efficient procedures for classifying or manipulating specific genetic variants.

During the past year about 75 seed samples of new stock acquisitions were received for increase and addition to the stock center inventory. We anticipate that a much larger number of stocks will be submitted within the next few years.

E. B. Patterson
S-116 Turner Hall
Agronomy Department
University of Illinois
Urbana, IL 61801
Phone (217)-333-9641
(217)-333-6631 (Lab)

Chromosome 1

101A sr zb4 P-WW **
101B sr P-WR **
101C sr P-WW **
101D sr P-RR **
102A sr P-WR an gs bm2 **
102B sr P-WR an bm2
102C sr P-RW ad bm2 **
103C sr P-WR bm2 **
103D vp5
103E zb4 ms17 P-WW
103G sr P-RR bm2 **
104B zb4 ts2 P-WW bm2
105C zb4 P-WW br **
105E ms17
106A zb4 P-WW bm2
106B ts2 P-RR
106C ts2 P-WW bm2
106D ts2 P-WW br bm2
107A P-CR **
107B P-RR **
107C P-RW **
107D P-CW **
107E P-MO **
107F P-VV **
108C P-RR br f an gs bm2
109A P-RR an ad bm2
109B P-RR an 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 **
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
112B P-WW br f bm2 **
112C P-WW br f an gs bm2
112E as
112H P-WW br **
113A as br2
113B rd
113C br f **
113E br f Kn
114A br f Kn Ts6
114B br f Kn bm2
114D Vg
115A Vg an bm2
115B Vg br2 bm2
115D bz2-m; A A2 C Pr **
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 **
117D tb-8963 **
117E Kn
118A Kn Ts6
118B Kn bm2
118C lw
118D Kn Ts6 bm2
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
122B TB-1Sb
125A Les2

Chromosome 2

201A ws3 lg gl2 B **
201B ws3 lg gl2 B sk
201E ws3 lg gl2 B ts
201F ws3 lg gl2 b **
202C ws3 lg gl2 fl v4 **
202E ws3 lg gl2 b v4 **
203B al **
203D al lg
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 **
207A lg gl2 b gl11 **
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 **
209E lg gl2 b sk
209F lg gl2 b sk fl
209G lg gl2 sk fl v4
210A lg gl2 b sk v4
211A lg gl2 b fl **
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 **
213B lg gl2 wt **
213C lg gl2 w3
213D lg gl2 w3 Ch
213E lg gl2 b Ch **
214B lg gl2 Ch
214C d5
214D B gl11
214E B ts
215A gl14
215B gl11 **
215C wt
215E fl **
216A fl v4 Ch
216B fl v4 Ht
216C fl v4 Ht Ch
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 ba2 w3
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 whp

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

(continued)

305A d Lg3
305D d Rg
306A d Rg ts4 lg2
306D d Rg ts4
307A d pm **
307C pm **
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
317C ts4 lg2 a-m 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 **
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 C R 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 **
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
325G a3 **
326A sh2 **
326B vp
326C Rp3 **
326D te **
327A TB-3La
327B TB-3Sb
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

** Some of these stocks are available in homozygous state.

Chromosome 4

401B Ga **
401C Ga su **
401D Ga-S **
401E Ga-S; y
401F Ga-S; A A2 C R **
402A st **
402B st Ts5
402C st fl2 **
402D Ts5
403A Ts5 fl2
403B Ts5 su
403C Ts5 la su gl3
405B la
405D la su gl3 **
406C fl2 **
406D fl2 su **
407B fl2 su bm3 **
407C fl2 su Tu gl4
407D su **
407E su-am **
407F su-am du
408B su bm3 **
408C su zb6 **
409A su zb6 Tu **
410A su zb6 gl3 dp **
410D su zb6 gl3 **
411A su gl4 j2
412E su j2 gl3 **
414B gl4 **
414C gl4 o **
412A su gl4 o Tu **
412B su j2
412C su gl3 **
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
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 Dt6 gl3; a1 **
420A Dt4 su; a-m A2 C R
420B TB-9Sb-4L6504
420H Dt4; a-m A2 C R
420I TB-9Sb-4L6222
421A TB-4Sa
421C TB-7Lb-4L4698
422A Primary Trisomic 4
423A TB-4La
423B TB-4Lc
423C TB-4Ld
423D TB-4Le
423E TB-4Lf

Chromosome 5

501A am a2; A A2 C R
501B lu **
501C lu sh4
501D ms13
501E gl17
501H gl17 a2 bt; A C R
501K gl17 v3
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
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 **

(continued)

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
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
603A y l10
603B y l11
603C y l12
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 l10
608B Y l12
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
611D Pt
611E w
611F Pl sm Pt;P-RR

(continued)

612A w14
612B ms6
612C l*-4923
612D oro
613A 2NOR ; a2 bm pr v2
613A TB-6Lb
614B TB-6Sa
614C TB-6Lc
615A Primary Trisomic 6

Chromosome 7

701A Hs o2 v5 ra gl
501B 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 **
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 **
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
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
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

** Some of these stocks are available in homozygous state.

Chromosome 9

901B yg2 C sh bz; A A2 R
901C yg2 C sh bz wx; A A2 R **
901D yg2 C-I 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-I; 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 **
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 **
910D c; A A2 R **
910E c sh wx gl15 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 (Other alleles from O. Nelson avail.) **
915B wx-a **
915C w11
915D wx pg12 bm4; y pg11
916A wx v **
916C wx bk2 **
916D wx bk2 bm4 **
917A wx Bf **
917C v **
917D ms2
917E gl15 **
917F d3
918A gl15 Bf **
918B gl15 bm4 **
918D Wc **
918E Wx bk2 bm4 **
918F Wx Bf **
919A bm4 **
919B Bf bm4 **
919C 16
919D 17
920A yel*-034-16
920B w*-4889
920C w*-8889
920E w*-8950
920F w*-9000
920G Tp9 N9 N3 Df3
921A TB-9La
921B TB-9Sb
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 **
X04A Og du R; A C R **
X04B ms11
X04C ms11 bf2
X04D bf2 **
X04E Og; C-I 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
X10A du **
x10C du o7 **
X10D du g r; A C R **
X10F zn **
X10G du v18 **
X11A zn g **
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; **
X12D v18 g R-g sr2 ; A A2 C
X12E g R;A A2 C **
X12F g r-ch K10;A A2 C **
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 **
X14A Ej r-r; A A2 C **
X14B v18 g R ;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 **
X18G R-scm2; bz2 A A2 **
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 **
X20F yel*-8721
X21A TB-10La
X22A TB-10Sc
X21B TB-10L19
X23A Primary Trisomic 10

** Some of these stocks are available in homozygous state.

Unplaced Genes

U235A dv
U235B dy
U335A el
U435A l4
U635A Rs
U535A vl3
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
M342A 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 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
E642C Knobless Wilbur's Flint
E442A Gaspe Flint
E642B Gourdseed
E742A Maiz Chapalote
E942B Missouri Cob Corn
E742B Papago Flour Corn
E742C Parker s Flint
E842A Tama Flint
E942A Winnebago Flint
E842B Zapalote Chico

Tetraploid Stocks

N103A P-RR
N103B P-VV
N102C 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
N107C Synthetic B
N107B W23 conversion
N104C su wx
N106D sh Wx ; Y
N106E sh wx; y

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
WX04A wx 2-9c (2S.49 ; 9S.33) * Sx
WX05A wx 2-9b (2S.18 ; 9L.22) * Sx
WX06A wx 2-9d (2L.83 ; 9L.27)
WX07A wx 3-98447 (3S.44 ; 9L.14)
WX08A wx 3-9c (3L.09 ; 9L.12) * Sx
WX10A wx 4-9e (4S.53 ; 9L.26) * Sx
WX11A wx 4-9g (4S.27 ; 9L.27) * Sx
WX12A wx 4-95657 (4L.33 ; 9S.25) * Sx
WX13A wx 4-9b (4L.90 ; 9L.29) * Sx
WX14A wx 5-9c (5S.07 ; 9L.10) * Sx
WX15A wx 5-94817 (5L.06 ; 9S.07)
WX16A wx 5-9d (5L.14 ; 9L.10)
WX17A wx 5-9a (5L.69 ; 9S.17) * Sx
WX18A wx 6-94778 (6S.80 ; 9L.30)
WX20A wx y 6-9b (6L.10 ; 9S.37) * Sx
WX21A wx 6-94505 (6L.13 ; 9ctr.)
WX22A wx 7-94363 (7ctr. ; 9ctr.) * Sx
WX23A wx 7-9a (7L.63 ; 9S.07) * Sx
WX24A wx 8-9d (8L.09 ; 9L.16) * Sx
WX25A wx 8-96673 (8L.35 ; 9S.31) * Sx
WX26A wx 9-108630 (9S.28 ; 10L.37)
WX27A wx 9-10b (9S.13 ; 10S.40) * Sx
WX28A wx 5-98386 (5L.87 ; 9S.13)

Non-Waxy Reciprocal Translocations

WX30A Wx 1-9c (1S.48 ; 9L.22) * 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) *
WX31B Wx 2-9b (2S.18 ; 9L.22) * Sx
WX32A Wx 3-98447 (3S.44 ; 9L.14) * Sx
WX32B Wx 3-98562 (3L.65 ; 9L.22) * Sx
WX32C Wx 3-9c (3L.09 ; 9L.12) * Sx
WX33A Wx 4-9e (4S.53 ; 9L.26) * Sx
WX33B Wx 4-95657 (4L.33 ; 9S.25) * Sx
WX33C Wx 4-9g (4S.27 ; 9L.27)
WX34A Wx 5-9c (5S.07 ; 9L.10) * Sx
WX34B Wx 5-94817 (5L.06 ; 9S.07) M14 only
WX34C Wx 4-9b (4L.90 ; 9L.29)
WX34D Wx 5-9a (5L.69 ; 9S.17)
WX34E Wx 5-9d (5L.14 ; 9L.10)
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 (7ctr. ; 9ctr.) *
WX37C Wx 6-94505 (6L.13 ; 9ctr.)
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 ; 10L37) M14 only
WX39B Wx 9-10b (9S.13 ; 10 S.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 1 5131-10 (1L.46-L.82)
I444A Inv 2a (2S.70-L.80)
I243A Inv 2 8865 (2S.06-L.05)
I243B Inv 2 5392-4 (2L.13-L.51)
I343A Inv 3a (3L.38-L.95)
I343B Inv 3L (3L.19-L.72)
I343C Inv 3 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 7 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

Mapping of EMS-induced mutants provided by M.G. Neuffer:

white*-4956 and white*-5716 are albino lethals that have been shown to be allelic by complementation tests (MGNL 61:114, 1987). The following data show that this locus is linked to wxT1-9(8389) [1L.74] but not to wxT1-9(035-10) [1L.89]. From an F2 progeny, wx and white in repulsion:

wxT1-9(8389) vs. white*-4956 and white*-5716 (combined data)

+ +	+ wh*	wx +	wx wh*	Chi-Square = 381
2597	1091	1021	5	p <.0000
				r = 0.078 ±0.014

wxT1-9(035-10) vs. white*-4956

+ +	+ wh*	wx +	wx wh*	Chi-Square = 1.28
243	36	68	6	p = .26

zebra7 (= zb*101) is a non-lethal mutant that has a clear, "zebra-stripe" phenotype in seedlings. The data below indicate the locus is about 5 map units proximal to green-stripe 1 on 1-L. The locus is linked to wxT1-9(8389) [1L.74] but not to wxT1-9(035-10) [1L.89]. One zb,gs,bm2 plant was recovered and outcrossed in the 1987 nursery for more precise mapping. Because zb7 is an easily scorable, non-lethal seedling marker, it should probably replace gs1 as a standard marker for 1-L.

F2 data, (bz2,gs,bm2 x zb7) selfed (Total = 660 progeny):

Class	Number	Class	Number	Class	Number	Class	Number
+,+,+,+	205	+,+,+,bm2	69	+,gs,zb7,+	0	bz2,gs,+,bm2	34
bz2,+,+,+	70	bz2,gs,+,+	41	+,gs,+,bm2	45	+,gs,zb7,bm2	1
+,gs,+,+	24	bz2,+,zb7,+	8	+,+,zb7,bm2	10	bz2,+,zb7,bm2	0
+,+,zb7,+	139	bz2,+,+,bm2	13	bz2,gs,zb7,+	0	bz2,gs,zb7,bm2	0

Maximum likelihood estimates by the computer program Linkage-1 (K.A. Suiter, J.F. Wendel, and J.S. Cass, version 3.50, 1987) are:

bz2-gs1 = 29 centimorgans (cm) ±4. The 1987 working map of maize also estimates the bz2-gs1 distance as 29 cm (MNL 61:129,1987).
gs1-bm2 = 28 cm ±4 The 1987 working map of maize estimates the gs1-bm2 map distance as 26 cm.
bz2-zb7 = 23 cm ±4 zb7-gs1 = 9 cm ±4 zb7-bm2 = 26 cm ±4

Our tentative map from the above data is:

bz2 - 24 - zb7 - 5 - gs1 - 26 - bm2

Testcross data, zb7 and:

	+,T	+,Normal	zb,T	zb,Normal	Linkage
wxT1-9(8389) [1L.74]:	161	14	14	149	8 cm ±1.5
wxT1-9(035-10) [1L.89]:	96	58	84	58	Not linked

adherent*-582 is a non-lethal mutant with a phenotype similar to adherent-1 on 1-L. The mutant is not allelic with ad1, however. Data from our 1987 summer nursery showed no linkage between ad*-582 and several 1-L marker stocks [(br1,bz2,gs1,bm2, wxT1-9(4995), wxT1-9(8389), and wxT1-9(035-10)]. TB1-La did not uncover the locus, also. It is thus likely that ad*-582 is not on 1-L.

Lesion-7 (= Les*-1461) is a mutant causing small, chlorotic spotting on leaves that had been reported to be linked to wxT1-9(8389) on 1-L (MNL 58:82, 1984). We have not been able to reproduce that result (data given below) and have found no linkage with br2, br1, bz2, gs1, bm2, or wxT1-(035-10). The 1984 report had also found no linkage between Les7 and br1, sn, gs, or bm2. It is thus likely that Les7 is not on 1-L.

Data from 1987 summer nursery, Raleigh:

Progeny of (wxT1-9(8389) x Les7) x (wxB14 x wxOh43); wx kernels planted, allowed to open-pollinate:

+, semi-sterile = 157	+, normal seed set = 2
Les, semi-sterile = 154	Les, normal seed set = 4

Conclusions: No linkage between Les7 and wxT1-9(8389);
Cross-over between wx and T = 6/317 = 1.9%

dek22 (= cp*-1113A) is not linked to wxT1-9(4995) [1L.19]. No distortions of an expected 3:1 ratio were found in a segregating F2 progeny.

Other unmapped morphological markers:

brachytic-2 is closely linked to hm1 in the proximal region of 1-L. In the 1987 summer nursery, one hm1,br2 plant was obtained among 1500 br2 plants in a segregating F2 progeny (hm1 x br2) selfed. The hm1,br2 plant was outcrossed to ms14, Va1 and f1 to determine gene order and map distance. Thanks to Dr. Steve Leath of the USDA-ARS for providing inoculum of Helminthosporium carbonum for identification of the hm gene.

reduced-1 produces an overall small statured plant. We have found the phenotype difficult to score in a non-uniform background. A bz2,gs1,rd1 stock has been sufficiently backcrossed to A632 to make linkage tests possible. Data from 36 testcross progeny is give below. A much larger sample will be scored next summer:

+,+,+ = 16	+,gs1,rd1 = 3	+,+,rd1 = 3	+,gs1,+ = 0	
bz2,gs1,rd1 = 9	bz2,+,+ = 3	bz2,gs1,+ = 2	bz2,+,rd1 = 0	

This indicates a gene order of bz2 - gs1 - rd1.

male-sterile-12 is reported to be somewhere on Chromosome 1, but the phenotype is "leaky", making linkage tests difficult (Beadle, G.W., Genetics 17:413-431, 1932). We have been backcrossing it into A632, where it appears to have stable, Mendelian segregation in our Raleigh summer nursery. Hopefully, this will allow accurate mapping.

Waxy translocation stocks:

We have been using wxT1-9(4995) [1L.19], wxT1-(8389) [1L.74], and wxT1-9(035-10) [1L.89] for localizing new genes, especially lethals. Testcrosses to be scored next summer should indicate where the breakpoints of these reciprocal translocations are in relation to the standard morphological markers of 1-L. The breakpoint of wxT1-9(8389) is somewhere near bz2 and gs1, since genes linked to the translocation are usually linked to at least one of the two morphological markers (see above data on zb7, for example.) We are also developing new waxy translocation stocks for 1-L: wxT1-9(5622) [1L.10] for the most proximal part of 1-L and wxT1-9(8886) [1L.33] for the region between wxT1-9(4995) and wxT1-9(8389). The most distal reciprocal translocation we have been able to recover from the Maize Genetics Cooperative stocks is T1-4g (1L.95), which we will also be mapping to morphological markers.

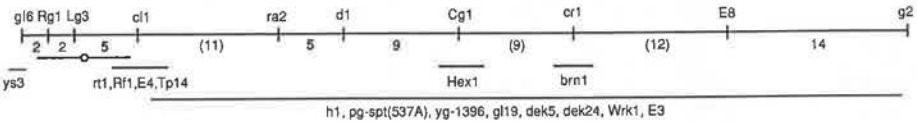
Isozyme-morphological marker mapping:

br1 bz2 vs. Amp1 Mdh4 Pgm1: Data from 231 testcross progeny give the following approximate map distances (see MNL 62:____):
Centromere - br1 (2.6) Amp1 (13.8) Mdh4 (1.7) bz2 (20.6) Pgm1

Paul H. Sisco

CHROMOSOME 3S

Shown below is the current status of the map for the short arm of chromosome 3. This map is based on previously published reports, as well as new data for Cg1, ra1, ys3, and E8 presented in this Newsletter. Map distances in parentheses are estimates. Using TB-3Sb, I have confirmed Neuffer's previous assignment of pgspt*-537A and yg*-1396 to 3S, and have demonstrated that hl is also located on this arm. yg*-1396 is not allelic to g2.



R. S. Poethig

CHROMOSOME 4L

A major recombination effort is going on to map the zein genes on chromosome 4 with Curtis Wilson to order them with conventional genes (i.e., dp1, c2, gl3, o1, gl4, and su1). Several translocations have been used for this purpose, too. A male sterile from Neuffer has been crossed for localization. A delay factor in Mo17, which gives large ears in heterozygotes, is on chromosome 4. Extensive crossing and zein analysis will place this gene in 1988. Also, we will attempt to locate the 4L knob genetically.

Dale M. Steffensen

CHROMOSOME 5S

Due to crop failure, we were unable to obtain seed from AB-translocation tests for chromosome location of putative new Spm-induced mutants on chromosome arm 5S (see last year's report). We obtained two new mutants on chromosome arm 5S from M. G. Neuffer and have made crosses to 5S linkage testers.

Mary Polacco

CHROMOSOME 10S

Two recessives, gl21 and l19, are reported to be on 10S (Neuffer & Beckett, MNL 61:50). Current data for this arm that we have accumulated need more tests to establish linkage relationships.

Marc C. Albertsen

GENELIST AND LINKAGE MAP OF MAIZE (*Zea mays* L.) - FEBRUARY, 1988

David A. Hoisington, Edward H. Coe, Jr. and M.G. Neuffer
Curtis Hall, University of Missouri, Columbia, Missouri 65211

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.

The linkage map represents the order and recombinational distances, in centimorgans (1% recombination = 1 cM), for those genes for which sufficient information is available to make a reasonable judgment of their location. Each chromosome is arranged beginning with the most distal gene in the short arm. Locations of the centromeres are indicated according to the best available data from cytogenetic studies. The physical map of each chromosome, to the left of each linkage map, is drawn with the length of each arm in proportion to the ratio of the length of that arm to the length on chromosome 1. Locations of the B-A translocations, which generate hemizygous segments, are shown as TB-.....; placement on the physical map is in accordance with observed breakpoints; placement on the linkage map is in relation to genes uncovered or not uncovered. The vertical line associated with simple B-A translocations represents the segment within which the breakpoint is located (genes distal to the line on that arm should be uncovered). In the case of compound translocations, the associated vertical line on the linkage map for the first arm involved (e.g., 1L of TB-1La-5S8041) defines the segment within which the second breakpoint is located (genes distal to the line are not uncovered). On the map of the second arm involved (5S, in the example), genes distal to the associated line are uncovered (as they are with simple B-A translocations). TB's shown as spanning one or more genes may or may not uncover the indicated gene or genes. Immediately to the right of the linkage map are those genes that have some information leading to a "rough" placement on the map, either near a gene already on the map or to a region of the map. Further to the right are those genes which have been placed to the chromosome (represented by the vertical line with arrows at both ends) or to one arm (represented by a vertical line running from near the centromere to the end of the arm).

To the left of each chromosome's linkage map is the current version of the restriction fragment length polymorphism map being developed by D. Hoisington at the University of Missouri. Distances between each locus are expressed in centimorgans with Haldane's mapping function being applied to the determined recombination value. Additional RFLP maps are being developed in other laboratories and efforts are underway to compile and analyze the data necessary to produce a integrated map of RFLP loci and other markers.

The authors greatly appreciate the corrections supplied by fellow maize co-operators and encourage all those interested in maize genetics to make suggestions and/or corrections to this list.

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
a1	3L-149.0	anthocyaninless: colorless aleurone, green or brown plant, brown pericarp with *P1-RR*	S	P	74
a2	5S-35	anthocyaninless: like *a1*, but red pericarp with *P1-RR*	S	P	142
a3	3L-132	anthocyanin: recessive intensifier of expression of *R1* and *B1* in plant tissues	S	P	181
Ac		activator: designator for autonomous transposable elements; regulates *Ds* transposition and dissociation; ex. *Ac9* designates element isolated from *wx1-m9*		P	201
Ac2		activator: similar to *Ac*			60
Aco1	4S	aconitase: electrophoretic mobility; monomeric			347
Aco2	-	aconitase: electrophoretic mobility			347
Aco3	-	aconitase: electrophoretic mobility			347
Aco4	-	aconitase: electrophoretic mobility; monomeric			347
Acp1	9	acid phosphatase (was *Ap1*, *Acph1*, *Phos*): electrophoretic mobility; cytosolic; dimeric			116
Acp2	-	acid phosphatase (was *Ap2*): electrophoretic mobility; dimeric			116
Acp4	1L-176	acid phosphatase: electrophoretic mobility; monomeric			152
ad1	1L-108	adherent: seedling leaves, tassel branches, and occasionally top leaves adhere	S	P	155
Adh1	1L-128	alcohol dehydrogenase: electrophoretic mobility; null allele is known; dimeric; intra/interlocus hybrid bands occur	S		302
Adh2	4S-46	alcohol dehydrogenase: electrophoretic mobility; null allele is known; dimeric; intra/interlocus hybrid bands occur			300
Adk1	6S-0	adenylate kinase: electrophoretic mobility; plastidial			348
Adr1	-	alcohol dehydrogenase regulator			166
ae1	5L-57	amylose extender: glassy, tarnished endosperm; high amylose content; starch branching enzyme IIb	S	P	346
afd1	-	absence of first division: male and female sterility; anaphase I equatorial			112
agt1	-	ageotropic: primary root unresponsive to gravity			67
al1	2S-4	albescent plant: erratic development of chlorophyll; pale yellow endosperm, some alleles viviparous	S	P	257
alh1	1L-near *bm2*	histone Ia (was *H1a*): electrophoretic mobility			332
Alpha		*A1* locus component (see *Beta*): determines reduced aleurone and plant color, brown pericarp			170
am1	5S-20	ameiotic: male and female sterility; anaphase I equatorial	S	P	248 271
Amp1	1L-near *f1*	aminopeptidase: electrophoretic mobility; cytosolic; monomeric			246
Amp2	1-near *hm1*	aminopeptidase: electrophoretic mobility; monomeric			246
Amp3	5S-near *a2*	aminopeptidase: electrophoretic mobility; monomeric			246
Amp4	-	aminopeptidase: electrophoretic mobility; monomeric			246
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; *an1-6923* includes deletion of *Bz2*	S	P	71 81
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; husk leaf tips striped	S	P	85
as1	1-56	asynaptic: synaptic failure in male and female	S	P	15
Asr1	4S-19	absence of seminal roots			212
Atc1		(see *Zb8*)			
ats1	8	atrazine susceptible: lacks glutathione S-transferase			119
B1	2S-49	colored plant: anthocyanin in major plant tissues; some alleles affect aleurone and embryo color	S	P	77
B chr		B chromosome: supernumerary chromosome		P	265
ba1	3L-102	barren stalk: ear shoots and most tassel branches and spikelets absent	S	P	128
ba2	2-near *ts1*	barren stalk: like *ba1*, but tassel more normal	S		128
baf1	9S-near *w11*	barren stalk fastigiate (was *ba*-s*): ear shoots often absent; tassel branches erect			45
bd1	7L-109	branched silkless: branched ear and tassel; silks absent	S	P	158
beta		*A1* locus component (see alpha): determines aleurone and plant color, red pericarp			170
Bf1	9L-137	blue fluorescent: homozygous seedlings (homozygous or heterozygous anthers) fluoresce blue under ultraviolet; anthranilic acid present	S	P	338
bf2	10L-30	blue fluorescent: similar to *Bf1* in expression; shows earlier, stronger seedling fluorescence than *Bf1*	S		2
Bg		Bergamo: regulatory element mediating *o2-mr*			286

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Bh1	6L-50	blotched: colored patches on colorless (*c1*) aleurone	S	P	76
Bif1	8	barren inflorescence (was *Bif*-1440*): spikelets absent from ear and tassel; tassel branching reduced			237
bk2	9L-82	brittle stalk: brittle plant parts after 4-leaf stage	S	P	169
bm1	5S-41	brown midrib: brown pigment over vascular bundles of leaf sheath, midrib, and blade	S	P	84
bm2	1L-161	brown midrib: like *bm1*	S		34
bm3	4-near *bt2*	brown midrib: like *bm1* (C.R. Burnham, unpublished)	S		164 222
bm4	9L-141	brown midrib: like *bm1*	S		31
Bn1	7L-71	brown aleurone: yellowish brown aleurone color	S		162
BNL		Brookhaven National Laboratory: designator for loci defined by restriction fragment polymorphisms			
br1	1L-81	brachytic: short internodes, short plant; no response to gibberellins	S	P	154 157
br2	1L-near *hm1*	brachytic: like *br1*	S		172
br3	5	brachytic: like *br1*	S		313
brn1	3S-near *cr1*	brown aleurone: brown kernel, brown embryo; seedling lethal			284
bs1	-	barren sterile			205
bt1	5L-42	brittle endosperm: mature kernel collapsed, angular, often translucent and brittle; affects starch-granule-bound phospho-oligosaccharide synthase	S	P	192 350
bt2	4S-67	brittle endosperm: like *bt1*; ADP glucose pyrophosphorylase electrophoretic mobility; (compare *sh2*) (G.F. Sprague, unpublished)	S		339
btn1	-	brittle node			153
bu1	-	leaf burn: leaves show burning, sometimes horizontal bands, accentuated by high temperature			99
bv1	5L-47	brevis plant: short internodes, short plant	S		174
bv2	-	brevis plant: plant height 30-50% of normal			258
Bx1	4S	benzoxazin: blue color reaction of crushed root tip with FeCl3, indicating cyclic hydroxamates present; inhibits growth of *Helminthosporium turcicum*, correlated with resistance to ECB (*Ostrinia nubilalis*)			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; allele *bz1-m4 = sh1-bz1-m4*	S	P	270
bz2	1L-106	bronze: like *bz1*; anthers not fluorescent; *an1-6923* mutation includes deletion for *Bz2*	S	P	244
C1	9S-26	colored aleurone: *c1* colorless; *C1-I* dominant colorless; *c1-p* pigment inducible by light	S	P	69
c2	4L-117	colorless: colorless aleurone, reduced plant color; chalcone synthase; *C2-Idf* dominant inhibitor	S	P	42
Car1	1S	catalase regulator: enzyme activity level increased			292
Cat1	5S-near *Mdh5*	catalase: electrophoretic mobility; cytosolic/glyoxysomal; tetrameric; intra/interlocus hybrid bands occur			18
Cat2	1S	catalase: electrophoretic mobility; null allele is known; cytosolic/glyoxysomal; tetrameric; intra/interlocus hybrid bands occur			289
Cat3	-	catalase: electrophoretic mobility; null allele is known; mitochondrial; tetrameric; no hybrid bands			291
Cdh1	-	cinnamyl alcohol dehydrogenase: electrophoretic mobility			93
Ce1	-	curled entangled: rolled leaves tend to be entangled			39 249
cfl2	-	complementary to *fl2*			247
Cg1	3S-35	corngrass: narrow leaves, extreme tillering	S	P	312
Cg2	-	corngrass: like *Cg1*; mutable			186
Ch1	2L-155	chocolate pericarp: dark brown pericarp	S	P	5
Cin		Cinteotl corn insert: repetitive sequences dispersed in the genome			305
cl1	3S-60	chlorophyll: white to green seedlings, depending upon *Clm1*; pale yellow endosperm	S		83
clh1	-	histone Ic: electrophoretic mobility			332
Clm1	8	modifier of *cl1*: greens *cl1* seedlings; does not restore endosperm carotenoids	S		83
Clt1	8	clumped tassel (was *Clt*-985*): variable dwarfing, developmental anomalies			102 236
cm1	10L-near *R1*	chloroplast mutator: like *ij1*	S		334
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*			149 151
cms-T		cytoplasmic male sterility: female-transmitted male sterility, Texas type; restored by *Rf1 Rf2*			149 151
cp1	7S-near *vp9*	collapsed: endosperm collapsed and partially defective			184
cp2	7S-near *vp9*	collapsed: endosperm rough, collapsed, partially defective; seedling very light green with darker streaks; lethal			240
cr1	3S-26	crinkly leaves: plant short; leaves broad, crinkled	S	P	78
Css1	9	sucrose synthase: sucrose synthase-2 of embryo and other tissues; (compare *sh1*)			198
ct1	8	compact plant: semi-dwarf plant, ear furcated			228
ct2	1S	compact plant: semi-dwarf plant with club tassel			105
ctDNA		chloroplast DNA: sequences or loci in chloroplast genome			
cto1	-	cob turned out: ear inverted to a sheet or tube, kernels internally placed; variable expression			345
Cx1	10L-near *bf2*	catechol oxidase: electrophoretic mobility; null allele is known; monomeric; no hybrid bands			264
Cy	5L-near *pr1*	regulatory element mediating *bz1-rcy*			294
d1	3S-44	dwarf plant: plant andromonoecious, short, compact; responds to gibberellins; *d1-1* intermediate in height	S	P	71
d2	3	dwarf plant: like *d1*			336
d3	9S-59	dwarf plant: like *d1*	S		57
d5	2S-34	dwarf plant: like *d1*	S		336
D8	1L-133	dwarf plant: dominant, resembles *d1*; not responsive to gibberellins; (compare *Mpl1*)	S	P	255
da1	9	dilute aleurone: aleurone color diluted	S		89
db1	-	dichotomously branching plants (= *dib*): variable location of dichotomy, usually at 4-8th node (possible association with aneuploidy)	S		204 205
dek1	1S-27	defective kernel (was *clf1, gay1, clf*-792*): germless; floury endosperm; anthocyanins and carotenoids absent; cultured embryos not obtained			238 239
dek2	1L	defective kernel (was *dsc*-1315A*): discolored, scarred endosperm; lethal; cultured embryos green			238 239
dek3	2S	defective kernel (was *gm*-1289*): germless; cultured embryos white with green stripe			238 239
dek4	2L	defective kernel (was *clf*-1024A*): germless; floury endosperm; cultured embryos green, narrow leaved			238 239
dek5	3S	defective kernel (was *sh*-874A*): shrunken endosperm; white seedling with green stripes			238 239
dek6	3L	defective kernel (was *sh*-627D*): shrunken endosperm; lethal; cultured embryos normal			238 239
dek7	4S	defective kernel (was *su*-211C*): shrunken sugary endosperm; white seedling with green stripes			238 239
dek8	4L	defective kernel (was *sh*-1156A*): shrunken endosperm; lethal; cultured embryos green, small			238 239
dek9	5L	defective kernel (was *crp*-1365*): crumpled endosperm; lethal; anthocyanins and carotenoids reduced; cultured embryos not obtained			238 239
dek10	4L	defective kernel (was *cp*-1176A*): collapsed endosperm; lethal; cultured embryos green, curled, stubby			238 239
dek11	7L	defective kernel (was *et*-788*): etched endosperm; lethal; cultured embryos white with green stripes			238 239
dek12	9S	defective kernel (was *cp*-873*): collapsed endosperm; lethal; cultured embryos green, narrow-leaved, curled			238 239
dek13	9L	defective kernel (was *o*-744*): defective opaque endosperm; lethal; cultured embryos pale green with green stripes			238 239
dek14	10S	defective kernel (was *cp*-1435*): collapsed endosperm; lethal; cultured embryos yellow-green			238 239
dek15	10L	defective kernel (was *cp*-1427A*): collapsed floury endosperm; lethal; cultured embryos green			238 239
dek16	2L	defective kernel (was *fl*-1414*): floury endosperm; lethal; cultured embryos normal			306
dek17	3L	defective kernel (was *cp*-330D*): collapsed endosperm; lethal; cultured embryos not obtained			306
dek18	5S	defective kernel (was *cp*-931A*): collapsed endosperm; lethal; cultured embryos green, narrow-leaved			306

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
dek19	6L	defective kernel (was *o*-1296A*): collapsed opaque endosperm; lethal; cultured embryos green			306
dek20	8L	defective kernel (was *cp*-1392A*): collapsed endosperm; lethal; cultured embryos green			306
dek21	10L	defective kernel (was *msc*-1330*): aleurone mosaic of reduced anthocyanins; reduced carotenoids; lethal; cultured embryos white; (compare *w2*)			306
dek22	1L	defective kernel (was *cp*-1113A*): collapsed endosperm; lethal; cultured embryos not obtained			41 307
dek23	2L	defective kernel (was *dcr*-1428*): defective crown; lethal; cultured embryos not obtained			41 307
dek24	3S	defective kernel (was *cp*-1283*): collapsed endosperm; lethal; cultured embryos normal			307
dek25	4S	defective kernel (was *sh*-1167A*): shrunken endosperm; lethal; cultured embryos normal			307
dek26	5L	defective kernel (was *cp*-1331*): collapsed endosperm; lethal; cultured embryos normal			307
dek27	5L	defective kernel (was *cp*-1380A*): collapsed endosperm; lethal; cultured embryos green			307
dek28	6S	defective kernel (was *o*-1307A*): opaque endosperm			307
dek29	8L	defective kernel (was *cp*-1387A*): collapsed endosperm; viable; cultured embryos green, narrow-leaved			307
dek30	9L	defective kernel (was *fl*-1391*): floury endosperm; lethal; cultured embryos green, narrow-leaved			307
dep1	6	defective pistils			206
Df		deficiency: general symbol for loss of segments of chromosome			
Dia1	2S	diaphorase: electrophoretic mobility; cytosolic; monomeric			347
Dia2	1L-near *bm2*	diaphorase: electrophoretic mobility; cytosolic; dimeric			347
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	201
dSpm		defective suppressor-mutator: designator for transposable factors regulated by *Spm*			293
dsy1	-	desynaptic: male and female sterility; synaptic failure			110
dsy2	-	desynaptic: like *dsy1*			109
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	266
Dt2	6L-44	dotted: like *Dt1*	S		245
Dt3	7L	dotted: like *Dt1*, but expression variable	S		245
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 *su1*	dotted: like *Dt1*	S		324
du1	10L-28	dull endosperm: glassy, tarnished endosperm; affects soluble starch synthase and branching enzyme IIa (P.C. Mangelsdorf, unpublished)	S		193
dv1	-	divergent spindle: chromosomes unoriented at metaphase I; partial male and female sterility	S		40
dy1	-	desynaptic: chromosomes unpaired in microsporocytes; partial male and female sterility	S		227
E1	7L	esterase: electrophoretic mobility; null allele is known; dimeric; intralocus hybrid bands occur			297
E2	-	esterase: presence-absence			299
E3	3S	esterase: electrophoretic mobility; dimeric; intralocus hybrid bands occur			298
E4	3S-near *cl1*	esterase (was *Est4*): electrophoretic mobility; null allele is known; monomeric			123
E5-I	-	esterase (duplicate factor with *E5-II*): electrophoretic mobility			188
E5-II	-	esterase (duplicate factor with *E5-I*): electrophoretic mobility			188
E6	-	esterase: presence-absence			188
E7	-	esterase: presence-absence			188
E8	3S-14	esterase: electrophoretic mobility; null allele is known; dimeric; intralocus hybrid bands occur			188
E9	-	esterase: electrophoretic mobility; null allele is known			188
E10	-	esterase: electrophoretic mobility			188
eg1	5L	expanded glumes: glumes open at right angle	S		32
Ej1		(= *Isr1*)			
el1	8L	elongate: chromosomes uncoiled during meiotic metaphase and anaphase in male and female; frequent unreduced gametes	S	P	271
En		enhancer: transposable element (equivalent to *Spm*); autonomous, regulates I transposition (e.g. at *g2-m* = *pg-m* = *pg14-m*)		P	253
Enp1	6L-near *y1*	endopeptidase: electrophoretic mobility; null allele is known; monomeric			203
et1	3L-161	etched: pitted, scarred endosperm; virescent seedling	S	P	328
fl	1L-86	fine stripe: virescent seedling, fine white stripes on base and margin of older leaves	S	P	178
Fcu		factor Cuna: controlling element of *r1-cu*			114
fl1	2S-68	floury endosperm (= *o4*): endosperm opaque, soft; dosage effect	S	P	127
fl2	4S-58	floury: endosperm opaque, soft; dosage effect (W.J. Mumm, unpublished)	S		231
fl3	8L-0	floury: endosperm opaque, soft; dosage effect	S		224
Flt		flint: designator for factors determining flint endosperm type			216
g1	10L-47	golden plant: seedling and plant with distinct yellow cast	S	P	71 73
g2	3S-0	golden plant (= *g5* = *pg14* = *v19*): like *g1*, but more extreme; sheaths whitish yellow-green	S		139
g5		(= *g2*)			
G6	9S-near *l7*	golden plant (was *G*-1585*): like *g1*; lighter yellowish sheaths			241
Ga1	4S-32	gametophyte factor (= *ga9*): *Ga1* pollen grains competitively superior to *ga1* on *Ga1* silks; *Ga1-S* super-gametophyte	S		150
ga2	5L-55	gametophyte factor: *Ga2* pollen grains competitively superior to *ga2*			30
ga7	3L-167	gametophyte factor: *ga7* pollen from heterozygotes 10-15% functional regardless of silk genotype			268
ga8	9S-near *lo2*	gametophyte factor: *Ga8* pollen grains competitively superior to *ga8* on *Ga8* silks			296
ga9		(= *ga1*)			
ga10	5	gametophyte factor			113
Gdh1	1L-near *vp8*	glutamic dehydrogenase: electrophoretic mobility; null allele is known; intra/interlocus hybrid bands occur			262
Gdh2	10	glutamic dehydrogenase: electrophoretic mobility; intralocus hybrid bands occur			115
Ger		glucoside earworm resistance: designator for earworm resistance factors from Cateto Palha Roxa			216
gl1	7L-36	glossy: cuticle wax altered; leaf surface bright, water adheres	S	P	162
gl2	2S-30	glossy: like *gl1*	S	P	125
gl3	4L-112	glossy: like *gl1*	S		125
gl4	4L-81	glossy (= *gl16*): like *gl1* (G.F. Sprague, unpublished)	S		
gl5	-	glossy (was *gl5-1*, duplicate factor with *gl20*): like *gl1* (G.F. Sprague, unpublished)			325
gl6	3L-69	glossy: like *gl1* (G.F. Sprague, unpublished)	S		
gl7	3L	glossy (= *gl12*): like *gl1* (G.F. Sprague, unpublished)			
gl8	5L-68	glossy (= *gl10*): like *gl1* (G.F. Sprague, unpublished)	S		
gl9	-	glossy: expression poor (G.F. Sprague, unpublished)			
gl10		(= *gl8*)			
gl11	2S-near *B1*	glossy: like *gl1*; abnormal seedling morphology	S		321
gl12		(= *gl7*)			
gl14	2	glossy: like *gl1*	S		323
gl15	9L-66	glossy: like *gl1*; expressed after 3rd leaf (G.F. Sprague, unpublished)	S	P	3
gl16		(= *gl4*)			
gl17	5S-34	glossy: like *gl1*, but semi-dwarf with necrotic crossbands on leaves	S		272

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
gl18	8L-near *fl3*	glossy: like *gl1*; expression poor	S		4
gl19	3S	glossy (was *gl*-169*): like *gl1*; lethal			233
gl20	-	glossy (was *gl5-2*, duplicate factor with *gl5*): like *gl1* (G.F. Sprague, unpublished)			325
gl21	10S	glossy (was *gl*-4786B*): like *gl1*			233
Glu1	10L-near *bf2*	beta glucosidase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intralocus hybrid bands occur			263
Got1	3L-near *Me1*	glutamate-oxaloacetate transaminase (possibly = *Ta1*): electrophoretic mobility; null allele is known; glyoxysomal; dimeric; intralocus hybrid bands occur			290
Got2	5L-96	glutamate-oxaloacetate transaminase: electrophoretic mobility; null allele is known; plastidial; dimeric; intralocus hybrid bands occur			117
Got3	5S-near *a2*	glutamate-oxaloacetic transaminase: electrophoretic mobility; null allele is known; mitochondrial; dimeric; intralocus hybrid bands occur			117
grt1	5L	green tip (was *grt*-1308B*): pale yellow seedling with green first leaf tip; lethal			233
gs1	1L-135	green stripe: grayish green stripes between vascular bundles on leaves; tissue wilts	S	P	76 210
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*			233
gt1	1	grassy tillers: numerous basal branches; vegetatively totipotent in combination with *id1* and *pe1*			303
h1	3	soft starch: endosperm soft, opaque	S		223
hcf1	2L	high chlorophyll fluorescence: affects NADP+ oxidoreductase; green seedling			207
hcf2	1L	high chlorophyll fluorescence: missing cytochrome f/b6 complex; yellow-green seedling			207
hcf3	1S	high chlorophyll fluorescence (= *hcf9*): missing PSII thylakoid membrane core complex; green seedling			207
hcf4	1L	high chlorophyll fluorescence: affects CO2 fixation; green seedling			208
hcf5	6S	high chlorophyll fluorescence: affects PSII reaction; green seedling			209
hcf6	1S	high chlorophyll fluorescence: missing cytochrome f/b6 complex; green seedling			173
hcf9		(= *hcf3*)			
hcf12	1L	high chlorophyll fluorescence			173
hcf13	1L	high chlorophyll fluorescence: affects CO2 fixation; green seedling			173 208
hcf15	2L	high chlorophyll fluorescence: affects photophosphorylation; yellow-green seedling			173
hcf18	5L-near *pr1*	high chlorophyll fluorescence (= *hcf43*): major loss of PSI; other thylakoid complexes reduced; yellow-green seedling			208
hcf19	3L	high chlorophyll fluorescence: affects PSII thylakoid membrane core complex; green/yellow-green seedling			173 208
hcf21	5L	high chlorophyll fluorescence: affects CO2 fixation, Rubisco; green seedling			208
hcf23	4S	high chlorophyll fluorescence: affects photophosphorylation; green seedling			173 208
hcf26	6S	high chlorophyll fluorescence: affects electron transport; yellow-green, viable seedling			173 208
hcf28	10L	high chlorophyll fluorescence: affects CO2 fixation; green seedling			209
hcf31	1S	high chlorophyll fluorescence: missing chlorophyll a/b binding protein; yellow-green seedling			209
hcf34	6L	high chlorophyll fluorescence: affects photophosphorylation; yellow-green seedling			173 208
hcf36	6L	high chlorophyll fluorescence: affects electron transport; green seedling			209
hcf38	5L	high chlorophyll fluorescence: affects cytochrome f/b6 complex, alpha and beta components of CF1; green seedling			173
hcf41	1L	high chlorophyll fluorescence: affects PSII thylakoid membrane core complex; green seedling			173 208
hcf42	9L	high chlorophyll fluorescence: affects Rubisco; green/yellow-green seedling			208
hcf43		(= *hcf18*)			
hcf44	1L	high chlorophyll fluorescence: affects PSI membrane core complex; pale-green seedling			208
hcf46	3L	high chlorophyll fluorescence			173
hcf47	10S	high chlorophyll fluorescence: affects cytochromes; yellow-green seedling			209
hcf48	6L	high chlorophyll fluorescence: affects electron transport; yellow-green seedling			209
hcf50	1L	high chlorophyll fluorescence: missing PSI thylakoid membrane core complex; green seedling			208
hcf101	7L	high chlorophyll fluorescence (was *Mu-5**): affects PSI thylakoid membrane core complex			209
hcf102	8L	high chlorophyll fluorescence: affects cytochrome f/b6 complex (D. Miles, unpublished)			
hcf316	10S	high chlorophyll fluorescence: affects chlorophyll a/b binding protein; yellow-green seedling			209
hcf323	6S	high chlorophyll fluorescence: affects photophosphorylation, coupling factor; green seedling			209
hcf408	6L	high chlorophyll fluorescence: affects chlorophyll a/b binding protein; yellow-green seedling			209
Hex1	3S-near *cg1*	hexokinase: electrophoretic mobility; null allele is known; cytosolic; monomeric			349
Hex2	6L-near *Pt1*	hexokinase: electrophoretic mobility; null allele is known; cytosolic; monomeric			349
hm1	1L-64	*Helminthosporium carbonum* susceptibility: disease lesions vs. yellowish flecks (resistant) on leaves with race 1	S	P	343
hm2	9L-near *bk2*	*Helminthosporium carbonum* susceptibility: like *hm1*; masked by *Hm1*			230
Hs1	7S-0	hairy sheath: abundant hairs on leaf sheath	S	P	337
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		132
Ht2	-	*Helminthosporium turcicum* resistance			133
Ht3	-	*Helminthosporium turcicum* resistance: (from *Tripsacum floridanum*)			134
I		inhibitor (= *C1-I*, inhibitor allele at *C1* locus): also commonly used as a general symbol for inhibition and for 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		311
Idh1	8L	isocitrate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			117
Idh2	6L-near *w14*	isocitrate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			117
ig1	3L-90	indeterminate gametophyte: polyembryony, heterofertilization, polyploidy, androgenesis (male and female affected)	S		159
ij1	7L-52	iojap striping: many variable white stripes on leaves; conditions chloroplast defects that are cytoplasmically inherited	S	P	138
ij2	1L	iojap striping: like *ij1*; chloroplast inheritance unknown			233
in1	7S-20	intensifier: intensifies aleurone anthocyanin pigments; *In1-D* dominant dilute	S	P	91
Inv		Inversion: general symbol for inversion of a segment of chromosome	S	P	
is1	-	cupulate interspace			96
Isr1	10L-near *R1*	inhibitor of striate (was *Ej1*): reduces expression of *sr2* and other leaf-striping factors	S		160
j1	8L-42	japonica striping: white stripes on leaf and sheath; not expressed in seedling	S	P	73
j2	4L-106	japonica striping: extreme white striping of leaves, etc. (R.A. Emerson, unpublished)	S	P	82
K		knob: general symbol for constitutive heterochromatic elements			
K3L	3L-115	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	183
Kn1	1L-near *Adh1*	knotted: localized proliferation of tissue at vascular bundles on leaf	S	P	28
Krn		kernel row number: designator for factors determining kernel row number			216
l1	10L-near *R1*	luteus: yellow pigment in white tissue of chlorophyll mutants *w1*, *w2*, *j1*, *ij1*, etc.	S	P	176 177

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
l4	-	luteus: lethal yellow seedling	S	P	144
l6	9S-near *bz1*	luteus: like *l4* (W.H. Eyster, unpublished)	S		
l7	9S-42	luteus: yellow seedling and plant; lethal	S		89
l10	6L-19	luteus: like *l4*; fails to convert protochlorophyllide to chlorophyllide	S		279
l11	6S	luteus (was *l*-4120*): yellow seedling with green leaf tips; lethal	S		8
l12	6L-16	luteus (was *l*-4902*): like *l11*	S		53
l13	10L-91	luteus (was *l*-59A*, *l*-Neuffer2*): dark yellow, lethal seedling; fails to convert protoporphyrin IX to Mg-protoporphyrin			194 233
l15	6L-30	luteus (was *l*-Blandy3*, *l*-Brawn*): like *l4*			283
l16	1S	luteus (was *l*-515*): like *l4*; leaves bleach to paler yellow in patches			233
l17	1L	luteus (was *l*-544*): like *l4*; leaves with lighter yellow crossbands			233
l18	2L	luteus (was *l*-1940*): like *l4*			233
l19	10S	luteus (was *l*-425*): like *l4*			233
la1	4S-55	lazy plant: prostrate growth habit	S	P	145
Lc1	10L-65	red leaf color: anthocyanin in coleoptile, nodes, auricle, leaf blade, etc.; (compare *Sn1*)	S		66
Lcs1	-	thylakoid membrane polypeptide: electrophoretic mobility			219
Lct1	-	thylakoid membrane polypeptide: electrophoretic mobility			219
lct2	-	thylakoid membrane polypeptide: presence-absence			219
Les1	2S-58	lesion (was *Les*-843*): large necrotic lesions resembling disease lesions formed by fungal infections on susceptible lines	S		234
Les2	1S-near *sr1*	lesion (was *Les*-845A*): small white lesions resembling disease lesions formed by fungal infections on resistant lines	S		234
Les3	10	lesion: like *Les1*			7
Les4	2L	lesion (was *Les*-1375*): late expression of large necrotic lesions			130
Les5	1S	lesion (was *Les*-1449*): like *Les2*			130
Les6	10S	lesion (was *Les*-1451*): like *Les4*			130
Les7	?	lesion (was *Les*-1461*): late expression of small chlorotic lesions			130
Les8	9S-near *lo2*	lesion (was *Les*-2005*): late expression of small, pale green lesions			130
Les9	7L-near *ra1*	lesion (was *Les*-2008*): late expression of small necrotic lesions			130
Les10	2-near *v4*	lesion (was *Les*-A607*): like *Les1*			131
Lfy1	-	leafy: increased number of leaves			304
lg1	2S-11	liguleless: ligule and auricle missing; leaves upright, enveloping	S	P	71 72
lg2	3L-101	liguleless: like *lg1*, less extreme	S	P	23
Lg3	3-65	liguleless: dominant, no ligule; leaves upright, broad, often concave and pleated	S	P	250
li1	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		344
ln1	6	linoleic acid: lower ratio of oleate to linoleate in kernel			54
lo2	9S-50	lethal ovule: ovules containing *lo2* gametophyte abort	S		227
loc1	-	low oil content in kernel: associated with albino seedlings			259
lp1	4	lethal pollen: *lp1* pollen fails in competition with *Lp1*			226
lte1	2	latente: drought, heat, aluminum tolerance; frost resistance; from Michoacan 21; dominance varies			213
Lte2	10L-near *g1*	latente: drought, heat, aluminum tolerance; from Cateto; epistatic to *lte1*			214
lty1	-	light yellow endosperm			63
lty2	-	light yellow endosperm			63
lu1	5S-29	lutescent: pale yellow green leaves	S		309
lw1	1L-near *Adh1*	lemon white: white seedling, pale yellow endosperm	S		341
lw2	5L-near *pr1*	lemon white: like *lw1*	S	P	341
lw3	5L-near *v2*	lemon white (duplicate factor with *lw4*): like *lw1*			341
lw4	4-near *zb6*	lemon white (duplicate factor with *lw3*): like *lw1*			341
lyc1		(= *ps1-lyc*)			
mal1	9	multiple aleurone layering: recessive interacts with two complementary dominants *Mal2* and an unnamed factor, giving multiple cell layers			211
Mal2	4	multiple aleurone layering: (see *mal1*)			211
Mc1	-	mucronate: opaque endosperm			287
Mdh1	8	malate dehydrogenase: electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur			242
Mdh2	6L-near *w14*	malate dehydrogenase: electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur			242
Mdh3	3L-146	malate dehydrogenase: electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur			242
Mdh4	1L-near *an1*	malate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			242
Mdh5	5S-17	malate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			242
Me1	3L-125	malic enzyme: electrophoretic mobility; null allele is known; tetrameric			117
Mei1	-	meiosis: chromosomes sticky in metaphase I; male sterile			107 108
mep1	5L	modifier of endosperm protein: affects quantities of *Prot1* protein forms			301
Mer		Maya earworm resistance: designator for earworm resistance factors from IAC Maya			215
mg1	-	miniature germ (replaces *mg* of Wentz): germ 1/4 to 1/3 of normal; viable			167
mi1	1	midget plant: small plant (H.S. Perry, unpublished)	S		
mmm1	1L-near *an1*	modifier of mitochondrial malate dehydrogenases: mobilities			242
mn1	2-near *fl1*	miniature seed: small, somewhat defective kernel; fully viable	S	P	185
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*			25
Mpl1	1L-near *Adh1*	miniplant: dominant, andromonoecious, intermediate dwarf; probable allele of *D8*; not responsive to gibberellins (M. Freeling, unpublished)			121
Mr	9S-near *l7*	mutator of *R-m*: transposable factor, regulates *R1-m* mutation		P	35
Mrh		mutator: controlling element of *a1-m-rh*			273
ms1	6L-near *si1*	male sterile: anthers shriveled, not usually exserted; affected at microspore vacuolation	S		314
ms2	9L-64	male sterile: like *ms1*; affected between vacuolation and pore formation	S		87 89
ms3	3	male sterile: anthers shrivelled; not usually exserted			87 89
ms4		(= *po1*)			
ms5	5-near *v3*	male sterile: anthers not exserted; affected at microspore mitosis	S		13
ms7	7L-near *ra1*	male sterile: like *ms2*	S		13
ms8	8L-28	male sterile: like *ms5*; affected in meiosis	S	P	13
ms9	1S-near *P1*	male sterile: like *ms5*; affected in meiosis	S		13
ms10	10L-near *bf2*	male sterile: like *ms5*; affected at microspore vacuolation	S		13
ms11	10	male sterile: like *ms5*; affected at microspore mitosis	S		13

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
ms12	1	male sterile: like *ms1*; affected at microspore vacuolation	S		13
ms13	5S	male sterile: like *ms5*; affected at microspore vacuolation	S		13
ms14	1-near *as1*	male sterile: like *ms5*; affected at microspore mitosis	S		13
ms17	1S-23	male sterile: like *ms1*; affected variably in meiosis	S		79
ms20	-	male sterile			89
Ms21	6	male sterile: pollen grains developing in presence of *Ms21* are defective and nonfunctional if *sks1*, normal if *Sks1*			171 295
ms22	-	male sterile: affected in meiosis			351
ms23	3L	male sterile (allelic to *ms*-Bear7*): affected in meiosis			351
ms24	-	male sterile: like *ms1*; affected in microspore mitosis			351
ms28	-	male sterile: anaphase I disturbed, spindle persists			108
Ms41	4L	male sterile (was *Ms*-1995*)			241
ms43	8L	male sterile: anaphase I impaired			107 108
Msc1	1L	mosaic (was *Msc*-791A*): aleurone mosaic for anthocyanin color			241
Msc2	5S	mosaic (was *Msc*-1124B*): aleurone mosaic for anthocyanin color			241
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*			282
Mut	2S-near *gl2*	mutator: controlling element for *bz1-m-rh*			274
Mv1	-	resistance to maize mosaic virus I ("corn stripe")			21
na1	3L-113	nana plant: short, erect dwarf; no response to gibberellins	S	P	137 175
na2	5S-near *bt1*	nana plant: like *na1* (H.S. Perry, unpublished)	S		
NCS1		nonchromosomal stripe: maternally inherited light green leaf striping			310
NCS2		nonchromosomal stripe: maternally inherited pale green and depressed striping; mitochondrial	S		43
NCS3		nonchromosomal stripe: maternally inherited striations, distorted plants; mitochondrial	S		43
nec1	8L-near *fl3*	necrotic (was *nec*-6697*, *sienna*-7748*): chlorotic seedling that stays rolled, wilts and dies	S		195
nec2	1S-34	necrotic (was *nec*-8147*, *olive-necrotic-8147*, *ON-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		232
nec4	2S-near *d5*	necrotic (was *nec*-516B*): seedling yellow, leaf tips necrotic; lethal			129
nec5	4L	necrotic (was *nec*-642A*): pale green seedling becoming necrotic; dark brown exudate; lethal			233
nec6	5S-near *a2*	necrotic (was *nec*-493*): like *nec3*			233
nec7	5L	necrotic (was *nec*-756B*): seedling becoming necrotic in crossbands; lethal			233
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		199
NPI		Native Plants, Inc.: designator for loci defined by restriction fragment polymorphisms			
o1	4L-near *gl3*	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; regulates b32 protein (see *pro*); reduced lysine degradation (lysine-ketoglutaric reductase) (W.R. Singleton and D.F. Jones, unpublished)	S	P	
o4		(= *fl1*)			
o5	7L-near *ra1*	opaque endosperm: like *o1*; virescent to yellow or white seedlings	S		278
o6		(= *pro1*)			
o7	10L-87	opaque: like *o1*; high lysine content	S		217
o9	-	opaque endosperm: crown opaque and light in color, frequently with a cavity; base or abgerminal side of kernel often corneous	S		225
o10	-	opaque endosperm: like *o1*	S		225
o11	-	opaque endosperm: thin, opaque, somewhat shrunken kernels with greyish cast	S		225
o12	-	opaque endosperm: thin, etched or scarred kernels, variable in size; plants chlorophyll deficient and small, with pollen but few ears			225
o13	-	opaque endosperm: opaque, etched kernels with rim of corneous starch on abgerminal side	S		225
Og1	10S-16	old gold stripe: variable bright yellow stripes on leaf blade	S	P	181
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 *Orom1*; fails to convert Mg-protoporphyrin monomethyl ester to protochlorophyllide	S		194
oro2	-	orobanche: like *oro1*			194
Orom1	-	orobanche modifier: partially corrects chlorophyll loss in *oro1*			194
orp1	4S	orange pericarp (duplicate factor with *orp2*): pericarp orange over *orp1 orp2* kernels; lethal			235
orp2	10L	orange pericarp (duplicate factor with *orp1*)			235
oy1	10S-12	oil yellow: seedling oily greenish-yellow; viable; fails to convert protoporphyrin IX to Mg-protoporphyrin; *oy1-t* tinged green; *oy1-1039*, *oy1-1040* lethal; *Oy1-700* dominant yellow-green	S	P	88
P		plant color component at *R1*: anthocyanin pigmentation in seedling leaf tip, coleoptile, anthers			329 330
P1	1S-26	pericarp color: red pigment in cob and pericarp	S	P	70 182
pam1	-	plural abnormalities of meiosis: desynchronized meiotic divisions and premeiotic mitosis; male sterile, incompletely female sterile			111
pam2	-	plural abnormalities of meiosis: like *pam1*			109
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			168
Pdf1	-	thylakoid membrane polypeptide: dominant increase in electrophoretic mobility			220
pe1	-	perennialism: vegetatively totipotent in combinations with *gt1* and *id1*			303
pg11	6L-38	pale green (duplicate factor with *pg12*): seedling light yellowish green; mature plant pale and vigorous	S	P	269
pg12	9-61	pale green (duplicate factor with *pg11*)	S		269
pg13	-	pale green: seedling light yellowish green; stunted growth			308
pg14		(= *g2*)	S	P	253
pg15	1S	pale green (was *ppg*-340B*): seedling light yellowish green; bleaches to near white in patches; lethal			233
pg16	1L	pale green (was *pg*-219*): seedling light yellowish green			233
Pgd1	6-near *rgd1*	6-phosphogluconate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			117
Pgd2	3L-near *ts4*	6-phosphogluconate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			117
Pgm1	1L-near *Prot1*	phosphoglucomutase: electrophoretic mobility; null allele is known; cytosolic; monomeric			117
Pgm2	5S-0	phosphoglucomutase: electrophoretic mobility; null allele is known; cytosolic; monomeric			117
Ph1	4S-0	pith abscission: cob disarticulation			97
Phi1	1L-149	phosphohexose isomerase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intralocus hybrid bands occur			117
pi1	-	pistillate florets (duplicate factor with *pi2*): secondary florets develop ("Country Gentlemen" or "Shoe Peg" expression) in *pi1 pi2* ears			135

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
pi2	-	pistillate florets (duplicate factor with *pi1*)			135
PIO		Pioneer Hi-Bred, International: designator for loci defined by restriction fragment polymorphisms			
Pl1	6L-49	purple plant: sunlight-independent purple pigment in plant	S	P	77
pm1	3L-near *ts4*	pale midrib: midrib and adjacent tissue lighter green	S	P	24
Pn1	7L-112	papyrescent glumes: long, thin papery glumes on ear and tassel	S	P	98
po1	6S-4	polymitotic (= *ms4*): repeats 2nd meiotic division in male and female	S	P	11
ppg1	5L	pale pale green (was *cb*-199A*): white seedling with faint green; white necrotic crossbands; lethal			233
pr1	5L-67	red aleurone: changes purple aleurone to red; flavonoid 3'-hydroxylase	S	P	69
pro1	8L-near *fl3*	proline requiring (= *o6*): crumpled opaque kernel; b32 protein isoforms and null; green-striped lethal seedling			100
Prot1	1L-121	protein: embryo protein mobility variations; null allele is known			301
ps1	5S-39	pink scutellum (= *vp7*): viviparous; endosperm and scutellum pink, seedling white with pink flush; *ps1-lyc* not viviparous	S	P	320
Pt1	6L-60	polytypic ear: proliferation produces irregular growth on ear and tassel	S	P	229
Px1	2L	peroxidase: electrophoretic mobility; null allele is known; monomeric			120
Px2	-	peroxidase: electrophoretic mobility; monomeric			187
Px3	7L-near *Pn1*	peroxidase: electrophoretic mobility; monomeric			187
Px4	-	peroxidase: electrophoretic mobility; null allele is known; monomeric			187
Px5	-	peroxidase: presence-absence			187
Px6	-	peroxidase: presence-absence			187
Px7	-	peroxidase: electrophoretic mobility; null allele is known; monomeric			187
Px8	-	peroxidase: electrophoretic mobility; monomeric			22
Px9	-	peroxidase: electrophoretic mobility; null allele is known; monomeric			22
py1	6L-69	pigmy plant: leaves short, pointed; fine white streaks	S	P	336
py2	1L	pigmy: like *py1*			233
pyd1	9S-near *yg2*	pale yellow deficiency: pale yellow seedling; deficiency for short terminal segment of chromosome arm; lethal			200
R1	10L-61	colored: red or purple color in aleurone and/or anthers, leaf tip, brace roots, etc.	S	P	69
ra1	7L-32	ramosa: ear branched, tassel conical	S	P	13 103
ra2	3S-49	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		228
rd2	6L	reduced plant: like *rd1*, but not as extreme			106
rDNA		ribosomal DNA: rDNA5.8S, rDNA18S and rDNA25S located in NOR on 6S; rDNA5S on 2L near *Ht1*			
rDt		receptor of Dotted			318
Rf1	3S-near *Lg3*	fertility restorer: restores fertility to cms-T; complementary to *Rf2*	S		148
Rf2	9-near *wx1*	fertility restorer: see *Rf1*	S		68
Rf3	2L	fertility restorer: restores fertility to cms-S			29
Rf4	2	fertility restorer: restores fertility to cms-C			118
Rg1	3-67	ragged leaves: defective tissue between veins of older leaves, causing holes and tearing	S	P	26
rgd1	6-8	ragged seedling: seedling leaves narrow, thread-like, have difficulty in emerging	S	P	163
Rgd2	5	ragged leaves (was *Rgd*-1445*): leaves narrow and distorted; tillering			237
rgo1	-	reversed germ orientation: embryo faces base of ear; variable frequency, maternal trait			285
rhm1	6-near *rgd1*	resistance to *Helminthosporium maydis*: chlorotic-lesion reaction with race O	S		315
Ri1	4S-27	rind abscission: cob disarticulation			97
Rp1	10S-0	resistance to *Puccinia sorghi*		P	189 190
Rp3	3-near *gl6*	resistance to *Puccinia sorghi*	S		352
Rp4	4S-24	resistance to *Puccinia sorghi*	S		352
Rp5	10S-near *Rp1*	resistance to *Puccinia sorghi*			288
Rp6	10S-near *Rp1*	resistance to *Puccinia sorghi*			352
Rpp9	10S-near *Rp1*	resistance to *Puccinia polysora* and *P. sorghi*			342
Rs1	-	rough sheath: extreme ligule disorganization	S		161
rs2	1-near *as1*	rough sheath	S		161
rt1	3S-near *Cg1*	rootless: secondary roots few or absent	S	P	141
S		seed color component at *R1*: anthocyanin pigmentation in aleurone			329
Sad1	10L-near *bf2*	shikimate dehydrogenase: electrophoretic mobility; plastidial; monomeric			347
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 Ill677a			90
sen1	3	soft endosperm (duplicate factor with *sen2*): endosperm soft, opaque			331
sen2	7	soft endosperm (duplicate factor with *sen1*)			331
sen3	1	soft endosperm (duplicate factor with *sen1*): like *sen1*			331
sen4	-	soft endosperm (duplicate factor with *sen3*)			331
sen5	2	soft endosperm (duplicate factor with *sen6*): like *sen1*			331
sen6	5	soft endosperm (duplicate factor with *sen5*)			331
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	95
sh1	9S-29	shrunken: inflated endosperm collapses on drying, forming smoothly indented kernels; sucrose synthase-1 of endosperm (compare *Css1*); homotetramer	S	P	136
sh2	3L-149.2	shrunken: inflated, transparent, sweet kernels collapse on drying, becoming angular and brittle; ADPG pyrophosphorylase (compare *bt2*)	S	P	191
sh4	5L	shrunken: collapsed, chalky endosperm	S		340
si1	6L-20	silky (= *ms-si*): multiple silks in ear; sterile tassel with silks	S		92
sk1	2S-56	silkless ears: pistils abort, no silks	S		147
Sks1	2L-near *v4*	suppressor of sterility: pollen grains developing in presence of *Ms21* are defective and nonfunctional if *sks1*, normal if *Sks1*			171 295
sl1	7L-50	slashed leaves: leaves slit longitudinally by necrotic streaks	S		125
sm1	6L-59	salmon silks: silks salmon color with *P1-RR*, brown in *P1-WW*	S	P	1
Sn1	10L-near *R1*	scutellar node color: anthocyanin in coleoptile, nodes, auricle, leaf blade, etc. (compare *Lc1*)			101
Sod1	-	superoxide dismutase: electrophoretic mobility; plastidial; dimeric; intralocus hybrid bands occur			9
Sod3	-	superoxide dismutase: electrophoretic mobility; mitochondrial; tetrameric; intralocus hybrid bands occur			9
Sod4	-	superoxide dismutase: electrophoretic mobility; cytosolic; dimeric; intralocus hybrid bands occur			9
Spc1	3L-near *ig1*	speckled (was *Spc*-1376*, *Les*-1376*): brown speckling on leaves and sheath at flowering; supporting tissues weak			237
spc2	1L	speckled (was *spc*-262A*): green seedling with light green speckles			233
spc3	3L	speckled (was *pg*-553C*): green seedling with dark and light green speckles			233
Spm		suppressor-mutator: autonomous transposable element (equivalent to *En*); regulates *dSpm* transposition and function at *a1-m1*, *a1-m2*, *bz1-m13*, etc.			202
spt1	2L	spotted (was *spt*-464*): pale green seedling with dark green spots			233
spt2	4S	spotted (was *pg spt*-1269A*): like *spt1*			233
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	146

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
sr3	10S	striate leaves: virescent and striate to striped	S	P	105
Ss2		(see *Css1*)			
st1	4S-62	sticky chromosome: small plant, striate leaves, pitted kernels resulting from sticky chromosomes; *st1-e* heightened by high temperature	S	P	14
su1	4S-66	sugary: endosperm wrinkled and translucent when dry; sweet at milk stage; starch debranching enzyme I; *su1-am* sugary-amylaceous; *su1-st* recessive starchy	S	P	51
su2	6L-58	sugary: endosperm glassy, translucent, sometimes wrinkled	S		89
Sup1	-	suppressor: modifies *o2* kernels to semi-transparent			196
sy1	-	yellow scutellum			319
T		reciprocal translocation: general symbol for exchange of parts between two nonhomologous chromosomes	S	P	
Ta1	-	transaminase (possibly = *Got1*): electrophoretic mobility; dimeric; intralocus hybrid bands occur			187
tb1	1L-near *Adh1*	teosinte branched: many tillers; ear branches tassel-like	S		33
td1	5-near *bt1*	thick tassel dwarf: (E.G. Anderson, unpublished)	S		
te1	3	terminal ear: stalked ear appendages at tip; varying to infolded ears	S		197
Thc1	-	thiocarbamate sensitive: sensitive to Eradicane			254
tl1	-	tasselless			205
Tlr1	1L	tillered (was *Tlr*-1590*): extreme tillering			241
Tp1	7L-46	teopod: many tillers, narrow leaves, many small partially podded ears, tassel simple	S	P	180
Tp2	10L-45	teopod: like *Tp1*	S	P	252
Tpi1	-	triose phosphate isomerase: electrophoretic mobility; plastidial; dimeric			347
Tpi2	-	triose phosphate isomerase: electrophoretic mobility; plastidial; dimeric			347
Tpi3	8	triose phosphate isomerase: electrophoretic mobility; cytosolic; monomeric			347
Tpi4	3L-near *Rg1*	triose phosphate isomerase: electrophoretic mobility; cytosolic; monomeric			349
tpm1	-	thylakoid peptide modifier: dominant decrease in electrophoretic mobility			218
tr1	-	two-ranked ear: distichous vs. decussate phyllotaxy in ear axis			168
ts1	2S-74	tassel seed: tassel pistillate and pendant; if removed, small ear with irregular kernel placement develops	S		75
ts2	1S-24	tassel seed: like *ts1*, but branches pendant rather than whole tassel	S	P	75
ts4	3L-73	tassel seed: tassel compact, upright, with pistillate and staminate florets	S	P	256
Ts5	4S-53	tassel seed: nearly normal tassel with scattered, short silks	S		80
Ts6	1L-158	tassel seed: tassel pistillate to mixed, compact; ear with irregular kernel placement	S	P	243
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	240
Ufo1	-	unstable factor for orange: anthers, silks, and most other plant parts orange with *P1-WR* or *P1-RR*; growth retarded			335
UMC		University of Missouri, Columbia: designator for loci defined by restriction fragment polymorphisms			
Uq		ubiquitous: controlling element mediating *a1-ruq*			94
v1	9L-63	virescent: yellowish white seedling, greens rapidly; low temperature exaggerates	S	P	56
v2	5L-107	virescent: like *v1*, but greens slowly; low temperature exaggerates	S	P	73
v3	5L-45	virescent: light yellow seedling, greens rapidly; low temperature exaggerates	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*; lethal	S		57
v12	5L-near *ys1*	virescent: like *v3*	S		257
v13	-	virescent: first leaf with green tip; greens slowly	S		257
v16	8L-14	virescent: like *v2*	S		257
v17	4	virescent: like *v1*, but greening from base to tip	S		257
v18	10	virescent: like *v1*	S		257
v19		(= *g2*)			
v21	8L	virescent (was *v*-25*, *v*-A552*): grainy virescent, greening from tips and margins inward	S		17
v22	1L-near *an1*	virescent (was *v*-18983*): like *v1* (E.G. Anderson, unpublished)	S		
v23	4-near *su1*	virescent (was *v*-8914*): like *v1* (E.G. Anderson, unpublished)	S		
v24	2L	virescent (was *v*-424*): like *v1*			233
v25	1S	virescent (was *v*-17*): greenish white seedling; greens from base upward			233
v26	2S	virescent (was *v*-453*): yellowish white seedling with green leaf tip and midrib			233
v27	7L	virescent (was *v*-590A*): like *v1*			233
v28	9S	virescent (was *v*-27*): like *v1*			233
v29	10L	virescent (was *v*-418*): grainy virescent			233
v30	9L-87	virescent (was *v*-8587*): like *v1*			44
va1	7L-near *ij1*	variable sterile: variable male and female fertility; cytokinesis fails in anaphase I	S		12
Vg1	1L-85	vestigial glume: glumes very small, cob and anthers exposed	S	P	322
vp1	3L-near *ts4*	viviparous: embryo fails to become dormant, viable if transplanted; some alleles dormant; chlorophyll and carotenoids unaffected; anthocyanins in aleurone suppressed	S		86
vp2	5S-38	viviparous: embryo fails to become dormant; white endosperm, white seedling; anthocyanins unaffected	S	P	86
vp5	1S-1	viviparous: like *vp2*	S	P	275
vp7		(= *ps1*)			
vp8	1L-154	viviparous: embryo fails to become dormant; chlorophyll and carotenoids unaffected; small, pointed-leaf seedlings	S		276
vp9	7S-25	viviparous (also known as *y7*): like *vp2*; *vp9-4889* dormant, pale aleurone, pale green seedling	S	P	276
Vsr1	10L	virescent striped (was *Vsr*-1446*): virescent seedling; greens to white and yellow striped plant			241
w1	6L-near *w14*	white: white seedling	S		71 72 177
w2	10L-77	white: white seedling; endosperm pitted and spotted (compare *dek21*)	S		179
w3	2L-111	white: like *vp2*; *w3-8686* dominant, pale endosperm, pale green seedling in dim light	S	P	179
w11	9S-54	white: like *w1*	S		57
w14	6L-78	white (was *w*-8657*): like *w1*	S		53
w15	6L-13	white (was *w*-8896*): like *w1*; fails to convert protochlorophyllide to chlorophyllide	S		53
w16	7S-near *vp9*	white: like *w1*			221
w17	7S-near *Hs1*	white: like *w1*			221
Wc1	9L-107	white cap: kernel with white crown and pale yellow endosperm	S		165
wd1	9S-near *yg2*	white deficiency: white seedling: deficiency for distal half of first chromomere of short arm	S	P	200
wgs1	5L	white green sectors (was *sct*-206B*): white seedling with green sectors			233
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		261
wlu1	3L	white luteus (was *wl*-28*): pale yellow seedling; lethal			233
wlu2	7L	white luteus (was *wl*-643A*): like *wlu1*			233
wlu3	8L	white luteus (was *wl*-203A*): like *wlu1*			233
wlu4	9L	white luteus (was *wl*-41A*): like *wlu1*			233
Wrk1	3S	wrinkled kernel (was *Wr*-1020*): kernels small and wrinkled			241
ws1	-	white sheath: light yellow leaf sheaths; duplicate factor with *ws2*	S		156
ws2	-	white sheath: see *ws1*	S		156

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
ws3	2S-0	white sheath: white leaf sheath, culm, husks	S	P	267
wsp		weak striped plant: maternally inherited pale striping			27
wt1	2S-60	white tip: tip of first leaf white and blunt	S		327
wt2	4S	white tip (was *cb*-10*): seedling with white leaf tip and crossbands on first 2 leaves			233
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			221
y1	6L-17	white endosperm: reduced carotenoid pigments in endosperm; some alleles affect chlorophyll in seedlings (e.g. *y1-8549*)	S	P	51
y3	2S-near *al1*	white endosperm: like *y1*			251
y7		(= *vp9-y7*)			
y8	7S-18	white endosperm: pale yellow endosperm	S		143
y9	10S-24	white endosperm: pale yellow endosperm, slightly viviparous; green to pale green seedlings and plants	S		281
y10	3L	white endosperm (was *w*-7748*): pale yellow endosperm; white seedling; lethal	S		277
y11	-	white endosperm: pale yellow endosperm; green seedling			326
y12	-	white endosperm: like *y11*			326
yd2	3L-near *lg2*	yellow dwarf			280
yg1	5L-near *v2*	yellow-green: yellow-green seedling and plant	S		84
yg2	9S-7	yellow-green: like *yg1*	S	P	140
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			260
ys3	3L-near *Rg1*	yellow stripe: like *ys1*	S		353
Ysk1	4-near *su1*	yellow streaked (was *Ysk*-844*): longitudinal yellow streaks top 3rd of mature leaves			237
z1		(= *vp9-z* = *y7-z*)			
zb1	-	zebra crossbands: yellowish crossbands on older leaves	S		55
zb2	-	zebra crossbands: crossbands on seedling leaves	S		333
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	124
zb6	4-79	zebra crossbands: regularly spaced crossbands on earlier leaves; enhanced by cool temperatures	S		126
zb7	1L-near *Adh1*	zebra crossbands (was *zb*-101*): lighter green crossbands on seedlings; glossy			233
Zb8	9-near *wx1*	zebra crossbands (was *Atc1*, *Cb*-1443*): yellow-green crossbands on older leaves; strong anthocyanin expression in leaf tip and blade			237 241
Zer		Zapalote Chico earworm resistance: designator for earworm resistance factors from Zapalote Chico			215
zn1	10L-26	zebra necrotic: necrotic tissue appears between veins in transverse leaf bands on half-grown or older plants	S	P	122
zn2	-	zebra necrotic: like *zn1*	S		104
Zp		zein polypeptide: designator for loci determining zein polypeptides			316 317
zpg1	-	zebra-stripe pale green			63

REFERENCES TO ORIGINAL DESCRIPTIONS AND DESIGNATIONS

(gene symbols referenced are included within brackets)
(MNL = Maize Genetics Cooperation News Letter)

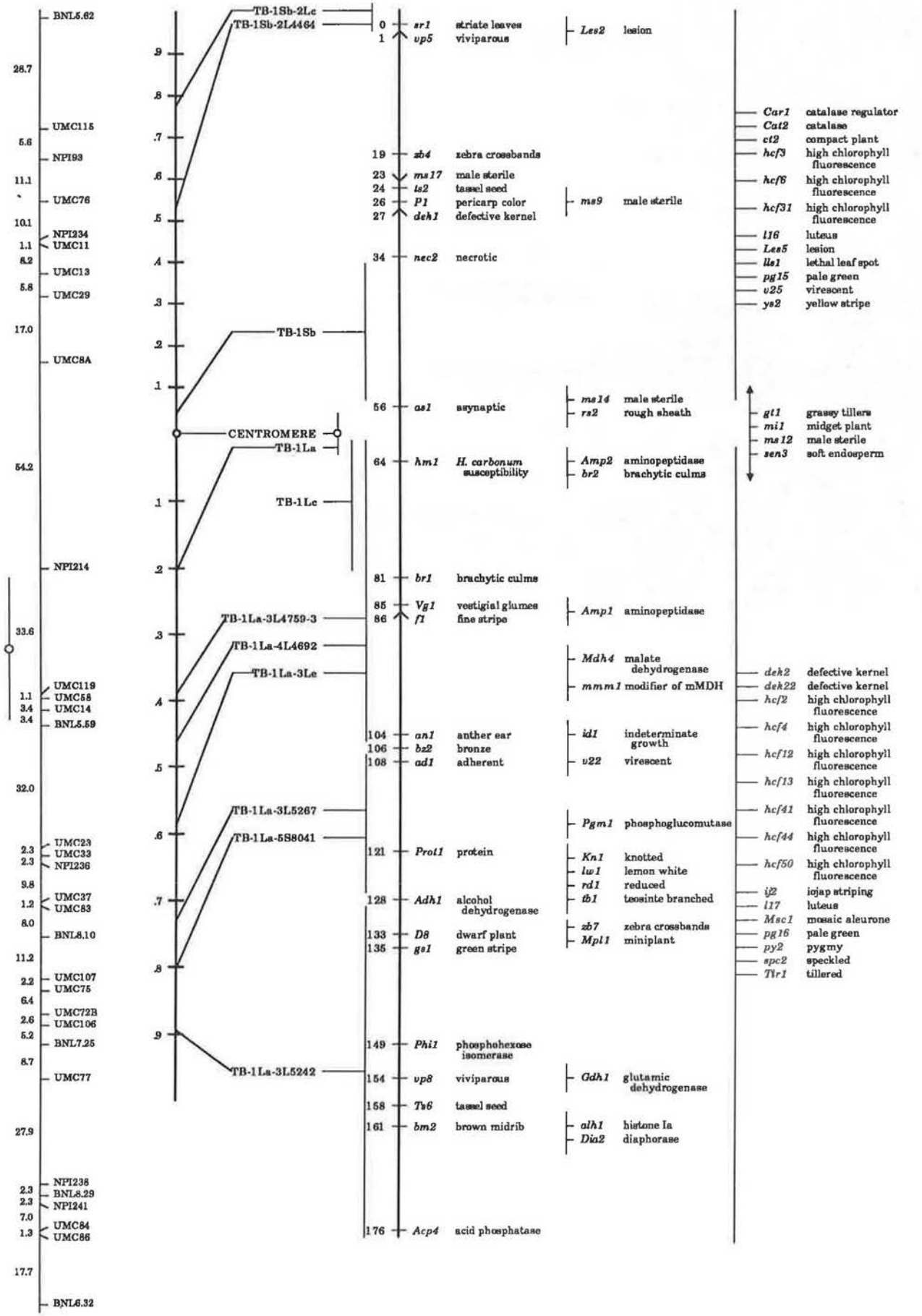
1. Anderson, E.G. 1921. Cornell Univ. Agr. Exp. Sta. Memoir 48:533-554. (*sm1*)
2. Anderson, E.G. 1953. MNL 27:5. (*bf2*)
3. Anderson, E.G. 1955. MNL 29:5. (*gl15*)
4. Anderson, E.G. 1955. MNL 29:6. (*gl18*)
5. Anderson, E.G. and R.A. Emerson. 1931. Amer. Nat. 65:253-257. (*Ch1*)
6. Ashman, R.B. 1960. Genetics 45:19-34. (*Mst1*)
7. Ashman, R.B. and A.J. Ullstrup. 1976. J. Hered. 67:220-222. (*Les3*)
8. Bachmann, M.D., et al. 1973. J. Ultrastruct. Res. 45:384-406. (*l11*)
9. Baum, J.A. and J.G. Scandalios. 1982. J. Hered. 73:95-100. (*Sod1 Sod3 Sod4*)
10. Beadle, G.W. 1929. Amer. Nat. 63:189-192. (*ys1*)
11. Beadle, G.W. 1931. Cornell Univ. Agr. Exp. Sta. Memoir 135:1-12. (*po1*)
12. Beadle, G.W. 1932. Cytologia 3:142-155. (*va1*)
13. Beadle, G.W. 1932. Genetics 17:413-431. (*ms5 ms7 ms8 ms9 ms10 ms11 ms12 ms13 ms14 ra1*)
14. Beadle, G.W. 1932. Ztschr. ind. Abst. Vererbungsl. 63:195-217. (*st1*)
15. Beadle, G.W. and B. McClintock. 1928. Science 68:433. (*as1*)
16. Beckett, J.B. 1971. Crop Science 11:724-727. (cms-C)
17. Beckett, J.B. and M.G. Neuffer. 1973. MNL 47:147. (*v21*)
18. Beckman, L., et al. 1964. Science 146:1174-1175. (*Cat1*)
19. Bird, R.M. and M.G. Neuffer. 1985. MNL 59:42. (*Sdw1*)
20. Bird, R.M. and M.G. Neuffer. 1986. Pp. 818-822 in M. Freeling, ed., Plant Genetics, Alan R. Liss, N.Y. (*Hsf1*)
21. Brewbaker, J.L. 1974. Proc. 29th Ann. Corn and Sorghum Res. Conf. pp. 118-133. (*Mu1*)
22. Brewbaker, J.L. and Y. Hasegawa. 1974. MNL 48:35-37. (*Px8 Px9*)
23. Brink, R.A. 1933. J. Hered. 24:325-326. (*lg2*)
24. Brink, R.A. 1935. J. Hered. 26:249-251. (*pm1*)
25. Brink, R.A. and R.A. Nilan. 1952. Genetics 37:519-544. (*Mp*)
26. Brink, R.A. and P.H. Senn. 1931. J. Hered. 22:155-161. (*Rg1*)
27. Brown, W.L. and D.N. Duvick. 1958. MNL 32:120-121. (*wsp*)
28. Bryan, A.A. and J.E. Sass. 1941. J. Hered. 32:342-346. (*Kn1*)
29. Buchert, J.G. 1961. Proc. Nat. Acad. Sci. 47:1436-1440. (*Rf3*)
30. Burnham, C.R. 1936. J. Amer. Soc. Agron. 28:968-975. (*ga2*)
31. Burnham, C.R. 1947. MNL 21:36. (*bm4*)
32. Burnham, C.R. 1958. MNL 32:93. (*eg1*)
33. Burnham, C.R. 1961. MNL 35:87. (*tb1*)
34. Burnham, C.R. and R.A. Brink. 1932. J. Amer. Soc. Agron. 24:960-963. (*bm2*)
35. Chang, M.T. and M.G. Neuffer. 1987. J. Hered. 78:163-170. (*Mr*)
36. Chang, S-H. and J.L. Brewbaker. 1976. MNL 50:31-32. (*aph1*)
37. Chao, S.E. and J.G. Scandalios. 1969. Biochem. Genet. 3:537-547. (*Amy2*)
38. Chao, S.E. and J.G. Scandalios. 1971. Genetics 69:47-61. (*Amy1*)
39. Chourey, P.S. and C. Mouli. 1975. Genetics 77:s11. (*Ce1*)
40. Clark, F.J. 1940. Amer. J. Bot. 27:547-559. (*dv1*)
41. Clark, J.K. and W.F. Sheridan. 1985. J. Hered. 77:83-92. (*dek22 dek23*)
42. Coe, E.H., Jr. 1958. MNL 32:102. (*c2*)
43. Coe, E.H. 1983. Maydica 28:151-168. (NCS2 NCS3)
44. Coe, E.H. 1987. MNL 61:47. (*anl1 v30*)

45. Coe, E.H. and J.B. Beckett. 1987. MNL 61:46-47. (*baf1*)
46. Coe, E.H., et al. 1981. J. Hered. 72:318-320. (*whp1*)
47. Collins, G.N. 1909. U.S. Dept. Agr., Pl. Indus. Bur. Bull. 161:1-30. (*wx1*)
48. Collins, G.N. 1917. J. Agr. Res. 9:383-395. (*Tu1*)
49. Collins, G.N. 1917. Proc. Nat. Acad. Sci. 3:345-349. (*Tu1*)
50. Collins, G.N. and J.H. Kempton. 1920. J. Hered. 11:3-6. (*li1*)
51. Correns, C. 1901. Bibliotheca Botanica 53:1-161. (*su1 y1*)
52. Couture, R.M., et al. 1971. Phys. Pl. Path. 1:515-521. (*Bx1*)
53. Cox, E.L. and D.B. Dickinson. 1971. Biochem. Genet. 5:15-25. (*l12 w14 w15*)
54. de la Roche, I.A., et al. 1971. Crop Science 11:856-859. (*In1*)
55. Demerec, M. 1921. J. Hered. 12:406-407. (*zb1*)
56. Demerec, M. 1924. Cornell Univ. Agr. Exp. Sta. Memoir 84. (*v1 v3 v4 v5*)
57. Demerec, M. 1926. Amer. Nat. 60:172-176. (*d3 v8 w11*)
58. Demerec, M. 1926. J. Hered. 17:301-306. (*pb1 pb4*)
59. Dempsey, E.D. 1971. MNL 45:58. (K3L)
60. Dempsey, E.D. 1985. Pp. 311-316 in M. Freeling, ed., Plant Genetics, Alan R. Liss, Inc., N.Y. (*Ac2*)
61. Doerschug, E.B. 1973. Theor. Appl. Genet. 43:182-189. (*Dt4 Dt5*)
62. Dollinger, E.J. 1984. MNL 58:209-210. (*ora2*)
63. Dollinger, E.J. 1985. Crop Sci. 25:819-821. (*lty1 lty2 ora3 zpg1*)
64. Dollinger, E.J. 1987. MNL 61:103. (*sft1*)
65. Dooner, H.K. and J.L. Kermicle. 1971. Genetics 67:427-436. (*I*)
66. Dooner, H.K. and J.L. Kermicle. 1976. Genetics 93:309-322. (*Lc1*)
67. Doyle, G.G. 1978. MNL 52:77. (*agt1*)
68. Duvick, D.N. 1965. Adv. Genet. 13:1-56. (*Rf2*)
69. East, E.M. and H.K. Hayes. 1911. Conn. Agr. Exp. Sta. Bull. 167. (*C1 pr1 R1*)
70. Emerson, R.A. 1911. Nebr. Agr. Exp. Sta. Ann. Rept. 24:59-90. (*P1*)
71. Emerson, R.A. 1912. Amer. Breeders Assoc. Ann. Rept. 8:385-399. (*an1 d1 g1 lg1 w1*)
72. Emerson, R.A. 1912. Nebr. Agr. Exp. Sta. Ann. Rept. 25:81-88. (*lg1 w1*)
73. Emerson, R.A. 1912. Nebr. Agr. Exp. Sta. Ann. Rept. 25:89-105. (*g1 j1 v2*)
74. Emerson, R.A. 1918. Cornell Univ. Agr. Exp. Sta. Memoir 16. (*a1*)
75. Emerson, R.A. 1920. J. Hered. 11:65-76. (*ts1 ts2*)
76. Emerson, R.A. 1921. Amer. J. Bot. 8:411-424. (*Bh1 gs1*)
77. Emerson, R.A. 1921. Cornell Univ. Agr. Exp. Sta. Memoir 39. (*B1 Pl1*)
78. Emerson, R.A. 1921. J. Hered. 12:267-270. (*cr1*)
79. Emerson, R.A. 1932. Science 75:566. (*ms17*)
80. Emerson, R.A. 1932. Sixth Int. Congress Genet. Proc. 1:141-152. (*Ts5*)
81. Emerson, R.A. and S.H. Emerson. 1922. Genetics 7:203-236. (*an1*)
82. Emerson, R.A., et al. 1935. Cornell Univ. Agr. Exp. Sta. Memoir 180. (*j2*)
83. Everett, H.L. 1949. Proc. Nat. Acad. Sci. 35:628-634. (*cl1 Clm1*)
84. Eyster, W.H. 1926. Science 64:22. (*bm1 yg1*)
85. Eyster, W.H. 1929. Ztschr. ind. Abst. Vererbungsl. 49:105-130. (*ar1*)
86. Eyster, W.H. 1931. Genetics 16:574-590. (*vp1 vp2*)
87. Eyster, W.H. 1931. J. Hered. 22:99-102. (*ms2 ms3*)
88. Eyster, W.H. 1933. Amer. Nat. 67:75. (*oy1*)
89. Eyster, W.H. 1934. Bibliographia Genetica. 11:187-392. (*da1 l7 ms2 ms3 ms20 su2*)
90. Ferguson, J.E., et al. 1978. J. Hered. 69:377-380. (*se1*)
91. Fraser, A.C. 1924. J. Hered. 15:119-123. (*in1*)
92. Fraser, A.C. 1933. J. Hered. 24:41-46. (*si1*)
93. Freeling, M. and J.C. Woodman. 1978. MNL 52:9-10. (*Cdh1*)
94. Friedemann, P. and P.A. Peterson. 1982. Mol. Gen. Genet. 187:19-29. (*Uq*)
95. Galinat, W.C. 1969. Mass. Agr. Exp. Sta. Bull. 577:1-19. (*Sg1*)
96. Galinat, W.C. 1971. MNL 45:98-99. (*is1*)
97. Galinat, W.C. 1975. MNL 49:100-102. (*Ph1 Ri1*)
98. Galinat, W.C. and P.C. Mangelsdorf. 1957. MNL 31:67. (*Pn1*)
99. Galinat, W.C., et al. 1978. MNL 52:58. (*bu1*)
100. Gavazzi, G., et al. 1975. Theor. Appl. Genet. 46:339-346. (*pro1*)
101. Gavazzi, G., et al. 1986. Pp. 91-103 in G.M. Reddy and E.H. Coe, eds., Gene Structure and Function in Higher Plants, Oxford-IBH, New Delhi. (*Sn1*)
102. Gelinas, D.A., et al. 1966. Amer. J. Bot. 53:615. (*Clt1*)
103. Gernert, W.B. 1912. Amer. Nat. 46:616-622. (*ra1*)
104. Giesbrecht, J. 1965. J. Hered. 56:118, 130. (*zn2*)
105. Glover, D.V. 1968. MNL 42:151. (*ct2 sr3*)
106. Glover, D.V. 1970. Crop Science 10:611-612. (*rd2*)
107. Golubovskaya, I.N. 1979. Int. Rev. Cytol. 58:247-290. (*Mei1 ms43*)
108. Golubovskaya, I.N. and D.V. Sitnikova. 1980. Genetika 16:656-666. (*Mei1 ms28 ms43*)
109. Golubovskaya, I.N. and N.B. Khristolyubova. 1986. Pp. 723-738 in M. Freeling, ed., Plant Genetics, Alan R. Liss, N.Y. (*dsy2 pam2*)
110. Golubovskaya, I.N. and A.S. Mashnenkov. 1976. Genetika. 12:7-14. (*dsy1*)
111. Golubovskaya, I.N. and A.S. Mashnenkov. 1977. Genetika. 13:1910-1921. (*pam1*)
112. Golubovskaya, I.N., et al. 1975. Genetika. 11:11-17. (*afd1*)
113. Gonella, J.A. and P.A. Peterson. 1975. MNL 49:71-73. (*ga10*)
114. Gonella, J.A. and P.A. Peterson. 1977. Genetics 85:629-645. (*Fcu*)
115. Goodman, M.M. and C.W. Stuber. 1982. MNL 56:125. (*Gdh2*)
116. Goodman, M.M. and C.W. Stuber. 1983. Maydica 28:169-188. (*Acp1 Acp2*)
117. Goodman, M.M., et al. 1980. Genetics 96:697-700. (*Got2 Got3 Idh1 Idh2 Me1 Pgd1 Pgd2 Pgm1 Pgm2 Phi1*)
118. Gracen, V.E., et al. 1979. Proc. 34th Ann. Corn and Sorghum Res. Conf. pp. 76-91. (*Rf4*)
119. Grogan, C.O., et al. 1963. Crop Sci. 3:451. (*ats1*)
120. Hamill, D.E. 1968. MNL 42:36-37. (*Px1*)
121. Harberd, N., et al. 1987. MNL61:23-24. (*Mpl1*)
122. Harovitz, S. 1948. MNL 22:42. (*zn1*)
123. Harris, J.W. 1968. Genetics 60:186-187. (*E4*)
124. Hayes, H.K. 1932. J. Hered. 23:415-419. (*zb4*)
125. Hayes, H.K. and H.E. Brewbaker. 1928. Amer. Nat. 62:228-235. (*gl2 gl3 sl1*)
126. Hayes, H.K. and M.S. Chang. 1938. MNL 12:8. (*zb6*)
127. Hayes, H.K. and E.M. East. 1915. Conn. Agr. Exp. Sta. Bull. 188:1-31. (*fl1*)
128. Hofmeyr, J.D.J. 1930. Unpub. thesis, Cornell. (*ba1 ba2*)
129. Hoisington, D.A. 1983. MNL 57:159-160. (*nec4*)
130. Hoisington, D.A. 1985. MNL 60:50-51. (*Les4 Les5 Les6 Les7 Les8 Les9*)
131. Hoisington, D.A. 1987. MNL 61:48-49. (*Les10*)
132. Hooker, A.L. 1963. Crop Science 3:381-383. (*Ht1*)
133. Hooker, A.L. 1977. Crop Science 17:132-135. (*Ht2*)

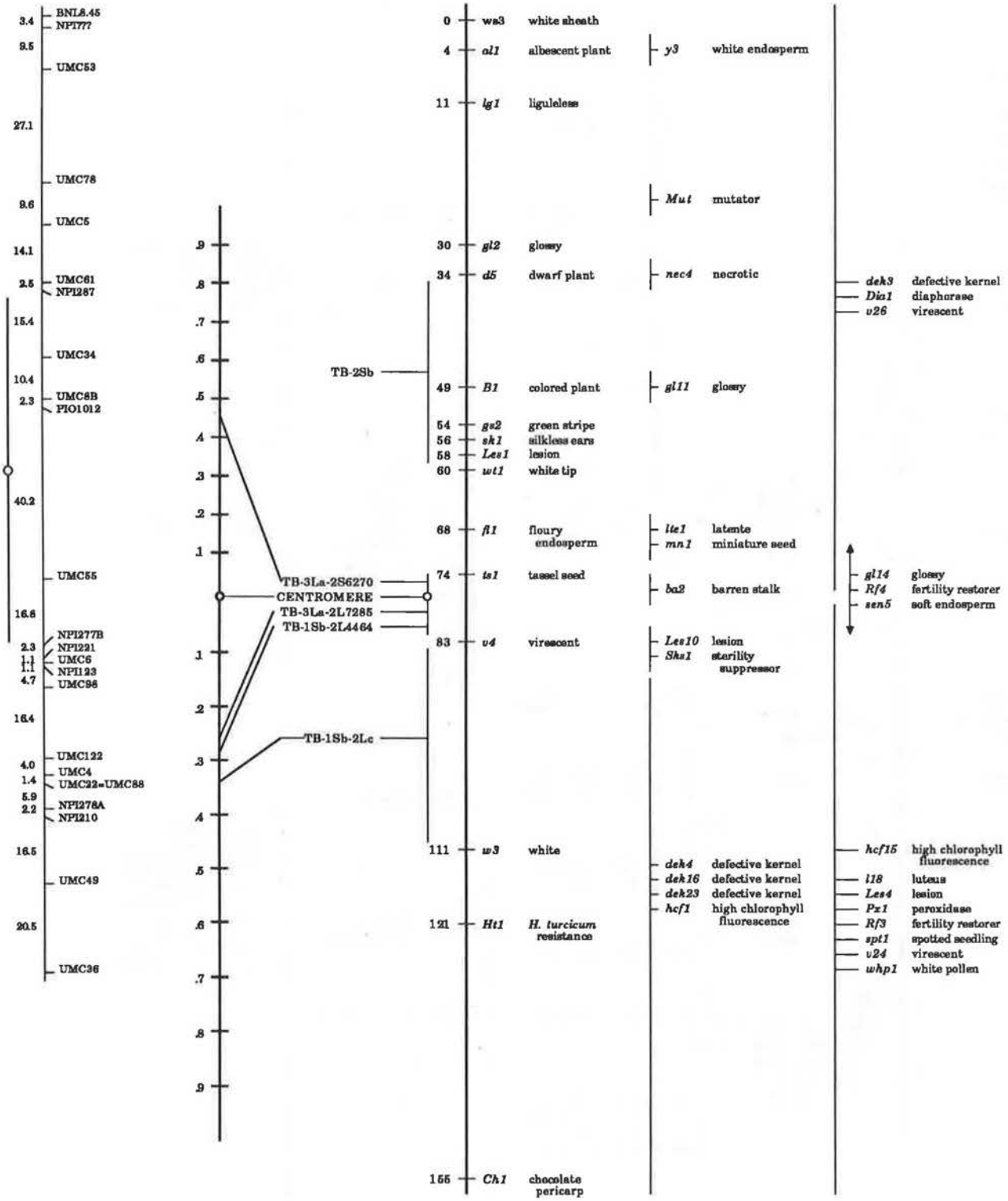
134. Hooker, A.L. 1981. MNL 55:87-88. (*Ht3*)
135. Huelsen, W.A. and M.C. Gillis. 1929. Ill. Agr. Exp. Sta. Bull. 320. (*pi1 pi2*)
136. Hutchison, C.B. 1921. J. Hered. 12:76-83. (*sh1*)
137. Hutchison, C.B. 1922. Cornell Univ. Agr. Exp. Sta. Memoir 60:1419-1473. (*na1*)
138. Jenkins, M.T. 1924. J. Hered. 15:467-472. (*ij1*)
139. Jenkins, M.T. 1926. Amer. Nat. 60:484-488. (*g2*)
140. Jenkins, M.T. 1927. Genetics 12:492-518. (*yg2*)
141. Jenkins, M.T. 1930. J. Hered. 21:79-80. (*rt1*)
142. Jenkins, M.T. 1932. J. Agr. Res. 44:495-502. (*a2*)
143. Jenkins, M.T. 1947. MNL 21:33. (*y8*)
144. Jenkins, M.T. and M.A. Bell. 1930. Genetics 15:253-282. (*l4*)
145. Jenkins, M.T. and F. Gerhardt. 1931. Iowa Agr. Exp. Sta. Res. Bull. 138:121-151. (*la1*)
146. Joachim, G. and C.R. Burnham. 1953. MNL 27:66. (*sr2*)
147. Jones, D.F. 1925. J. Hered. 16:339-341. (*sk1*)
148. Jones, D.F. 1951. Proc. Nat. Acad. Sci. 37:408-410. (*Rf1*)
149. Jones, D.F. 1954. Proc. IX Int. Genet. Cong. 1225-1237. (cms-S cms-T)
150. Jones, D.F. and P.C. Mangelsdorf. 1925. Anat. Rec. 31:351. (*Ga1*)
151. Josephson, L.M. 1955. Empire J. Exp. Agr. 23(89):1-10 (cms-S cms-T)
152. Kahler, A.L. 1983. J. Hered. 74:239-246. (*Acp4*)
153. Kang, M.S. 1981. MNL 55:26. (*btn1*)
154. Kempton, J.H. 1920. J. Hered. 11:111-115. (*br1*)
155. Kempton, J.H. 1920. J. Hered. 11:317-322. (*ad1*)
156. Kempton, J.H. 1921. Amer. Nat. 56:461-464. (*ws1 ws2*)
157. Kempton, J.H. 1921. U.S. Dept. Agr. Bull. 925:1-28. (*br1*)
158. Kempton, J.H. 1934. J. Hered. 25:29-32. (*bd1*)
159. Kermicle, J.L. 1969. Science 166:1422-1424. (*ig1*)
160. Kermicle, J.L. and J.D. Axtell. 1981. Maydica 26:185-197. (*Isr1*)
161. Khadzhinov, M.I. 1937. Bull. Appl. Bot. Gen. Pl. Breed. Ser. II. 7:247-258. (*Rs1 rs2*)
162. Kvakan, P. 1924. Cornell Univ. Agr. Exp. Sta. Memoir 83:1-22. (*Bn1 gl1*)
163. Kramer, H.H. 1957. MNL 31:120. (*rgd1*)
164. Kuc, J., et al. 1968. Phytochemistry 7:1435-1436. (*bm3*)
165. Kulkarni, C.G. 1927. Mich. Acad. Sci. Arts and Letters Papers. 6:253-273. (*Wc1*)
166. Lai, Y-K. and J.G. Scandalios. 1980. Devel. Genet. 1:311-324. (*Adr1*)
167. Lambert, R.J. and G.F. Sprague. 1987. MNL61:96. (*mg1*)
168. Langham, D.G. 1940. Genetics 25:88-107. (*pd1 tr1*)
169. Langham, D.G. 1940. MNL 14:21. (*bk2*)
170. Laughnan, J.R. 1949. Proc. Nat. Acad. Sci. 35:167-178. (*Alpha Beta*)
171. Leng, E.R. and L.F. Bauman. 1955. Agron. J. 47:189-191. (*Ms21 Sks1*)
172. Leng, E.R. and M.L. Vineyard. 1951. MNL 25:31-32. (*br2*)
173. Leto, K.J. 1982. Pp. 317-325 in W.F. Sheridan, ed., Maize for Biological Research, Plant Mol. Biol. Assoc., Charlottesville, VA. (*hcf6 hcf12 hcf13 hcf15 hcf19 hcf23 hcf26 hcf34 hcf38 hcf41 hcf46*)
174. Li, H.W. 1931. J. Hered. 22:14-16. (*bv1*)
175. Li, H.W. 1937. J. Hered. 24:279-281. (*na1*)
176. Lindstrom, E.W. 1917. Amer. Nat. 51:225-237. (*l1*)
177. Lindstrom, E.W. 1918. Cornell Univ. Agr. Exp. Sta. Memoir 13:1-68. (*l1 w1*)
178. Lindstrom, E.W. 1921. Genetics 6:91-110. (*f1*)
179. Lindstrom, E.W. 1924. Genetics 9:305-326. (*w2 w3*)
180. Lindstrom, E.W. 1925. J. Hered. 16:135-140. (*Tp1*)
181. Lindstrom, E.W. 1935. Iowa St. Coll. J. Sci. 9:451-459. (*a3 Og1*)
182. Lock, R.H. 1906. Roy. Bot. Gard. Annals 3:95-184. (*P1*)
183. Longley, A.E. 1932. J. Agr. Res. 54:835-862. (K10)
184. Lorenzoni, C., et al. 1974. MNL 48:19-20. (*cp1*)
185. Lowe, J. and O.E. Nelson, Jr. 1946. Genetics 31:525-533. (*mn1*)
186. Lysikov, V.N., et al. 1984. Sov. Genet. 20:72-80. (*Cg2*)
187. MacDonald, T. and J.L. Brewbaker. 1972. J. Hered. 63:11-14. (*Px2 Px3 Px4 Px5 Px6 Px7 Ta1*)
188. MacDonald, T. and J.L. Brewbaker. 1974. J. Hered. 65:37-42. (*E5-I E5-II E6 E7 E8 E9 E10*)
189. Mains, E.B. 1926. J Hered. 17:313-325. (*Rp1*)
190. Mains, E.B. 1931. J. Agr. Res. 43:419-430. (*Rp1*)
191. Mains, E.B. 1949. J. Hered. 40:21-24. (*sh2*)
192. Mangelsdorf, P.C. 1926. Conn. Agr. Exp. Sta. Bull. 279:509-614. (*bt1*)
193. Mangelsdorf, P.C. 1947. Genetics 32:448-458. (*du1*)
194. Mascia, P.N. 1978. Molec. Gen. Genet. 161:237-244. (*l13 oro1 oro2 Orom1*)
195. Mascia, P.N. and D.S. Robertson. 1980. J. Hered. 71:19-24. (*nec1*)
196. Mashnenkov, A.S. and M.I. Khadjinov. 1979. Proc. IX Eucarpia Corn and Sorghum Sect. pp. 447-450. (*Sup1*)
197. Matthews, D.L., et al. 1974. J. Agr. Sci. 82:433-435. (*te1*)
198. McCarty, D.P., et al. 1986. Proc. Nat. Acad. Sci. 83:9099-9103. (*Css1*)
199. McClintock, B. 1934. Z. Zellforsch. Mikrosk. Anat. 21:294-328. (NOR)
200. McClintock, B. 1944. Genetics 29:478-502. (*pyd1 wd1*)
201. McClintock, B. 1950. Proc. Nat. Acad. Sci. 36:344-355. (*Ac Ds*)
202. McClintock, B. 1956. Brookhaven Symp. Biol. 8:58-74. (*Spm*)
203. Melville, J.C. and J.G. Scandalios, J.G. 1972. Biochem. Genet. 7:15-31. (*Enp1*)
204. Micu, V. 1980. MNL 54:63-64. (*db1*)
205. Micu, V. 1981. Genetical Studies of Maize. Shtiintsa, Kishinev, Mold. SSR (*bs1 db1 tl1*)
206. Micu, V. and S.I. Mustyatsa. 1978. Genetika. 14:365-368. (*dep1*)
207. Miles, C.D. and D.J. Daniel. 1974. Pl. Phys. 53:589-595. (*hcf1 hcf2 hcf3*)
208. Miles, D. 1982. Pp. 75-107 in M. Edelman, et al., eds., Methods in Chloroplast Molecular Biology, Elsevier, Amsterdam. (*hcf4 hcf13 hcf18 hcf19 hcf21 hcf23 hcf26 hcf34 hcf41 hcf42 hcf44 hcf50*)
209. Miles, D., et al. 1986. Pp. 361-365 in K.E. Steinbeck, et al., eds., Molecular Biology of the Photosynthetic Apparatus, Cold Spring Harbor Laboratory. (*hcf5 hcf28 hcf31 hcf36 hcf47 hcf48 hcf101 hcf316 hcf323 hcf408*)
210. Miles, F.C. 1915. J. Genetics 4:193-214. (*gs1*)
211. Miranda, L.T. de. 1980. MNL 54:15-18. (*mal1 Mal2*)
212. Miranda, L.T. de. 1980. MNL 54:19. (*Asr1*)
213. Miranda, L.T. de. 1981. MNL 55:18-19. (*lte1*)
214. Miranda, L.T. de, et al. 1982. MNL 56:28-30. (*Lte2*)
215. Miranda, L.T. de, et al. 1982. MNL 56:30-32. (Mer Zer)
216. Miranda, L.T. de, et al. 1984. MNL 58:38-46. (Ger Flt Krn)
217. Misra, P.S., et al. 1972. Science 176:1425-1427. (*o7*)
218. Modena, S.A. 1983. MNL 57:38. (*tpm1*)
219. Modena, S.A. 1984. MNL 58:79-82. (*Lcs1 Lct1 lct2*)
220. Modena, S.A. 1984. MNL 58:211-212. (*Pdf1*)

221. Motto, M., et al. 1983. Theor. Appl. Genet. 64:41-46. (*w16 w17 wyg1*)
222. Muller, L.D., et al. 1971. Crop Sci. 11:413-415. (*bm3*)
223. Mumm, W.J. 1929. Anat. Rec. 44:279. (*h1*)
224. Nelson, O.E. 1976. MNL 50:114. (*fl3*)
225. Nelson, O.E. 1981. MNL 55:68. (*o9 o10 o11 o12 o13*)
226. Nelson, O.E. 1981. MNL 55:73. (*lp1*)
227. Nelson, O.E. and G.B. Clary. 1952. J. Hered. 43:205-210. (*dy1 lo2*)
228. Nelson, O.E. and A.J. Ohlrogge. 1957. Science 125:1200. (*ct1 rd1*)
229. Nelson, O.E. and S.N. Postlethwait. 1954. Amer. J. Bot. 41:739-748. (*Pt1*)
230. Nelson, O.E. and A.J. Ullstrup. 1964. J. Hered. 55:194-199. (*hm2*)
231. Nelson, O.E., et al. 1965. Science 150:1469-1470. (*fl2*)
232. Neuffer, M.G. 1973. MNL 47:150. (*nec3*)
233. Neuffer, M.G. and J.B. Beckett. 1987. MNL 61:50. (*gl19 gl21 grt1 gs3 ij2 l13 l16 l17 l18 l19 nec5 nec6 nec7 pg15 pg16 ppg1 py2 spc2 spc3 spt1 spt2 v24 v25 v26 v27 v28 v29 wgs1 wlu1 wlu2 wlu3 wlu4 wt2 zb7*)
234. Neuffer, M.G. and O.H. Calvert. 1975. J. Hered. 66:265-270. (*Les1 Les2*)
235. Neuffer, M.G. and M.T. Chang. 1986. MNL 60:55. (*orp1 orp2*)
236. Neuffer, M.G. and D. England. 1984. MNL 58:77-78. (*Clt1*)
237. Neuffer, M.G. and K.A. Sheridan. 1977. MNL 51:60. (*Bif1 Rgd2 Spc1 Ysk1 Zb8*)
238. Neuffer, M.G. and W.F. Sheridan. 1980. Genetics 95:929-944. (*dek1 dek2 dek3 dek4 dek5 dek6 dek7 dek8 dek9 dek10 dek11 dek12 dek13 dek14 dek15*)
239. Neuffer, M.G. and W.F. Sheridan. 1980. MNL 55:29-30. (*dek1 dek2 dek3 dek4 dek5 dek6 dek7 dek8 dek9 dek10 dek11 dek12 dek13 dek14 dek15*)
240. Neuffer, M.G., et al. 1968. The mutants of maize. Crop Sci. Soc. Amer., Madison, Wisc. (*cp2 ub1*)
241. Neuffer, M.G., et al. 1987. MNL 61:50-51. (*G6 Ms41 Msc1 Msc2 Tlr1 Vsr1 Wrk1 Zb8*)
242. Newton, K.J. and D. Schwartz. 1980. Genetics 95:425-442. (*Mdh1 Mdh2 Mdh3 Mdh4 Mdh5 mmm1*)
243. Nickerson, N.H. and E.E. Dale. 1955. Ann. Mo. Bot. Gard. 42:195-212. (*Ts6*)
244. Nuffer, M.G. 1954. MNL 28:63-64. (*bz2*)
245. Nuffer, M.G. 1955. Science 121:399-400. (*Dt2 Dt3*)
246. Ott, L. and J.G. Scandalios. 1978. Genetics 89:137-146. (*Amp1 Amp2 Amp3 Amp4*)
247. Paliy, A.F. and A.I. Rotar. 1979. Genetika 15:478-481. (*cfl2*)
248. Palmer, R.G. 1971. Chromosoma 35:233-246. (*am1*)
249. Pawar, S.E. and C. Mouli. 1973. MNL 47:17. (*Ce1*)
250. Perry, H.S. 1939. MNL 13:7. (*Lg3*)
251. Perry, H.S. and C.F. Sprague. 1936. J. Amer. Soc. Agron. 28:990-996. (*y3*)
252. Peterson, H. 1959. MNL 33:41. (*Tp2*)
253. Peterson, P.A. 1960. Genetics 45:115-133. (*En pg14*)
254. Pfund, J.H. and C.W. Crum. 1977. Agron. Abst. p. 66. (*Thc1*)
255. Phinney, B.O. 1956. Proc. Nat. Acad. Sci. 42:185-189. (*D8*)
256. Phipps, I.F. 1928. J. Hered. 19:399-404. (*ts4*)
257. Phipps, I.F. 1929. Cornell Univ. Agr. Exp. Sta. Memoir 125:1-63. (*al1 v12 v13 v16 v17 v18*)
258. Piovarci, A. 1982. MNL 56:157. (*bv2*)
259. Plewa, M. 1979. MNL 53:93-96. (*loc1*)
260. Pogna, N.E., et al. 1982. MNL 56:153. (*ys2*)
261. Postlethwait, S.N. and O.E. Nelson, Jr. 1957. Amer. J. Bot. 44:628-633. (*wi1*)
262. Pryor, A.J. 1974. Heredity 32:397-401. (*Gdh1*)
263. Pryor, A.J. 1976. MNL 50:15-16. (*Glu1*)
264. Pryor, T. and D. Schwartz. 1973. Genetics 75:75-92. (*Cx1*)
265. Randolph, L.F. 1928. Anat. Rec. 41:102. (B chr)
266. Rhoades, M.M. 1935. Amer. Nat. 69:74-75. (*Dt1*)
267. Rhoades, M.M. 1939. Genetics 24:62. (*ws3*)
268. Rhoades, M.M. 1948. MNL 22:9. (*ga7*)
269. Rhoades, M.M. 1951. Amer. Nat. 85:105-110. (*pg11 pg12*)
270. Rhoades, M.M. 1952. Amer. Nat. 86:105-106. (*bz1*)
271. Rhoades, M.M. 1956. MNL 30:38-42. (*am1 el1*)
272. Rhoades, M.M. and E. Dempsey. 1954. MNL 28:58. (*gl17*)
273. Rhoades, M.M. and E. Dempsey. 1982. MNL 56:21. (*Mrh*)
274. Rhoades, M.M. and E. Dempsey. 1982. MNL 56:22. (*Mut*)
275. Robertson, D.S. 1952. Proc. Nat. Acad. Sci. 38:580-583. (*vp5*)
276. Robertson, D.S. 1955. Genetics 40:745-760. (*vp8 vp9*)
277. Robertson, D.S. 1961. Genetics 46:649-662. (*y10*)
278. Robertson, D.S. 1967. MNL 41:94. (*o5*)
279. Robertson, D.S. 1973. MNL 47:82. (*l10*)
280. Robertson, D.S. 1974. MNL 48:70. (*yd2*)
281. Robertson, D.S. 1975. J. Hered. 66:127-130. (*y9*)
282. Robertson, D.S. 1978. Mutat. Res. 51:21-28. (*Mu*)
283. Robertson, D.S. 1981. MNL 55:115. (*l15*)
284. Robertson, D.S. 1984. MNL 58:18. (*brn1*)
285. Sachan, J.K. and K.R. Sarkar. 1978. MNL 52:119-120. (*rgo1*)
286. Salamini, F. 1980. Cold Spr. Harb. Symp. 45:467-476. (*Bg*)
287. Salamini, F., et al. 1983. Theor. Appl. Genet. 65:123-128. (*Mc1*)
288. Saxena, K.M.S. and A.L. Hooker. 1968. Proc. Nat. Acad. Sci. 61:1300-1305. (*Rp5*)
289. Scandalios, J.G., et al. 1972. Arch. Biochem. Biophys. 153:695-705. (*Cat2*)
290. Scandalios, J.G., et al. 1975. Biochem. Genet. 13:759-769. (*Got1*)
291. Scandalios, J.G., et al. 1980. Mol. Gen. Genet. 179:33-41. (*Cat3*)
292. Scandalios, J.G., et al. 1980. Proc. Nat. Acad. Sci. 77:5360-5364. (*Car1*)
293. Schiefelbein, J.W., et al. 1985. Proc. Nat. Acad. Sci. 82:4783-4787. (*dSpm*)
294. Schnable, P.S. and P.A. Peterson. 1986. Maydica 31:59-82. (*Cy*)
295. Schwartz, D. 1951. Genetics 36:676-696. (*Ms21 Sks1*)
296. Schwartz, D. 1951. MNL 25:30. (*ga8*)
297. Schwartz, D. 1960. Proc. Nat. Acad. Sci. 46:1210-1215. (*E1*)
298. Schwartz, D. 1964. Proc. Nat. Acad. Sci. 51:602-605. (*E3*)
299. Schwartz, D. 1965. Proc. XI Int. Genet. Cong. 2:131-135. (*E2*)
300. Schwartz, D. 1966. Proc. Nat. Acad. Sci. 56:1431-1436. (*Adh2*)
301. Schwartz, D. 1979. Mol. Gen. Genet. 174:233-240. (*mep1 Prot1*)
302. Schwartz, D. and T. Endo. 1966. Genetics 53:709-715. (*Adh1*)
303. Shaver, D.L. 1967. J. Hered. 58:270-273. (*gt1 pe1*)
304. Shaver, D. 1983. Proc. Annu. Corn Sorghum Res. Conf. 38:161-180. (*Lfy1*)
305. Shepherd, N.S., et al. 1982. Mol. Gen. Genet. 188:266-271. (*Cin*)
306. Sheridan, W.F., et al. 1984. MNL 58:98-99. (*dek16 dek17 dek18 dek19 dek20 dek21*)
307. Sheridan, W.F., et al. 1986. MNL 60:64. (*dek22 dek23 dek24 dek25 dek26 dek27 dek28 dek29 dek30*)
308. Shortess, D.K. and R.P. Amby. 1979. Maydica 24:215-221. (*pg13*)

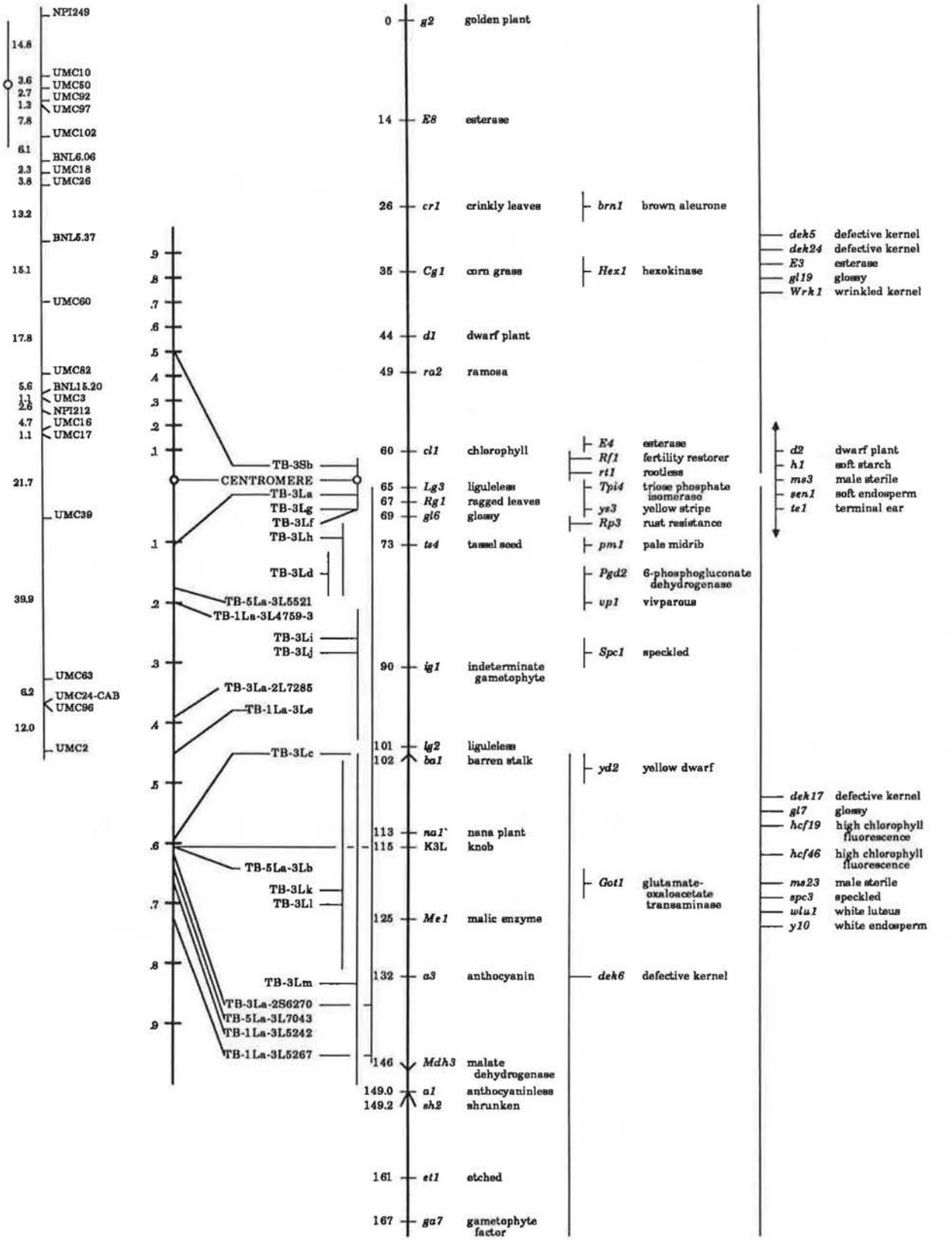
309. Shortess, D.K., et al. 1968. Genetics 58:227-235. (*lu1*)
310. Shumway, L.K. and L.F. Bauman. 1967. Genetics 55:33-38. (NCS1)
311. Singleton, W.R. 1946. J. Hered. 37:61-64. (*id1*)
312. Singleton, W.R. 1951. Amer. Nat. 85:81-96. (*Cg1*)
313. Singleton, W.R. 1959. MNL 33:3. (*br3*)
314. Singleton, W.R. and D.F. Jones. 1930. J. Hered. 21:266-268. (*ms1*)
315. Smith, D.R. and A.L. Hooker. 1973. Crop Science 13:330-331. (*rhm1*)
316. Soave, C., et al. 1978. Theor. Appl. Genet. 52:263-267. (Zp)
317. Soave, C., et al. 1981. Genetics 97:363-377. (Zp)
318. Sorrentino, J.J., et al. 1987. MNL 61:103. (*rDt*)
319. Sprague, G.F. 1932. U.S. Tech. Bull. 292:1-43. (*sy1*)
320. Sprague, G.F. 1936. J. Amer. Soc. Agron. 28:472-478. (*ps1*)
321. Sprague, G.F. 1938. MNL 12:2. (*gl11*)
322. Sprague, G.F. 1939. J. Hered. 30:143-145. (*Vg1*)
323. Sprague, G.F. 1955. MNL 29:6. (*gl14*)
324. Sprague, G.F. 1984. MNL 58:197. (*Dt6*)
325. Sprague, G.F. 1987. MNL 61:96. (*gl5 gl20*)
326. Sprague, G.F. 1987. MNL 61:96. (*y11, y12*)
327. Sprague, G.F., et al. 1965. MNL 39:164. (*wt1*)
328. Stadler, L.J. 1940. MNL 14:26. (*et1*)
329. Stadler, L.J. and M.H. Emmerling. 1956. Genetics 41:124-137. (*P S*)
330. Stadler, L.J. and M.G. Nuffer. 1953. Science 117:471-472. (*P*)
331. Stierwalt, T.R. and P.L. Crane. 1974. MNL 48:139. (*sen1 sen2 sen3 sen4 sen5 sen6*)
332. Stout, J.T. and R.L. Phillips. 1973. Proc. Nat. Acad. Sci. 70:3043-3047. (*alh1 clh1*)
333. Stroman, G.N. 1924. Genetics 9:493-512. (*zb2*)
334. Stroup, D. 1970. J. Hered. 61:139-141. (*cm1*)
335. Styles, E.D., et al. 1987. MNL 61:100. (*Ufo1*)
336. Suttle, A.D. . 1924. Unpub. thesis, Cornell. (*d2 d5 py1*)
337. Tavcar, A. 1932. Jugoslav. Akad. Znanosti i Umjetnosti Prestampo. 244:74-93. (*Hs1*)
338. Teas, H.J. and E.G. Anderson. 1951. Proc. Nat. Acad. Sci. 37:645-649. (*Bf1*)
339. Teas, H.J. and A.N. Teas. 1953. J.Hered. 44:156-158. (*bt2*)
340. Tsai, C. and O.E. Nelson. 1968. Genetics 61:813-821. (*sh4*)
341. Tulpule, S.H. 1954. Amer. J. Bot. 41:294-301. (*lw1 lw2 lw3 lw4*)
342. Ullstrup, A.J. 1965. Phytopath. 55:425-428. (*Rpp9*)
343. Ullstrup, A.J. and A.M. Brunson. 1947. J. Amer. Soc. Agron. 39:606-609. (*hm1*)
344. Ullstrup, A.J. and A.F. Troyer. 1968. Phytopath. 57:1252-1283. (*lls1*)
345. Vahrusheva, E.I. 1975. MNL 49:95-96. (*cto1*)
346. Vineyard, M.L. and R.P. Bear. 1952. MNL 26:5. (*ae1*)
347. Wendel, J.F., et al. 1985. MNL 59:87-88. (*Aco1 Aco2 Aco3 Aco4 Dia1 Dia2 Sad1 Tpi1 Tpi2 Tpi3*)
348. Wendel, J.F., et al. 1986. MNL 60:109. (*Adk1*)
349. Wendel, J.F., et al. 1986. Theor. Appl. Genet. 72:178-185. (*Hex1 Hex2 Tpi4*)
350. Wentz, J.B. 1926. J. Hered. 17:327-329. (*bt1*)
351. West, D.P. and M.C. Albertsen. 1985. MNL 59:87. (*ms22 ms23 ms24*)
352. Wilkinson, D.R. and A.L. Hooker. 1968. Phytopath. 58:605-608. (*Rp3 Rp4 Rp6*)
353. Wright, J.E. 1961. MNL 35:111. (*ys3*)


BNL5.62
28.7
UMC115
5.6
NPI193
11.1
UMC76
10.1
NPI234
1.1
UMC11
8.2
UMC13
5.8
UMC29
17.0
UMC8A
54.2
NPI214
33.6
UMC119
1.1
UMC58
3.4
UMC14
3.4
BNL5.59
32.0
UMC23
2.3
UMC33
2.3
NPI236
9.8
UMC37
1.2
UMC83
8.0
BNL8.10
11.2
UMC107
2.2
UMC75
6.4
UMC72B
2.6
UMC106
5.2
BNL7.25
8.7
UMC77
27.9
NPI238
2.3
BNL8.29
2.3
NPI241
7.0
UMC84
1.3
UMC86
17.7
BNL6.32
TB-1Sb-2Lc
TB-1Sb-2L4464
TB-1Sb
CENTROMERE
TB-1La
TB-1Lc
TB-1La-3L4759-3
TB-1La-4L4692
TB-1La-3Lc
TB-1La-3L5267
TB-1La-5S8041
TB-1La-3L5242
0 sr1 striate leaves
1 vp5 viviparous
19 zb4 zebra crossbands
23 ms17 male sterile
24 ts2 tassel seed
26 P1 pericarp color
27 deh1 defective kernel
34 nec2 necrotic
56 as1 asynaptic
64 hm1 H. carbonum susceptibility
81 br1 brachytic culms
85 Vg1 vestigial glumes
86 f1 fine stripe
104 an1 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
Les2 lesion
ms9 male sterile
ms14 male sterile
rs2 rough sheath
Amp2 aminopeptidase
br2 brachytic culms
Amp1 aminopeptidase
Mdh4 malate dehydrogenase
mmm1 modifier of mMDH
id1 indeterminate growth
v22 virescent
Pgm1 phosphoglucomutase
Kn1 knotted
lw1 lemon white
rd1 reduced
tb1 teosinte branched
zb7 zebra crossbands
Mpl1 miniplant
Gdh1 glutamic dehydrogenase
alh1 histone Ia
Dia2 diaphorase
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
pg15 pale green
v25 virescent
ys2 yellow stripe
gt1 grassy tillers
mi1 midget plant
ms12 male sterile
sen3 soft endosperm
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
Msc1 mosaic aleurone
pg16 pale green
py2 pygmy
spc2 speckled
Tlr1 tillered

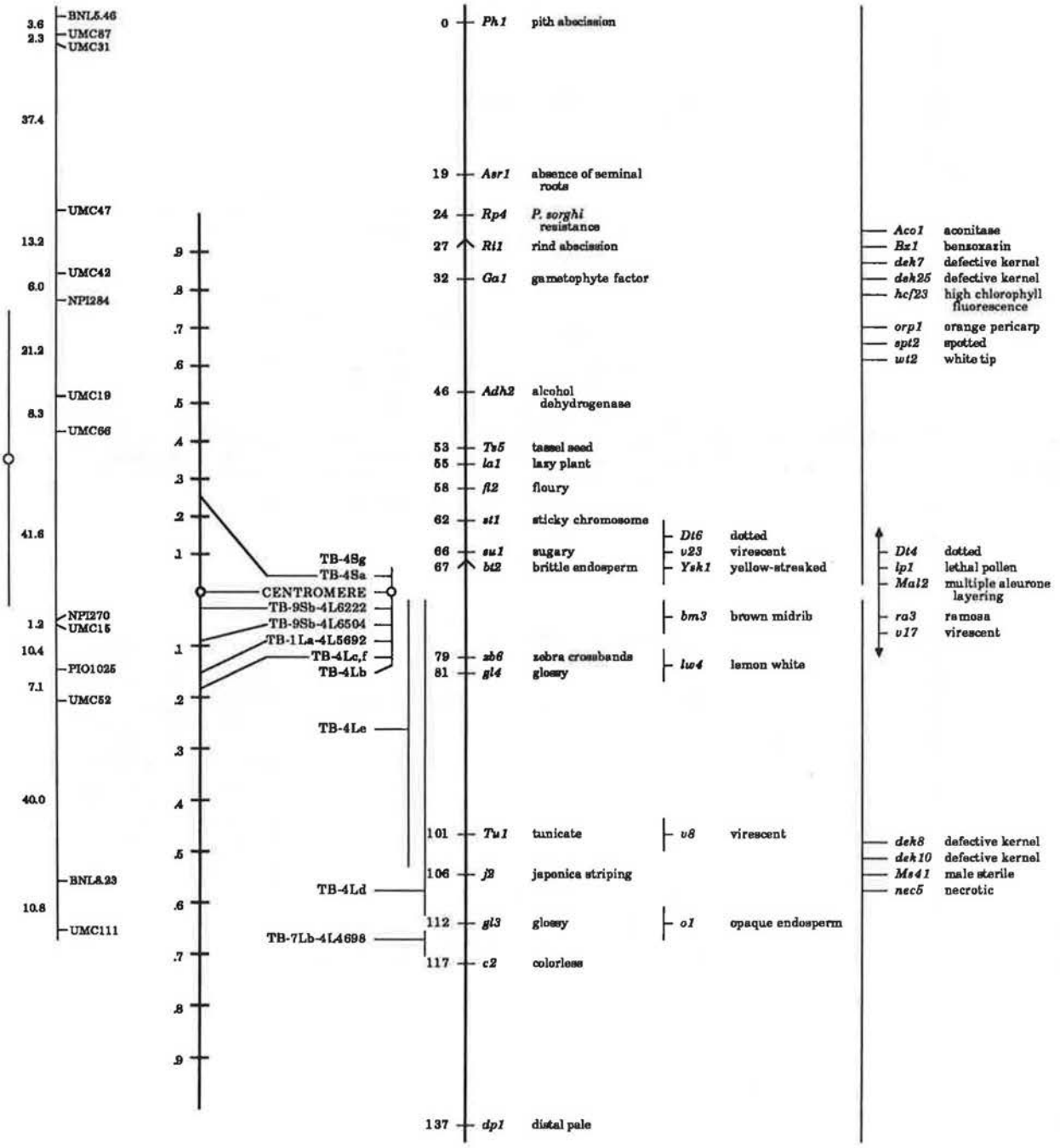
CHROMOSOME 2



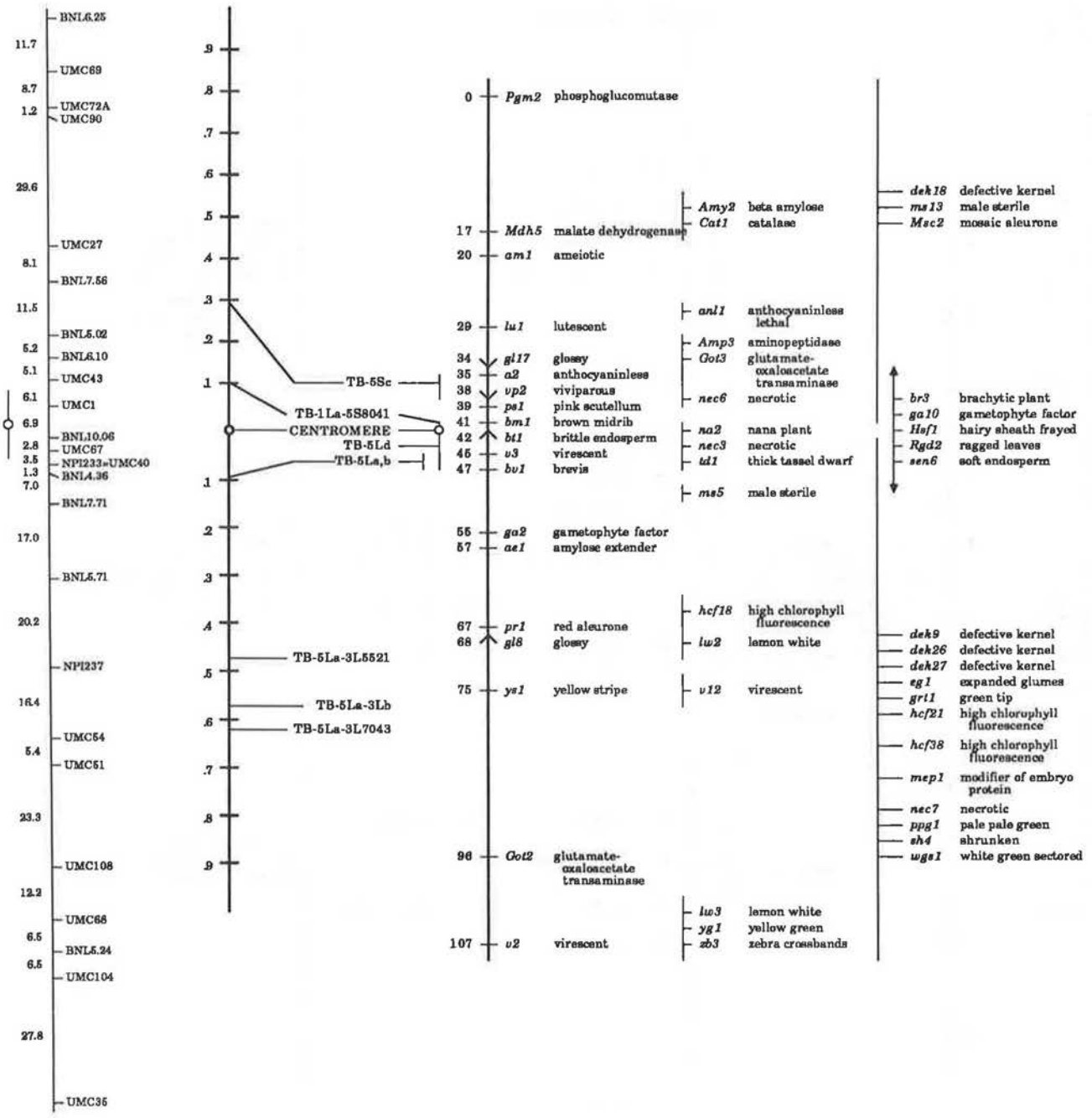
CHROMOSOME 3



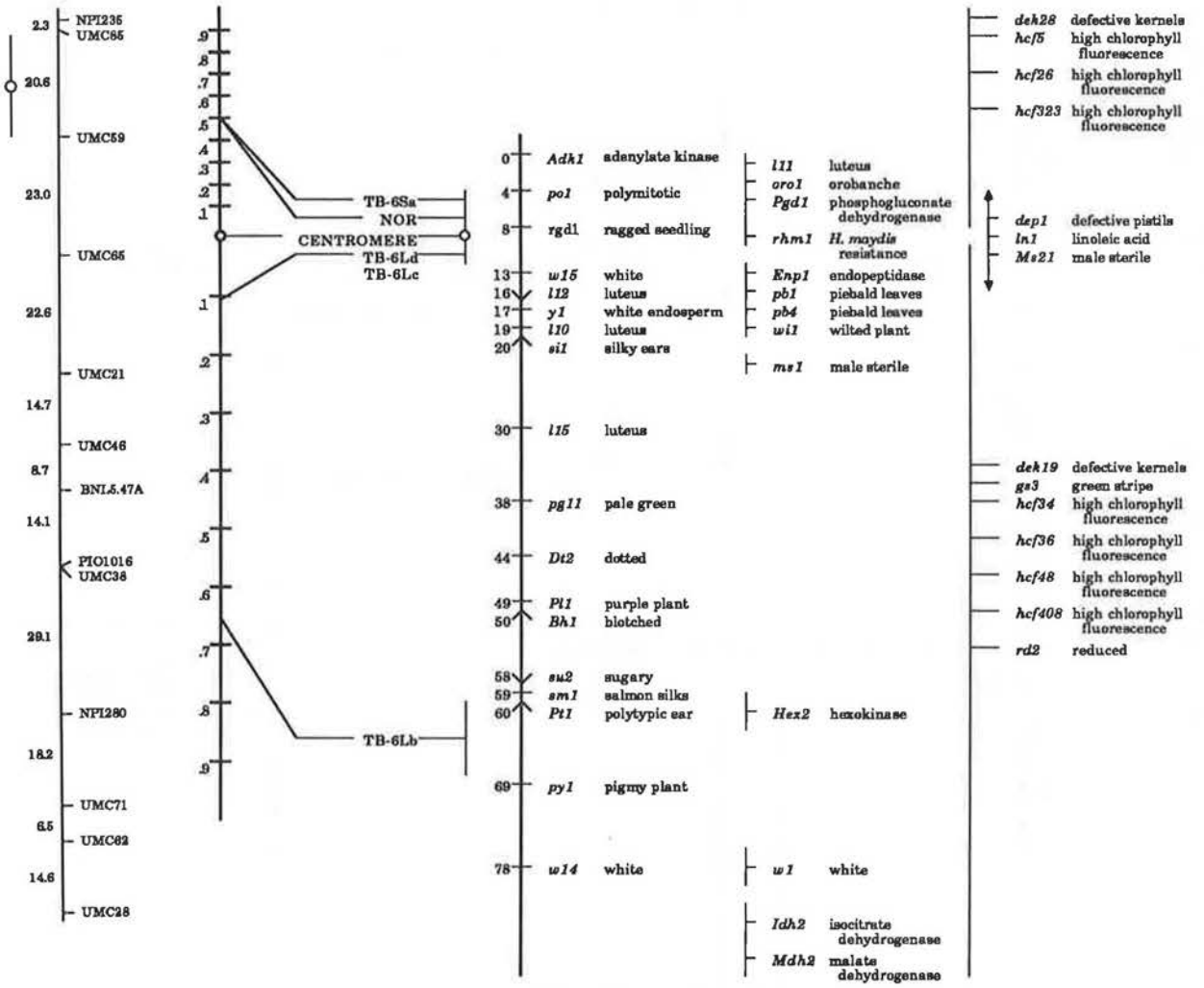
CHROMOSOME 4



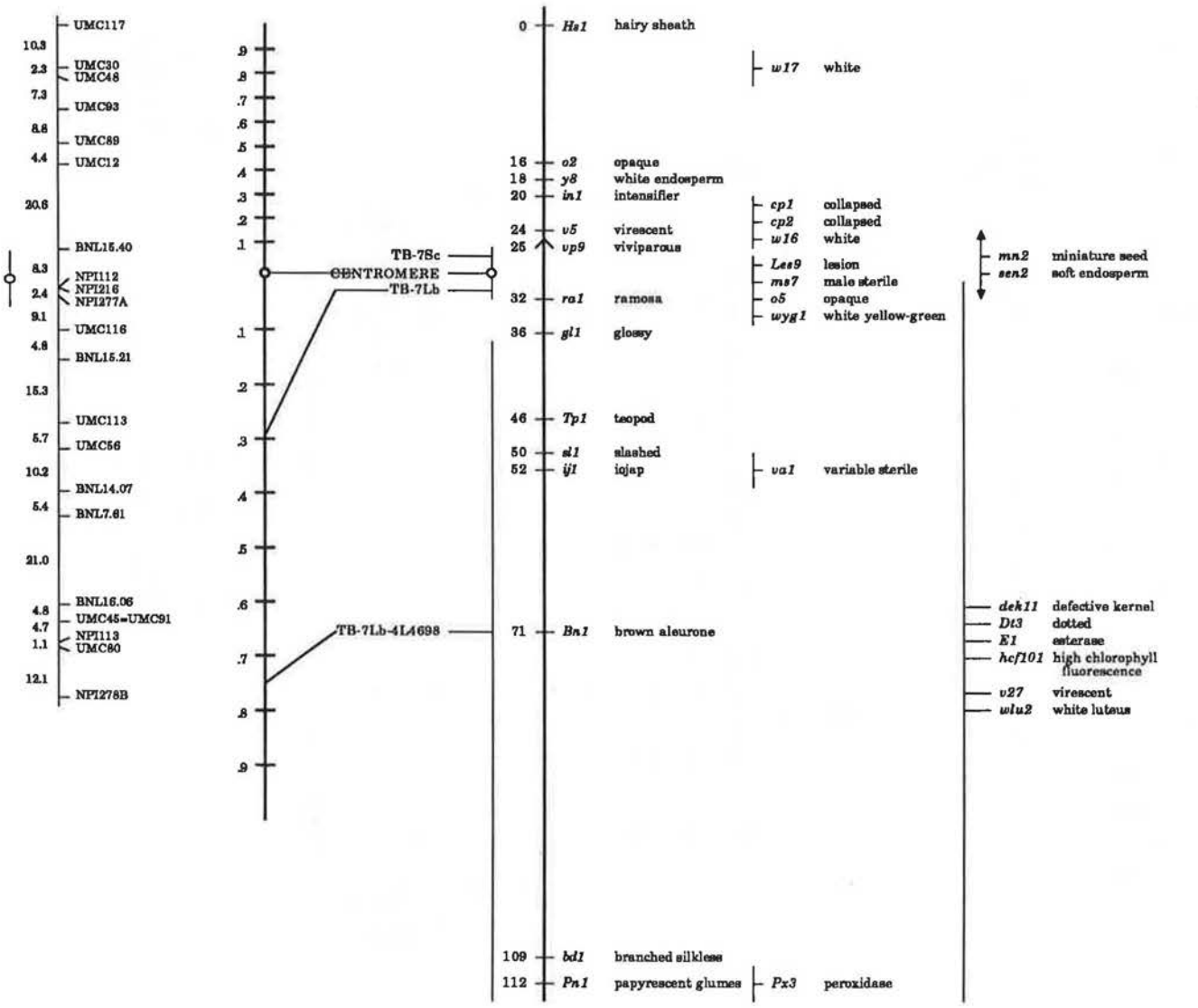
CHROMOSOME 5



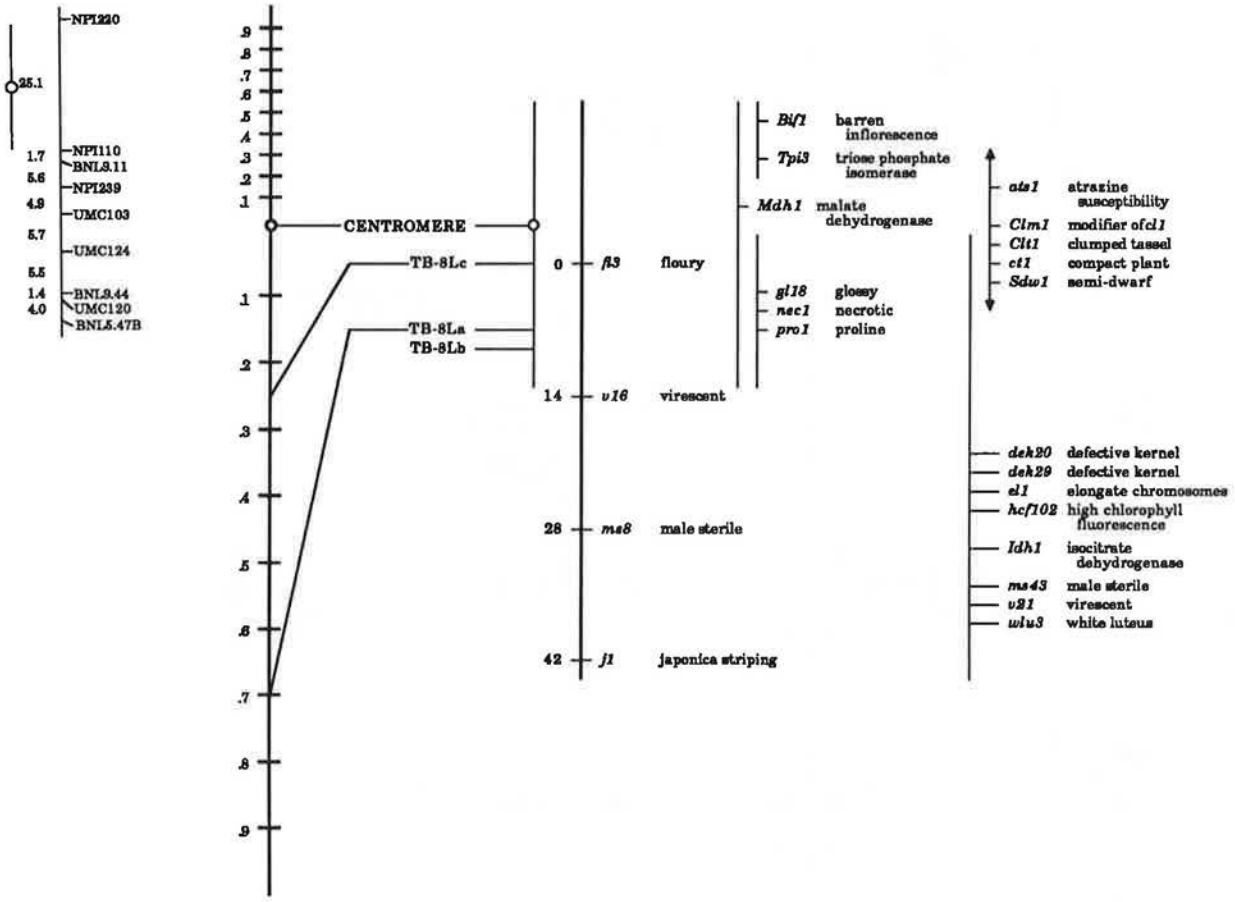
CHROMOSOME 6



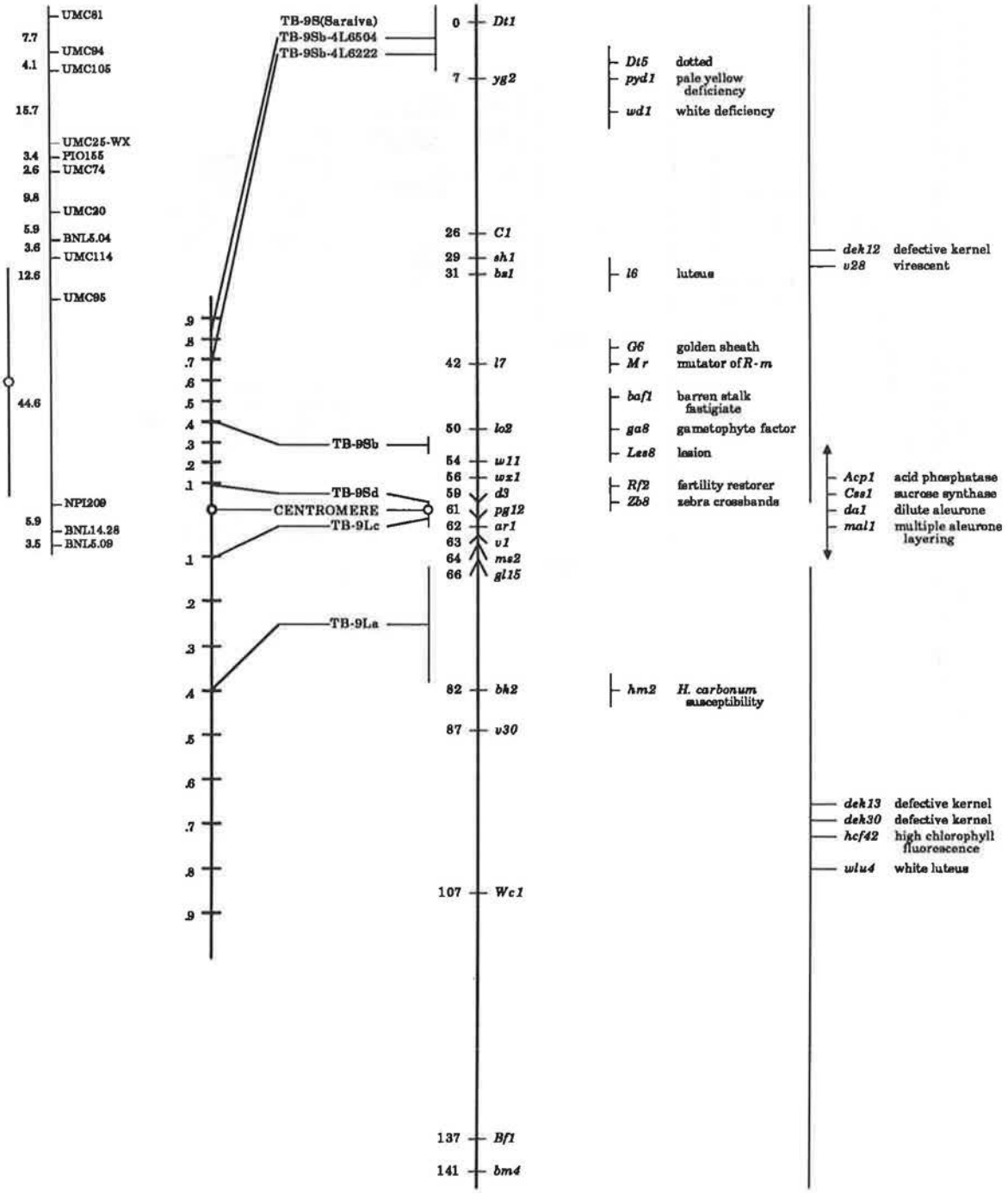
CHROMOSOME 7



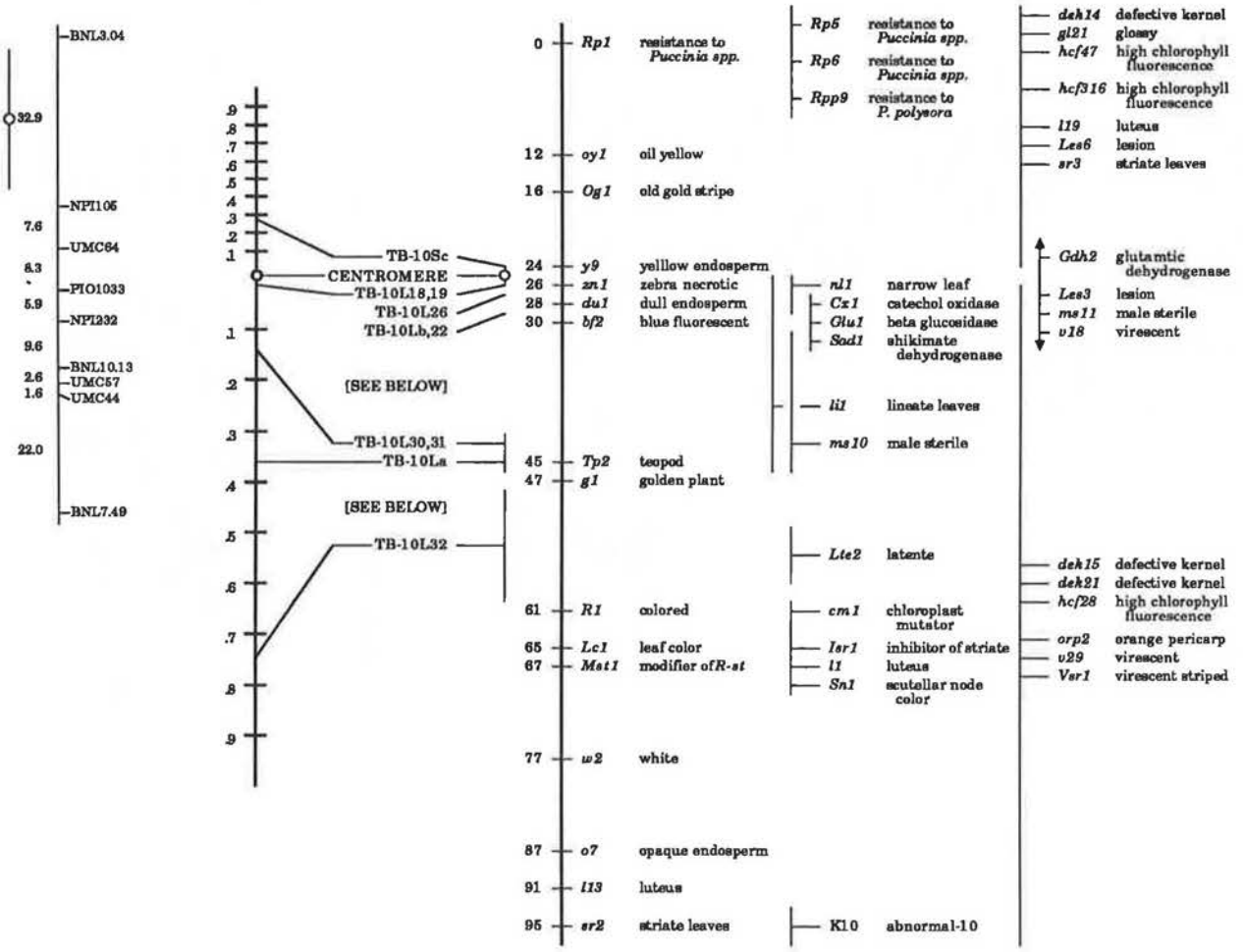
CHROMOSOME 8

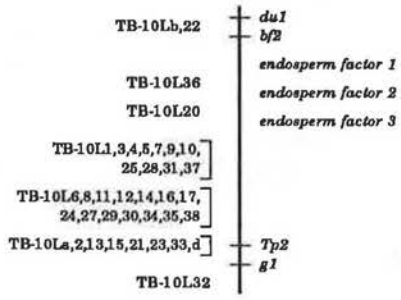


CHROMOSOME 9



CHROMOSOME 10

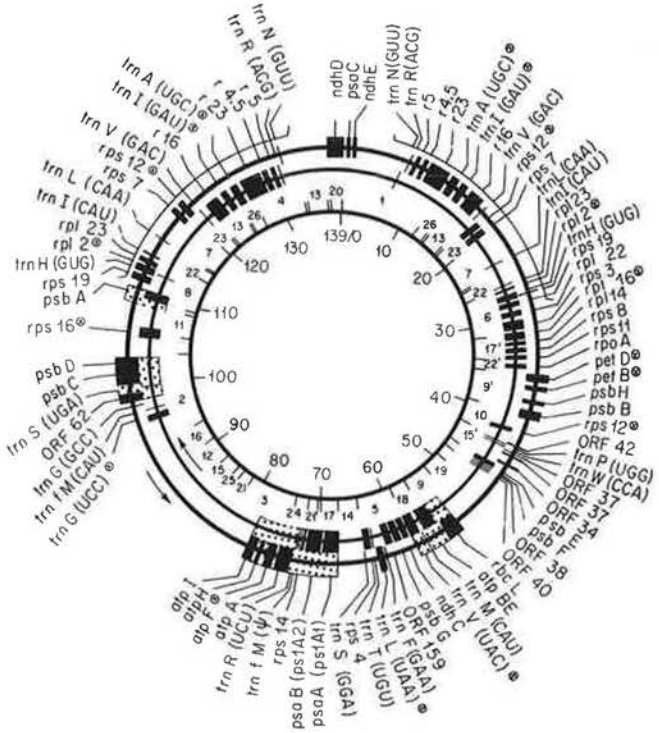




GENETIC MAP OF THE ZEA MAYS PLASTID CHROMOSOME

Steven Rodermel and Lawrence Bogorad, Department of Cellular and Developmental Biology, Harvard University, Cambridge, Massachusetts, USA 02138

The 139 kbp circular maize plastid chromosome has been physically mapped by Bedbrook and Bogorad (1976) and Larrinua et al. (1983). The genetic map of the chromosome is presented below, and includes genes sequenced through about December, 1987. The coordinate system of Larrinua et al. (1983) is shown on the innermost circle (inward pointing slashes); the "0/139" kbp coordinate begins with BamHI fragment 1 in the small single copy region of the chromosome (12 o'clock). The outward pointing slashes on the innermost circle delimit the 26 largest BamHI restriction fragments of the chromosome (designated by arabic numerals). The next two circles show the location of sequenced genes. Those genes on the middle circle are transcribed clockwise, while those on the outer circle are transcribed counterclockwise. The brackets outside the outer circle indicate the extent of the two large (22.5 kbp) inverted repeats (from coordinates 5.5 - 28.0, and 111.0 - 133.5) which divide the chromsome into two single copy (unique) DNA segments- a small single copy region (11 kbp) and a large single copy region (83 kbp). Stippled boxes represent those regions of the chromosome containing strongly photoregulated genes-- i.e., genes whose transcripts increase appreciably in abundance during light-induced plastid development (Bedbrook et al., 1978; Rodermel and Bogorad, 1985). Unidentified open reading frames are designated by "ORF" followed by the number of amino acids in the putative polypeptide product encoded by that particular gene. Those genes containing introns are followed by a star (*). The nomenclature for maize plastid genes follows the conventions of Hallick and Bottomley (1983). However, due to confusion in the literature arising largely from the discovery of new plastid-encoded components, a separate table has been provided containing a description of the gene product encoded by each maize locus.



ZEA MAYS PLASTID GENES AND GENE PRODUCTS

Gene Product	Gene	Reference
Ribosomal RNAs:		
4.5S rRNA	rr4.5	Edwards et al., 1981; Strittmatter and Kossel, 1984
5S rRNA	rr5	Strittmatter and Kossel, 1984
16S rRNA	rr16	Schwarz and Kossel, 1979, 1980;
23S rRNA	rr23	Edwards and Kossel, 1981
Transfer RNAs:		
Alanine	trnA (UGC)	Koch et al., 1981
Arginine	trnR (UCU)	Rodermel et al., 1987
	trnR (ACG)	Dormann-Przybyl et al., 1986
Asparagine	trnN (GUU)	Dormann-Przybyl et al., 1986
Glycine	trnG (GCC)	S. Rodermel and L. Bogorad, unpublished
	trnG (UCC)	S. Rodermel and L. Bogorad, unpublished
Histidine	trnH (GUG)	Schwarz et al., 1981; Larrinua and McLaughlin, 1987
Isoleucine	trnI (CAU)	Koch et al., 1981
	trnI (CAU)	I. Larrinua, unpublished
Leucine	trnL (CAA)	Steinmetz et al., 1983
	trnL (UAA)	Steinmetz et al., 1982
Methionine- initiator	trnfM (CAU)	S. Rodermel and L. Bogorad, unpublished
Methionine- initiator (pseudogene)	trnfM (Ψ)	Rodermel et al., 1987
Methionine- elongator	trnM (CAU)	Steinmetz et al., 1983
Phenylalanine	trnF (GAA)	Steinmetz et al., 1983
Proline	trnP (UGG)	J. Lukens and L. Bogorad, unpublished
Serine	trnS (GGA)	Steinmetz et al., 1983
	trnS (UGA)	Krebbers et al., 1984
Threonine	trnT (UGU)	Steinmetz et al., 1983
Tryptophan	trnW (CCA)	J. Lukens and L. Bogorad, unpublished
Valine	trnV (UAC)	Krebbers et al., 1984
	trnV (GAC)	Schwarz et al., 1981
Photosystem I Components:		
P700 chlorophyll a apoprotein A1	psaA (psiA1)	Fish et al., 1985; Fish and Bogorad, 1986
P700 chlorophyll a apoprotein A2	psaB (psiA2)	Fish et al., 1985; Fish and Bogorad, 1986
Apoprotein of iron-sulfur centers A and B	psaC	R. Schantz and L. Bogorad, unpublished
Photosystem II Components:		
32 kd herbicide-binding polypeptide ("D-1" protein)	psbA	L. McIntosh and L. Bogorad, unpublished
51 kd P680 chlorophyll a apoprotein	psbB	Rock et al., 1987
44 kd chlorophyll a apoprotein	psbC	E.T. Krebbers, K. Muskavitch, H. Roy, D. Russell, and L. Bogorad, unpublished
"D-2" protein	psbD	E.T. Krebbers, K. Muskavitch, H. Roy, D. Russell, and L. Bogorad, unpublished
Apocytochrome b-559, 9 kd subunit	psbE	J. Lukens and L. Bogorad, unpublished
Apocytochrome b-559, 4 kd subunit	psbF	J. Lukens and L. Bogorad, unpublished
24 kd membrane-associated protein	psbG	Steinmetz et al., 1986
10 kd phosphoprotein	psbH	Rock et al., 1987
Cytochrome b6/f Components:		
Apocytochrome b6	petB	Rock et al., 1987
Subunit IV	petD	Rock et al., 1987
ATP Synthetase Components:		
CF_1: alpha subunit	atpA	Rodermel and Bogorad, 1987
beta and epsilon subunits	atpBE (fused gene)	Krebbers et al., 1982
CF_0: subunit I	atpF	S. Rodermel and L. Bogorad, unpublished
subunit III (proteolipid)	atpH	Rodermel and Bogorad, 1987
subunit IV	atpI	S. Rodermel and L. Bogorad, unpublished

70S **Ribosomal Proteins**:

Proteins homologous to E. coli 30S ribosomal subunit proteins:

S3	rps3	McLaughlin and Larrinua, 1987c
S4	rps4	Subramanian et al., 1983
S7	rps7	A. Subramanian, unpublished
S8	rps8	U. Markmann-Mulisch and A. Subramanian, submitted
S11	rps11	A. Subramanian, unpublished
S12	rps12	A. Subramanian, unpublished
S14	rps14	Rodermel et al., 1987
S16	rps16	A. Subramanian, unpublished
S19	rps19	McLaughlin and Larrinua, 1987a

Proteins homologous to E. coli 50S ribosomal subunit proteins:

L2	rpl2	Larrinua and McLaughlin, 1987
L14	rpl14	U. Markmann-Mulisch and A. Subramanian, submitted
L16	rpl16	Gold et al., 1987; McLaughlin and Larrinua, 1987d
L22	rpl22	McLaughlin and Larrinua, 1987b
L23	rpl23	Larrinua and McLaughlin, 1987

Other stromal **proteins**:

Large subunit of ribulose bisphosphate carboxylase	rbcL	McIntosh et al., 1980; Poulsen, 1981
Protein homologous to alpha subunit of E. coli RNA polymerase	rpoA	D. Russell, D. Zaitlin, and L. Bogorad, unpublished

Other **membrane proteins**:

Proteins homologous to human and bovine mitochondrial NADH dehydrogenase complex proteins:

ND3	ndhC	A. Steinmetz and L. Bogorad, unpublished
ND4	ndhD	R. Schantz and L. Bogorad, unpublished
ND4L	ndhE	R. Schantz and L. Bogorad, unpublished

REFERENCES

Bedbrook, J.R. and L. Bogorad. 1976. Endonuclease recognition sites mapped on Zea mays chloroplast DNA. Proc. Natl. Acad. Sci. USA 73: 4309-4313.

Bedbrook, J.R., G. Link, D.M. Coen, L. Bogorad and A. Rich. 1978. Maize plastid gene expressed during photoregulated development. Proc. Natl. Acad. Sci. USA 75: 3060-3064.

Dormann-Przybyl, D., G. Strittmatter and H. Kossel. 1986. The region distal to the rRNA operon from chloroplasts of maize contains genes coding for $tRNA^{Arg}$ (ACG) and $tRNA^{Asn}$ (GUU). Plant Mol. Biol. 7: 419-431.

Edwards, K., J. Bedbrook, T. Dyer and H. Kossel. 1981. 4.5S rRNA from Zea mays chloroplasts shows structural homology with the 3' end of procaryotic 23S rRNA. Biochem. International 2: 533-538.

Edwards, K. and H. Kossel. 1981. The rRNA operon from Zea mays chloroplasts: nucleotide sequence of 23S rDNA and its homology with E. coli 23S rDNA. Nucleic Acids Res. 9: 2853-2869.

Fish, L.E. and L. Bogorad. 1986. Identification and analysis of the maize P700 chlorophyll a apoproteins PSI-A1 and PSI-A2 by high pressure liquid chromatography analysis and partial sequence determination. J. Biol. Chem. 261: 8134-8139.

Fish, L.E., U. Kuck and L. Bogorad. 1985. Two partially homologous adjacent light-inducible maize chloroplast genes encoding polypeptides of the P700 chlorophyll a-protein complex of photosystem I. J. Biol. Chem. 260: 1413-1421.

Gold, B. N. Carrillo, K.K. Tewari and L. Bogorad. 1987. Nucleotide sequence of a preferred maize chloroplast genome template for in vitro DNA synthesis. Proc. Natl. Acad. Sci. USA. 84: 194-198.

Hallick, R.B. and W. Bottomley. 1983. Proposals for the naming of chloroplast genes. Plant Mol. Biol. Reporter 1: 38-43.

Koch, W., K. Edwards and H. Kossel. 1981. Sequencing of the 16S-23S spacer in a ribosomal RNA operon of Zea mays chloroplast DNA reveals two split tRNA genes. Cell 25: 203-213.

Krebbers, E.T., I.M. Larrinua, L. McIntosh and L. Bogorad. 1982. The maize chloroplast genes for the beta and epsilon subunits of the photosynthetic coupling factor CF_1 are fused. Nucleic Acids Res. 10: 4985-5002.

Krebbers, E., A. Steinmetz and L. Bogorad. 1984. DNA sequences for the Zea mays tRNA genes tV-UAC and tS-UGA: tV-UAC contains a large intron. Plant Mol. Biol. 3: 13-20.

Larrinua, I.M., K.M.T. Muskavitch, E.J. Gubbins and L. Bogorad. 1983. A detailed restriction endonuclease site map of the Zea mays plastid genome. Plant Mol. Biol. 2: 129-140.

Larrinua, I.M. and W.E. McLaughlin. 1987. A gene cluster in the Z. mays plastid genome is homologous to part of the S10 operon of E. coli. In **Progress in Photosynthesis Research**. Vol. IV (ed. J. Biggens); Martinus Nijhoff Publishers, Dordrecht, the Netherlands.

Markmann-Mulisch, U. and A.R. Subramanian. Nucleotide sequence and linkage map position of the genes for ribosomal proteins L14 and S8 in the maize chloroplast genome. Submitted.

McIntosh, L., C. Poulsen and L. Bogorad. 1980. Chloroplast gene sequence for the large subunit of ribulose bisphosphate carboxylase of maize. Nature (London) 288: 556-560.

McLaughlin, W.E. and I.M. Larrinua. 1987a. The sequence of the maize rps19 locus and of the inverted repeat/unique region junctions. Nucleic Acids Res. 15: 3932.

McLaughlin, W.E. and I.M. Larrinua. 1987b. The sequence of the maize plastid encoded rpl22 locus. Nucleic Acids Res. 15: 4356.

McLaughlin, W.E. and I.M. Larrinua. 1987c. The sequence of the maize plastid encoded rps3 locus. Nucleic Acids Res. 15: 4689.

McLaughlin, W.E. and I.M. Larrinua. 1987d. The sequence of the first exon and part of the intron of the maize plastid encoded rpl16 locus. Nucleic Acids Res. 15: 5896.

Poulsen, C. 1981. Comments on the structure and function of the large subunit of the enzyme ribulose bisphosphate carboxylase-oxygenase. Carlsberg Res. Commun. 46: 259-278.

Rock, C.D., A. Barkan and W.C. Taylor. 1987. The maize plastid psbB - psbF - petB - petD gene cluster: spliced and unspliced petB and petD RNAs encode alternative products. Curr Genet. 12: 69-77.

Rodermel, S.R. and L. Bogorad. 1985. Maize plastid photogenes: mapping and photoregulation of transcript levels during light-induced development. J. Cell Biol. 100: 463-476.

Rodermel, S.R. and L. Bogorad. 1987. Molecular evolution and nucleotide sequences of the maize plastid genes for the alpha subunit of CF_1 (atpA) and the proteolipid subunit of CF_0 (atpH). Genetics 116: 127-139.

Rodermel, S., P. Orlin and L. Bogorad. 1987. The transcription termination region between two convergently-transcribed photoregulated operons in the maize plastid chromosome contains rps14, trnR (UCU) and a putative trnfM pseudogene. Nucleic Acids Res. 15: 5493.

Schwarz, Zs., S.O. Jolly, A.A. Steinmetz and L. Bogorad. 1981. Overlapping divergent genes in the maize chloroplast chromosome and in vitro transcription of the gene for $tRNA^{His}$. Proc. Natl. Acad. Sci. USA 78: 3423-3427.

Schwarz, Zs. and H. Kossel. 1979. Sequencing of the 3'-terminal region of a 16S rRNA gene from Zea mays chloroplast reveals homology with E. coli 16S rRNA. Nature (London) 279: 520-522.

Schwarz, Zs. and H. Kossel. 1980. The primary structure of 16S rDNA from Zea mays chloroplast is homologous to E. coli 16S rRNA. Nature (London) 283: 739-742.

Schwarz, Zs., H. Kossel, E. Schwarz and L. Bogorad. 1981. A gene coding for $tRNA^{Val}$ is located near 5' terminus of 16S rRNA gene in Zea mays chloroplast genome. Proc. Natl. Acad. Sci. USA 78: 4748-4752.

Steinmetz, A.A., M. Castroviejo, R.T. Sayre and L. Bogorad. 1986. Protein PSII-G. An additional component of photosystem II identified through its plastid gene in maize. J. Biol. Chem. 261: 2485-2488.

Steinmetz, A., E.J. Gubbins and L. Bogorad. 1982. The anticodon of the maize chloroplast gene for $tRNA^{Leu}$ (UAA) is split by a large intron. Nucleic Acids Res. 10: 3027-3037.

Steinmetz, A.A., E.T. Krebbers, Zs. Schwarz, E.J. Gubbins and L. Bogorad. 1983. Nucleotide sequences of five maize chloroplast transfer RNA genes and their flanking regions. J. Biol. Chem. 258: 5503-5511.

Strittmatter, G. and H. Kossel. 1984. Cotranscription and processing of 23S, 4.5S and 5S rRNA in chloroplasts from Zea mays. Nucleic Acids Res. 12: 7633-7647.

Subramanian, A.R., A. Steinmetz and L. Bogorad. 1983. Maize chloroplast DNA encodes a protein sequence homologous to the bacterial ribosome assembly protein S4. Nucleic Acids Res. 11: 5277-5286.

Abbott, Albert G., 338 Long Hall, Clemson University, Clemson, SC, 29634-1903
Agrawal, B. D., G. B. Pant Univ Agr Tech, Pantnagar 263145, India
Agrema, Hesham A., 42 Mumphis Street, Camp Caesar, Alexandria, Egypt
Ainsworth, Thomas C., Ainsworth Seed Company, Mason City, IL, 62664, 217-482-3281
Aken'Ova, M. E., Department of Agronomy, University of Ibadan, Ibadan, Nigeria
Albertsen, M. C., Pioneer Hi-Bred Internatl, P. O. Box 38, Johnston, IA, 50131, 515-270-3648
Albrecht, Bruno, CIBA-GEIGY S.p.A., Via per Soresina, Casalmorano, CR 26020, Italy
Alexander, D. E., Dept Agron, S-110 Turner Hall, Univ Illinois, 1102 S. Goodwin, Urbana, IL, 61801, 217-333-4254
Ali, S. Mahboob, Genetics & Plant Breed, College of Agriculture, Rajendranagar, Hyderabad-500 030, India
Alleman, M., Department of Genetics, University of Wisconsin, Madison, WI, 53706, 608-262-3286
Allen, Jim, Laboratory of Genetics, University of Wisconsin, Madison, WI, 53706, 608-262-3286
Altschuler, Marsha I., Dept. of Biology, Washington University, St. Louis, MO, 63130
Alvarez, Angel, Departamento de Genetica, Estacion Exp de Aula Dei, Apartado 202, 50080 Zaragoza, Spain
Alvey, David, FFR Cooperative, 4112 East State Road 225, W. Lafayette, IN, 47906, 317-567-2115
Amano, Etsuo, Inst of Radiation Breeding, PO Box 3, Ohmiya-machi, Naka-gun, Ibaraki-Ken 319-22, Japan
Anderson, E. L., Bldg 007, Rm 251, BARC-West, Beltsville, MD, 20705, 301-344-3148
Anderson, Joe, Room 8 Curtiss Hall, Department of Genetics, Iowa State University, Ames, IA, 50011, 515-294-0339
Andrew, R. H., Department of Agronomy, University of Wisconsin, Madison, WI, 53706, 608-262-2587
Arikoglu, Osman, Sakarya Zirai Arastirma Inst, P.K. 25, Adapazari, Turkey
Armstrong, Charles L., Agrigenetics Corporation, Advanced Research Division, 5649 E. Buckeye Road, Madison, WI, 53716, 608-221-5000
Arntzen, C. J., MSU-DOE Plant Res Lab, Michigan State Univ, East Lansing, MI, 48824, 517-353-2270
Aromose, O. S. A., Dept Biol Sci, Univ of Benin, Benin City, Nigeria
Ashman, R. B., Dept of Botany, Purdue University, W. Lafayette, IN, 47907, 317 494-4634
Athma, Prasanna, Dept Plant Biol, 289 Morrill, University of Illinois, 505 S. Goodwin, Urbana, IL, 61801, 217-333-2919
Auger, Donald L., Biology Department, Box 8238, University Station, University of North Dakota, Grand Forks, ND, 58202, 701-777-3674
Avila L., Gonzalo, Centro Eco-Genetico De, Pairumani, Casilla 128, Cochabamba, Bolivia
Axtell, John D., Department of Agronomy, Purdue University, W. Lafayette, IN, 47907, 317-494-8056
Ayala-Osuna, Juan, Univ Ext Paul Julio Mesq F, Fac Med Vet Agron, 14870 Jaboticabal SP, Brazil, 0163-220260
Aycock, Harold S., Dairyland Research Internatl, Route #1, Box 51, Clinton, WI, 53525, 608-676-2237
Ayers, J. E., Dept Plant Pathology, 211 Buckhout Lab, Pennsylvania State Univ, University Park, PA, 16802, 814-865-7776
Badenhorst, Coerie, Saffola, Box 36, Boskop 2528, South Africa
Baenziger, P. Stephen, Department of Agronomy, University of Nebraska, Lincoln, NE, 68583-0915
Bahadur, Khan, NWFP Maize & Millets Res, Nowshera Cantt, Pakistan
Baier, John W., 1255 Fifield Hall, University of Florida, Gainesville, FL, 32611
Bailey, Zeno E., Botany Dept, Eastern Illinois Univ, Charleston, IL, 61920, 267-581-3525
Baker, Barbara, USDA, Plant Gene Expression Center, 800 Buchanan Street, Albany, CA, 94710, 415-559-5912
Baker, Raymond, Pioneer Hybrid Seed Co, Johnston, IA, 50131, 515-278-2108
Baldi, G., Centro di Ricerche Sul Riso, C. P. 99, 27036 Mortara (PV), Italy
Balint, Andor, Agrar Egyetem, Novenynemesitestani, Tanszek, Godollo H-2103, Hungary
Banchero, Carlos, Santa Ursula, SA, Paraguay 577.P.1, 1057 Buenos Aires, Argentina, 311-0446-0510-4803-4846
Banerjee, U. C., Dept Biol, Box 19692, North Carolina Central Univ, Durham, NC, 27707, 919-683-6170
Bansal, R. K., Crop Research Substation, D.S.I.R., Cronin Road/R.D.1, Pukekohe, New Zealand
Banuett, F., Dept Biochem Biophys, Univ of California, San Francisco, CA, 94143, 415-666-4985
Bar-Tsur, Avri, Dept Maize Breed/Genet, Newe Yaar Exper Sta 31999, PO Haifa, Israel, 04-933-186
Baracco, Nestor P., Compania Continental S.A., C.C. 294, 2600 Venado Tuerto, Santa Fe, Argentina, 2815
Barbosa, Helio M., Dept Biologia Geral, Univ Vicosa, 36570-Vicosa, Minas Gerais, Brazil, 891-1790 R. 157
Barghoorn, E. S., Biological Laboratories, Harvard University, 16 Divinity Avenue, Cambridge, MA, 02138, 617-495-5224
Barry, Edward G., Department of Biology, Univ of North Carolina, Chapel Hill, NC, 27514, 919-962-6933
Baszczynski, C. L., Allelix Inc, 6850 Goreway Drive, Mississauga, Ont L4V 1P1, Canada, 416-677-0831
Bates, Lynn S., LSB Products, 731 McCall Road, Manhattan, KS, 66502, 913-537-9773
Baxter, Donald Ray, Dept Biol Sci, Stanford University, Stanford, CA, 94305, 415-723-3902
Bayliss, Michael W., Imperial Chem Ind PLC, Internatl Seeds Business, Jealott's Hill Research Stn, Bracknell Berks RG12 6EY, U K, 0344-424701
Bayne, Peter D., PO Box 11596, Shorewood, WI, 53211, 414-263-4111
Bdliya, Paul Mari, Department of Agronomy, Iowa State University, Ames, IA, 50011
Beach, Larry R., Pioneer Hi-Bred Internatl, Department of Biotechnology, Box 38, Johnston, IA, 50131, 515-270-3633
Bear, Robert P., Bear Hybrid Corn Company, PO Box 950, Decatur, IL, 62525, 217-422-5621
Beckett, Jack, Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-8214
Bedinger, Patricia, Dept of Biol, 010A Coker Hall, U of North Carolina, Chapel Hill, NC, 27599-3280, 919-962-6943
Bendbow, Evelyn, Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-8214
Benner, Michael, Dept. Agron. & Plant Genetics, Univ. of Minnesota, St. Paul, MN, 55108
Bennett, Clark, Genetics Department, University of California, Berkeley, CA, 94720, 415-642-7085
Bennett, J. H., Biology Dept, Brookhaven National Lab, Upton, NY, 11973
Bennett, M. D., Plant Breeding Institute, Maris Lane, Trumpington, Cambridge CB2 2LQ, U K, 0223 840411
Bennetzen, Jeff, Department Biological Science, Purdue University, W. Lafayette, IN, 47907, 317-494-4763
Benson, Dirk L., CIMMYT Maize Program, Londres 40 Apdo Postal 6-641, 06600, Mexico D. F., Mexico
Benton, W. D., ARCO Plant Cell Research Inst, 6560 Trinity Court, Dublin, CA, 94568, 415-833-3455
Benzion, G., Central Research Department, E.I. Dupont Exp Sta, Bldg E402, Room 4238, Wilmington, DE, 19898, 302-772-7031
Bergquist, Richard R., Plant Pathologist, 401 East Sixth Street, El Paso, IL, 61738, 309-527-6000
Berndt, Ken, RBA Seeds Inc, Rainbow Lane, Olivia, MN, 56277
Berville, A., INRA, Station d'Amelior des Plantes, Lab de Genet et Mutag, BV 1540, 21034 Dijon Cedex, France
Bhat, B. K., RRL BR Srinagar, PIN-190005 J + K, India
Bianchi, Angelo, Ist Sper Cerealicoltura, Via Cassia, 176, 00191 Rome, Italy, 06-3285705
Birchler, J., Room 290, The Biological Labs, Harvard University, 16 Divinity Avenue, Cambridge, MA, 02138, 617-495-4272
Bird, Robert McK., Inst Study Plants, Food & Man , P.O. Box 5142, Raleigh, NC, 27650
Bjarnason, Magni, CIMMYT, Apdo. Postal 6-641, 06600 Mexico, D.F., Mexico
Blackwood, Margaret, School of Botany, Univ of Melbourne, Parkville, Victoria 3052, Australia, 03-341-7157
Blake, Tom, Department of Plant & Soil Sci, Montana State University, Bozeman, MT, 59717-0002, 406-994-4601
Blakey, Cynthia Ann, L.F. Peterson Bldg., Rm. 202, Texas A&M University, College Station, TX, 77843, 409-845-1126
Blanco, J. L., Inst Biol Barcelona (Genet), C/Jorge Girona Salgado S/NO, Barcelona 34, Spain, 217-35-73
Blanco, M. H., McCurdy Seed Company, Fremont, IA, 52561, 515-933-4291
Bocanski, Jan, Faculty of Agriculture, University of Novi Sad, 21000 Novi Sad, Yugoslavia
Bocanski, Jan, Faculty of Agriculture, 21000 Novi Sad, Yugoslavia
Bogorad, L., Biol Labs, Harvard Univ, 16 Divinity Ave, Cambridge, MA, 02138, 617-495-4292
Bojanowska, Constance, Inst Plant Breeding, Biochemical Lab Box 1019, 00-950 Warsaw, Poland
Bokde, S., UNDP-FAO, C. P. 179, Bissau, Guine Bissau, West Africa
Bolen, P. L., NRRL-USDA, 1815 N. Univ St, Peoria, IL, 61614, 309-685-4011 EXT 272
Bommineni, V. R., Department of Plant Sciences, University of Western Ontario, London, Ontario N6A 5B7, Canada, 519-679-2908
Boothe, Joe G., Plant Research Institute, Saskatoon S7N 0W0, Canada
Borner, T., Sekt Biol, Humboldt Univ, Invalidenstr 43 DDR-1040, Berlin, E Germany
Bosch, L., Casablancas 108 2-2A, Sabadell, Barcelona, Spain, 93-7255689
Boston, Rebecca S., Box 7612, Dept Botany, North Carolina State Univ, Raleigh, NC, 27695-7612
Bouchard, Robert A., Dept of Biol, The College of Wooster, Wooster, OH, 44691, 216-263-2026
Boyer, C., Dept Hort Penn State Univ, 103 Tyson Building, University Park, PA, 16802, 814-863-3371
Brady, Kevin P., Department of Biology, Indiana University, Bloomington, IN, 47405, 812-335-4234
Brakke, M., Dept of Plant Path, Univ of Nebraska-Lincoln, Lincoln, NE, 68583, 402-472-3166
Brar, G. S., Agracetus Corp, 2208 Parview Road, Middleton, WI, 53562, 608-836-7300
Brawn, R. I., CIBA-GEIGY AG, Rosental, AG 5.1, CH-4002, Basel, Switzerland
Brettell, R., CSIRO Div Plant Industry, P.O. Box 1600, Canberra City ACT 2601, Australia, 062-46-5581
Brewbaker, James, Horticulture, Univ of Hawaii, 3190 Maile Way, Honolulu, HI, 96822, 808-948-7985
Briggs, Robert W., Funk Seeds International, 1300 W Washington Street, Bloomington, IL, 61701, 309-829-9461
Briggs, Steven, Cold Spring Harbor Lab, P.O. Box 100, Cold Spring Harbor, NY, 11724

Brown, D., Dept of Biology, Bishops University, Lennoxville Quebec, Canada, 819-569-9551 EXT 289
Brown, G. L., Allelix Inc, 6850 Goreway Drive, Mississauga, Ont L4V 1P1, Canada, 416-677-0831
Brown, R. P., Ohio Foundation Seeds, Croton, OH, 43013, 614-893-2501
Brown, William L., Pioneer Hibred Internatl, 6800 Pioneer Parkway, Johnston, IA, 50131, 515-270-3155
Brown, Willis, Dept Biol Sci, Purdue University, W. Lafayette, IN, 47907
Browne, C., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-2674
Buckner, Brent, Dept Genetics, Iowa State Univ, Ames, IA, 50011, 515-294-4618
Budiyanto, Tumin, D/A P. Kuru, Pegawai Stasiun, Banyuwangi, Jawa Timur, Indonesia
Buendgen, Mike, Department of Agronomy, University of Wisconsin, 1575 Linden Drive, Madison, WI, 53706
Buescher, Pat, Custom Farm Seed, Box 160, Momence, IL, 60954, 815-472-2433
Burgess, Diane, Advanced Genetic Sciences, 6701 San Pablo Avenue, Oakland, CA, 94608, 415-547-2395
Burmood, T., Research, Jacques Seed Company, Prescott, WI, 54022, 715-262-3223
Burnham, C. R., Agronomy & Plant Genetics, University of Minnesota, St. Paul, MN, 55108, 612-644-7797
Burr, Benjamin, Biology Dept, Brookhaven National Lab, Upton, NY, 11973, 516-282-3396
Bussard, James B., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-3410
Butler, Edward, Dept Agron, Curtis Hall, University of Missouri, Columbia, MO, 65211
Byrne, P. F., CIMMYT, Lisboa 27, Apdo. Postal 6-641, Col. Juarez, Deleg. Cuauhtemoc, 06600 Mexico, D.F., Mexico
Cabulea, I., Plant Breeding Laboratory, Str Agriculturii Nr 27 Jud, Cluj, Turda, Romania
Callis, Judy, Horticulture Dept, Univ Wisconsin, Madison, WI, 53706
Camara-Hernandez, J., Altolaguirre 1295, Buenos Aires 1427, Argentina, 51-6464
Cameron, James, Dept of Botany & Plant Sci, University of California, Riverside, CA, 92521, 714-686-0801
Cantrell, Ronald P., CIMMYT, Maize Program, Apartado Postal 6-641, 06600 Mexico, D.F., Mexico
Carlson, G., North Central Region, USDA-ARS MWA, 1815 N. University, Peoria, IL, 61604
Carlson, John E., Allelix, 6850 Goreway Drive, Mississauga L4V 1P1, Ont., Canada, 416-677-0831
Carlson, Lawrence A., 7 N. Winthrop Street, St. Paul, MN, 55119, 612-735-3423
Carlson, Wayne, Botany Department, University of Iowa, Iowa City, IA, 52240, 319-335-1316
Caruso, John L., Dept Biol Sci, University of Cincinnati, Cincinnati, OH, 45221
Cassals, Gladys, Department of Biology, Washington University, St. Louis, MO, 63130, 314-889-6813
Catcheside, D. G., Waite Agr Res Inst, Glen Osmond 5064 S, Australia, 08-797901 EXT 389
Chandlee, Joel M., USDA-PGGI, Seed Research Laboratory, Building 006, Beltsville, MD, 20708
Chandler, Vicki, Department of Biology, Institute of Molecular Biology, University of Oregon, Eugene, OR, 97403, 503-686-5136
Chandravadana, P., Sub Exp Sta, Univ Mass, 240 Beaver Street, Waltham, MA, 02154, 617-891-0650 EXT 67
Chang, Ming-Tang, Garst Seed Company, Highway 210, P.O. Box 500, Slater, IA, 50244, 515-685-3574
Chang, S. C., Known-You Seed Co. Ltd., 55 Min-Yi Rd., Yen-Pu Hsiang, Pingtung Hsieng, 904, Taiwan
Chao, Chuan-Ying, Tunghai University, Box 917, Taichung, Taiwan
Chao, Eddie, Center For Laboratories & Rsch, NYS Health Dept, Empire St Plaza, Albany, NY, 12201, 518-473-7333
Chaoxi, Dai, Department of Agronomy, Gansu Univ of Agriculture, Wuwei, Gansu 733006, China
Chase, Christine D., Plant Pathol Dept, Univ of Florida, Gainesville, FL, 32611, 904-392-3631
Chase, Sherret S., Chase Road, P.O. Box 193, Shokan, NY, 12481, 914-657-2392
Chaudhry, A. R., Punjab Maize + Millets Res, Yousafwala Sahiwal, Pakistan
Chen, Che-Hong, Section Plant Biol, Plant Sci Bldg, Cornell Univ, Ithaca, NY, 14853, 607-255-8423
Chen, Dexing, Dept of Agronomy, Shanghai Agric College, Shanghai, China
Chen, Jychian, Dept Biol, Yale Univ, P.O. Box 6666, New Haven, CT, 06511
Chen, Shu-Mei, 7841 Arcadia, Morton Grove, IL, 60053
Chen, Weicheng, Dept of Agronomy, Henan Agric College, Zhengzhou, China
Cheng, Andrew S., Sungene Technologies Corp , 2050 Concourse Drive, San Jose, CA, 95131, 408-433-9800
Cheng, David S. K., Sungene Technologies Corp, 2050 Concourse Drive, San Jose, CA, 95131
Cheng, P. C., 26-224, Dept of Biophysics, IBM T. J. Watson Research Ctr, Yorktown Heights, NY, 10598, 914-945-2070
Choe, Bong-Ho, Agronomy Dept, College of Agriculture, Chungnam National University, Dae-Jon 300-31, Korea
Chojecki, Jan, Imperial Chem Industries PLC, Jealott's Hill Research Sta, Bracknell Berks RG12 6EY, U K
Chomet, Paul, P.O. Box 100, Cold Spring Harbor Lab, Cold Spring Harbor, NY, 11724, 516-367-8484
Chou, Tau-San, Geo. J. Ball, Inc., P.O. Box 335, West Chicago, IL, 60185, 312-231-3600
Choudhary, V. K., Dept Genetics, Rajendra Agr Univ, Bihar, Pusa (Samastipur)-848125, India
Chourey, Prem, USDA ARS, Plant Pathology Department, University of Florida, Gainesville, FL, 32611, 904-392-7237
Christensen, Dean W., Funk Seeds Internatl, P.O. 477, Algona, IA, 50511, 515-295-9223
Christian, J. A., Dept Bot & Bact, Box 3158, Tech Station, Ruston, LA, 71272, 318-257-4772
Chung, J. H., (C) Yaounde, Agency for Internatl Develop, Washington, DC, 20523
Churchill, Gerald A., Asgrow Seed Company, P.O. Box 1235, Twin Falls, ID, 83301
Clark, E. M., Dept Botany & Microbiol, Auburn University, Auburn, AL, 36830, 205-826-4830
Clark, Jan, Department of Biology, Box 8238, University Station, University of North Dakota, Grand Forks, ND, 58202, 701-777-4479
Clark, Raymond L., USDA Plant Introduction Sta, Iowa State University, Ames, IA, 50010, 515-292-6506
Coe, E. H., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-2768
Coe, E. H., 206 Heather Lane, Columbia, MO, 65203
Cohen, Jerry, Bldg 50 HH4 BARC , BARC-West, Beltsville, MD, 20705
Cohen, Joel, Dekalb-Pfizer Genetics Inc, RR #2, Box 275L, Palmyra, PA, 17078, 717-964-3642
Colbert, Terry, Funk Seeds International, P.O. Box 921, Union City, TN, 38261, 901-885-7538
Colless, J. M., Agric Research Sta, Grafton, NSW, Australia, 066-447210
Compton, W. A., 317 Keim Hall, University of Nebraska, Lincoln, NE, 68583-0915, 402-472-2811
Cone, Karen C., Biology Department, Building 463, Brookhaven National Lab, Upton, NY, 11973, 516-282-3396
Cook, Bill, 105 Tucker Hall #11, University of Missouri, Columbia, MO, 65211, 314-882-3240
Coors, James G., Department of Agronomy, University of Wisconsin, 1575 Linden Drive, Madison, WI, 53706, 608-262-1390
Cormack, Jean, 27 Agronomy, Iowa State University, Ames, IA, 50011, 515-294-5076
Cornu, A., Sta d'Amelior des Plantes, B V 1540, 21034 Dijon, France, 80-237221
Costa-Rodrigues, L., Estacao Agron. Nacional, 2780 Oeiras, Portugal
Cowen, Neil, United AgriSeeds, P.O. Box 4011, Champaign, IL, 61820, 217-373-5300
Crane, Paul L., Dept of Agronomy, Purdue University, W. Lafayette, IN, 47907, 317-494-8088
Crawford, C. G., Northern Regional Res Lab, USDA-ARS, 1815 N. University Street, Peoria, IL, 61604, FTS 360-4011
Creech, Roy G., Agronomy, PO Box 5248, Mississippi State University, Mississippi State, MS, 39762, 601-325-2459 EXT 2311
Cress, Dean, Agricultural Biotechnology, Rohm and Hass Company, 727 Norristown Road, Spring House, PA, 19477
Cresse, April, Dept Biol Sci, Purdue University, W. Lafayette, IN, 47907
Crosbie, T. M., Garst Seed Co., 2515 Elwood Dr., Ames, IA, 50010, 515-296-2550
Cross, Harold, Dept of Agronomy, North Dakota State Univ, Fargo, ND, 58102, 701-237-8138
Crowe, Todd G., Department of Plant Sci, Biol & Geol Bldg, Rm 231, London, Ontario N6A 5B7, Canada
Cuany, Robin L., Department of Agronomy, Colorado State University, Fort Collins, CO, 80523, 303-491-6832
Culley, David, 352 Alderman Hall, Department of Hort Sci, University of Minnesota, St. Paul, MN, 55108, 612-373-1010
Cullis, Christopher A., Department of Biology, Case Western Reserve, Cleveland, OH, 44106, 216-368-5362
Cummings, D. P., Dekalb-Pfizer Genetics, Box 367, Windfall, IN, 46076, 317-945-7121
Curtis, Christine A., Department of Genetics, The Weizmann Inst Sci, Rehovot 76 100, Israel
Cutter, Gary L., Cargill, Inc, 324 Troy Street, Covington, OH, 45318, 513-473-5265
Da Silva, W. J., Genet Evol, Univ Campinas, Cidad Univ Barao Geraldo, Campinas SP, Brazil
Dahmen, Wayne J., Merck, Sharp & Dohme, Bldg 33-Ag Chem, Rahway, NJ, 07065, 201-574-4176
Dallmier, Donald J., Sturdy Grow Hybrids Inc, Box 94, Arcola, IL, 61910, 217-268-3838
Dankov, Toma, Ivan Assen Str.-93, Sofia 1504, Bulgaria, 43-82-73
Darrah, L., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-7707
Davidson, D., McMaster University, Biology Department, Hamilton, Ontario L8S 4K1, Canada, 416-525-9140 EXT 4588
Davis, David W., Laboratory of Plant Hardiness, 286 Alderman Hall-Univ Minn, 1970 Folwell Avenue, St. Paul, MN, 55108
Dawe, R. Kelly, Dept Genetics, University of California, Berkeley, CA, 94720, 415-642-8058
Day, Janet, Dept Plant Biol, 289 Morrill, University of Illinois, 505 S. Goodwin, Urbana, IL, 61801, 217-333-2919
Day, Peter R., Plant Breeding Institute, Maris Lane, Trumpington, Cambridge CB2 2LQ, UK, 0223-840411
De Aguiar-Perecin, Margarida L R, Esc Sup Agr Luiz de Queiroz, Inst de Genetica, C.P. 83, 13400 Piracicaba SP, Brazil
De Araujo, S. M. C., Fac Med Vet Agron, Univ Est Paul Julio Mesq F, 14870 Jaboticabal SP, Brazil, 0163-221798
De Jong, A. W., Zelder B. V., Postbus 26, 6590 AA GENNEP, Nederland
De Leon, Carlos, Apartado Postal 6-641, Mexico 6 D F, Mexico

De Leon, J. L., Vilela 103, 2600 Venado Tuerto, Santa Fe, Argentina
Dellaporta, Steve, Department of Biology, P.O. Box 6666, Yale University, New Haven, CT, 06511
Demopulos, J. T., Department of Zoology, Arizona State University, Tempe, AZ, 85287
Dempsey, Ellen, Biology Dept, Indiana University, Bloomington, IN, 47401, 812-335-4061
Dennis, Elizabeth, CSIRO, Division of Plant Industry, P.O. Box 1600, Canberra, ACT 2601, Australia
Dey, S. K., Department of Plant Breeding, Punjab Agric University, Ludhiana 141004, Punjab, India
Dhawan, N. L., Ford Foundation, 1, Sharia, Osiris, PO Box 2344, Garden City, Cairo, Egypt
Dhillon, B. S., Dept of Plant Breeding, Punjab Agric University, Ludhiana 141004, India
Di Fonzo, Natale, Ist Sper Cereal, Via Stezzano 24, P.O. 164, 24100 Bergamo, Italy, 035-248823
Dicke, F. F., 1430 Harding Avenue, Ames, IA, 50010, 515-232-3140
Dickinson, D. B., Horticulture Department, University of Illinois, 1103 West Dorner Drive, Urbana, IL, 61801, 217-367-2756
Diedrick, T., 9612-18-2, The Upjohn Company, Kalamazoo, MI, 49001, 616-385-8409
Dierks-Ventling, C., Friedrich Miescher-Inst, R W60 5.26, PO Box 273, 4002 Basel, Switzerland
Dilworth, Machi, USDA ARS OGPS, Room 112, W. Auditors Bldg., 15th & Independence Ave., SW, Washington, DC, 20251
Dodd, J. L., P-A-G Seeds, Box 470, Aurora, IL, 60507, 312-892-4331
Doebley, John F., Biology Department, Texas A&M University, College Station, TX, 77843, 919-737-2292
Dolfini, Silvana Faccio, Dipartimento di Genetica, University of Milano, Via Celoria, 26, 20133 Milano, Italy
Dollinger, E. J., Ohio Agric Res & Devel Center, Wooster, OH, 44691, 216-263-3893
Dooner, Hugo K., Advanced Genetic Sciences Inc, 6701 San Pablo Avenue, Oakland, CA, 94608, 415-547-2395
Doring, H.-P., Inst Genetik, Univ Koln, D-5000 Koln 41, Weyertal 121, W Germany, 0221-4702465
Dowling, Edward J., C/O Jacques Seed Co, P.O. Box 370, Lincoln, IL, 62656, 217-735-2770
Doyle, G. G., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-8215
Drolsom, Paul, Department of Agronomy, University of Wisconsin, Madison, WI, 53706, 608-262-0876
du Plessis, J. G., Summer Grain Centre, Grain Crops Research Inst, Private Bag X1251, Potchefstroom 2520, Republic of South Africa, 01481-27211
Duclos, Zachary M., Rt 3, Box 132, Leesburg, GA, 31763, 912-759-2161
Duke, Stephen O., S W S L, P.O. Box 225, Stoneville, MS, 38776
Dumanovic, Janko, Maize Res Inst, P.O. Box 89, 11081 Zemun, Yugoslavia
Duncan, Dave, Dept Agron, S-332 Turner Hall, University of Illinois, 1102 S. Goodwin, Urbana, IL, 61801, 217-333-9465
Dunlop, Joan, Plant Sciences Dept, Univ of Western Ontario, London, Ontario N6A 5E7, Canada
Dunn, G. M., Plant Sci Nesmith Hall, Univ of New Hampshire, Durham, NH, 03824, 603-868-2104
Duvick, Donald N., Pioneer Hi-Bred Internat, 400 Locust Street, 700 Capitol Square, Des Moines, IA, 50309, 515-245-3591
Duvick, Jonathan P., Pioneer Hi-Bred Internatl, Dept of Biotechnology Res, 7300 N.W. 62nd Ave, Box 38, Johnston, IA, 50131, 515-270-3176
Earle, Elizabeth D., Dept Plant Breed & Biom, Cornell University, 516 Bradfield, Ithaca, NY, 14853, 607-256-3102
Eastin, John A., Box 389, Grant, NE, 69140, 308-352-4819
Eberhart, Steve, USDA ARS Natl Seed Storage Lab, Colorado State Univ, Fort Collins, CO, 80523
Echt, Craig, Plant Pathology Department, Forbes Hall, University of Arizona, Tucson, AZ, 85721, 602-621-5490
Edwards, Marlin, Green Giant/Pillsbury, 1201 N. 4th Street, Le Sueur, MN, 56058, 612-665-3515
Eichholtz, David A., Monsanto, AA3E, 700 Chesterfield Village Pkwy, St. Louis, MO, 63198, 314-537-6227
El-Ard, Raghad Shiekh, Malki-Abdul Monem, Regad St Build 77 #7, Damascus, Syria
Empig, L. T., U.S.A.I.D./NCRE, Yaounde, Cameroon
Endo, Toru, National Inst of Genetics, Misima 411, Japan, 0559-75-0771 EXT 319
England, Dan, Curtis Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-7818
Esen, Asim, Dept Biology, Va Polytech Inst & State Univ, Blacksburg, VA, 24061, 703-961-5894
Eubanks, Mary W., Dept of Crop Science, N Carolina State U., Box 7620, Raleigh, NC, 27695-7620, 919-737-2647
Everett, Herbert L., Cornell University, 252 Emerson Hall, Ithaca, NY, 14853, 607-256-3103
Evola, S. V., CIBA-GEIGY Corporation, P.O. Box 12257, Research Triangle Park, NC, 27709-2257, 919-549-8164
Faberge, A. C., Department of Zoology, University of Texas, Austin, TX, 78711, 512-471-7442
Fahey, Ted, Crop Genetic International, 7170 Standard Drive, Hanover, MD, 21076, 301-796-4633
Fajen, Ava, Dept Agron, Curtis Hall, Univ Missouri, Columbia, MO, 65211
Farish, Guy, Department of Biology, University of North Dakota, Box 8238 University Station, Grand Forks, ND, 58202, 701-777-3676
Fauron, Christiane, University of Utah, 743 Wintrobe Building, Salt Lake City, UT, 84132, 801-581-6517
Feazel, Rhonda, RR1, Box 56, Washburn, IL, 61570
Fedoroff, Nina, Carnegie Inst Wash, 115 West Univ Parkway, Baltimore, MD, 21210, 301-467-1414
Feiler, Heidi, 16562 Drexel Avenue, South Holland, IL, 60423, 312-333-0937
Feix, G., Institut Fur Biologie III, Universitat Freiburg, Freiburg, W Germany, 0761-2032724
Ferguson, V., Custom Farm Seed, 2761 N Main, Decatur, IL, 62526, 217-875-2826
Ferl, R. J., Dept Bot, Univ Florida, Gainesville, FL, 32611, 904-392-2370
Ficsor, G., Dept of Biology, Western Mich Univ, Kalamazoo, MI, 49008, 616-383-1820
Figoni, Richard, IPRI, 830 Bransten Road, San Carlos, CA, 94070, 415-595-5335
Filion, W. G., Erindale College, University of Toronto, 3359 Mississauga Road, Mississauga, Ontario, Canada, 416-828-5326
Fincher, Robert R., Pioneer Hi-Bred Internatl, Dept of Biotechnology Research, P.O. Box 38, Johnston, IA, 50131, 515-270-3650
Findley, William R., Agronomy Department, Ohio Agri Res & Dev Center, Wooster, OH, 44691, 216-263-3894
Fitzgerald, P. J., USDA ARS, 2000 W Pioneer Parkway, Peoria, IL, 61614, 309-671-7176
Fleming, A. A., Miller Bldg, University of Georgia, Athens, GA, 30602, 404-542-2461
Fletcher, G., Agronomy Dept, S-122 Turner, University of Illinois, 1102 S. Goodwin, Urbana, IL, 61801, 217-333-9644
Floren, Catalina, CIMMYT, Londres 40, Apdo Postal 6-641, 06600 Mexico, D.F., Mexico
Foley, Terry, Holden's Foundation Seeds Inc, P.O. Box 839, Williamsburg, IA, 52361, 319-668-1100
Fong, Franklin, Dept Soil & Crop Sci, Texas A&M University, College Station, TX, 77843-2474
Foss, Jette, Department of Genetics, 8 Curtiss Hall, Iowa State University, Ames, IA, 50010, 515-292-3984
Foster, David J., Garst Seed Co/Research Dept, 2515 Elwood, Box 2, Ames, IA, 50010, 515-296-2550
Fourie, A. P., Dep Landb Vis, Highveld, Privaatsak X1251, Potchefstroom 2520, S Africa, 01481-25504
Fowler, Robert G., Dept Biological Sciences, San Jose State University, San Jose, CA, 95192, 408-277-2355
Fraley, Robb, Monsanto, AA3E, 700 Chesterfield Village Pkwy, St. Louis, MO, 63198, 314-537-6204
Francis, T. R., Northrup King Co., R. R. 1, Arva, NOM 1CO, Canada, 519-452-3019
Freeling, Michael, Dept of Genetics, Univ of California, Berkeley, CA, 94720, 415-642-7085
Frisch, David, Department of Agronomy, University of Minnesota, St. Paul, MN, 55108, 612-376-2648
Fromm, Michael, USDA ARS PGEC, 800 Buchanan Street, Albany, CA, 94710, 415-559-5908
Funk, Don E., Edward Funk and Sons, P.O. Box 67, Kentland, IN, 47951
Furtek, Douglas, Lab of Genetics, Univ of Wisconsin, Madison, WI, 53706, 608-262-3287
Gabay-Laughnan, S., Plant Biology/289 Morrill Hall, University of Illinois, 505 S. Goodwin Avenue, Urbana, IL, 61801, 217-333-2919
Galili, Gad, Dept Bot & Plant Path, Lilly Hall of Life Sciences, Purdue University, W. Lafayette, IN, 47907, 317-494-5877
Galinat, Walton C., Sub Exp Sta, Univ Mass, 240 Beaver Street, Waltham, MA, 02154, 617-891-0650
Gallais, A., G.I.S. du Moulon, Ferme du Moulon, 91190 Gif sur Yvette, France
Gamble, Edwin E., Dept of Crop Science, University of Guelph, Guelph, Ontario N1G 2W1, Canada, 519-824-4120 X3574
Gamborg, Oluf, United Energy Corporation, 420 Lincoln Centre Drive, Foster City, CA, 94404, 415-952-1000 EXT 6313
Garber, E. D., Barnes Lab, 5630 S Ingleside Avenue, Chicago, IL, 60637, 312-753-2671
Garber, Robert C., Department of Plant Pathology, 334 Plant Science Building, Cornell University, Ithaca, NY, 14853, 607-256-3243
Garcia, Ramon, EniChem Americas, Biotech Lab/Lewis Thomas Lab, Princeton University, Princeton, NJ, 08544
Garcia-Olmedo, F., Dept Biochemistry, E T S Ingenieros Agronomos, Madrid-3, Spain, 1-24444807
Gardner, C. A. C., Pioneer Hybrid Internatl, Highway 65 South, PO Box 218, Carrollton, MO, 64633, 816-542-3780
Gardner, C. O., Department of Agronomy, University of Nebraska, Lincoln, NE, 68583-0915
Garwood, D. L., Garwood Seed Company, RR 1, Box 20, Stonington, IL, 62567, 217-325-3715
Garzonio, Dennis, PAG Seeds, PO Box 467, Monticello, IL, 61856, 217-762-2711
Gavazzi, Giuseppe, Universita de Milano, Dept of Genetics & Microbiol, Via Celoria 26, 20133 Milano, Italy, 02-230823
Gay, Philippe, CIBA-GEIGY Ltd, Agricultural Division, CH-4002 Basel, Switzerland, 061-37-44-57
Geadelmann, J. L., Holden's Foundation Seeds, Inc, P.O. Box 839, Williamsburg, IA, 52361, 319-668-1100
Gebauer, Juan E., Corn Breeding, Casilla 190, Buin, Chile
Geerthsen, J. M. P., Dept of Genetics, University of Pretoria, Pretoria 0181, S Africa, 012-436381
Geiger, Hartwig H., Univ Hohenheim, Inst Pflanzenzucht, Postfach 700562(350), D-7000 Stuttgart 70, W Germany
Geng, Qing-Han, Dept of Agronomy, Inner Mongolia Agric College, Inner Mongolia Aut Reg, China
Gengenbach, B.G., Agron & Plant Genetics, Univ of Minnesota, St Paul, MN, 55108, 612-373-1371
Georgiev, Trifon, Technical University, "A. Kanchev", Rousse 7004, Bulgaria, 082-23469
Gerats, Tom, Department of Genetics, Free University, De Boelelaan 1087, 108 MV Amsterdam, Nederland, 020-68-05-51

Gerrish, Everett E., Box 774, Grinnell, IA, 50112, 515-236-4911
Ghidoni, Achille, Istituto di Genetica, Universita di Milano, Via Celoria 26, Milano 20133, Italy, 02-230823
Ghobrial, Helmy K., 4100 Valentine Crest Rd, St. Paul, MN, 55112
Ghosh, P., Dept of Botany, Kalyani Univ, Kalyani, Nadia, W Bengal, India, 220/ext 9
Giesbrecht, John, Res Sta Canada Agr, P.O. Box 3001, Morden, Manitoba, Canada, 822-4471
Gillies, Christopher, Botany Bldg A12, Univ of Sydney, Sydney NSW 2006, Australia, 02-692-2688
Glover, David V., Dept of Agronomy, Purdue University, W. Lafayette, IN, 47907, 317-494-8067
Godshalk, E. Brent, 1561 Agronomy, Iowa State University, Ames, IA, 50011, 515-294-3052
Goertzen, Kenneth L., Goertzen Seed Res Inc, R R 2 Box 48, Scott City, KS, 67871, 316-872-2807
Goldman, Stephen, Dept of Biology, University of Toledo, Toledo, OH, 43606, 419-537-4197
Golubovskaya, I. N., Inst Genet & Cytol, Siberian Branch Akad Sci, Novosibirsk, USSR
Gonella, Jaime, CIBA-GEIGY, 26020 Casalmorano (CR), Italy, 0374-74144
Good, Ronald L., Funk Seeds International, PO Box 417, Bluffton, IN, 47714, 219-824-5056
Goodman, Major M., Department of Crop Sciences, North Carolina State Univ, P.O. Box 7620, Raleigh, NC, 27695, 919-737-2704
Gordon, P. N., Department of Biology, 918 Kline Biology Tower, Yale Univ, P.O. Box 6666, New Haven, CT, 06511-8112
Gottlieb, Leslie D., Dept of Genetics, Univ of Calif-Davis, Davis, CA, 95616, 916-752-0268
Grabau, Beth, Dept Agronomy, Univ Minnesota, St. Paul, MN, 55108, 612-625-5215
Gracen, V. E., Cargill Hi-Bred Seed, Box 9300, Minneapolis, MN, 55440, 612-475-7244
Graham, K. M., Genetics Department, Faculty of Life Sciences, Natl Univ of Malaysia, Bangi, Malaysia
Grant, David, Pioneer Hi-Bred Internatl, Box 38, 7300 N.W. 62nd Avenue, Johnston, IA, 50131, 515-270-3503
Gray, Dennis J., University of Florida, Agricultural Research Center, P.O. Box 388, Leesburg, FL, 32749-0388, 904-787-3423
Greder, Rod, 2106 Orchard St #302, Urbana, IL, 61801, 217-333-7585
Green, C. E., Molecular Genetics Inc., 10320 Bren Road East, Minnetonka, MN, 55343, 612-935-7335
Green, M. M., Dept of Genetics, University of California, Davis, CA, 95616, 916-752-1189 EXT 6295
Greenblatt, I. M., Genetics & Cell Biology, U-131, Univ of Connecticut, Storrs, CT, 06268, 203-486-2915
Greyson, R. I., Dept of Pl Sci-Faculty of Sci, Univ of Western Ontario, London, Ontario N6A 5B7, Canada, 519-679-3107
Grier, Stephen L., Agrigenetics Corp, Vegetable Products Group, 1120-220th St West, Farmington, MN, 55024, 612-463-4646
Grobman, Alexander, Grobman Genotecnica SA, Apartado 5161, Lima 1, Peru, 234464
Gross, D. F., Dekalb-Pfizer Genetics, Corn Research Center, 3100 Sycamore Road, Dekalb, IL, 60115, 815-756-7333
Grula, John, Phytogen, 101 Waverly Drive, Pasadena, CA, 91105, 818-792-1802
Grumbles, Robert M., Dept Molec Biophys & Biochem, 260 Whitney Avenue, P.O. Box 6666, New Haven, CT, 06511, 203-432-2606
Gu, Mingguang, Boston College, Department of Biology, Chestnut Hill, MA, 02167, 617-969-0100 EXT 3551
Haag, Dr. Wayne L., CIMMYT, c/o CIAT, 1380 NW 78th Ave, Miami, FL, 33126
Haag, Wayne L., CIMMYT, P. O. Box 120 Yenimahalle, Ankara, Turkey
Hack, Ethan, Curtiss Hall, Genetics Department, Iowa State University , Ames, IA, 50011
Hagan, William, Del Monte Corp, 850 Thornton St, PO Box 36, San Leandro, CA, 94577, 415-351-2661
Hagemann, R., Dept Genetics, Martin Luther University, Domplatz 1, 4020 Halle/S., E Germany
Hague, Donald R., Department of Biology, University of Oregon, Eugene, OR, 97403, 503-686-4545
Hake, S., USDA-ARS-PGEC, 800 Buchanan Street, Albany, CA, 94710, 415-559-5907
Hallauer, Arnel R., Agronomy Building, Iowa State University, Ames, IA, 50010, 515-294-3052
Hallberg, Boone, SEP, Centro de Graduados, Calzs . Technologico Y, Gral. Wilfrido Massieu S/N., Oaxaca, Oax. C.P. 68000, Mexico
Hamilton, R. H., Buckhout Laboratory, Pennsylvania State Univ, University Park, PA, 16802, 814-865-8800
Han, Changdeok, Curtis Hall, University of Missouri, Columbia, MO, 65211
Hannah, L. C., Veg Crops Dept, Univ of Florida, IFAS, Gainesville, FL, 32611, 904-392-1794
Hansel, W. C., Hansel Cons & Mgmt, Box 283, Carrollton, MO, 64633, 816-542-1616
Hansen, Dale J., Agrigenetics Res Corp, 3375 Mitchell Lane, Boulder, CO, 80301, 303-443-5900 EXT 230
Hanson, Maureen R., Dept Biol, Gilmer Hall, Univ Virginia, Charlottesville, VA, 22901, 804-924-0706
Hanson, W. D., Dept of Genetics, North Carolina State Univ, Box 7614, Raleigh, NC, 27695-7614, 919-737-2289
Harberd, Nicholas, Dept Genetics, 345 Mulford Hall, University of California, Berkeley, CA, 94720, 415-642-8058
Harlan, Jack, Agronomy Department, University of Illinois, 1102 S. Goodwin Avenue, Urbana, IL, 61801, 217-333-4373
Harn, Chee, Dept Botany & Plant Path, Oregon State University, Corvallis, OR, 97331, 503-754-3451
Harper, David C., Holden's Foundation Seeds Inc., P.O. Box 839, Williamsburg, IA, 52361, 319-668-1100
Harris, John W., Dept of Biology, Tennessee Tech Univ, Cookeville, TN, 38505, 615-528-3143
Harris, Robert M., Committee on Genetics, University of Arizona, Tucson, AZ, 85721, 602-621-3584
Harris-Cramer, Jane, Agrigenetics, 5649 East Buckeye Road, Madison, WI, 53716, 608-221-5000
Harrison, Andrew, Laboratory of Genetics, Room 218, University of Wisconsin, Madison, WI, 53706
Hartman, Carl J., 155 South St. Rd. 2, Valparaiso, IN, 46383
Harvey, P. H., Dept Crop Sci, NC State Univ, Raleigh, NC, 27650, 919-787-6054
Hashizume, Ken, Snow Brand Seed Co, Hokkaido Research Station, Horonai, Naganuma-cho, Hokkaido 069-14, Japan
Haskins, F. A., Dept of Agronomy, Univ of Nebraska, Lincoln, NE, 68583, 402-472-1567
Hauptli, Holly, Calgene Inc, 1910 Fifth Street, Davis, CA, 95616, 916-753-6313
Hawk, James A., Dept Plant Science, University of Delaware, Newark, DE, 19711, 302-738-2531
Hayden, D. B., Dept of Botany, Univ of Western Ontario, London, Ontario, Canada, 519-679-6804
Headrick, John, Plant Pathology Department, Univ Ill, N-519 Turner Hall, 1102 S. Goodwin Avenue, Urbana, IL, 61801
Heidrich-Sobrinho, E., Dept Genetica Ib Ufgrs, Caixa Postal, 1953, Porto Alegre Rs, Brazil
Helentjaris, Tim, NPI, 417 Wakara Way, Salt Lake City, UT, 84108, 801-582-0144
Helgason, S. B., Dept of Plant Science, Univ of Manitoba, Winnipeg, Man. R3T 1N1, Canada, 474-8221
Helm, James, Agronomy Department, North Dakota State University, Fargo, ND, 58105
Helsel, D., 2-64 Agriculture Building, Univ of Missouri, Columbia, MO, 65211, 314-882-8301
Henderson, Clarion B., Illinois Foundation Seeds, PO Box 722, Champaign, IL, 61820, 217-485-6260
Henke, Randolph R., Department of Botany, University of Tennessee, Knoxville, TN, 37996, 615-974-6227
Herrmann, R., Botany Department, Ludwig-Maximilans Univ Munich, Manzenger Str 67, 8 Munchen 19, W. Germany
Herskowitz, I., Dept Biochem & Biophys, Univ of California, San Francisco, CA, 94143, 415-666-4977
Hibberd, K. A., Molecular Genetics Inc, 10320 Bren Road East, Minnetonka, MN, 55343, 612-935-7335
Hicks, Glenn R., Sungene Technologies Corp, 2050 Concourse Drive, San Jose, CA, 95131
History file, Curtis Hall, University of Missouri, Columbia, MO, 65211
Ho, David, Department of Biology, Washington University, St. Louis, MO, 63130, 314-889-4632
Hoagland, R. E., USDA ARS SWSL, PO Box 225, Stoneville, MS, 38776, 601-686-2311
Hodges, Tom, Botany & Plant Pathology, Purdue University, W. Lafayette, IN, 47907
Hoegemeyer, Thomas C., Hoegemeyer Hybrids Inc, Rt 2, Hooper, NE, 68031, 402-654-3399
Hoisington, David, Curtis Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-2033
Holden, A., Corn States Hybrid Serv, PO Box 2706, Des Moines, IA, 50315, 515-285-3091
Hole, David, Soil & Crop Sci. Dept., Plant Sci. Bldg., Texas A&M University, College Station, TX, 77843, 409-845-1126
Hooker, A. L., 39W749 Deerhaven Trail, St. Charles, IL, 60174, 312-377-1786
Hopkins, W. G., Dept of Plant Sciences, Univ of Western Ontario, London, Ontario, Canada
Horne, Shelley M., Biology Department, P.O. Box 8238, University of North Dakota, Grand Forks, ND, 58202-8238, 701-777-4479
Hourcade, Dennis, Monsanto, GG4I, 700 Chesterfield Village Pkwy, Chesterfield, MO, 63198, 314-537-6783
Hsieh, S. C., Taichung District, Agricultural Improvement Sta, Tienyang Village, Tatsuen Hsiang, Changhua, Taiwan, 048-358-130
Hsu, Lilian M., Carr Lab, Mount Holyoke College, South Hadley, MA, 01075, 413-538-2609
Hu, Wilma Wei-Lin, Dept of Crop Science, North Carolina State Univ, Raleigh, NC, 27607
Huang, Yi Xiang, Crop Breed & Cult Res Inst, Sichuan Academy Agr Sci, Chengdu, Sichuan Province, China
Hunsperger, John P., Dept Genet & Cell Biol, University of Minnesota, St. Paul, MN, 55108
Hunt, Marjorie, Div Biol Sci, Tucker Hall, Univ Missouri, Columbia, MO, 65211
Hunter, Brenda, Dept Genet Cell B, Univ Minnesota, St. Paul, MN, 55108
Hunter, R. B., Funk Seeds International , 1300 West Washington, Bloomington, IL, 61701
Iltis, Hugh H., Dept of Botany, Univ of Wisconsin, Madison, WI, 53706, 608-256-7247
Inglett, George, USDA-NRRC, 1815 N University Ave, Peoria, IL, 61604, 309-685-4011
Ingram, David S., Botany Department, University of Cambridge, Downing Street, Cambridge CB2 3EA, U K, 0223-61414
Inoue, Yasuaki, National Grassland Res Inst, 768 Nishinasuno, Tochigi 329-27, Japan
Irish, Erin, Department of Biology, Leidy Lab/G7, University of Pennsylvania, Philadelphia, PA, 19104
Isaac, Peter, The Nickerson Seed Co Ltd, Cambridge Science Park, Milton Road, Cambridge CB4 4WE, UK
Isenhour, David J., Department of Entomology, Univ Ga, Coastal Plain Exp Sta, P.O. Box 748, Tifton, GA, 31793-0748, 912-386-3374
Ishige, Teruo, Department of Cell Biology, Natl Inst Agrobiol Resources , Yatabe Tsukuba, Ibaraki 305, Japan

Isola, Narayana R., Department of Plant Pathology, University of Florida, Gainesville, FL, 32611, 904-392-7237
Jackson, R. C., Biology Department, Texas Tech University, Lubbock, TX, 79409, 806-742-2717
Jacobs, M., Plant Genetics Univ Brussels, 65 Paardenstraat, B-1640st Genesius-Rode, Belgium, 2-358-65-22
Jakob, Karl M., Department of Plant Genetics, Weizmann Inst Sci, Rehovot, Israel, 054-83301
Jalani, B. S., Nat Univ of Malaysia, Bangi-Selangor, Malaysia
James, Martha G., Genetics Department, 8 Curtiss Hall, Iowa State University, Ames, IA, 50010, 515-292-3984
Jan-Orn, Jinda, Division of Field Crops, Department of Agriculture, Bangkok 10903, Thailand
Jauhar, Prem P., USDA ARS, Forage & Range Res Lab, Utah State Univ, Logan, UT, 84322-6300, 801-750-3074
Jaynes, Jesse, Department of Biology, 322 Choppin Hall, Louisiana State University, Baton Rouge, LA, 70803
Jenkins, M. T., 1200 Mira Mar Avenue, Apt 826, Medford, OR, 97504, 503-776-5255
Jennings, Paul H., Plant & Soil Sciences, Univ of Massachusetts, Amherst, MA, 01003
Jewell, David C., CIMMYT, Londres 40, Apdo Postal 6-641, Mexico D.F. 06600, Mexico, 585-4355 EXT 1110
Jimenez, J., Biology Dept, Purdue Univ, Fort Wayne, IN, 46805, 219-482-5253
Jin, Young Kwan, Dept Biol Sci, Purdue University, W. Lafayette, IN, 47907
Johns, Mitrick A., Dept Biological Sciences, Northern Illinois University, DeKalb, IL, 60115, 815-753-7836
Johnson, Alice, 412 Bradfield Hall, Cornell Univ, Ithaca, NY, 14853, 607-256-3104
Johnson, E. C., IPRI, Internatl Plant Res, 830 Bransten Road, San Carlos, CA, 94070, 415-595-5335
Johnson, Elizabeth, Box 371, Kaunakakai, HI, 96748, 808-553-5468
Johnson, Ken, Sigco Research, Box 289, Breckenridge, MN, 56520, 218-643-2155
Johnson, M. W., Dept of Agronomy, Pennsylvania State Univ, University Park, PA, 16802, 814-865-0324
Johnson, Scott, Dept Agronomy, Curtis Hall, Univ Missouri, Columbia, MO, 65211, 314-882-2674
Johri, M. M., Molecular Biol Unit, Tata Inst Fundamental Res, Bombay 400 005, India, 219111/ext 338
Jondle, Doug, Department of Agronomy, University of Wisconsin, 1575 Linden Drive, Madison, WI, 53706
Jones, J. K., Department of Agricultural Bot, University of Reading, Whiteknights, Reading RG6 2AS, UK, 0734-875123 EXT 7905
Jones, John Edward, 8008 Daniel Drive, Forestville, MD, 20747
Jordan, Berry, Dept Botany, Miller Plant Science, Univ Georgia, Athens, GA, 30602
Josephson, L. M., 350 Plant Sci Bldg, Univ of Tennessee, Knoxville, TN, 37916, 615-974-7391
Jotshi, P. N., Regional Research Lab (Branch), Council Sci & Indus Res, Sanat Nagar 190005, Srinagar, India
Julstrom, P., Dekalb-Pfizer Genetics, 3100 Sycamore Road, Dekalb, IL, 60115
Jupe, Eldon R., Department of Biochemistry, 322 Choppin Hall, Louisiana State University, Baton Rouge, LA, 70803, 504-388-1554
Juvik, J. A., Dept of Horticulture, University of Illinois, 1103 West Dorner Drive, Urbana, IL, 61801, 217-333-1966
Kaan, F., INRA ENSA, Place Viala 34060, Montpellier Cedex, France, 67-63-43-20
Kahler, A. L., Garst Seed Company, Highway 210, P.O. Box 500, Slater, IA, 50244, 515-685-3574
Kalia, V., Regional Research Station, Dhaulakuan-173001, Distt Sirmur (H. P.), India
Kaltsikes, P. J., Dept Plant Breed & Biometry, Athens School of Agr Sci, Iera Odos 75, Athens T.T.301, Greece, 347-1504
Kamimura, Shoji, Kunisada Plant Breed Stn, 1211 Ichiba, Akabori-mura, Sawa-gun, Gunma-ken 379-22, Japan
Kaneko, Koji, National Agr Res Center, Yatabe-Cho, Tsukuba-Gun, Ibaraki-Ken 305, Japan
Kaneko, Tatsuo, Snow Brand Seed Co, Hokkaido Research Station, Horonai, Naganuma-cho, Hokkaido 069-14, Japan
Kang, M. S., Department of Agronomy, Louisiana State University, Baton Rouge, LA, 70803, 504-388-2110
Kannenberg, L. M., Dept of Crop Science, University of Guelph, Guelph, Ontario N1G 2W1, Canada, 519-824-4120 EXT 2506
Karazawa, Missae, Department of Agronomy, Room 20, Iowa State University, Ames, IA, 50010, 515-294-5076
Karpoff, A. J., Dept of Biology, Univ of Louisville, Louisville, KY, 40208, 502-588-6771
Kasha, K. J., Dept of Crop Science, Univ of Guelph, Guelph, Ontario N1G 2W1, Canada, 824-4120 EXT 2507
Kato Y., T. A., Centro de Genetica, Colegio de Postgraduados, Chapingo 56230, Mexico, (5)5-85-07-28
Keil, R. L., Yale U/Biology Dpt/PO Box 6666, Kline Biol Tower Rm 812, New Haven, CT, 06511, 203-436-2266
Keim, Kent, Cargill Seed Division, Box 467, 1020 S. Market Street, Monticello, IL, 61856, 217-762-2711
Keim, Kent R., 1211 W. Daniel, Champaign, IL, 61821, 217-356-2688
Keller, Patrick, Biochemistry Dept, Univ of Rhode Island, Kingston, RI, 02881, 401-295-0298
Kelley, Phil, Dept Microbiol & Immunol, 6643 Medical Sci Bldg II, University of Michigan, Ann Arbor, MI, 48109, 313-763-3531
Kemble, R. J., Allelix Inc, 6850 Goreway Drive, Mississauga, Ont. L4V 1P1, Canada, 416-677-0831
Kennell, Jack, Department of Plant Pathology, Fifield Hall, University of Florida, Gainesville, FL, 32611, 904-392-3639
Kermicle, Jerry, Dept of Genetics, University of Wisconsin, Madison, WI, 53706, 608-262-1253
Kerr, E. A., Stokes Seeds Limited, 39 James Street, P.O. Box 10, St. Catharines, Ontario L2R 6R6, Canada, 416-688-4300
Keyes, Carol A. Lemke, Campus Box 1137, Washington University, St. Louis, MO, 63130
Khavkin, Emil E., All-Union Res Institute of, Agricultural Biotechnology , 12 Pskovskaya str., bld. 4, 127253 Moscow, USSR
Khehra, A. S., Punjab Agr University, Department of Plant Breeding, Ludhiana, India
Kim, Byung-Dong, Department of Plant Science, University of Rhode Island, Kingston, RI, 02881, 401-792-2529
Kim, Hwa Yeong, University of Wisconsin, Lab of Genetics, 445 Henry Mall, Madison, WI, 53706, 608-262-3287
Kim, Soon Kwon, Internatl Inst Tropical Agr, PMB 5320, Ibadan, Nigeria, 413244
Kind, K., Curtis Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-2674
Kindiger, Bryan, Wilson Hybrids, Inc, P.O. Box 391, Harlan, IA, 51537, 712-755-3841
King, Patrick J., Friedrich Miescher-Inst, Postfach 2543, CH-4002 Basel, Switzerland, 61-375583
Kinugawa, Kenjiro, 4-23-3, Takakuradai, Univ of Osaka Prefecture, Sakai, Osaka 590-01, Japan, 0722-91-5497
Kirihara, Julie, Department of Biochemistry, University of Minnesota, 1479 Gortner Avenue, St. Paul, MN, 55108, 612-376-9273
Kiss, Charles, Northrup King Semences, 18 Avenue Gollieni, 49130 Les Ponts de C, France, (41) 663831
Kleese, Roger, Molecular Genetics, 10320 Bren Road East, Minnetonka, MN, 55343, 612-935-7335
Klein, Anita S., Department of Biochemistry, Spaulding Life Science Bldg, University of New Hampshire, Durham, NH, 03824, 603-862-2473
Kodrzycki, R., Dept Bot & Plant Pathol, Lilly Hall, Purdue Univ, W. Lafayette, IN, 47907
Kohashi, Josue, Dept of Botany, Colegio de Postgrad, ENA, Chapingo, Edo de Mex, Mexico
Konstantinov, Kosana, Maize Research Institute, P.O. Box 89, 11081 Zemun, Yugoslavia
Konstantinov, Yu., Siberian Inst Plant Phys Bioch, USSR Academy of Sciences, P.O. Box 1243, Irkutsk, USSR
Konzak, Calvin F., Dept of Agronomy & Soils, Washington State Univ, Pullman, WA, 99164-6420, 509-335-3635
Kovacs, Istvan, Agriculture Research Instit, Hungarian Acad Sci, 2462 Martonvasar, Hungary, 26-45774
Kowles, R. V., Biology Department, St Marys College, Winona, MN, 55987, 507-452-4430 EXT 1554
Krapovickas, Antonio, Dept Bot y Ecol, Casilla Correo 209, 3400 Corrientes, Argentina
Kratochvil, D. E., Sokota Hybrid Producers, Brookings, SD, 57006, 605-692-6258
Krill-Shadley, Gwen, Dept Biology, Ill State Univ, Normal, IL, 61761, 309-438-2685
Kriz, A. L., Dept Agronomy, Turner Hall, Univ Illinois, Urbana, IL, 61801
Kuehnle, Adelheid, Dept Plant Breed & Biometry, 252 Emerson Hall, Cornell University, Ithaca, NY, 14853, 607-256-2180
Kumar, D., Div Plant Br & Genet JARI, PO Barrackpore D 24 Pargana, West Bengal, India, 24-4806
Kumar, Santosh, Professors' Colony, Aghoriachowk, PO Ramna, Muzaffarpur-842002 BIHAR, India
Kuranda, K., Genetics Institute, 87 Cambridge Park Drive, Cambridge, MA, 02140, 617-876-1170
Laible, Charles A., Research Department, Funk Seeds International, Bloomington, IL, 61701, 309-829-9461
Lambert, A., Semences Nickerson, 49160 Longue, Jumelles, France
Lambert, R. J., Agronomy Dpt/S-118 Turner Hall, University of Illinois, 1102 S. Goodwin Avenue, Urbana, IL, 61801, 217-333-4254
Lamkey, Kendall, Agronomy Building, Iowa State University, Ames, IA, 50010, 515-294-7826
Lampoh, E., Crops Res Inst, PO Box 20, KPEVE, Volta Reg, Ghana, 2 ext 5
Lander, Eric S., Whitehead Institute, 9 Cambridge Center, Cambridge, MA, 02142, 617-258-5192
Lane, Barbara, Genetics Department, University of California, Berkeley, CA, 94720, 415-642-8058
Langdale, Jane, Biology Department, P.O. Box 6666, Yale University, New Haven, CT, 06511, 203-436-3752
Larkins, B., Dept Bot & Plant Path, Purdue Univ, W. Lafayette, IN, 47907, 317-494-4624
Larson, Russell L., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-3410
Laughnan, John R., Plant Biology/289 Morrill Hall, U of Illinois/505 S Goodwin Av, Urbana, IL, 61801, 217-333-2919
Laughner, Bill, Dept Biology, Hiram College, Hiram, OH, 44234, 216-569-3211 EXT 260
Laurie, David, Plant Breeding Institute, Maris Lane, Trumpington, Cambridge CB2 2LQ, UK, 0223-840411
Lavapaurya, Tavat, Dept Horticulture, Kasetsart University, Bangkok 10900, Thailand
Law, Colin, Plant Breed Inst, Maris Lane, Trumpington, Cambridge CB2 2LQ, UK
Leaver, C. J., Dept Botany, Kings Bldgs, U. of Edinburgh/Mayfield Road, Edinburgh EH9 3JH, Scotland, 031-667-1081 EXT 3304
Lee, Chong, Oil Palm Res Station, PO Box 207, Banting, Selangor, Malaysia
Lee, Elizabeth, Dept Agron, Iowa State Univ, Ames, IA, 50011
Lee, Michael, Department of Agronomy, Iowa State University, Ames, IA, 50011, 515-294-3052
Lee, Sharon K., Department of Botany, University of Rhode Island, Kingston, RI, 02881, 401-792-2625

Lee, Wayne, 108 S. Grant, Milford, IL, 60953, 815-889-4426
LeFord, Duane, Pfister Hybrid Corn Co, El Paso, IL, 61738, 309-527-6008
Leja, Carol, Dept Plant Biol, 289 Morrill, University of Illinois, 505 S. Goodwin, Urbana, IL, 61801, 217-333-2919
Leland, Tim, Funk Seeds International, 1300 West Washington, Bloomington, IL, 61701, 309-829-9461
Leonard, Kurt, USDA-ARS, Box 7616, Raleigh, NC, 27695-7616, 919-737-2751
Leto, K. J., Central Research Dept, E. I. Du Pont Exp Sta, Bldg 328, Rm 259, Wilmington, DE, 19898, 302-772-1525
Levings, C. S., Department of Genetics, North Carolina State Univ, Box 7614, Raleigh, NC, 27695-7614, 919-737-2289
Levites, E. V., Inst Cytol Genetics, Novosibirsk, USSR
Lewis, Herman, Div of Phys, Cell & Mol Biol, National Science Foundation, Washington, DC, 20550, 202-357-7647
Li, Ching Hsiung, Chinese Acad Agric Sci, Department of Crop Science, Beijing, China
Li, H. W., Institute of Botany, Academia Sinica, Taipei, Taiwan
Li, Jigeng, Institute of Genetics, Academia Sinica, Beijing, China
Liang, George H., Dept of Agronomy, Kansas State University, Manhattan, KS, 66502, 913-532-7255
Lim, Yong Pyo, Dept Biol, Yale Univ, P.O. Box 6666, New Haven, CT, 06511
Lin, Bor-yaw, Genetics Institute, 87 Cambridge Park Drive, Cambridge, MA, 02140, 617-876-1170
Lin, Chi C., Lilly Research Lab, Box 708, Greenfield, IN, 46140
Lin, Ji-Zhou, Institute of Crops, Shanxi Acad Agric Sci, Shanxi Province, China
Lindsey, M. F., Dekalb-Pfizer Genetics, Box 408, Dayton, IA, 50530, 515-547-2550
Liu, Edwin H., Savannah River Ecology Lab, P.O. Drawer E, Aiken, SC, 29801, 803-725-2472
Liu, Enxun, Dept of Agronomy, Laiyang Agric College, Laiyang, Shandong, China
Liu, Jilin, Dept of Agronomy, Huazhung Agric College, Wuhan, Hubei Province, China
Loeffel, F. A., NAPB Res. Center, Rural Route #2, Brookston, IN, 47923, 317-563-3111
Lonsdale, D. M., Plant Breeding Institute, Maris Lane, Trumpington, Cambridge CB2 2LQ, UK
Lorenzoni, C., Istituto di Genetica Vegetale, Universita Cattolica, 29100 Piacenza, Italy
Lorz, Horst, Max-Planck-Institut Fur, Zuchtungsforschung, 05000 Koln 30, W Germany
Lower, W., 235 Environ Trace Ctr, Univ of Missouri, Columbia, MO, 65211, 314-445-8595
Lu, Yingtang, Dept Plant Molec Physiol, University of Hawaii, 3190 Maile Way, Honolulu, HI, 96822, 808-948-8384
Ludwig, Steven, Department of Botany, University of Georgia, Athens, GA, 30602
Ma, Te-Hsiu, Dept Biol Sciences, Western Illinois University, Macomb, IL, 61455, 309-298-1457
Ma, Yu, Agrigenetics, 5649 East Buckeye Road, Madison, WI, 53716, 608-221-5000
Mackenzie, David, Harris Moran Seed Company, 1832 Garrity Blvd, P.O. Box 842, Nampa, ID, 83651, 208-467-6341
Maggini, Fabio, Ist di Biologia Agraria, Cattedra di Botanica, Via Riello, 01100 Viterbo, Italy
Magoja, J. L., Inst Fitotec Santa Catalina, Llavallol, Argentina, 243-0233
Maguire, Marjorie, Zoology Department, University of Texas, Austin, TX, 78712, 512-471-7451
Maheshwari, J. K., National Botanical Res Inst, Lucknow-226001, India
Majeste, A, Coop Agric Cereales, Ave Gaston Phoebus, 64230 Lescar, France, 59-328446
Maletsky, S. I., Tereshkova St, House 6, Flat 49, Novosibirsk 630090, USSR
Malone, Carl, 205 McKinley Avenue, Milford, IL, 60953, 815-889-4079
Maloney, Alan, Biol Sci Dept, Stanford Univ, Stanford, CA, 94305, 415-497-2609
Mangelsdorf, P. C., Apt. 1200, Carol Woods, Chapel Hill, NC, 27514, 919-942-4396
Manrique, Antonio, Univ Agraria Progr, Coop, Inv Maiz, La Molina, Apt 456 Lima, Peru, 312746
Mans, Rusty J., Dept of Biochem/J245 JHMHC, Univ of Florida, Gainesville, FL, 32610, 904-392-2332
Manzocchi, Lucia A., Consiglio Nazionale Richerche, Istituto Biosintesi Vegetali, Via Bassini 15, 20133 Milano, Italy
Mapping file, Curtis Hall, University of Missouri, Columbia, MO, 65211
Marchand, J. L., IRAT, Ligne Paradis, 97610 St. Pierre, Reunion
Mariani, Giuseppe, Ist Sper Cerealicoltura, Via Cassia 176, Roma 00191, Italy
Marks, Michael D., Zoecon Corporation, 975 California Avenue, Palo Alto, CA, 94304, 408-996-9134
Martin, I. F., Research Station, PO Box 27, Kairi, Q 4872, Australia
Martin Sanchez, J. A., Esc T Sup Ingenieros Agron, Carretera de Huesca Km 3, Lerida, Spain, 973-233841
Martineau, Belinda, Department of Genetics, 345 Mulford Hall, University of California, Berkeley, CA, 94720, 415-642-7948
Marton, Csaba Lajos, 2462 Martonvasar, P.O.B. 19, Hungary, 26-45774
Mascarenhas, Joseph P., Department of Biol Sci, State University of New York, Albany, NY, 12222, 518-457-3300
Mascia, P., Cargill Hi-Bred Seed Division, RR 3, Box 751, Aurora, IL, 60506, 312-892-4331
Mashnenkov, A. S., Krasnodar Res Inst Agric, Krasnodar, USSR
Masterson, Robert, Genetics Department, 8 Curtiss Hall, Iowa State University, Ames, IA, 50010, 515-292-3984
Mathur, D. S., 126, NRL, Indian Agr Res Inst, New Delhi, India
Matthews, David L., Agway Inc, Box 4933, Syracuse, NY, 13221, 315-477-6391
Maunder, A. Bruce, Dekalb-Pfizer Genetics, Route 2, Lubbock, TX, 79415, 806-763-3336
Mazoti, Luis B., C. Croce 145, 1832 Lomas de Zamora, Argentina
McBride, A. C., Phytotech, 624 South 775 E, Lafayette, IN, 47905, 317-296-3497
McCarty, Donald R., Vegetable Crops Department, 1255 Fifield Hall, University of Florida, Gainesville, FL, 32611, 904-392-1794
McClain, Eugene F., Dept Agronomy & Soils, Clemson University, Clemson, SC, 29634-0359, 803-656-3102
McClane, John M., P.O. Box 158, Ag. Alumni Seed Imp. Assn., Romney, IN, 47981, 317-538-3145
McClintock, Barbara, Cold Spring Harbor Laboratory, Cold Spring Harbor, NY, 11724, 516-367-8385
McCormick, Sheila, USDA-ARS-PGEC, 800 Buchanan Street, Albany, CA, 94710, 415-559-5906
McCoy, T., Department of Plant Sciences, University of Arizona, Tucson, AZ, 85721, 602-621-1014
McCullough, Andrew, Department of Agronomy, University of Minnesota, St. Paul, MN, 55108, 612-371-1371
McDaniel, R. G., Dept of Plant Sciences, University of Arizona, Tucson, AZ, 85721, 602-261-3357
McElfresh, Kevin C., Department of Genetics, University of Georgia, Athens, GA, 30602, 404-542-7980
McMillin, David E., Department of Biology, Georgia State University, Atlanta, GA, 30303
McMullen, Michael, USDA ARS, Department of Agronomy, OARDC, Wooster, OH, 44691, 216-263-3894
McWhirter, K. S., Dept of Agric Genet & Biometry, University of Sydney, Sydney NSW 2006, Australia, 02-692-2938
Meinke, D., Dept Botany & Microbiology, Oklahoma State University, Stillwater, OK, 74078, 405-624-5559
Mello-Sampayo, Tristao, Centro di Biologia, Inst Gulbenkian de Ciancia, Oeiras, Portugal
Mertens, Thomas R., Dept of Biology, Ball State University, Muncie, IN, 47306, 317-285-1639
Mettin, Dieter, Akad der Wissenschaften DDR, Zentralinstitut fur Genetik, Correnssstrasse 3, 4235 Gatersleben, E Germany
Metzger, Robert, 255 Crops Science, Oregon State College, Corvallis, OR, 97331, 503-757-4449
Michel, Kenneth, Department of Biology, Slippery Rock University, Slippery Rock, PA, 16057, 412-794-7326
Micu, V. E., Moldav Inst Maize & Sorghum, Kriulianskii Raion Krcv-Nova, 278335 Moldavian SSR, USSR
Miernyk, Jan, Northern Regional Res Lab, USDA-ARS, 1815 N. University Street, Peoria, IL, 61604, FTS 360-4011
Migus, Wally, Brandon Research Station, Agriculture Canada, P.O. Box 610, Brandon, Manitoba R7A 5Z5, Canada, 204-728-7234
Miksche, J. P., USDA NPS, Bldg 005, Rm 239, BARC-W, Beltsville, MD, 20705
Mikula, Bernard C., Defiance College, Defiance, OH, 43512, 419-784-4010 EXT 426
Miles, Donald, Tucker Hall, University of Missouri, Columbia, MO, 65211, 314-882-6675
Miranda, L. T. De, Instituto Agronomico, Caixa Postal, 28, 13.100 Campinas SP, Brazil, 0192-420235
Mock, James J., Northrup King Company, 1500 Jackson St NE, PO Box 959, Minneapolis, MN, 55440
Modena, Stephen, Dept. of Crop Science, North Carolina State Univ., Box 7620, Raleigh, NC, 27695, 919-737-2704
Mogford, D. J., Asgrow Research Sta, P.O. Box 152, Bronkhorstpruit 1020, S Africa, (012) 982400
Mohamed, A. H., Department of Biology, University of Missouri, Kansas City, MO, 64110, 816-276-1385
Mohl, Leo, PO Box 825, Colorado Springs, CO, 80901, 303-633-0683
Molina, Marcia del Carmen, Inst Fitotec Santa Catalina, Llavallol, Argentina
Moll, Robert H., Department of Genetics, North Carolina State Univ, Box 7614, Raleigh, NC, 27695-7614, 919-737-2289
Molnar, S. J., Agriculture Canada, Gen Eng Ottawa Res Sta, Ottawa, Ontario K1A 0C6, Canada, 613-995-3700 EXT 313
Moore, Carol W., J910 Microbiology, City Univ New York Med School, 138th Street & Convent Avenue, New York, NY, 10031, 212-690-6926
Mora, G., GIE Pioneer-France, Epuiseau, 41290 Oucques, France
Morocz, Sandor, Cereal Research Institute, Alsokikoto sor 9, Szeged, H-6726, Hungary
Morris, David, Dept Genetics, Iowa State Univ, Ames, IA, 50011, 515-294-4618
Morris, Rosalind, Department of Agronomy, University of Nebraska, Lincoln, NE, 68583-0915, 402-472-1534
Mottinger, John, Department of Botany, University of Rhode Island, Kingston, RI, 02881, 401-792-2625
Mourad, George, 5, Mostafa M. Ismail, Mazarita, Alexandria, Egypt, 011-203-4823745
Muchena, S. C., Ministry of Agriculture, Private Bag 7701, Causeway Harare, Zimbabwe, 706081
Muenchrath, Deborah A., Dept Agron & Plant Genet, Univ Minnesota, 1991 Buford Circle, St. Paul, MN, 55108, 612-625-1208

Mukai, Yasushi, 1-11-10, Tachibana-Higashi, 880 Miyazaki, Japan, 0985-27-1838
Mukherjee, B. K., Cummings Lab, Indian Agr Res Inst, New Delhi 110012, India, 586198
Mulcahy, David, Botany Department, Univ of Massachusetts, Amherst, MA, 01003, 413-545-2238
Mumm, W. J., Box 306, Milford, IL, 60953, 815-889-4550
Murphy, C. F., USDA NPS, Bldg 005, Rm. 239, BARC-W, Beltsville, MD, 20705, 301-344-1560
Murphy, T. C., Northrup King Co, Stanton, MN, 55081, 507-645-5621
Murray, Michael G., Agrigenetics, 5649 E Buckeye Rd, Madison, WI, 53716, 608-221-5000
Muszynski, Michael G., Department of Genetics, 8 Curtiss Hall, Iowa State University, Ames, IA, 50010, 515-292-3984
Myers, Jr, Oval, Dept of Plant & Soil Science, Southern Illinois Univ, Carbondale, IL, 62901, 618-453-2496
Nagle, B. J., Dekalb-Pfizer Genetics, Box 33, Mason City, IL, 62664, 217-482-3213
Narayan, Kishen, Maize Res Station, Amberpet, Hyderabad 500013 A.P., India
Nasser, DeLill S., Eukaryotic Genetics Program, Division of Cell Biology, National Science Foundation, Washington, DC, 20550, 202-357-0112
Natarajan, Gurumurti, Pioneer Seed Co, Ltd, 8-1-39 Toli Chowki, Hyderabad, India 500 008
Natti, Thomas A., Robson Seed Farms Corp, PO Box 270, Hall, NY, 14463, 716-526-5879
Nault, Lowell R, Dept Entomology, Ohio Agr R&D Ctr, Wooster, OH, 44691
Ndegwa, C., P.O. Box 553, Kitale, Kenya, 20941
Nebiolo, Christine M., Dept of Biology, Allegheny College, Meadville, PA, 16335, 814-724-5360
Neely, J. Winston, Cokers Pedigreed Seed Co, Hartsville, SC, 29550
Nel, P. M., Dept of Genetics, Univ of The Witwatersrand, Johannesburg 2001, S Africa, 011-716-2134
Nelson, S. W., CIBA-GEIGY Ltd, PO Box 92, Isando, 1600, S Africa, 011-706-7257
Nelson, Timothy, Department of Biology, Kline Biol Tower, PO Box 6666, Yale University, New Haven, CT, 06511-8112, 203-436-2571
Nelson, Jr., O. E., Department of Genetics, University of Wisconsin, Madison, WI, 53706, 608-262-3344
Nemeth, J., Gabonater Kutatointezet, Szeged, Also Kikoto sor 9, Hungary, 62-17-025
Nesticky, Milan, Maize Research Institute, 917 52 Trnava, Czechoslovakia
Neuffer, M. G., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-7735
Newhouse, Keith, American Cyanamid Co, P.O. Box 600, Princeton, NJ, 08540, 609-799-0400 ext 2471
Newton, K., Dept Biol Sci, University of Missouri, Columbia, MO, 65211, 314-882-4049
Nguyen, Henry T., Dept Plant & Soil Sci, Box 4169, Texas Tech University, Lubbock, TX, 79409-4169, 806-742-2837
Nickerson, N. H., Department of Biology, Tufts University, Medford, MA, 02155, 617-381-3544
Nider, Fabio, Dekalb Argentina Saciai, C.C. 25, 2741 Salto, Buenos Aires, Argentina, 0474-22350
Nieto-Sotelo, Jorge, Department of Biology, Campus Box 1137, Washington University, St. Louis, MO, 63130, 314-889-4632
Nitsch, C., CNRS, Genet Phys Devel Plantes, 91190 Gif-Sur-Yvette, France, 6-907-7828 EXT 387
Noble, Jr., Stephen W., Dept of Corn Breeding, Pioneer Hi-Bred Internat Inc, Johnston, IA, 50131, 515-270-3318
Norman, Penny, 1819 Addison #7, Berkeley, CA, 94704, 415-644-1688
Notani, N. K., Bhabha Atomic Res Centre, Biology Division, Mod Lab, Trombay, Bombay 400085, India, 5510323
Novak, F. J., Czech Acad Sci, Inst Exp Bot, Sokolovska 6 772 00, Olomouc, Czechoslovakia, 068-29521
O'Reilly, Catherine, Sunderland Polytechnic, Biology Department, Chester Road, Sunderland SR1 3SD, U K, 0783-76231
Oaks, Ann, Biology Department, McMaster University, Hamilton, Ontario L8S 4K1, Canada, 416-525-9140 EXT 4377
Oberthur, Elizabeth E., 27, Cytogenetics, Agronomy Department, Iowa State University, Ames, IA, 50010, 515-294-1360
Ochieng, Joseph A. W., NARS-Kitale, P.O. Box 450, Kitale, Kenya
Ogle, Charles W., RR 1, Box 162, Sugar Grove, IL, 60554, 312-466-4742
Ohta, Y., Inst Agr & Forestry, Univ of Tsukuba, Sakura-Mura, Ibaraki-Ken 305, Japan, 0298-53-6691
Oishi, Karen, Molec & Cell Biol Dept, University of Arizona, Tucson, AZ, 85721, 602-621-3381
Okabe, Takashi, Hokkaido Nat Agr Exper Sta, Hitsujigaoka, Sapporo, Japan
Okuno, K., Div Genetics, NIAS, Yatabe, Tsukuba 305, Japan
Openshaw, Stephen J., Agronomy & Plant Genetics, University of Minnesota, St. Paul, MN, 55108
Openshaw, Steve, Dekalb Pfizer Genetics, 3100 Sycamore Road, Dekalb, IL, 60115, 815-756-3671
Osborne, Jane, 509 BioSciences West, Univ Arizona, Tucson, AZ, 85721, 602-621-3381
Osler, R. D., CIMMYT, Apartado Postal 6-641, 06600 Mexico D.F., Mexico, (905) 5-85-43-55
Osterman, John, School of Life Sciences, University of Nebraska-Lincoln, Lincoln, NE, 68588, 402-472-5129
Ottaviano, E., Dip de Biol Sez di Genet, e Microbiol, Via Celoria 26, 20133 Milano, Italy, 02-230823
Paez, Alix V., PO Box 316, Pioneer Hybrid Intern Inc, 6800 Pioneer Pkwy, Johnston, IA, 50131, 515-270-3145
Palmer, Reid, Agronomy Department, Iowa State University, Ames, IA, 50010, 515-294-7378
Palmer, Susan E., Department of Pediatrics, University of Iowa Hospital, Iowa City, IA, 52242
Pan, David, Department of Genetics, University of Wisconsin, Madison, WI, 53706
Pan, Yong-Bao, 714 Pammel Court, Ames, IA, 50010, 515-292-4782
Pareddy, Dayakar R., United AgriSeeds, Inc., P.O. Box 4011, Champaign, IL, 61820
Park, Keun Yong, Crop Experiment Station, Office of Rural Development, Suwon 170, Korea
Pataky, Jerald K., Department of Plant Pathology, N-519 Turner, Univ Ill, 1102 S. Goodwin Avenue, Urbana, IL, 61801, 217-333-3171
Paterniani, Ernesto, Univ Sao Paulo Esc Sup Agr, Luiz de Queiroz, Piracicaba, Brazil
Patterson, Earl, Agron Dept, S-116 Turner Hall, University of Illinois, 1102 S. Goodwin Avenue, Urbana, IL, 61801, 217-333-9641
Paul, Anna-Lisa, Department of Botany, University of Florida, Gainesville, FL, 32611, 904-392-2370
Pauly, Michael H., Rogers Brothers Seed Co., P.O. Box 1069, Nampa, ID, 83653
Pawar, S. E., Biol/Agric Div, Bhabha Atomic Research Center, Trombay Bombay 40085, India, 5514910 ext 2340
Peacock, W. J., Division of Plant Industry, CSIRO, Canberra ACT 2601, Australia, 062-465250
Pearson, Mark L., E. I. Du Pont de Nemours & Co, Cent Res & Develop Dept, Exp Sta, E328/241, Wilmington, DE, 19898, 302-772-2140
Pego, Silas, R. Simoes de Almeida 27, 4700 Braga, Portugal
Pelham, John, Inst of Terrestrial Ecol, Bush Estate, Penicuik, Midlothian EH26 0QB, Scotland
Penny, L., Asgrow Seed Co, 634 E. Lincoln Way, Ames, IA, 50010, 515-232-7170
Pereira, Andy, 27, Cytogenetics, Agronomy Department, Iowa State University, Ames, IA, 50010
Perkins, K. W., Carolina Bio Supply Co, Burlington, NC, 27215, 919-584-0381
Peschke, Virginia M., Dept. Agron. & Plant Genetics, Univ. of Minnesota, St. Paul, MN, 55108
Peters, D. W., O's Gold Seed Co, PO Drawer D, Farmer City, IL, 61842, 309-928-9445
Peterson, Karl, Box 187, Marshalltown, IA, 50158, 515-752-2170
Peterson, Peter A., Dept of Agronomy, Iowa State University, Ames, IA, 50011, 515-294-9652
Peterson, Thomas A., Cold Spg. Harbor Lab, P.O. Box 100, Cold Spg. Harbor, NY, 11724, 516-367-8825
Pfahler, P. L., Agronomy Dept Univ Fl, 304 Newell Hall, Gainesville, FL, 32611, 904-392-6186
Pfister, Walter, Pfister Hybrid Corn Co, El Paso, IL, 61738
Phillips, Ronald, Agronomy & Pl Genetics, University of Minnesota, St. Paul, MN, 55108, 612-373-1366
Phinney, Bernard O., Division of Botany, University of Calif, Los Angeles, CA, 90024, 213-825-3177
Pienaar, R. de V., Dept Genet & Plant Breeding, University of Stellenbosch, 7600 Stellenbosch, S. Africa
Pinter, Lajos, Crop Science Dept, Univ Keszthely, P.O. Box 71, H-8361 Keszthely, Hungary
Plewa, Michael, 302 Environ Res-1005 W Western, University of Illinois, Urbana, IL, 61801, 217-333-3614
Poethig, R. Scott, Biology Department, Leidy Labs/G7, Univ Penn, 415 University Ave, Philadelphia, PA, 19104, 215-898-8915
Poff, Kenneth L., MSU-DOE Plant Res Lab, Michigan State University, East Lansing, MI, 48824-1312
Pogna, N. E., Exp Inst Cer Res, 20079 S. Angelo, Lodigiano, Italy
Pohlman, Bob, Colgate University, Biology Department, Hamilton, NY, 13346, 315-824-1000 EXT 344
Polacco, Mary, Biochemistry, 107 Schweitzer Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-6484
Pollacsek, M., Station D Amelior Des Pl-INRA, 63039 Clermont Ferrand, France, 73-92-45-30
Pollak, Linda, Dept of Agronomy, Iowa State Univ, Ames, IA, 50011, 515-294-3052
Pollmer, W. G., Universitat Hohenheim Lh, Postf 700562-304, Garbenstrasse 9, 7000 Stuttgart 70, W Germany
Polowick, Patricia L., Department of Biology, Univ of Saskatchewan, Saskatoon, Sask. S7N 0W0, Canada, 343-5280
Poneleit, Charles G., Agronomy, University of Kentucky, Lexington, KY, 40546-0091, 606-257-4934
Popow, G., Dubendorfstrasse 38, CH-8602 Wangen (ZH), Switzerland, 01-833-2456
Potts, Yvonne, NPI, 417 Wakara Way, Salt Lake City, UT, 84108, 801-582-0144
Pradhan, K., West Bengal Haringhata Farm, PO Mohanpur, Dist Nadia, West Bengal, India
Pratt, Richard C., Agronomy Department, OSU/OARDC, 1680 Madison Avenue, Wooster, OH, 44691
Prensky, Wolf, Braton Biotechnical Inc, Box 234, Hawthorne, NY, 10532, 914-747-1522
Price, Carl, Waksman Inst, Piscataway, NJ, 08855-0759, 201-932-2920
Price, S. C., Standard Oil Co, 3092 Broadway, Cleveland, OH, 44115, 216-271-8192
Pring, D. R., Dept of Plant Pathology, University of Florida, Gainesville, FL, 32611, 904-392-3638
Pryor, A. J., Div Plant Ind CSIRO, PO Box 1600, Canberra Act 2601, Australia, 062-46-5896

Qin, Minmin, 1630 Lenden Drive, Madison, WI, 53706, 618-263-2098
Qin, Tai-chen, Department of Agronomy, Jiangsu Agriculture College, Jiangsu, Yangchou, China
Quail, Peter H., Department of Botany, University of Wisconsin, 132 Birge Hall, Madison, WI, 53706, 608-262-1057
Raab, Quinton J., Asgrow Seed Company, 205 N. Michigan, Rt. 1 Box 22, Oxford, IN, 47971, 317-385-2201
Raboy, Victor, USDA/ARS, Plant & Soil Science Dept, MT State University, Bozeman, MT, 59717, 406-994-5054
Rabson, Robert, Biological Energy Sciences, Office of Basic Energy Sci, U.S. Dept of Energy, ER-17 GTN, Washington, DC, 20545, 301-353-2873
Racchi, M. L., Univ di Milano, Dept Genetics & Microbiology, Via Celoria 26, 20133 Milan, Italy
Rakha, Farouk A., Dep Genetics Fac Agric, Alexandria University, Alexandria, Egypt, 966386
Ralston, Ed, Advanced Genetic Sciences, 6701 San Pablo, Oakland, CA, 94608, 415-547-2395
Raman, Rajhi, 114, NRL, Indian Agr Res Inst, New Delhi 110012, India, 588783
Ramsey, Ann, RR2, Box 15, Rochester, IL, 62563, 217-498-9374
Rao, Arelli P., 16-11-417/6, Mosarambagh, Hyderabad-500036 AP, India
Rao, P. N., Dept of Botany, Andhra University, Visakhapatnam 530003, India, 64871 ext 390
Reddy, A. R., School of Life Sci, Univ of Hyderabad, Hyderabad-500001, India
Reddy, G. M., Department of Genetics, Osmania University, Hyderabad-500007, India
Reddy, Lekkala V., Biochemistry Department, University of Iowa, Iowa City, IA, 52242, 319-353-7333
Redei, G. P., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-6434
Reed, J. E., Pfizer Genetics, Box 367, Windfall, IN, 46076, 317-945-7121
Rees, Carol, Plant Sciences Dept, Univ of Western Ontario, London, Ontario, Canada, 519-679-2908
Rees, Hubert, Agricultural Botany Dept, Univ College of Wales, Aberystwyth, Wales, U K, 0790-3111
Reid, W. R., O's Gold Seed Company, P.O. Box 460, Parkersburg, IA, 50665
Reksodihardjo, S., Pioneer Agricultura Ltda, Caixa Postal 131, 86100 Londrina PR, Brazil, 0432 271512
Remley, Frank M., Cargill Inc, Seed Div, PO Box 5645, Minneapolis, MN, 55440, 612-890-1852
Reyes, Pedro, Departamento de Agronomi, Sucursal de Correos J., Monterrey, N.L., Mexico, 83-59-24-40
Rhoades, M. M., Biology Dept, Indiana University, Bloomington, IN, 47401, 812-335-9319
Rhodes, C., Calgene Inc, 1920 Fifth St, Suite F, Davis, CA, 95616, 916-753-6313
Riccelli, Mauricio, Apartado Postal 236, Maracay 2101A, Venezuela, 43-23793
Rice, Thomas B., Pfizer Inc, Eastern Point Road, Groton, CT, 06340, 203-441-4576
Riedel, Rod, Genetics Institute, 87 Cambridge Park Drive, Cambridge, MA, 02140, 617-876-1170
Rieffel, Larry D., 598 East 2nd Street, El Paso, IL, 61738, 309-527-2676
Rinke, E. H., 9505 Glen Oaks CR., Sun City, AZ, 85351, 602-933-9646
Rivin, Carol, Dept. of Botany, Oregon State University, Corvallis, OR, 97331, 503-754-4433
Robbelen, G., Inst fur Pflanzenbau, 34 Gottingen, Vonsiebold Strasse 8, W Germany
Roberts, Jean L., Lilly Research Labs, Greenfield, IN, 46140, 317-467-4788
Robertson, Dominique, Dept Plant Breeding, Cornell University, Ithaca, NY, 14853
Robertson, Donald S., Department of Genetics, Iowa State University, Ames, IA, 50010, 515-294-3908
Robichaud, C., Dept of Biology, Yale Univ, New Haven, CT, 06511, 203-432-4636
Robison, Glenn, Dekalb-Pfizer Genetics, Sycamore Road, Dekalb, IL, 60115, 815-756-3671 EXT 265
Rocheford, Torbert, 313 Keim Hall, Department of Agronomy, University of Nebraska, Lincoln, NE, 68583-0915
Rockwell, Bruce H., Box 988, 3600 Chestnut Street, Philadelphia, PA, 19104
Rodermel, S., The Biological Laboratories, Harvard University, 16 Divinity Avenue, Cambridge, MA, 02138, 617-495-4260
Rodrigues, Delmo Diogo, Sem Cargill Ltda, Sitio Sao Jao Barao Geraldo, Caixa Postal, 6553, Campinas SP 13.100, Brazil, 0192-39-13-81
Romero-Severson, Jeanne, Agrigenetics, 5649 E. Buckeye Road, Madison, WI, 53716, 608-221-5000
Roper, J. A., Dept of Genetics, The University, Sheffield 10, U K, 0742-78555
Rossman, E. C., Dept of Crop & Soil Sci, Michigan State University, East Lansing, MI, 48823, 517-355-2289
Roth, Bradley, USDA ARS, Plant Gene Expression Center, 800 Buchanan Street, Albany, CA, 94710, 415-486-3717
Roussell, Deborah L., Div Biol Sci, Tucker Hall, Univ of Missouri, Columbia, MO, 65211, 314-882-8033
Rubenstein, Irwin, Dept Genetics & Cell Biol, University of Minnesota, St Paul, MN, 55108, 612-624-2716
Russell, W. A., Dept of Agronomy, Iowa State University, Ames, IA, 50010
Ryan, G. S., E.E.A. INTA, 5988 Manfredi, (Cordoba), Argentina
Saad, Shawky Farid abooel, SIDS Agriculture Research Stn, Beni-Suef, Egypt
Sachan, J. K. S., 114, NRL Div Genet, IARI, New Delhi-12 110012, India, 588783
Sachdev, Vishal, Dept Biochemistry, Louisiana State University, Baton Rouge, LA, 70803-1806, 504-388-1556
Sachs, M. M., Department of Biology, Campus Box 1137, Washington University, St. Louis, MO, 63130, 314-889-4631
Sadek, Sadek el Shahat, Gemmeiza Agriculture Res Stn, Gharbia, Egypt
Saedler, H., Max-Planck Inst Zuchtungsforsc, Egelspfad, D 5000 Koln 30 Vogelsang, W Germany, 221-5062200
Sager, Ruth, DANA Cancer Ctr, 44 Binney Street, Boston, MA, 02115, 617-732-3380
Saghai, Mohammad, Garst Seed Company, Hwy 210, P.O. Box 500, Slater, IA, 50244, 515-685-3574
Saha, B. C., Agriculture College, PO Sabour Dist Bhagalpur, Bihar, India 813210
Salama, Fawzy A., Nubaria Research Station, El-Naser North Tahrir Sector, El-Ameria, Egypt
Salamini, Francesco, Ist Sper Cerealicoltura, Via Stezzano 24 (PO Box 164), 24100 Bergamo, Italy, 035-248823
Salerno, Juan C., Dept Genetica, INTA, Castelar, C.C. 25, 1712-, Castelar- Buenos Aires, Argentina
Sanchez, F. M., Colegio de Postgraduados, Chapingo, Mexico, 585-0728
Sano, Y., Nat Inst Genetics, Misima, Japan
Santos, M. A., Dep Biol Molec Agro, Centro Invest Desarrollo, 08034 Barcelona, Spain
Saraiva, L. S., Genetica Dept Biol Geral, Univ Fed Vicosa, 36570 Minas Gerais, Brazil
Sarkar, K. R., Room 114, Nuclear Research Lab, Indian Agric Res Inst, New Delhi-12, India
Sarma, J. S. P., Dept of Botany, C.S.R. Sarma College, Ongole-523 001, Andhra Pradesh, India, 2684
Sastry, G. R. K., Dept of Genetics, University of Leeds, Leeds-2, U K, 0532-431751 EXT 598
Sauvaire, D., CIRAD - IRAT, B.P. 910, Bobo-Dioulasso, Burkina Faso, 99-03-57
Saxena, V. K., Dept Plant Breed, Punjab Agric Univ, Ludhiana 141004, India
Sayed, Sayed Galal, PO Box 953, Cairo, Egypt, 847718
Scandalios, J. G., Dept of Genetics, N C State Univ, Box 7614, Raleigh, NC, 27695-7614, 919-237-2291
Schaeffer, Jurgen R., Dept of Plant + Soil Sci, Montana State University, Bozeman, MT, 59715, 406-994-5066
Schell, J., Max-Planck-Institut, 5000 Koln 30, W. Germany
Schertz, K. F., Dept Crop & Soil Sci, Texas A & M Univ, College Station, TX, 77843, 409-260-9252
Schiefelbein, John W., B14 Genetics, 445 Henry Mall, University of Wisconsin, Madison, WI, 53706, 608-262-3287
Schillinger, John, Asgrow Seed Company, 634 E. Lincolnway, Ames, IA, 50010, 515-232-7170
Schmidt, Daria, Department of Agronomy, University of Wisconsin, 1575 Linden Drive, Madison, WI, 53706
Schmidt, Robert J., Biology Department, Building 463, Brookhaven National Lab, Upton, NY, 11973, 516-282-3396
Schnable, Patrick, Max Planck Institute, Egelspfad, D5000 Koln-Vogelsang 30, W Germany
Schnell, F. W., Windhalmweg 19, 7000 Stuttgart 70, W Germany, 0711-453367
Schon, Astrid, Dept Mol Biophys Biochem, 260 Whitney Ave, P.O. Box 6666, New Haven, CT, 06511
Schulman, Alan H., Helsinki University, Department of Genetics, Arkadiankatu 7, 00100 Helsinki, Finland, 011-358-0-402-7286
Schwartz, Drew, Biology Dept, Indiana University, Bloomington, IN, 47401, 812-335-6060
Schwer, Joseph F., Lilly Greenfield Labs, P.O. Box 708, Greenfield, IN, 46140
Schy, W. E., 112 Environ Res-1005 W Western, Univ of Illinois, Urbana, IL, 61801, 217-333-8018
Scoles, Graham J., Crop Sci Dept, Univ of Saskatchewan, Saskatoon S7N 0W0, Canada, 306-343-5708
Scott, Gene E., Agronomy Dept, Box 5248, Mississippi State University, Mississippi State, MS, 39762, 601-325-2311
Sehgal, S. M., Pioneer Hi-Bred Intnl Inc, 6800 Pioneer Pkwy, Johnston, IA, 50131, 515-270-3103
Setter, Tim L., Department of Agronomy, Cornell University, Ithaca, NY, 14853
Sevilla P., Ricardo, Programa de Maiz, Univ Nacional Agraria, Aptdo 456, La Molina, Lima, Peru
Shadley, Jeff D., Illinois State University, Dept Biol Sciences, Normal, IL, 61761, 309-438-2685
Shafer, John, 3031 N Roselawn Drive, Logansport, IN, 46947, 219-753-5547
Shands, Henry, Natl Program Staff, USDA-ARS, Beltsville, MD, 20705, 301-344-3311
Sharma, C. B. S. R., Dept Environ, School Energy Environ Nat Res, Madurai Kamaraj University, Madurai 625021, India
Sharma, N. D., Division of Biochemistry, Indian Agric Res Inst, New Delhi 110012, India
Shaver, D. L., Plant Br Dept, Cornnuts, 24 Winham St, Salinas, CA, 93901, 408-424-1023
Shcherbak, V. S., Krasnodar Res Inst Agric, Krasnodar, USSR
Sheen, Jen, Molecular Biology Dept, Wellman 8, MGH, Boston, MA, 02114, 617-726-5916
Shepherd, Nancy, DuPont E328/246A, Wilmington, DE, 19898, 302-772-1519
Sheridan, W. F., Biology Department, Univ of North Dakota, Grand Forks, ND, 58202, 701-777-4479 or 777-4206
Short, Kent E., CIMMYT, Lisboa 27, Apdo. Postal 6-641, 06600 Mexico, D.F., Mexico

Shortess, David K., Department of Biology, New Mexico Inst M + T, Socorro, NM, 87801, 505-835-5608
Shukla, P. T., Department of Agr Botany, Gujarat Agr University, Junagadh Campus, Junagadh-362001, India, 93 EXT 51
Shure, M., Carnegie Inst Wash, 115 W Univ Parkway, Baltimore, MD, 21210, 301-467-1414
Siddiqui, Khushnood A., Plant Genetics Division, A.E.A.R.C., Tandojam Sind, Pakistan
Sidhu, G., Dept Plant Path, Univ of Nebraska, Lincoln, NE, 68583, 402-472-6024
Simchen, Giora, Department of Genetics, The Hebrew University, Jerusalem 91904, Israel 91904
Simcox, K., Dept Biological Sciences, Purdue University, West Lafayette, IN, 47907
Simon, R. D., Dept of Biology, State Univ Coll A + S, Geneseo, NY, 14454, 716-245-5301
Simonds, S. Caprice, 1253 Fifield Hall, University of Florida, Gainesville, FL, 32611, 904-392-9087
Sims, Lynne E., Pioneer Hi-Bred Internatl, Dept Biotechnology, P.O. Box 38, Johnston, IA, 50131
Sinclair, John, Biology Department, Indiana University, Bloomington, IN, 47405, 812-335-1201
Singh, I. S., Dept of Plant Breeding, G. B. Pant Univ, Agr & Tech, Pantnagar, Nainital, India
Singh, Joginder, Cereal Research Lab, Indian Agricultural Res Inst, New Delhi 110012, India, 582372
Singh, S. V., Dept of Genetics and Pl Breed, Institute of Agric Sciences, Banaras Hindu University, Varanasi-221005 (U.P.), India
Singleton, Katya, 243 Smith Street, Apartment 1704, Providence, RI, 02908
Sisco, P. H., Dept Crop Science, North Carolina State Univ, P.O. Box 7620, Raleigh, NC, 27695-7620, 919-737-2704
Skogen-Hagenson, Mary Jane, Department of Genetics, Rm. 10 Curtiss Hall, Iowa State University, Ames, IA, 50011, 515-294-4618
Smeltzer, Dale, The Rockefeller Found, GPO Box 2453, Bangkok, Thailand
Smith, James D., PO Box 2132, Department Soil & Crop Sci, Texas A & M University, College Station, TX, 77843, 409-845-8276
Smith, Jane, ENI-Chem Americas, 2000 Princeton Park, Corporation Center, Monmouth Junction, NJ, 08852
Smith, Oscar S., Pioneer Hi-Bred Internatl, Plant Breeding Division, P.O. Box 85, Johnston, IA, 50131, 515-270-3300
Smith, S. E., Dept Plant Sciences, University of Arizona, Tucson, AZ, 85721
Smith, Steve, Pioneer Hi-Bred Internatl, Plant Breeding Division, 7301 N.W. 62nd Avenue, Johnston, IA, 50131, 515-270-3353
Snape, John W., Plant Breeding Institute, Maris Lane, Trumpington, Cambridge CB2 2LQ, UK, 0223-840411
Snope, Andrew, Div of Science + Math, Essex Community College, Essex, MD, 21237, 301-522-1280
Snyder, John R., United Agriseeds, 103 Tomaras, Savoy, IL, 61874, 217-373-5300
Soave, C., Instituto Biologia Agraria, Univ. della Basilicata, 85100 Potenga, Italy
Soledad, S. V., Tagum, Northdavao 9401, Philippines
Soll, Dieter, Dept Molec Biophys & Biochem, 260 Whitney Avenue, Box 6666, Yale University, New Haven, CT, 06511
Somers, David A., Dept Agron & Plant Genet, University of Minnesota, St. Paul, MN, 55108, 612-373-1373
Song, Maoshan, Shanxi Acad Agric Sci, Wugong, Shanxi, China
Soong, Tom, Monsanto, GG4I, 700 Chesterfield Village Pkwy, St. Louis, MO, 63198, 314-537-6601
Sorenson, John C., Experimental Ag Sci, The Upjohn Co, Kalamazoo, MI, 49001, 616-385-4582
Sorrentino, Jennifer, Cold Spring Harbor Lab, Cold Spring Harbor, NY, 11724
Sprague, G. F., Dept Agron, S-120 Turner Hall, University of Illinois, 1102 S. Goodwin Avenue, Urbana, IL, 61801, 217-333-4254
Springer, Patricia, Dept Biol Sci, Purdue University, W. Lafayette, IN, 47907
Springer, Warren, Northrup King Company, Stanton, MN, 55081, 507-645-5621
Srinivasan, C., HyClone, Inc, P.O. Box 3190, Controe, TX, 77305, 409-756-0520
Stack, Stephen, Department of Botany, Colorado State University, Fort Collins, CO, 80523
Stadler, Joan, 8 Curtiss Hall, Genetics Dept, Iowa State University, Ames, IA, 50011, 515-294-0337
Stark, Frank, Illinois Foundation Seeds Inc, 6959 River Road, Deforest, WI, 53532, 608-846-3066
Starlinger, P., Inst Genetik, Univ Koln, D-5000 Koln 41, Weyertal 121, W Germany, 221-470-2465
Start, William G., Plant Genetics Department, Pfizer Central Research, Groton, CT, 06340, 203-441-4766
Staub, Rick W., Dept of Biology, Carleton College, Northfield, MN, 55057, 507-663-4394
Steffensen, D. M., 515 Morrill Hall, Univ of Illinois, Urbana, IL, 61801, 217-333-3087
Stein, Otto L., Dept of Botany, Univ of Massachusetts, Amherst, MA, 01003, 413-545-0861
Stern, Herbert, Dept of Biology, Univ of Calif-San Diego, La Jolla, CA, 92037, 619-452-3746
Stiles, J. I., Dept. of Botany, Univ. Hawaii at Manoa, Honolulu, HI, 96822
Still, Gerald, Western Regional Res Ctr, 800 Buchanan Street, Berkeley, CA, 94710
Stinard, Philip, Dept Genetics, Iowa State Univ, Ames, IA, 50010
Stout, John T., Upjohn Company, 9602-18-2, Kalamazoo, MI, 49001, 616-385-4583
Strissel, Jerry, Wilson Hybrids, Inc, P.O. Box 391, Harlan, IA, 51537, 712-755-3841
Strommer, Judith, Dept of Genetics, Univ of Georgia, Athens, GA, 30602, 404-542-7980
Stroup, D., 4740 Conn. Ave. NW #613, Washington, DC, 20008
Stuber, Charles W., Dept of Genetics, North Carolina State Univ, Box 7614, Raleigh, NC, 27695-7614, 919-737-2289 FTS672-4564
Stucker, Robert, Dept of Agron + Pl Gen, University of Minnesota, St Paul, MN, 55108
Styles, E. D., Biology, Univ of Victoria, Victoria, British Columbia V8W 2Y2, Canada, 604-721-7101
Sundaresan, V., Cold Spring Harbor Lab, P.O. Box 100, Cold Spring Harbor, NY, 11724
Sundberg, Marshall D., Department of Botany, 508 Life Science Bldg, Louisiana State University, Baton Rouge, LA, 70803-1705, 504-388-8563
Sung, Tong Ming, Dept of Agronomy, Beijing Agric Univ, Beijing, China, 28-5831
Suprasanna, P., Department of Genetics, Osmania University, Hyderabad-500 007, A.P., India
Sussex, I. M., Dept Biol, PO Box 6666, Yale University, New Haven, CT, 06511, 203-432-4636
Swaminathan, M. S., International Rice Res Inst, Los Banos, Philippines
Tadic, Bojana, Maize Research Institute, P.O. Box 89, 11081 Zemun, Yugoslavia
Talbert, Luther, Institute of Molecular Biology, University of Oregon, Eugene, OR, 97402, 503-686-5136
Tang, Ching-Yan, Maize Research Program, IITA, Oyo Road, PMB 5320, Ibadan, Nigeria
Tantravahi, R. V., Sub Exp Sta, Univ Mass, 240 Beaver Street, Waltham, MA, 02154
Taylor, Brian H., CSIRO, Division Plant Industry, Clunies Ross St, Acton, ACT, Australia
Taylor, Loverine P., Dept Plant Biol, Carnegie Inst Wash, 290 Panama Street, Stanford, CA, 94305, 415-325-1521
Taylor, W. C., CSIRO, Division of Plant Industry, P.O. Box 1600, Canberra ACT 2601, Australia
Thompson, D., 191 Sumner Blvd, Collinsville, IL, 62234, 618-345-0426
Thompson, G. A., Dept Bot & Plant Pathol, Lilly Hall, Purdue Univ, W. Lafayette, IN, 47907
Thompson, Jack C., FFR Coop, P.O. Box 10, Providence Forge, VA, 23140, 804-966-2888
Thompson, M. E., Medical Department, Brookhaven National Lab, Upton, NY, 11973
Thompson, Steven A., United Agriseeds, 103 Tomaras, Savoy, IL, 61874, 217-373-5300
Thornbury, David W., S-305 Ag Science Center N, Dept Plant Path, University of Kentucky, Lexington, KY, 40546, 606-257-2681
Tierney, Mary L., Biotechnology Center, Rightmire Hall, 1060 Carmack Road, Columbus, OH, 43210-1002, 614-422-5670
Timothy, David H., Dept of Crop Science, North Carolina State Univ, Box 7620, Raleigh, NC, 27695-7620, 919-834-5161
Ting, Chu-Po, Shandong Agricultural College, Taian, Shandong, China
Ting, Yu-Chen, Biology, Boston College, Chestnut Hill 67, Boston, MA, 02167, 617-969-0100 EXT 3551
Tintorri, John A., 53 Hampshire Drive, Mendham, NJ, 07945
Tomov, Nicola, Institute for Maize, Kneja, Bulgaria
Tonelli, Chiara, Dept of Genetics & Microbiol, Univ Milano, Via Celoria 26, 20133 Milano, Italy, 02-230823
Tracy, W. F., Department of Agronomy, University of Wisconsin, Madison, WI, 53706, 608-262-1390
Treat, Calvin, Department of Agronomy, University of Wisconsin, 1575 Linden Drive, Madison, WI, 53706
Troyer, A. F., Dekalb-Pfizer Genetics, 3100 Sycamore Road, Dekalb, IL, 60115, 815-758-3461
Tsai, C. Y., Dept Bot Plant Pathol, Purdue University, W. Lafayette, IN, 47907, 317-494-4640
Tsuchiya, T., Dept of Agronomy, Colorado State University, Fort Collins, CO, 80523, 303-491-6648 EXT 6237
Uchimiya, H., Inst Biol Sciences, Univ Tsukuba, Sakura-Mura, Ibaraki-Ken, Japan, 0298-51-1384
Ukai, Yasuo, Inst Radiation Breed, Nias Maff, Ohmiya-Machi, Naka-Gun, Ibaraki 319-22, Japan, 02955-3-0807
Ulrich, Y., Div Plant & Soil Sci, West Virginia Univ, Morgantown, WV, 26506, 304-293-6258
Umbeck, P. F., Agracetus Corp, 2208 Parview Road, Middleton, WI, 53562, 608-836-7300
Vallejos, Ruben H., CEFOBI, Suipacha 531, 2000 Rosario, Argentina
Van Der Walt, W. J., c/o Asgrow South Africa (Pty), P.O. Box 912-653, Silverton 0127, S Africa
Van Schaik, N., Dept Genetics/U Witwatersrand, 1 Jan Smuts Avenue, Johannesburg 2001, S Africa, (011) 716-2125
Varagona, Rita, Department of Botany, University of Georgia, Athens, GA, 30605, 404-542-3732
Varner, Joseph E., Department of Biology, Washington University, St. Louis, MO, 63130, 314-889-6803
Vasal, S. K., Londres 40 1 Piso, Apartado Postal 6-641, Mexico 6, D.F., Mexico, 5-85-43-55 EXT 115
Vasil, Indra K., Dept Botany, 220 Bartram Hall, Univ Florida, Gainesville, FL, 32611, 904-392-1193
Vega, Pedro C., Instituto de Genetica, Univ Central Venezuela, Maracay, Venezuela
Veit, Bruce, USDA ARS PGEC, 800 Buchanan Street, Albany, CA, 94710, 415-559-5907
Verma, S. C., Botany Department, Panjab University, Chandigarh-160014, India
Vetturini, Mario, Via Marco Polo, 48, 40138 Bologna, Italy, 051 364181

Vierling, Richard, Plant and Soil Science, Box 4169, Lubbock, TX, 79409-4169
Vineyard, Marvin L., 695 Dogwood Drive, Arcola, IL, 61910, 217-268-3426
Violic, Alejandro D., CIMMYT, Apartado Postal 6-641, Mexico 6, D.F., Mexico, 5854355
Viotti, Angelo, Ist Biosintesi Vegetali, Via Bassini 15, 20133 Milano, Italy
Vodkin, Lila Ott, Dept Agron, S-110 Turner Hall, Univ Illinois, 1102 S. Goodwin, Urbana, IL, 61801
Vojnikov, V., Siber Inst Pl Phys & Biochem, USSR Academy of Sciences, P.O. Box 1243, Irkutsk-33, 664033, USSR
Volk, B., Agronomy Dept, 135 Mumford, Univ of Missouri, Columbia, MO, 65211, 314-882-2801
Von Bulow, J. F. W., Univ Brasilia, Cx Postal 15-2958, 70.910 Brasilia DF, Brazil, 274-0022 r.2429
Von Wettstein, D., Carlsberg Lab, Gamle Carlsbergvej 10 DK-2500, Copenhagen Valby, Denmark, 01-221022
Voris, Merle, Box 457, Windfall, IN, 46076, 317-945-7777
Vosa, Canio G., Botany School, Oxford University, Oxford OS1 3RA, U K, 53391 EXT 233
Vulchinkov, Stefan, Maize Research Institute, Kneja, Bulgaria
Wagner, Tom, Rt 2, Wathena, KS, 66090
Waines, J. Giles, Dept of Botany/Plant Sciences, University of California, Riverside, CA, 92502, 714-787-3706
Walbot, V., Dept Biol Sci, Stanford Univ, Stanford, CA, 94305, 415-723-2227
Walburg, Greg, Div Biol Sci, 108 Tucker Hall, Univ Missouri, Columbia, MO, 65211
Walden, D. B., Dept of Plant Sciences, Univ of Western Ontario, London N6A 5B7, Canada, 519-679-6757
Walker, John, Biol Sci, Univ of Missouri, Columbia, MO, 65211
Wallace, John C., Dept Botany & Plant Pathol, Purdue University, W. Lafayette, IN, 47907, 317-494-5877
Waller, Jerome H., Berea College, C P O Box 2325, Berea, KY, 40404
Walton, Jonathan, DOE Plant Research Lab, Michigan State University, East Lansing, MI, 48824, 517-353-4885
Wang, Lai-Weng, Institute of Maize, Shandong Acad Agric Sci, Jinan, Shandong Province, China
Wang, Mao-Hua, Dept Agronomy, Henan Agric College, Zhengshou, Henan Province, China
Wang, Yunxia, Department of Botany, 220 Biological Science Center, University of Minnesota, St. Paul, MN, 55108, 612-376-2648
Warren, Herman L., Dept Bot Plant Pathol, Purdue University, W. Lafayette, IN, 47907, 317-494-4630
Wassom, Clyde E., Agronomy Dept, Kansas State Univ, Manhattan, KS, 66506, 913-532-7253
Weber, David, Dept of Biological Sci, Illinois State Univ, Normal, IL, 61761, 309-438-2685
Weber, Evelyn J., USDA-ARS, Univ of Illinois, S-320 Turner Hall, 1102 S. Goodwin Avenue, Urbana, IL, 61801, 217-333-1117
Weck, Edward A., Dept Biol Sci, Purdue University, W. Lafayette, IN, 47907
Wei, Jia-Kun, Hebei Agric Reclaim Inst, Tangshan, Hebei Province, China
Wei, Lai-fan, Teacher's Office, Yantai Agricultural School, Yantai, Shandong Province, China
Weiser, Glen C., Edisto Experiment Station, P.O. Box 247, Blackville, SC, 29817
Weissinger, Arthur, Pioneer Hi-Bred Internatl, 7300 N.W. 62nd Avenue, Johnston, IA, 50131, 515-270-3647
Wendel, J. F., Department of Botany, Bessey Hall, Iowa State University, Ames, IA, 50011, 515-294-7172
Werr, Wolfgang, Institut fur Genetik, Universitat zu Koln, Weyertal 121, 5000 Koln 41, W. Germany, 0221-470-4580
Wessells, Catherine P. B., 106 Peter Coutts Circle, Stanford, CA, 94305, 415-497-2609
Wessler, Sue, Dept of Botany, Univ of Georgia, Athens, GA, 30602, 404-542-3732
West, D. P., Research Dept, Jacques Seed Company, Prescott, WI, 54021, 715-262-3162
West, Dennis R., Dept Plant and Soil Sci, Univ of Tennessee, Knoxville, TN, 37901, 615-974-7391
Whalen, R. H., Dept of Biology, South Dakota State Univ, Brookings, SD, 57007, 605-688-6141
Whitwood, W., Musser Seed Co., P.O. Box 787, Caldwell, ID, 83605, 208-459-0243
Wicks, Z. W., Dept Plant Sci, South Dakota State Univ, Brookings, SD, 57007, 605-688-5156
Widholm, J. M., Dept of Agronomy, U Illinois/1102 S. Goodwin, Urbana, IL, 61801, 217-333-1279
Widstrom, Neil W., Coastal Plain Exp Sta, PO Box 748, Tifton, GA, 31793, 912-382-6904
Wienand, Udo, Max Planck Institute, Egelspfad, D-5000 Koln-Vogelsang 30 , W Germany
Wiesmeyer, Herbert, Dept Molecular Biol, Vanderbilt University, Nashville, TN, 37235, 615-322-4737
Wilcox, Wesley C., Funk Seeds Internat, 1300 West Washington St, Bloomington, IL, 61701, 309-829-9461
Wilkes, H. Garrison, Biology-College II, Univ of Mass/Boston, Boston, MA, 02125-3393, 617-929-8448
Williams, A. F., Rt 4, Box 172, Mitchell, IN, 47446, 812-388-6385
Williams, Robert E., Columbiana Seed Company, PO Box 294, Pittsfield, IL, 62363, 217-285-2530
Williams, Terrill E., Pioneer Hi-Bred Internatl, P.O. Box 426, Windfall, IN, 46076, 317-945-8217
Williams, W. P., Crop Sci Res Lab, PO Box 5248, Miss. State, MS, 39762, 601-325-2311
Willman, Mark R., Beatrice/Hunt-Wesson Inc, 463 U.S. Hwy. 30 East, Valparaiso, IN, 46383
Wilmarth, C. E., Cargill Research, 515 River Ave North, Belmond, IA, 50421, 515-444-4488
Wilson, Curtis M., USDA ARS NRRC, 1815 North University Street, Peoria, IL, 61604
Wilson, Martin, Garst Seed Company, Hwy 210, P.O. Box 500, Slater, IA, 50244, 515-685-3574
Wise, Roger, Max-Planck-Institut Zuchtung., D-5000 Koln 30, West Germany
Wong, James R., DuPont Co, Agricultural Products, Experimental Stn 402/4235, Wilmington, DE, 19898
Woodman, James C., Molec Genetics Inc, 10320 Bren Road East, Minnetonka, MN, 55343, 612-935-7335
Wozniak, Chris A., 348 Manter Hall, Biological Sciences, University of Nebraska, Lincoln, NE, 68588-0118
Wright, Allen, Dept Agron, Curtis Hall, Univ Missouri, Columbia, MO, 65211
Wright, D., Brooklyn Botanic Garden, 712 Kitchawan Road, Ossining, NY, 10562
Wright, Steven, Dairyland Res Internat, R.R. #1, Box 51, Clinton, WI, 53525
Wurtzel, Eleanore, Dept Biol Sci, Davis Hall, Lehman College, City Univ New York, Bronx, NY, 10468, 212-960-8483
Xie, Daohong, Jilin Acad Agric Sci, Gongzhuling, Jilin, China
Xie, You-Ju, Dept of Agronomy, Beijing Agricultural Univ, Beijing, China
Xu, Qifeng, Dept of Agronomy, Beijing Agric Univ, Beijing, China
Xu, Wenwei, Inst Crop Germplasm Resources, Chinese Acad Agr Sci, No. 30 Bai Shi Qiao Road, Beijing 100081, China
Yabuno, T., Yamanouchi Moto Machi 13-2, Sumiyoshi-Ku, Osaka 558, Japan
Yaklich, Robert W., Seed Research Lab, PGGI, B-006, Rm 103, BARC-W, Beltsville, MD, 20705, 301-344-2491
Yamada, Minoru, Dept. Cell Biology, NIAR,, Kan-Nondai 1-2-1,, Yatabe,, Tsukuba Ibaraki 305, Japan, 02975-6-8372
Yan, Tang Ching, CIMMYT, Londres 40, Apdo Postal 6-641, 06600 Mexico, D.F., Mexico
Yates, Don, Downing Foundation Seeds, Inc, 2380 Harrison Road, New Madison, OH, 45346, 513-997-3813
Yatou, Osamu, Institute of Radiation Breed, P.O. Box 3, Ohmiya-machi, Naka-gun, Ibaraki-ken, 319-22, Japan
Yi, Bu-young, Inst Biological Chemistry, Washington State University, Pullman, WA, 99164-6340, 509-335-3956
Yoder, O. C., Dept Plant Path, Cornell Univ, Ithaca, NY, 14853, 607-256-3243
Yong, H. S., Dept Genetics, University of Malaya, Kuala Lumpur, Malaysia
Yu, Ming H., Agr Res Service, PO Box 5098, Salinas, CA, 93915, 408-443-2253
Zabala, Gracia, Plant Biol/289 Morrill Hall, Univ Illinois, 505 S. Goodwin Avenue, Urbana, IL, 61801
Zabulionis, Ray, Department of Plant Sci, B & G Building, University of Western Ontario, London, Ontario N6A 5B7, Canada
Zainal-Abidin, M. I., Dept Agron/Hort, Univ Agr Malaysia, Serdang, Selangor, Malaysia, 356101-326
Zanoni, Urs, S11 Turner Hall, University of Illinois, 1102 S. Goodwin, Urbana, IL, 61801
Zarowitz, Michael, IPRI, 830 Bransten Road, San Carlos, CA, 94070, 415-595-5335
Zeng, M. Q., Institute of Genetics, Academia Sinica, Beijing, China, 446551-556
Zhao, Ji-ping, Department of Agronomy, Shandong Agric University, Taian, Shandong Province, China
Zhao, Zuo-Yu, Department of Biological Sci, Illinois State University, Normal, IL, 61761, 309-438-3669
Zidwick, Mary Jo, Cargill Research Department, Box 9309, Minneapolis, MN, 55440
Zimmer, Elizabeth, Department of Biochemistry, 322 Choppin Hall, Louisiana State University, Baton Rouge, LA, 70803, 504-388-1556
Zimmering, Stanley, Dept of Biology, Brown University, Providence, RI, 02912
Zuber, M. S., Curtis Hall, University of Missouri, Columbia, MO, 65211, 314-882-7707

1. Adedzwa, D. K., El-Rouby, M. M., 1986. Estimates of genetic and environmental parameters for yield components and protein content in a composite population of maize. Samaru J. Agric. Res. 4:85-92.
2. Adeyemi, I. A., Osunami, A. T., Fakorede, M. A. B., 1987. Effect of corn varieties on ogi quality. J. Food Sci. 52:322-324.
3. Ahnert, D., Saraiva, L. S., 1987. Analysis of the phenomenon of preferential segregation involving a 9^B translocation in maize (Zea mays L.). Rev. Brasil. Genetica 10:75-86.
4. Akita, S., Mochizuki, N., Yamada, M., Tanaka, I., 1986. Variations of heterosis in leaf photosynthetic activity of maize (Zea mays L.) with growth stages. Jpn. J. Crop Sci. 55:404-407.
5. Albrecht, B., Dudley, J. W., 1987. Divergent selection for stalk quality and grain yield in an adapted x exotic maize population cross. Crop Sci. 27:487-494.
6. Albrecht, B., Dudley, J. W., 1987. Evaluation of 4 maize populations containing different proportions of exotic germplasm. Crop Sci. 27:480-486.
7. Alexander, R. J., 1987. Corn dry milling: processes, products, and applications. Pp. 351-376 in S. A. Watson and P. E. Ramstad, eds., (which see).
8. Ali, M. L., Tepora, N. M., 1986. Comparative performance of four types of testers for evaluating corn inbred lines from two populations. Philipp. J. Crop Sci. 11:175-180.
9. Ampofo, J. K. O., 1986. Maize stalk borer (Lepidoptera: Pyralidae) damage and plant resistance. Environ. Entomol. 15:1124-1129.
10. Ampofo, J. K. O., Kidiavai, E. L., 1987. Chilo partellus (Swinhoe)(Lepid, Pyralidae) larval movement and growth on maize plants in relation to plant age and resistance or susceptibility. J. Appl. Entomol. 103:483-488.
11. Ampofo, J. K. O., Saxena, K. N., Kibuka, J. G., Nyangiri, E. O., 1986. Evaluation of some maize cultivars for resistance to the stem borer Chilo partellus (Swinhoe) in western Kenya. Maydica 31:379-389.
12. Anderson, P., 1986. Cell culture selection of herbicide tolerant corn. Proc. Annu. Corn Sorghum Res. Conf. 41:48-55.
13. Anderson, R. C., 1987. Growth of eastern gamagrass (Tripsacum dactyloides L.)(Poaceae) under various inorganic nutrient conditions. Bull. Torrey Bot. Club 114:46-52.
14. Angelov, K., Lalova E., 1987. Growth period, grain moisture content and productivity of multi-ear inbred maize lines. Rasteniev'd Nauki 24:3-9.
15. Apostolakos, P., Galatis, B., 1987. Induction, polarity and spatial control of cytokinesis in some abnormal subsidiary cell mother cells of Zea mays. Protoplasma 140:26-42.
16. Aranda, G., Durlin, P., Gauvrit, C., 1987. Methomyl analogues with increased biological activity towards F_7T maize mitochondria. Phytochemistry 26:1909-1914.
17. Armond, P. A., Orr, P. M., 1987. Protogyny in Zea mays. U.S. Patent 4677246, June 30, 1987. Off. Gaz. U.S. Pat. Trademark Off. Pat. 1079:2876.
18. Ashcroft, R. G., Preston, C., Cleland, R. E., Critchley, C., 1986. Flow cytometry of isolated chloroplasts and thylakoids. Photobiochem.-Photobiophys. 13:1-14.
19. Ashraf, M., Vasil, V., Vasil, I. K., Ferl, R. J., 1987. Chromatin structure at the 5' promoter region of the maize Adh2 gene and its role in gene regulation. Mol. Gen. Genet. 208:185-190.
20. Aslam, M., Chaudhri, M. Y., Khan, A. R., 1985. Combining ability of maize (Zea mays) inbred lines. Pak. J. Agric. Res. 6:168-170.
21. Avato, P., 1987. Chemical genetics of epicuticular wax formation in maize. Plant Physiol. Biochem. 25:179-190.
22. Avato, P., Bianchi, G., Nayak, A., Salamini, F., Gentinetta, E., 1987. Epicuticular waxes of maize as affected by the interaction of mutant gl8 with gl3, gl4 and gl15. Lipids 22:11-16.
23. Baduapraku, B., Gracen, V. E., Bergstrom, G. C., 1987. A major gene for resistance to anthracnose leaf blight in maize. Plant Breed. 98:194-199.
24. Baduapraku, B., Gracen, V. E., Bergstrom, G. C., 1987. A major gene for resistance to anthracnose stalk rot in maize. Phytopathology 77:957-959.
25. Baduapraku, B., Gracen, V. E., Bergstrom, G. C., 1987. Inheritance of resistance of anthracnose stalk rot and leaf blight in a maize inbred derived from a temperate by tropical germplasm combination. Maydica 32:221-138.
26. Bagby, M. O., Widstrom, N. W., 1987. Biomass uses and conversion. Pp. 575-590 in S. A. Watson and P. E. Ramstad, eds., (which see).
27. Bailey, B. A., Schuh, W., Frederiksen, R. A., Bockholt, A. J., Smith, J. D., 1987. Identification of slow-rusting resistance to Puccinia polysora in maize inbreds and single crosses. Plant Dis. 71:518-521.
28. Baileyserres, J., Isaac, P. G., Small, I. D., Leaver, C. J., 1987. Mitochondrial genome rearrangements associated with the cytoplasmic male sterile phenotype in maize and sorghum. Pp. 53-72 in Developmental Mutants in Higher Plants, H. Thomas and D. Grierson, eds., Cambridge U. Press, Cambridge.
29. Baker, B., Coupland, G., Fedoroff, N. V., Starlinger, P., Schell, J., 1987. Phenotypic assay for excision of the maize controlling element Ac in tobacco. EMBO J. 6:1547-1554.
30. Baluska, F., Kubica, S., 1987. Changes in chromatin condensation during growth and differentiation of maize primary root cells. Biologia 42:9-16.
31. Baluska, F., Kubica, S., 1987. DNA content, nucleus size and chromatin structure in cells of the maize primary root. Biologia 42:409-418.
32. Barber, S. A., 1986. Root distribution and mineral uptake as influenced by hybrids, environment and fertilizer. Proc. Annu. Corn Sorghum Res. Conf. 41:56-69.
33. Barfield, C. S., Ashley, T. R., 1987. Effects of corn phenology and temperature on the life cycle of the fall armyworm, Spodoptera frugiperda (Lepidoptera: Noctuidae). Fla. Entomol. 70:110-116.
34. Barlow, P. W., 1987. Cellular packets, cell division and morphogenesis in the primary root meristem of Zea mays L. New Phytol. 105:27-56.
35. Barnabas, B., Fransz, P. F., Schel, J. H. N., 1987. Ultrastructural studies on pollen embryogenesis in maize (Zea mays L.). Plant Cell Rep. 6:212-215.
36. Baron, V. S., Daynard, T. B., Dupuis, G., Fairey, N. A., Major, D. J., Muldoon, J. F., White, R. P., 1987. Evaluation of early-maturing European and Canadian corn hybrids for grain and forage production in Canada. Maydica 32:33-48.
37. Barriere, Y., Gallais, A., Derieux, M., Panouille, A., 1987. Whole-plant value at harvest for silage of grain maize cultivars bred between 1950 and 1980. Agronomie 7:73-80.
38. Barry, D., Lillehoj, E. B., Widstrom, N. W., McMillian, W. W., Zuber, M. S., Kwolek, W. F., Guthrie, W. D., 1986. Effect of husk tightness and insect (Lepidoptera) infestation on aflatoxin contamination of preharvest maize. Environ. Entomol. 15:1116-1118.
39. Bassi, R., Simpson, D. J., 1986. Differential expression of LHCII genes in mesophyll and bundle sheath cells of maize. Carlsberg Res. Commun. 51:363-370.
40. Baszczynski, C. L., 1986. Immunochemical analysis of heat-shock protein synthesis in maize (Zea mays L.). Can. J. Genet. Cytol. 28:1076-1087.
41. Baszczynski, C. L., Kemble, R. J., 1987. Terminal protein association and sequence homology in linear mitochondrial plasmid-like DNAs of sorghum and maize. Plant Cell Rep. 6:127-130.
42. Beck, D. L., Darrah, L. L., Zuber, M. S., 1987. An improved technique for measuring resistance to root pulling in maize. Crop Sci. 27:356-358.
43. Bedinger, P., Hostos, E. L. de, Leon, P., Walbot, V., 1986. Cloning and characterization of a linear 2.3 kb mitochondrial plasmid of maize. Molec. Gen. Genet. 205:206-212.
44. Bell, R. R., 1986. Performance of hybrids as related to seedstock sources. Proc. Annu. Corn Sorghum Res. Conf. 41:160-170.
45. Belousov, A. A., 1987. Genetic analysis of modified endosperm texture in opaque-2 maize. Genetika 23:677-685.
46. Bennetzen, J. L., 1984. Genetic engineering for improved crop disease resistance. Pp. 491-524 in Applications of Genetic Engineering to Crop Improvement, G. B. Collins and J. G. Petolino, eds., Martinus Nijhof/Dr. W. Junk, Dordrecht.
47. Bennetzen, J. L., 1987. Covalent DNA modification and the regulation of Mutator element transposition in maize. Mol. Gen. Genet. 208:45-51.
48. Bennetzen, J. L., Cresse, A., Brown, W. E., Lee, L., 1987. Molecular cloning of maize genes by transposon tagging with mutator. Pp. 183-206 in Plant Gene Systems and Their Biology, J. L. Key and L. McIntosh, eds., Liss, New York.
49. Bennetzen, J. L., Fracasso, R. P., Morris, D. W., Robertson, D. S., Skogen-Hagenson, M. J., 1987. Concomitant regulation of Mul transposition and Mutator activity in maize. Mol. Gen. Genet. 208:57-62.
50. Benson, G. O., Pearce, R. B., 1987. Corn perspective and culture. Pp. 1-29 in S. A. Watson and P. E. Ramstad, eds., (which see).
51. Berriere, Y., Gallais, A., Derieux, M., Panouille, A., 1987. Whole-plant value at harvest for silage of grain maize cultivars bred between 1950 and 1980. Agronomie 7:73-80.
52. Bethards, L. A., Skadsen, R. W., Scandalios, J. G., 1987. Isolation and characterization of a cDNA clone for the Cat2 gene in maize and its homology with other catalases. Proc. Nat. Acad. Sci. USA 84:6830-6834.
53. Bino, R. J., Suurs, L. C. J. M., Hoop, S. J. d., Van Der Neut, A., Van Went, J. L., Van Marrewijk, G. A. M., 1986. Characterization of cytoplasmic male sterility in Petunia hybrida and Zea mays. Localization and activity of cytochrome c oxidase. Euphytica 35:905-918.
54. Birchler, J. A., Hart, J. R., 1987. Interaction of endosperm size factors in maize. Genetics 117:309-317.
55. Bird, R. McK., Neuffer, M. G., 1987. Induced mutations in maize. Pp. 139-180 in Plant Breeding Reviews, Vol. 5, J. Janick, ed., Van Nostrand Reinhold, New York.
56. Boetz, C., Panfil, C., 1986. The graphical analysis of genetic variance: modern method for genetic potential characterization in dihaploid lines of maize. Bul. Inst. Agron. Cluj-Napoca Ser. Agric. 40:53-58.
57. Bommineni, V. R., Greyson, R. I., 1987. In vitro culture of ear shoots of Zea mays and the effect of kinetin on sex expression. Amer. J. Bot. 74:883-890.
58. Bonham-Smith, P. C., Kapoor, M., Bewley, J. D., 1987. Establishment of thermotolerance in maize by exposure to stresses other than a heat shock does not require heat shock protein synthesis. Plant Physiol. 85:575-580.
59. Borges, O. L., 1987. Diallel analysis of maize resistance to sorghum downy mildew. Crop Sci. 27:178-180.
60. Borner, T., Mendel, R. R., Schiemann, J., 1986. Nitrate reductase is not accumulated in chloroplast-ribosome-deficient mutants of higher plants. Planta 169:202-207.
61. Boronat, A., Martinez, M. C., Reina, M., Puigdomenech, P., Palau, J., 1986. Isolation and sequencing of a 28 kD glutelin-2 gene from maize. Common elements in the 5' flanking regions among zein and glutelin genes. Plant Sci. 47:95-102.

62. Bosabalidis, A. M., Tsaftaris, A. S., 1987. Acatalasemic glyoxysomes during late post-germination in an inbred Cat2 catalase-null maize. Bio. Cell 60:151-154.
63. Bowman, D. T., 1987. Feasibility studies on planting corn trials to a stand. Crop Sci. 27:1231-1234.
64. Boyer, C. D., Shannon, J. C., 1987. Carbohydrates of the kernel. Pp. 253-272 in S. A. Watson and P. E. Ramstad, eds., (which see).
65. Brar, G. S., Nelson, O., 1987. Genic male-sterile maize, U.S. Patent 4654465. Off. Gaz. U. S. Pat. Trademark Off. Pat 1076:2848.
66. Brar, H. S., Sharma, R. K., Malhotra, V. V., Dhillon, B. S., Khehra, A. S., 1987. Cold tolerance of maize genotypes under different methods of sowing and plant densities. Crop Improv. 14:92-94.
67. Brar, N. K., Basra, A. S., Malik, C. P., 1987. Distribution of potassium and chloride ions during pollen-pistil interaction in maize. Curr. Sci. 56:1104-1105.
68. Brendel, V., Segel, L. A., 1987. On modes of recombination, replication and segregation of the higher plant mitochondrial genome. J. Theoret. Biol. 125:163-176.
69. Brenner, M. L., 1987. The role of hormones in photosynthate partitioning and seed filling. Pp. 474-493 in Plant Hormones and Their Role in Plant Growth and Development, P. J. Davies, ed., Martinus Nijhoff, Dordrecht.
70. Brettell, R. I. S., Pryor, A. J., 1986. Molecular approaches to plant and pathogen genes. Pp. 233-246 in Genetic Approach to Plant Biochemistry, A. D. Blonstein and P. J. King, eds., Springer-Verlag, Vienna.
71. Bretting, P. K., Goodman, M. M., Stuber, C. W., 1987. Karyological and isozyme variation in West Indian and allied American mainland races of maize. Am. J. Bot. 74:1601-1613.
72. Bridges, W. C., Jr., Gardner, C. O., 1987. Foundation populations for adapted by exotic crosses. Crop Sci. 27:501-505.
73. Brink, D. E., Dewet, J. M. J., Price, S. C., Woods, J. A., 1987. Similarities in sectional delimitation in Tripsacum and Zea (Gramineae). Evolution 41:445-448.
74. Burgess, D. G., Taylor, W. C., 1987. Chloroplast photo-oxidation affects the accumulation of cytosolic mRNAs encoding chloroplast proteins in maize. Planta 170:520-527.
75. Burkey, K. O., 1987. Chlorophyll-protein complex composition during chloroplast development: a species comparison. Photosynth. Res. 11:211-224.
76. Cadena-Gomez, G., Nicholson, R. L., 1987. Papilla formation and associated peroxidase activity: a non-specific response to attempted fungal penetration of maize. Physiol. Mol. Plant Pathol. 31:51-68.
77. Cannon, R. E., Scandalios, J. G., 1987. The superoxide dismutase-2 gene of maize. Pp. 73-82 in Isozymes: Current Topics in Biological and Medical Research, Vol. 14, M. C. Rattazzi et al., eds., Alan R. Liss, New York.
78. Cannon, R. E., White, J. A., Scandalios, J. G., 1987. Cloning of cDNA for maize superoxide dismutase 2 (SOD2). Proc. Nat. Acad. Sci. USA 84:179-183.
79. Cantrell, R. P., 1986. The past experience and future course of the CIMMYT maize program. Proc. Annu. Corn Sorghum Res. Conf. 41:134-154.
80. Carlone, M. R., Russell, W. A., 1987. Response to plant densities and nitrogen levels for 4 maize cultivars from different eras of breeding. Crop Sci. 27:465-470.
81. Carlson, W. R., Curtis, C., 1986. A new method for producing homozygous duplications in maize. Can. J. Genet. Cytol. 28:1034-1040.
82. Castleberry, R. M., 1986. Border height effects on two row corn plot yields. Proc. Annu Corn Sorghum Res. Conf. 41:171-179.
83. Cauderon, A., 1986. Genetics resources and botany. Bull. Soc. Bot. Fr.-Actual. Bot. 133:7-13.
84. Chalyk, S. T., 1986. Diallel analysis of the character for multiple ears in corn. Izv. Akad. Nauk Mold. S.S.R. Ser. Biol. Khim. Nauk (6):24-28.
85. Chambers, K. R., 1987. Stalk rot of maize--host-pathogen interaction. J. Phytopathol. 118:103-108.
86. Chang, M.-T., Neuffer, M. G., 1987. Mr R-m controlling element system in maize. J. Hered. 78:163-170.
87. Charlesworth, B., 1987. The population biology of transposable elements. Trends Ecol. Evol. 2:21-23.
88. Chen, C.-H., Oishi, K. K., Kloeckener-Gruissem, B., Freeling, M., 1987. Organ-specific expression of maize Adh1 is altered after a Mu transposon insertion. Genetics 116:469-477.
89. Chen, J., Greenblatt, I. M., Dellaporta, S. L., 1987. Transposition of Ac from the P locus of maize into unreplicated chromosomal sites. Genetics 117:109-116.
90. Cheng, D. S. K., 1987. Process for regenerating cereals. U.S. Patent-4666844. Off. Gaz. U. S. Pat. Trademark Off. Pat. 1078:1711.
91. Chiang, M. S., Hudon, M., Devaux, A., Ogilvie, I., 1987. Inheritance of resistance to Gibberella ear rot in maize. Phytoprotection 68:29-33.
92. Chomet, P. S., Wessler, S. R., Dellaporta, S. L., 1987. Inactivation of the maize transposable element Activator (Ac) is associated with its DNA modification. EMBO J. 6:295-302.
93. Chory, J., Voytas, D. F., Olszewski, N. E., Ausubel, F. M., 1987. Gibberellin-induced changes in the populations of translatable mRNAs and accumulated polypeptides in dwarfs of maize and pea. Plant Physiol. 83:15-23.
94. Chowdhury, V. K., Zehr, B. E., Widholm, J. M., 1986. Differential sensitivity of suspension cultures of maize, several soybean genotypes and Glycine canescens to the herbicide DPX-F6025, 2-(([4-chloro-6-methoxypyrimidine-2-yl)amino carbonyl]-amino sulfonyl)) benzoic acid, ethyl ester. Plant Sci. 46:207-211.
95. Chowdhury, V. K., Zehr, B. E., Widholm, J. M., 1987. Effect of herbicide DPX-F6025 (Classic), 2(([4-chloro-6-methyl-pyrimidine-2-yl) amino carbonyl] amino sulfonyl)) benzoic acid, ethyl ester, on cultured cells of corn and several genotypes of soyabean. Curr. Sci. 56:362-363.
96. Chughtai, S. R., Steffensen, D. M., 1987. Heterochromatic knob composition of commercial inbred lines of maize. Maydica 32:171-188.
97. Close, K. R., 1987. Process for regenerating corn. U.S. Patent 4665030. Off. Gaz. U.S. Pat. Trademark Off., Pat. 1078:1062.
98. Close, K. R., Ludeman, L. A., 1987. The effect of auxin-like plant growth regulators and osmotic regulation on induction of somatic embryogenesis from elite maize inbreds. Plant Sci. 52:81-90.
99. Clucas, C. P., Hallauer, A. R., 1986. Selection among and within S-1 lines of maize on S-2 line and testcross performance. Proc. Iowa Acad. Sci. 93:178-183.
100. Cobbina, J., Miller, M. H., 1987. Purpling in maize hybrids as influenced by temperature and soil phosphorus. Agron. J. 79:576-581.
101. Cocking, E. C., Davey, M. R., 1987. Gene transfer in cereals. Science 236:1259-1262.
102. Coe, E. H., Jr., Hoisington, D. A., Neuffer, M. G., 1987. Linkage map of corn (maize) (Zea mays L.). Pp. 685-707 in Genetic Maps 1987: A Compilation of Linkage and Restriction Maps of Genetically Studied Organisms, Vol. 4, S. J. O'Brien, ed., Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.
103. Compagno, C., Coraggio, I., Ranzi, B. M., Alberghina, L., Viotti, A., Martegani, E., 1987. Translational regulation of the expression of zein cloned in yeast under an inducible GAL promoter. Biochem. Biophys. Res. Commun. 146:809-814.
104. Cone, K. C., Burr, F. A., Burr, B., 1986. Molecular analysis of the maize anthocyanin regulatory locus C1. Proc. Nat. Acad. Sci. USA 83:9631-9635.
105. Conger, B. V., Novak, F. J., Afza, R., Erdelsky, K., 1987. Somatic embryogenesis from cultured leaf segments of Zea mays. Plant Cell Rep. 6:345-347.
106. Connell, T. R., Below, F. E., Hageman, R. H., Willman, M. R., 1987. Photosynthetic components associated with differential senescence of maize hybrids following ear removal. Field Crop Res. 17:55-62.
107. Cooper, P., Ho, T.-H. D., 1987. Intracellular localization of heat shock proteins in maize. Plant Physiol. 84:1197-1203.
108. Cornu, A., Vallade, J., 1987. A genic male sterility due to a structural abnormality of lodicules preventing anthesis. Maydica 32:1-7.
109. Cowen, N. M., 1986. Selection theory for selfed progenies. Theor. Appl. Genet. 73:182-189.
110. Cowen, N. M., 1987. Testcross selection theory. Theor. Appl. Genet. 73:371-378.
111. Crafts-Brandner, S. J., Poneleit, C. G., 1987. Carbon dioxide exchange rates, ribulose bisphosphate carboxylase/oxygenase and phosphoenolpyruvate carboxylase activities, and kernel growth characteristics of maize. Plant Physiol. 84:255-260.
112. Crafts-Brandner, S. J., Poneleit, C. G., 1987. Effect of ear removal on CO_2 exchange and activities of ribulose bisphosphate carboxylase/oxygenase and phosphoenolpyruvate carboxylase of maize hybrids and inbred lines. Plant Physiol. 84:261-265.
113. Crevecoeur, M., 1985. Acid phosphatase activity in radicle cells of dehydration-resistant and dehydration-sensitive maize embryos. Saussurea (16):7-13.
114. Cross, H. Z., 1987. Registration of ND259 and ND260 parental lines of maize. Crop Sci. 27:375.
115. Cross, H. Z., 1987. Registration of NDSBF maize germplasm. Crop Sci. 27:368-369.
116. Cross, H. Z., Chyle, J. R., Hammond, J. J., 1987. Divergent selection for ear moisture in early maize. Crop Sci. 27:914-918.
117. Cross, H. Z., Djava, K., 1987. Mass selection for kernel depth in early maize. Euphytica 36:81-90.
118. Cross, H. Z., Kamen, J. T., Brun, L., 1987. Plant density, maturity and prolificacy effects on early maize. Can. J. Plant Sci. 67:35-42.
119. Crossa, J., Gardner, C. O., 1987. Introgression of an exotic germplasm for improving an adapted maize population. Crop Sci. 27:187-190.
120. Crossa, J., Gardner, C. O., Mumm, R. F., 1987. Heterosis among populations of maize (Zea mays L.) with different levels of exotic germplasm. Theor. Appl. Genet. 73:445-450.
121. Crossland, L. D., Rodermel, S. R., Stirdivant, S. M., Bogorad, L., 1985. Transcription of maize plastid genes in vitro. Pp. 297-302 in Molecular Biology of the Photosynthetic Apparatus, K. E. Steinback et al., eds., Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.
122. Damerval, C., Hebert, Y., Vienne, D. de, 1987. Is the polymorphism of protein amounts related to phenotypic variability? A comparison of two-dimensional electrophoresis data with morphological traits in maize. Theor. Appl. Genet. 74:194-202.
123. Danian, H., Gu, M., Zhang, X., Guo, C., Cao, Z., 1987. The changes of nucleic acid and protein for clones of pollen callus of maize in subcultures. Acta Genet. Sin. 14:114-120.
124. Daniel, L., 1986. Evaluation of the applicability of Hayman's model of diallel analysis with crosses of two small grained inbred lines by selecting for small grain and xenia for grain size. Novenytermeles 35:377-381.
125. Dankov, T., 1986. A proposed designation of maize lines and hybrids on M and C type of male sterility. Rast. Nauki 23:20-29.
126. Dankov, T., Hao, F. S., 1987. Differences in the manifestations of the cytoplasmic male sterility and restoration of the maize fertility under conditions in Bulgaria and Vietnam. Genet. Sel. 20:136-142.
127. Darrah, L. L., 1987. Marcus S. Zuber--"A man with an idea." J. Agron. Educ. 16:97-98.

128. Darrah, L. L., Lillehoj, E. B., Zuber, M. S., Scott, G. E., Thompson, D., West, D. R., Widstrom, N. W., Fortnum, B. A., 1987. Inheritance of aflatoxin-B1 levels in maize kernels under modified natural inoculation with Aspergillus flavus. Crop Sci. 27:869-872.
129. Das, O. P., Messing, J. W., 1987. Allelic variation and differential expression at the 27-kilodalton zein locus in maize. Mol. Cell Biol. 7:4490-4497.
130. Davis, N. D., Currier, C. G., Diener, U. L., 1986. Aflatoxin contamination of corn hybrids in Alabama. Cereal Chem. 63:467-470.
131. Davydenko, O. G., Kruleva, M., Dankov, T., 1987. Effect of nucleus and cytoplasm on the ultrastructure of mitochondria from maize leaf mesophyll. Genet. Sel. 20:187-195.
132. Day, A. D., 1987. Registration of Arizona-8601 maize germplasm for saline environments. Crop Sci. 27:1096.
133. Day, P. R., 1985. The impact of biotechnology on agricultural research. Pp. 94-102 in Crop Productivity--Research Imperatives Revisited, M. Gibbs and C. Carlson, eds.
134. Delguidice, L., Manna, F., Massardo, D. R., Wolf, K., Motto, M., 1987. Cryptic copies of Mu1 transposon in Italian maize varieties. Maydica 32:189-200.
135. Delp, G., Igloi, G. L., Beck, C. F., Kossel, H., 1987. Functional in vivo verification in E. coli of promoter activities from the rDNA/tDNA$_{val}$ (GAC) leader region of Zea mays chloroplasts. Curr. Genetics 12:241-246.
136. Demarly, Y., 1986. Domestication and plant breeding. Bull. Soc. Bot. Fr.-Actual. Bot. 133:15-26.
137. Dennis, E. S., Brettell, R. I. S., Peacock, W. J., 1987. A tissue culture induced Adh1 null mutant of maize results from a single base change. Mol. Gen. Genet. 210:181-183.
138. Derieux, M., Darrigrand, M., Gallais, A., Barriere, Y., Bloc, D., Montalant, Y., 1987. Evaluation of the genetic gain in grain maize during the last 30 years in France. Agronomie 7:1-12.
139. Deron, A. M., Ordas, A., 1987. Genetic study of non-ear characters in maize. Plant Breed. 98:268-271.
140. Dewald, C. L., Burson, B. L., Dewet, J. M. J., Harlan, J. R., 1987. Morphology, inheritance, and evolutionary significance of sex reversal in Tripsacum dactyloides (Poaceae). Am. J. Bot. 74:1055-1059. ,11 90
141. DeWet, J. M. J., Prunty, S. A., Brink, D. E., 1987. Zeins as indicators of maize-Tripsacum introgression. Biochem. Syst. Ecol. 15:231-233.
142. Dewey, R. E., Hu, W. W. L., Levings, C. S. III, 1987. The URF13-T mitochondrial gene of cms-T maize. Pp. 65-74 in Plant Gene Systems and Their Biology, J. L. Key and L. McIntosh, eds., Liss, New York.
143. Dewey, R. E., Timothy, D. H., Levings, C. S. III, 1987. A mitochondrial protein associated with cytoplasmic male sterility in the T cytoplasm of maize. Proc. Nat. Acad. Sci. USA 84:5374-5378.
144. Dhillon, B. S., Khehra, A. S., Singh, M., 1987. Modified full-sib selection and estimation of genetic parameters. Theor. Appl. Genet. 73:672-674.
145. Djisbar, A., Brewbaker, J. L., 1987. Effects of the brachytic-2 gene on maize yield and its components. Maydica 32:107-123.
146. Doebley, J. F., Goodman, M. M., Stuber, C. W., 1987. Patterns of isozyme variation between maize and Mexican annual teosinte. Econ. Bot. 41:234-246.
147. Doebley, J. F., Ma, D. P., Renfroe, W. T., 1987. Insertion/deletion mutations in the Zea chloroplast genome. Curr. Genet. 11:617-624.
148. Doebley, J. F., Renfroe, W., Blanton, A., 1987. Restriction site variation in the Zea chloroplast genome. Genetics 117:139-147.
149. Dong, F. M., Rasco, B. A., Gazzaz, S. S., 1987. A protein quality assessment of wheat and corn distiller's dried grain with solubles. Cereal Chem. 64:327-331.
150. Donofrio, J. C., Kuchta, J., Moore, R., Kaczmarczyk, W., 1986. Properties of a solubilized replicase isolated from corn infected with maize dwarf mosaic virus. Can. J. Microbiol. 32:637-644. GA19:01
151. Dudley, J. W., 1987. Modification of methods for identifying inbred lines useful for improving parents of elite single crosses. Crop Sci. 27:944-947.
152. Duncan, D. R., Widholm, J. M., 1987. Proline accumulation and its implication in cold tolerance of regenerable maize callus. Plant Physiol. 83:703-708.
153. Earle, E. D., Gracen, V. E., Best, V. M., Batts, L. A., Smith, M. E., 1987. Fertile revertants from S-type male-sterile maize grown in vitro. Theor. Appl. Genet. 74:601-609.
154. Ebelhar, S. A., Kamprath, E. J., Moll, R. H., 1987. Effects of nitrogen and potassium on growth and cation composition of corn genotypes differing in average ear number. Agron. J. 79:875-881.
155. Echt, C. S., Polacco, M. L., Neuffer, M. G., 1987. A nuclear encoded chloroplast ATP synthase mutant of Zea mays L. Molec. Gen. Genet. 208:230-234.
156. Eckenrode, V. K., Levings, C. S. III, 1987. Maize mitochondrial genes and cytoplasmic male sterility. Pp. 69-84 in Tailoring Genes for Crop Improvement: An Agricultural Perspective, G. Bruening et al., eds., Plenum, New York.
157. Edwards, M. D., Stuber, C. W., Wendel, J. F., 1987. Molecular-marker-facilitated investigations of quantitative-trait loci in maize. I. Numbers, genomic distribution and types of gene action. Genetics 116:113-125.
158. Eggum, B. O., Dumanovic, J., Misevic, D., Denic, M., 1985. Nutritive quality and energy yield of high oil opaque-2 and waxy maize hybrids compared to normal maize hybrids. Qual. Plant., Plant Foods Hum. Nutr. 35:165-174. GA19:01
159. El-Itriby, H. A., 1986. Estimates of genetic parameters in control of grain yield in some varietal crosses of maize (Zea mays). Ann. Agric. Sci. 31:589-600.
160. Ellis, J. G., Llewellyn, D. J., Dennis, E. S., Peacock, W. J., 1987. Maize Adh1 promoter sequences control anaerobic regulation: addition of upstream promoter elements from constitutive genes is necessary for expression in tobacco. EMBO J. 6:11-16.
161. Ellis, J. G., Llewellyn, D. J., Walker, J. C., Dennis, E. S., Peacock, W. J., 1987. The ocs element: a 16 base pair palindrome essential for activity of the octopine synthase enhancer. EMBO J. 6:3203-3208.
162. Epp, M. D., Parthasarathy, M. V., 1987. Nuclear gene-induced plastome mutations in Oenothera hookeri. II. Phenotypic description with electron microscopy. Am. J. Bot. 74:143-151.
163. Esen, A., 1987. A proposed nomenclature for the alcohol-soluble proteins (zeins) of maize (Zea mays L.). J. Cereal Sci. 5:117-128.
164. Esen, A., Bietz, J. A., Paulis, J. W., Wall, J. S., 1987. A 23.8 kD alpha-zein with N-terminal sequence and immunological properties similar to 26.7 kD alpha-zeins. Plant Mol. Biol. 9:421-430.
165. Eubanks, M., 1987. Book Review: Gay, J. P., Fabuleux Mais: Histoire et Avenir d'une Plante. Econ. Bot. 41:120-121.
166. Fakorede, M. A. B., 1986. Factor analysis of genotype-X environment interaction in maize. Maydica 31:315-324.
167. Faulde, M., Nagl, W., 1987. Cultivar, organ, and herbicide treatment-specific variation of histone H1 in maize (Zea mays L.). J. Plant Physiol. 129:415-424.
168. Fauron, C. M.-R., Abbott, A. G., Brettell, R. I. S., Gesteland, R. F., 1987. Maize mitochondrial DNA rearrangements between the normal type, the Texas male sterile cytoplasm, and a fertile revertant cms-T regenerated plant. Curr. Genet. 11:339-346.
169. Fed'ko, I.A., Morshchatskii, A.A., Tereshchenko, B. A., 1986. On maize resistance to smut. S.-Kh. Biol. (8):100-103.
170. Fedoroff, N. V., 1985. Moving genes in maize. Pp. 70-75 in Engineered Organisms in the Environment: Scientific Issues, H. O. Halvorson et al., eds., Amer. Soc. Microbiol., Washington, D.C. GA19:05
171. Feiler, H. S., Newton, K. J., 1987. Altered mitochondrial gene expression in the nonchromosomal stripe 2 mutant of maize. EMBO J. 6:1535-1539.
172. Felker, F. C., 1987. Ultrastructure of maize endosperm suspension cultures. Am. J. Bot. 74:1912-1920.
173. Ferl, R. J., Nick, H. S., 1987. In vivo detection of regulatory factor binding sites in the 5' flanking region of maize Adh1. J. Biol. Chem. 262:7947-7950.
174. Ferl, R. J., Nick, H. S., Laughner, B. H., 1987. Architecture of a plant promoter: S1 nuclease hypersensitive features of maize Adh1. Plant Mol. Biol. 8:299-307.
175. Ferl, R. J., Paul, A. L., Ashraf, M., Bollinger, S., 1987. An analysis of chromatin structure and gene regulation. Pp. 47-58 in Tailoring Genes for Crop Improvement: An Agricultural Perspective, G. Bruening et al., eds., Plenum, New York.
176. Fischer, K. S., Edmeades, G. O., Johnson, E. C., 1987. Recurrent selection for reduced tassel branch number and reduced leaf area density above the ear in tropical maize populations. Crop Sci. 27:1150-1156.
177. Fish, L. E., Kuck, U., Bogorad, L., 1985. Analysis of the two partially homologous P700 chlorophyll a proteins of maize photosystem I: Predictions based on the primary sequences and features shared by other chlorophyll proteins. Pp. 111-120 in Molecular Biology of the Photosynthetic Apparatus, K. E. Steinback et al., eds., Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.
178. Frantzen, K. A., Daly, J. M., Knoche, H. W., 1987. The binding of host-selective toxin analogs to mitochondria from normal and 'Texas' male sterile cytoplasm maize. Plant Physiol. 83:863-868.
179. Frova, C., Binelli, G., Ottaviano, E., 1987. Isozyme and HSP gene expression during male gametophyte development in maize. Pp. 97-120 in Isozymes: Current Topics in Biological and Medical Research, Vol. 15, M. C. Rattazzi et al., eds., Alan R. Liss, New York.
180. Furlani, A. M. C., Bataglia, O. C., Lima, M., 1985. Differences in nitrogen uptake and use of corn inbred lines grown in nutrient solutions. Bragantia 44:599-618.
181. Furlani, P. R., Lima, M., Miranda, L. T. de, Miranda, L. E. C. de, Sawasaki, E., Magnavaca, R., 1986. Evaluation of inbred lines, commercial materials and two maize populations for aluminum tolerance. Pesq. Agrop. Brasil. 21:655-660.
182. Fuwa, H., Glover, D. V., Miyaura, K., Inouchi, N., Konishi, Y., Sugimoto, Y., 1987. Chain length distribution of amylopectins of double- and triple-mutants containing the waxy gene in the inbred Oh43 maize background. Starch 39:295-298.
183. Galal, A. A., Omar, F. M. El-D., Ismail, A. A., El-Zeir, F. A., 1985. Genetic analysis of resistance to late wilt disease in single crosses of maize. Egypt. J. Genet. Cytol. 14:309-317. GA19:11
184. Galeev, G. S., Gorbacheva, A. G., Palii, A. F., Solonetskaya, E. V., 1986. A comparative study of productivity of maize hybrids with various types of cytoplasmic male sterility. S.-Kh. Biol. (8):73-77.
185. Galili, G., Kawata, E. E., Larkins, B. A., 1987. Characterization of polypeptides corresponding to clones of maize zein mRNAs. Plant Physiol. 84:291-297.

186. Gangopadhyay, S., Lal, S., 1986. Changes in certain biochemical constituents in maize (Zea mays L.). Indian J. Plant Pathol. 4:9-16.
187. Gardner, C. A. C., Darrah, L. L., Zuber, M. S., Wallin, J. R., 1987. Genetic control of aflatoxin production in maize. Plant Dis. 71:426-429.
188. Gardner, H. W., Miernyk, J. A., Christianson, D. D., Khoo, U., 1987. Isolation and characterization of an amyloplast envelope-enriched fraction from immature maize endosperm. Physiol. Plant. 69:541-549.
189. Gatenby, A. A., Van der Vies, S. M., Rothstein, S. J., 1987. Co-expression of both the maize large and wheat small subunit genes of ribulose-bisphosphate carboxylase in Escherichia coli. Eur. J. Biochem. 168:227-231.
190. Gavazzi, G., Tonelli, C., 1987. La genetica dei pigmenti nelle piante. Quad. Biol. Ser. Verde 11 (79 pp.).
191. Gay, J. P., 1987. Maize. Recherche 18:539-543.
192. Gbadegesin, A., 1987. Predicting maize yield from plant characters in the savanna belt of Oyo State, Nigeria. Trop. Agr. 64:172-176.
193. Geisler, G., Thiraporn, R., Stamp, P., 1986. Leaf anatomy of maize inbred lines and crosses at extreme temperatures for active growth. J. Agron. Crop Sci. 157:255-263.
194. Gelie, B., Petitprez, M., Souvre, A., Albertini, L., 1987. Etude ultrastructurale des modifications enduites par Exserohilum turcicum chez le limbe de Zea mays--Effet du gene de resistance Ht-1. Can. J. Bot. 65:2061-2066.
195. Genov, M., 1987. Inheritance of quantitative characters in maize and variability of the genetic parameters. VIII. 1000 kernel weight yield and productivity of diploid and tetraploid hybrids diverse in earliness. Genet. Sel. 20:232-239.
196. Genova, I., 1987. The combining ability of introduced maize lines. Genet. Sel. 20:305-310.
197. Gerlach, W. L., Sachs, M. M., Llewellyn, D. J., Finnegan, E. J., Dennis, E. S., 1986. Maize alcohol dehydrogenase--A molecular perspective. Pp. 73-100 in Genetic Approach to Plant Biochemistry, A. D. Blonstein and P. J. King, eds., Springer-Verlag, Vienna.
198. Giese, K., Subramanian, A. R., Larrinua, I. M., Bogorad, L., 1987. Nucleotide sequence, promoter analysis, and linkage mapping of the unusually organized operon encoding ribosomal proteins S7 and S12 in maize chloroplast. J. Biol. Chem. 262:15251-15255.
199. Gigot, C., Chaubet, N., Chaboute, M.-E., Ehling, M., Philipps, G., 1987. Nucleotide sequences of two histone H3 and H4 genes of corn. Further insight into the structure and organization of the histone genes in higher plants. Plant Physiol. Biochem. 25:235-247.
200. Gilyazetdinov, Sh. Ya., Zimnitskii, A. N., 1986. Composition of poly(A)$^+$-RNA populations in roots of corn heterotic hybrids and their starting lines. Fiziol. Rast. 33:769-778; Sov. Plant Physiol. 33:590-597.
201. Gilyazetdinov, Sh. Ya., Zimnitskii, A. N., Yakhin, I. A., Bikbaeva, E. Sh., 1986. Modulation of repeated genes in the parental forms of heterotic maize hybrids. Dokl. Akad. Nauk. SSSR 289:224-227.
202. Girardin, P., Morel-Fourrier, B., Jordan, M.-O., Millet, B., 1987. Developpement des racines adventives chez le mais. Agronomie 7:353-360.
203. Gold, B., Carrillo, N., Tewari, K. K., Bogorad, L., 1987. Nucleotide sequence of a preferred maize chloroplast genome template for in vitro DNA synthesis. Proc. Nat. Acad. Sci. 84:194-198.
204. Golubovskaya, I. N., 1987. Location of the two mei-genes by B-A translocations in maize. Genetika 23:698-706.
205. Gomez, M. H., Rooney, L. W., Waniska, R. D., Pflugfelder, R. L., 1987. Dry corn masa flours for tortilla and snack food production. Cereal Food World 32:372-378.
206. Gontarovskii, V. A., Kirikashvili, L. V., 1986. Study of the response of self-pollinated corn lines to crossing with the Paraguayan type of cytoplasmic male sterility. Dokl. Vses. Ordena Lenina Ordena Trud. Krasnogo Znameni Akad. S.-Kh. Nauk Im V. I. Lenina (8):13-15.
207. Gorla, M. S., Villa, M., Ottaviano, E., 1987. Pollen irradiation and gene transfer in maize. Maydica 32:239-148.
208. Graebe, J. E., 1987. Gibberellin biosynthesis and control. Ann. Rev. Plant Physiol. 38:419-465.
209. Griffith, S. M., Jones, R. J., Brenner, M. L., 1987. In vitro sugar transport in Zea mays L. kernels. I. Characteristics of sugar absorption and metabolism by developing maize endosperm. Plant Physiol. 84:467-471.
210. Griffith, S. M., Jones, R. J., Brenner, M. L., 1987. In vitro sugar transport in Zea mays L. kernels. II. Characteristics of sugar absorption and metabolism by isolated developing embryos. Plant Physiol. 84:472-475.
211. Grula, J. W., Hudspeth, R. L., 1987. The phosphoenolpyruvate carboxylase gene family of maize. Pp. 207-216 in Plant Gene Systems and Their Biology, J. L. Key and L. McIntosh, eds., Liss, New York.
212. Gupta, H. O., Singh, J., Ram, P. C., Singh, R. P., 1987. Chemical and biochemical investigations on normal, chalky and modified opaque-2 strains of maize. J. Food Sci. Technol. 24:184-186.
213. Guthrie, W. D., Wilson, R. L., Coats, J. R., Robbins, J. C., Tseng, C. T., Jarvis, J. L., Russell, W. A., 1986. European corn borer (Lepidoptera: Pyralidae) leaf-feeding resistance and DIMBOA content in inbred lines of dent maize grown under field versus greenhouse conditions. J. Econ. Entomol. 79:1492-1496.
214. Gwynn, B., Dewey, R. E., Sederoff, R. R., Timothy, D. H., Levings, C. S. III, 1987. Sequence of the 18S-5S ribosomal gene region and the cytochrome oxidase II gene from mtDNA of Zea diploperennis. Theor. Appl. Genet. 74:781-788.
215. Hajibagheri, M. A., Harvey, D. M. R., Flowers, T. J., 1987. Quantitative ion distribution within root cells of salt-sensitive and salt-tolerant maize varieties. New Phytol. 105:367-379.
216. Hanson, W. D., 1987. Evaluating genetic changes associated with selection utilizing information from diallel mating designs. Crop Sci. 27:919-923.
217. Haraszti, E., Vetter, J., 1986. Trypsin inhibitor content of grains: differences among species and sorts. Magy. Allatorv. Lapja 41:625-628.
218. Hashimoto, T., 1987. Gibberellin structure-dependent interaction between gibberellins and deoxygibberellin C in the growth of dwarf maize seedlings. Plant Physiol. 83:910-914.
219. Hausen, G., Beiderbeck, R., 1987. The development of an in vitro host-pathogen system consisting of maize seedling segments infected with Ustilago maydis. J. Phytopathol. 118:97-102.
220. Headrick, J. M., Pataky, J. K., 1987. Expression of partial resistance to common rust in sweet corn hybrids at various host growth stages. Phytopathology 77:454-458.
221. Hebeda, R. E., 1987. Corn sweeteners. Pp. 501-534 in S. A. Watson and P. E. Ramstad, eds., (which see).
222. Helentjaris, T., 1987. A genetic linkage map for maize based on RFLPs. Trends Genet. 3:217-221.
223. Henzell, R. G., Martin, I. F., Cox, M. C., 1985. The contribution of science to Australian tropical agriculture. 5. Tropical coarse grains. J. Austral. Inst. Agr. Sci. 51:42-52.
224. Herich, R., 1985. Membrane impression and genome expression in the proliferation process of microspores. I. Primary vacuolization of microspores. Acta Fac. Rerum Nat. Univ. Comenianae Physiol. Plant. (21):1-10.
225. Herum, F. L., 1987. Harvesting and postharvest management. Pp. 83-123 in S. A. Watson and P. E. Ramstad, eds., (which see).
226. Hisajima, S., Arai, Y., Okazawa, N., 1987. Induction of ears from maize seeds in vitro and plant regeneration from ovaries of unfertilized ears. Agr. Biol. Chem. Tokyo 51:583-584.
227. Hockensmith, P. E., Devine, T. L., Legg, D. E., Rodriguez, J. G., 1986. Energy consumption and food utilization of the Indian meal moth (Lepidoptera: Pyralidae) on different corn genotypes. J. Kans. Entomol. Soc. 59:598-603.
228. Hodges, T., Botner, D., Sakamoto, C., Haug, J. H., 1987. Using the CERES-maize model to estimate production for the U.S. cornbelt. Agr. Forest Meteorol. 40:293-304.
229. Hodgins, R. R., Huystee, R. B. v., 1986. Porphyrin metabolism in chill stressed maize (Zea mays L.). J. Plant Physiol. 125:325-336.
230. Hodgins, R. R., Huystee, R. B. v., 1986. Rapid simultaneous estimation of protoporphyrin and Mg-porphyrins in higher plants. J. Plant Physiol. 125:311-323.
231. Hoffman, L. M., Donaldson, D. D., Bookland, R., Rashka, K., Herman, E. M., 1987. Synthesis and protein body deposition of maize 15-kd zein in transgenic tobacco seeds. EMBO J. 6:3213-3222.
232. Holden, M. J., Sze, H., 1987. Dissipation of the membrane potential in susceptible corn mitochondria by the toxin of Helminthosporium maydis, Race T, and toxin analogs. Plant Physiol. 84:670-676.
233. Howard, E. A., Walker, J. C., Dennis, E. S., Peacock, W. J., 1987. Regulated expression of an alcohol dehydrogenase 1 chimeric gene introduced into maize protoplasts. Planta 170:535-540.
234. Huber, S. J., Sautter, C., 1986. Immunofluorescence localization of conjugated atrazine in leaf pieces of corn (Zea mays). Z. Pflanzenkr. Pflanzenschutz 93:608-613.
235. Hudon, M., 1987. An example of the efficient control of a maize pest, the European corn borer. Rev. Entomol. Que. 31:23-32.
236. Humphreys, T. E., 1986. Apparent zero order kinetics for sucrose uptake in the maize scutellum. Pp. 67-75 in Phloem Transport, Alan R. Liss, N.Y.
237. Humphreys, T. E., 1987. Sucrose efflux and export from the maize scutellum. Plant Cell Environ. 10:259-266.
238. Hunsperger, M. H., Davis, D. W., 1987. Effects of the sugary-1 locus on plant and ear traits in corn. Crop Sci. 27:1173-1176.
239. Ignatova, A., Ignatov, B., 1986. Comparative trials of maize hybrids. Rast. Nauki 23:40-45.
240. Iken, J. E., Odulaja, A., 1987. The development and performance of composite varieties of maize in Nigeria (Zea mays L.). Maydica 32:163-169.
241. Imbrie-Milligan, C., Kamo, K. K., Hodges, T. K., 1987. Microcallus growth from maize protoplasts. Planta 171:58-64.
242. Inouchi, N., Glover, D. V., Fuwa, H., 1987. Chain length distribution of amylopectins of several single mutants and the normal counterpart, and Sugary-1 phytoglycogen in maize (Zea mays L.). Starch 39:259-266.
243. Inouchi, N., Glover, D. V., Fuwa, H., 1987. Properties of residual maize starches following acid hydrolysis. Starch 39:284-288.
244. Irish, E. E., Schwartz, D., 1987. Activation of low and null activity isozymes of maize alcohol dehydrogenase by antibodies. Mol. Gen. Genet. 208:271-278.
245. Ivanchenko, M., Georgieva, E., Uschewa, A., Avramova, Z., 1987. A study on the heterogeneity of histone H1 from dry maize embryos. Eur. J. Biochem. 162:339-344.
246. Jamil, F. F., Nicholson, R. L., 1987. Susceptibility of corn to isolates of Colletotrichum graminicola pathogenic to other grasses. Plant Dis. 71:809-811.
247. Janowiak, F., Markowski, A., 1987. Effect of chilling on germination, growth, survival and membrane permeability in seedlings of different breeding forms of maize (Zea mays L.). Acta Physiol. Plant. 9:77-88.

248. Jarvis, J. L., Clark, R. L., Block, C. C., Berry, E. C., Guthrie, W. D., 1987. Interaction between tillage systems, maize hybrids, European corn borers (Lepidoptera, Pyralidae), and stalk rot pathogens. Maydica 32:125-137.
249. Jeffree, C. E., 1987. The cuticle, epicuticular waxes and trichomes of plants, with reference to their structure, functions and evolution. Pp. 23-64 in Insects and the Plant Surface, B. Juniper and R. Southwood, eds., Edward Arnold Publ., London.
250. Jha, P. B., Khehra, A. S., Akhter, S. A., 1986. Stability analysis for grain development and yield in maize (Zea mays). Crop Improv. 13:16-19.
251. Jones, R. J., Brenner, M. L., 1987. Distribution of abscisic acid in maize kernel during grain filling. Plant Physiol. 83:905-909.
252. Jordan, M.-O., 1987. Growth and development of the corn root system. 1. Importance of some parameters concerning the plant. Agronomie 7:365-372.
253. Kalinnikov, D. D., 1987. Regularities of variations in the amino acidic composition of alcohol-soluble proteins of maize grain in selection for quality. Fiziol. Biokhim. Kult. Rast. 19:130-132.
254. Kamo, K. K., Chang, K. L., Lynn, M. E., Hodges, T. K., 1987. Embryogenic callus formation from maize protoplasts. Planta 172:245-251.
255. Kang, M. S., Pappelis, A. J., Zuber, M. S., 1986. Effect of stalk inoculation (Diplodia maydis) on parenchyma cell death in cob and shank internodes of maize. Cereal Res. Commun. 14:267-272.
256. Kasatkin, Y. A., Ostapenko, A. I., 1986. Inheritance of resistance to common smut in maize hybrids grown under irrigation. S.-Kh. Biol. (10)63-65.
257. Kayani, S. A., Rahman, M., 1987. Salt tolerance in corn (Zea mays L.) at the germination stage. Pakistan J. Bot. 19:9-16.
258. Keith, B., Rappaport, L., 1987. In vitro [H-3]gibberellin-Al binding to soluble proteins from GA-sensitive and GA-insensitive dwarf maize mutants. Pp. 289-298 in Molecular Biology of Plant Growth Control, J. E. Fox and M. Jacobs, eds., Alan R. Liss, New York.
259. Kelley, P. M., Tolan, D. R., 1986. The complete amino acid sequence for the anaerobically induced aldolase from maize derived from cDNA clones. Plant Physiol. 82:1076-1080.
260. Kennedy, G. G., Gould, F., Deponti, O. M. B., Stinner, R. E., 1987. Ecological, agricultural, genetic and commercial considerations in the deployment of insect-resistant germplasm. Environ. Entomol. 16:327-338.
261. Kerhoas, C., Gay, G., Dumas, C., 1987. A multidisciplinary approach to the study of the plasma membrane of Zea mays pollen during controlled dehydration. Planta 171:1-10.
262. Kerr, P. S., Kalt-Torres, W., Huber, S. C., 1987. Resolution of two molecular forms of sucrose-phosphate synthase from maize, soybean and spinach leaves. Planta 170:515-519.
263. Khehra, A. S., Dhillon, B. S., Malhi, N. S., Pal, S. S., Saxena, V. K., Malhotra, V. V., 1986. Reciprocal recurrent selection for multitrait indices in maize. Euphytica 35:817-822.
264. Khehra, A. S., Dhillon, B. S., Malhotra, V. V., Brar, H. S., Sharma, R. K., 1986. Response of different genotypes of maize to transplanting. Crop Improv. 13:196-199.
265. Khehra, A. S., Sharma, R. K., Dhillon, B. S., 1987. Laboratory studies on freezing injury in maize. Indian J. Agric. Sci. 57:176-178.
266. Khesin, R. B., 1986. Controlling elements in maize. Molec. Biol. 20:697-709.
267. Khorasani, G. R., Savage, G. P., 1987. Protein quality of different varieties of maize. Nutr. Rep. Int. 36:99-108.
268. Khotyleva, L. V., Razumovich, A. N., Titok, V. V., 1987. State of integral indices of energy metabolism in etiolated seedlings of self-pollinated lines of corn. Sov. Plant Physiol. 34:243-249.
269. Khotyleva, L. V., Tarutina, L. A., Kapusta, I. B., 1986. Genetic interpretation of the combining ability of maize lines based on quantitative characters when various crossing systems are used.. S.-Kh. Biol. (8):78-82.
270. Khristov, K., Khristova, P., 1986. Genetic foundations and speeded extension of C group cms with restoration of fertility. Rast. Nauki 23:14-19.
271. Khristov, K., Khristova, P., 1986. Mutation breeding in maize. III. Heterosis and its components in a mutant x initial line hybrid. Genet. Sel. 19:288-297.
272. Khristov, K., Khristova, P., 1986. The modified high protein maize hybrid Knezha MVP-556. Rast. Nauki 23:35-39.
273. Khuong, N. T., Schubert, I., 1986. Differential staining of mitotic chromosomes from inbred lines and hybrids of Zea mays L. Kulturpflanze 34:195-205.
274. Kidd, A. D., Francis, D., Bennett, M. D., 1987. Replicon size, mean rate of DNA replication and the duration of the cell cycle and its component phases in eight monocotyledonous species of contrasting DNA C values. Ann. Bot. 59:603-610.
275. Kim, H.-Y., Schiefelbein, J. W., Raboy, V., Furtek, D. B., Nelson, O. E., Jr., 1987. RNA splicing permits expression of a maize gene with a defective Suppressor-mutator transposable element insertion in an exon. Proc. Nat. Acad. Sci. USA 84:5863-5867.
276. Kim, S. K., Efron, Y., Khadr, F., Fajemisin, J., Lee, M. H., 1987. Registration of 16 maize-streak virus resistant tropical maize parental inbred lines. Crop Sci. 27:824-825.
277. Kleczkowski, L. A., Zeiher, C. A., Randall, D. D., 1987. Extrachloroplastic site of synthesis of three chloroplast proteins in maize (Zea mays). Z. Naturforsch. 42:1113-1115.
278. Klenke, J. R., Russell, W. A., Guthrie, W. D., Martinson, C. A., Pedersen, W. L., 1987. Disease resistance in five cycles of 'BS9' corn synthetic selected for resistance to two generations of European corn borer. Phytopathology 77:735-739.
279. Kling, J. G., Gardner, C. O., Thomas-Compton, M. A., 1987. Gene frequencies in mass-selected corn populations and in derived random inbred lines. Crop Sci. 27:190-194.
280. Klittich, C. J. R., Bronson, C. R., 1986. Reduced fitness associated with TOX1 of Cochliobolus heterostrophus. Phytopathology 76:1294-1298. GA19:05
281. Klymyuk, V. I., Karpenchuk, K. H., 1986. Plant histone variants as potential taxonomic markers. Ukr. Bot. Zh. 43:67-69.
282. Klyuchko, P. F., Asyka, Y. A., 1987. On the question of breeding maize for rapid grain drying during ripening. S.-Kh. Biol. (3):3-7.
283. Knake, R. P., Beckwith, W. A., Shen, S. C., 1986. Effect of pericarp damage and seed treatment formulations on cold test and field performance of corn. Proc. Annu. Corn Sorghum Res. Conf. 41:106-120.
284. Kobayashi, H., Bogorad, L., Miles, D., 1987. Nuclear gene-regulated expression of chloroplast genes for coupling factor one in maize. Plant Physiol. 85:757-767.
285. Koinuma, K., Mochizuki, N., 1986. Diallel analysis for reaction of eight inbred lines to Bipolaris maydis Race O in maize. Bull. Natl. Grassl. Res. Inst. (35):35-41.
286. Komives, L. T., Hulesch, A., Komives, A. V., Dutka, F., 1987. Effects of S-ethyl-N,N-dipropylthiocarbamate (EPTC) on normal and dwarf seedlings of Zea mays. Biochem. Physiol. Pflanz. 182:257-260.
287. Kovacsschneider, M., Balint, A., Geczki, I., Kovacs, G., 1986. Breeding of maize for oil and protein content of the caryopsis. Novenytermeles 35:383-390.
288. Kriz, A. L., Boston, R. S., Larkins, B. A., 1987. Structural and transcriptional analysis of DNA sequences flanking genes that encode 19 kilodalton zeins. Mol. Gen. Genet. 207:90-98.
289. Kriz, A. L., Schwartz, D., 1986. Synthesis of globulins in maize embryos. Plant Physiol. 82:1069-1075.
290. Krueger, B. R., Walker, C. E., Knutson, C. A., Inglett, G. E., 1987. Differential scanning calorimetry of raw and annealed starch isolated from normal and mutant maize genotypes. Cereal Chem. 64:187-190.
291. Kudoyarova, G. R., Veselov, S. Yu., Erkeev, M. I., Gyuli-zade, V. Z., Gimaeva, R. M., Zagidullin, N. V., Shakirova, F. M., 1986. Enzyme immunoassay of indoleacetic acid in corn seeds using labeled antibodies. Sov. Plant Physiol. 33:937-
292. Kumar, D., Sarkar, K. R., 1987. Genetic structure of the R-navajo allele in maize, Zea mays L. Theor. Appl. Genet. 74:476-479.
293. Kunze, R., Stochaj, U., Laufs, J., Starlinger, P., 1987. Transcription of transposable element Activator (Ac) of Zea mays L. EMBO J. 6:1555-1563.
294. Kutovoj, A. A., Kotova, G. P., 1987. Variability of amino acidic composition in proteins of maize grain in the process of its genetic breeding. Fiziol. Biokhim. Kult. Rast. 19:375-378.
295. Kutschera, U., Bergfeld, R., Schopfer, P., 1987. Cooperation of epidermis and inner tissues in auxin-mediated growth of maize coleoptiles. Planta 170:168-180.
296. Lamkey, K. R., Hallauer, A. R., 1987. Heritability estimated from recurrent selection experiments in maize. Maydica 32:61-78.
297. Lamkey, K. R., Hallauer, A. R., Kahler, A. L., 1987. Allelic differences at enzyme loci and hybrid performance in maize. J. Hered. 78:231-234.
298. Lamkey, K. R., Smith, O. S., 1987. Performance and inbreeding depression of populations representing 7 eras of maize breeding. Crop Sci. 27:695-699.
299. Lamoureux, G. L., Rusness, D. G., 1987. EPTC metabolism in corn, cotton, and soybean: identification of a novel metabolite derived from the metabolism of a glutathione conjugate. J. Agr. Food Chem. 35:1-7.
300. Landi, P., Camussi, A., Verderio, A., Lorenzoni, C., 1986. Combining ability of early maize (Zea mays L.) inbred lines as evaluated under conditions of double cropping. Maydica 31:369-377.
301. Landi, P., Frascaroli, E., Speranza, A., 1986. Pollen characteristics of maize (Zea mays L.) inbred lines selected for grain yield. Genet. Agrar. 40:397-404.
302. Landry, J., Paulis, J. W., Wall, J. S., 1987. Chromatographic and electrophoretic analysis of zein heterogeneity. J. Cereal Sci. 5:51-60.
303. Langdale, J. A., Metzler, M. C., Nelson, T., 1987. The argentia mutation delays normal development of photosynthetic cell types in Zea mays. Develop. Biol. 122:243-255.
304. Larkins, B. A., 1987. Modification of proteins encoded by seed storage protein genes. Pp. 163-167 in Tailoring Genes for Crop Improvement: An Agricultural Perspective, G. Bruening et al., eds., Plenum, New York.
305. Larrinua, I. M., McLaughlin, W. E., 1987. A gene cluster in the Z. mays plastid genome is homologous to part of the S10 operon of E. coli. Pp. 649-652 in Progress in Photosynthesis Research, Vol. IV, J. Biggens, ed., Martinus Nijhoff, Dordrecht.
306. Laurie, D. A., Bennett, M. D., 1987. The effect of the crossability loci Kr1 and Kr2 on fertilization frequency in hexaploid wheat x maize crosses. Theor. Appl. Genet. 73:403-409.

307. Leath, M. N., Hill, L. D., 1987. Economics of production, marketing and utilization. Pp. 201-252 in S. A. Watson and P. E. Ramstad, eds., (which see).
308. Lebrun, M., Waksman, G., Freyssinet, G., 1987. Nucleotide sequence of a gene encoding corn ribulose-1,5-bisphosphate carboxylase/oxygenase small subunit (rbcs). Nucl. Acid. Res. 15:4360.
309. Lee, L., Fenoll, C., Bennetzen, J. L., 1987. Construction and homologous expression of a maize Adh1 based NcoI cassette vector. Plant Physiol. 85:327-330.
310. Lee, M., Phillips, R. L., 1987. Genomic rearrangements in maize induced by tissue culture. Genome 29:122-128.
311. Lee, T.-C., Shieh, G.-J., Ho, C.-L., Juang, J.-R., 1987. Analysis of diallel sets of dent and flint maize inbreds for combining ability and heterosis. J. Agr. Res. China 35:145-164.
312. Legg, D. E., Barney, R. J., Tipping, P. W., Rodriguez, J. G., 1987. Factors influencing the distribution of maize weevil (Coleoptera Curculionidae) eggs on maize. Environ. Entomol. 16:809-813.
313. Lei, J. D., Agrios, G. N., 1986. Mechanisms of resistance in corn to maize dwarf mosaic virus. Phytopathology 76:1034-1040.
314. Leonardi, A., Damerval, C., De Vienne, D., 1987. Inheritance of protein amounts: comparison of two-dimensional electrophoresis patterns of leaf sheaths of two maize lines (Zea mays L.) and their hybrids. Genet. Res. 50:1-5.
315. Leong, S. A., Wang, J., Budde, A., Holden, D., Kinscherf, T., Smith, T., 1987. Molecular strategies for the analysis of the interaction of Ustilago maydis and maize. Pp. 95-106 in Molecular Strategies for Crop Protection, C. J. Arntzen and C. Ryan, eds., Alan R. Liss, New York.
316. Leto, K. J., Bell, E., McIntosh, L., 1985. Assembly of photosystem II: Accelerated turnover of chloroplast-encoded polypeptides in a nuclear mutant lacking photosystem II. Pp. 39-45 in Molecular Biology of the Photosynthetic Apparatus, K. E. Steinback et al., eds., Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.
317. Levites, E. V., Monastyreva, L. E., Reimers, F. E., Sukhorzhevskaya, T. B., 1986. Biochemical assessment of inbred corn lines used in Soviet breeding. Dokl. Akad. Nauk SSSR 290:984-988.
318. Li, J., Li, J., 1986. Chloroplast thylakoid membrane polypeptides and cytoplasmic male sterility. Acta Genet. Sin. 13:430-436.
319. Lidanski, T., Todorova, L., Velikova, Y., 1987. Correlation and path-coefficient analyses of productivity in intergeneric maize X teosinte hybrids. Genet. Sel. 20:35-44.
320. Likhachev, B. S., Dovbnya, O. M., 1987. Potential longevity of maize seeds of different genotypes. Dokl. Vses. Ordena Lenina Ordena Trud. Krasnogo Znameni Akad. S.-Kh. Nauk Im. V. I. Lenina (4):13-15.
321. Lin, S.-F., Chen, C., 1986. Studies on combining ability of major agronomic characters in maize (Zea mays L.). J. Agric. Assn. China-Taiwan 136:6-14.
322. Lindsey, M. F., 1987. Inbred corn line U.S. Patent 4654466. Off. Gaz. U. S. Pat. Trademark Off. Pat. 1076:2848.
323. Liu, K.-S., Chen, C., 1986. Effect of agronomic characters on the grain yield of corn hybrids derived from double chromosomal translocations. J. Agric. Res. China 35:424-436.
324. Lobler, M., Klambt, D., 1987. Purification and immunohistochemical localization of the auxin binding site-I protein from maize coleoptiles. Biol. Plant. 29:98-103.
325. Lonsdale, D. M., 1987. Cytoplasmic male sterility: a molecular perspective. Plant Physiol. Biochem. 25:265-271.
326. Lopatina, L. M., Papazov, D. Y., Dragavtsev, V. A., 1986. On the genetic mechanisms of the formation and variability of homeostasis parameters in crop varieties. Genetika 22:2295-2302.
327. Lopes, M. A., Magnavaca, R., Filho, A. F. C. B., Gama, E. E. G. e, 1987. Performance of maize populations and their crosses for aluminum tolerance in nutrient solution. Pesquisa Agr. Brasil. 22:257-263.
328. Lorenzoni, C., 1987. Book review: O. Dolstra and P. Miedema, eds., Breeding of Silage Maize. Maydica 32:79.
329. Lorz, H., 1987. In vitro culture and genetic transformation of cereal cells. Pp. 285-290 in Molecular Mechamisms in the Regulation of Cell Behavior: Modern Cell Biology, Vol. 5, C. Waymouth, ed., Liss, New York.
330. Losyeva, Z. I., Kapusta, I. B., 1986. Mitochondrial complementation and heterosis in corn. Vyestsi Akad. Navuk B.S.S.R. Syer. Biyal. Navuk (6):29-32.
331. Loza-Tavera, H., Serrano, B., Molina, J. D., Ortega-Delgado, M. L., Sanchez de Jimenez, E., 1987. CO_2 fixation enzymes in maize (Zea mays) and grain yield. Can. J. Bot. 65:607-611.
332. MacRae, W. D., Yoder, O. C., 1987. Iron specifically protects corn protoplasts from T-toxin of Cochliobolus heterostrophus. Plant Physiol. 84:1257-1264.
333. Madden, L. V., Louie, R., Knoke, J. K., 1987. Temporal and spatial analysis of maize dwarf mosaic epidemics. Phytopathology 77:143-156.
334. Maeda, J. A., Lago, A. A. do, Miranda, L. T. de, Tella, R. de, 1987. Storage of seeds of maize and sorghum cultivars with different environmental resistances. Pesquisa Agr. Brasil. 22:1-8.
335. Maier, U.-G., Brown, J. W. S., Toloczyki, C., Feix, G., 1987. Binding of a nuclear factor to a consensus sequence in the 5' flanking region of zein genes from maize. EMBO J. 6:17-22.
336. Maisch, W. F., 1987. Fermentation processes and products. Pp. 553-574 in S. A. Watson and P. E. Ramstad, eds., (which see).
337. Malhi, N. S., Khehra, A. S., 1987. Improvement of grain yield through direct selection and selection for harvest index in two maize composites. Crop Improv. 14:74-78.
338. Malhotra, V. V., Khehra, A. S., 1986. Genotypic variation and covariation in indigenous germplasm of maize. Indian J. Agr. Sci. 56:811-815.
339. Marechal, L., Guillemaut, P., Grienenberger, J.-M., Jeannin, G., Weil, J.-H., 1987. Erratum (Marechal et al., 1986, Sequences of initiator and elongator methionine tRNAs in bean mitochondria, Plant Molec. Biol. 7:245-253). Plant Molec. Biol. 8:193.
340. Marshall, S. W., 1987. Sweet corn. Pp. 431-445 in S. A. Watson and P. E. Ramstad, eds., (which see).
341. Martienssen, R. A., Barkan, A., Scriven, A., Taylor, W. C., 1987. Identification of a nuclear gene involved in thylakoid structure. Pp. 181-194 in Plant Membranes: Structure, Function, Biogenesis, C. Leaver and H. Sze, eds., Liss, New York.
342. Masson, P., Surosky, R., Kingsbury, J. A., Fedoroff, N. V., 1987. Genetic and molecular analysis of the Spm-dependent a-m2 alleles of the maize a locus. Genetics 117:117-137.
343. Matsuoka, M., Kano-Murakami, Y., Tanaka, Y., Ozeki, Y., Yamamoto, N., 1987. Nucleotide sequence of cDNA encoding the small subunit of ribulose-1,5-bisphosphate carboxylase. J. Biochem. Tokyo 102:673-676.
344. Matsuoka, M., Kano-Murakami, Y., Yamamoto, N., 1987. Nucleotide sequence of cDNA encoding the light-harvesting chlorophyll a/b binding protein from maize. Nucl. Acid. Res. 15:6302.
345. Matters, G. L., Scandalios, J. G., 1986. Changes in plant gene expression during stress. Devel. Genet. 7:167-176.
346. Matters, G. L., Scandalios, J. G., 1987. Synthesis of isozymes of superoxide dismutase in maize leaves in response to O_3, SO_2 and elevated O_2. J. Exp. Bot. 38:842-852.
347. Matthys-Rochon, E., Vergne, P., Detchepare, S., Dumas, C., 1987. Male germ unit isolation from three tricellular pollen species: Brassica oleracea, Zea mays, and Triticum aestivum. Plant Physiol. 83:464-466.
348. May, J. B., 1987. Wet milling: process and products. Pp. 377-397 in S. A. Watson and P. E. Ramstad, eds., (which see).
349. Mazoti, L. B., 1987. Zea mays L. knobs variation induced by an Euchlaena mexicana Schrader plasmon. Mendeliana 8:45-52.
350. McClure, P. R., Omholt, T. E., Pace, G. M., Bouthyette, P.-Y., 1987. Nitrate-induced changes in protein synthesis and translation of RNA in maize roots. Plant Physiol. 84:52-57.
351. McConchie, C. A., Hough, T., Knox, R. B., 1987. Ultrastructural analysis of the sperm cells of mature pollen of maize, Zea mays. Protoplasma 139:9-19.
352. McLaughlin, M., Walbot, V., 1987. Cloning of a mutable bz2 allele of maize by transposon tagging and differential hybridization. Genetics 117:771-776.
353. McLaughlin, W. E., Larrinua, I. M., 1987. The sequence of the maize plastid encoded rpl22 locus. Nucl. Acid Res. 15:4356.
354. McLaughlin, W. E., Larrinua, I. M., 1987. The sequence of the maize plastid encoded rps3 locus. Nucl. Acid Res. 15:4689.
355. McLaughlin, W. E., Larrinua, I. M., 1987. The sequence of the maize rps19 locus and of the inverted repeat/unique region junctions. Nucl. Acid Res. 15:3932.
356. McMillian, W. W., Widstrom, N. W., Wilson, D. M., 1987. Impact of husk type and species of infesting insects on aflatoxin contamination in preharvest corn at Tifton, Georgia. J. Entomol. Sci. 22:307-310.
357. Meinke, D. W., 1986. Embryo-lethal mutants and the study of plant embryo development. Oxford Surv. Plant Molec. Cell Biol. 3:122-165.
358. Melchinger, A. E., Geiger, H. H., Schmidt, G. A., 1986. Vertical root-pull resistance and its relationship to root lodging and forage traits in early maturing European inbred lines and F1 hybrids of maize. Maydica 31:335-348.
359. Melchinger, A. E., Geiger, H. H., Seitz, G., Schmidt, G. A., 1987. Epistasis in maize (Zea mays L.). 3. Comparison of single and 3-way crosses for forage traits. Plant Breed. 98:185-193.
360. Melchinger, A. E., Geiger, H. H., Seitz, G., Schmidt, G. A., 1987. Optimum prediction of three-way crosses from single crosses in forage maize (Zea mays L.). Theor. Appl. Genet. 74:339-345.
361. Menkir, A., Larter, E. N., 1987. Emergence and seedling growth of inbred lines of corn at suboptimal root-zone temperatures. Can. J. Plant Sci. 67:409-416.
362. Meuwly, P., Pilet, P.-E., 1987. Maize root growth and localized indol-3yl-acetic acid treatment. A new methodological approach. Plant Physiol. 84:1265-1269.
363. Meza-Basso, L., Pino, C. del, Cardenas, J., Rosas, A., 1986. Purification and properties of a ribonuclease from corn leaf tissues. Phytochemistry 25:2489-2492.
364. Miernyk, J. A., 1987. Extracellular secretion of acid hydrolases by maize endosperm cells grown in liquid medium. J. Plant Physiol. 129:19-32.
365. Miles, D., Leto, K. J., Neuffer, M. G., Polacco, M. L., Hanks, J. F., Hunt, M. A., 1985. Chromosome arm location of photosynthesis mutants in Zea mays L. using B-A translocations. Pp. 361-365 in Molecular Biology of the Photosynthetic Apparatus, K. E. Steinbeck et al., eds., Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.

366. Milinko, I., Gyulavari, O., Kovacs, G., Tatrai, J., 1986. Resistance of corn hybrids and inbred lines to MDMV and inheritance of resistance. Acta Phytopathol. Entomol. Hung. 21:23-28.
367. Mitev, S., Ivanova, I., 1986. Disease resistance of high lysine maize lines with mealy and modified endosperm type. Rasteniev'd Nauki 23:13-19.
368. Mladenova, J. I., V'Nkova-Radeva, R., Pavlova, A., 1987. Nitrate assimilation in parental maize inbred lines determined by in vitro NADH-nitrate reductase activity and nitrogen-15 technique. Genet. Sel. 20:3-10.
369. Molina, M. del C., 1986. Cytogenetic study of Zea perennis. Genet. Iber. 38:27-46.
370. Molina, M. del C., Naranjo, C. A., 1987. Cytogenetic studies in the genus Zea. 1. Evidence for five as the basic chromosome number. Theor. Appl. Genet. 73:542-550.
371. Moll, R. H., Kamprath, E. J., Jackson, W. A., 1987. Development of nitrogen efficient prolific hybrids of maize. Crop Sci. 27:181-186.
372. Moon, H. G., Choe, B. H., Park, K. Y., 1985. Inheritance of resistance to maize dwarf mosaic virus in corn (Zea mays L.). Korean J. Breed. 17:140-145. GA19:01
373. Moore, R., 1987. Root gravitropism in a cultivar of Zea mays whose columella cells contain starch-deficient amyloplasts. Ann. Bot. 59:661-666.
374. Moore, R., Fondren, W. M., Marcum, H., 1987. Characterization of root agravitropism induced by genetic, chemical and developmental constraints. Am. J. Bot. 74:329-336.
375. Moore, R., McClelen, C. E., Fondren, W. M., Wang, C.-L., 1987. Influence of microgravity on root-cap regeneration and the structure of columella cells in Zea mays. Am. J. Bot. 74:218-224.
376. Morell, M., Bloom, M., Larsen, R., Okita, T. W., Preiss, J., 1987. Biochemistry and molecular biology of starch synthesis. Pp. 227-242 in Plant Gene Systems and Their Biology, J. L. Key and L. McIntosh, eds., Liss, New York.
377. Motto, M., Difonzo, N., Soave, C., Salamini, F., 1987. Chromosomal and genic instabilities originating from revertant derivatives of the o2-m(r) mutable allele of maize. Maydica 32:249-
378. Moutot, F., Huet, J. C., Wuilleme, S., Lescure, J. C., Baudet, J., Pernollet, J. C., Morotgaudry, J. F., 1987. Biochemical aspect of photoassimilated carbon partitioning at late kernel fill in maize under climatic stress. Biochimie 69:563-568.
379. Mulcahy, D. L., Mulcahy, G. B., 1987. The effects of pollen competition. Amer. Scientist 75:44-50.
380. Neill, S. J., Horgan, R., Rees, A. F., 1987. Seed development and vivipary in Zea mays L. Planta 171:358-364.
381. Nelson, O. E., 1987. The waxy locus in maize twenty-five years later. Genetics 116:339-342.
382. Neuffer, M. G., Chang, M.-T., Clark, J. K., Sheridan, W. F., 1986. The genetic control of maize kernel development. Pp. 35-50 in Regulation of Carbon and Nitrogen Reduction and Utilization in Maize, J. C. Shannon et al., eds., Amer. Soc. Plant Phys.
383. Nevers, P., Shepherd, N. S., Saedler, H., 1986. Plant transposable elements. Adv. Bot. Res. 12:103-203.
384. Newhouse, K. E., Crosbie, T. M., 1987. Genotype by tillage interactions of S1 lines from 2 maize synthetics. Crop Sci. 27:440-445.
385. Nieto-Sotelo, J., Ho, T.-H. D., 1986. Effect of heat shock on the metabolism of glutathione in maize roots. Plant Physiol. 82:1031-1035.
386. Nieto-Sotelo, J., Ho, T.-H. D., 1987. Absence of heat shock protein synthesis in isolated mitochondria and plastids from maize. J. Biol. Chem. 262:12288-12292.
387. Noldin, J. A., Mundstock, C. M., 1987. Leaf development in three maize cultivars in two planting dates. Pesquisa Agr. Brasil. 22:187-194.
388. Ochor, T. E., Trevathan, L. E., King, S. B., 1987. Relationship of harvest date and host genotype to infection of maize kernels by Fusarium moniliforme. Plant Dis. 71:311-312.
389. Odhiambo, M. O., Compton, W. A., 1987. 20 cycles of divergent mass selection for seed size in corn. Crop Sci. 27:1113-1116.
390. Odiemah, M., 1986. Diallel analysis for seed reaction of four inbred lines of maize to Fusarium damping-off disease. Acta Agron. Hung. 35:271-278.
391. Oliveira, L. A. A. de, Groszman, A., Costa, R. A. da, 1987. Ear characters of maize cultivars in green stadium. Pesquisa Agr. Brasil. 22:587-592.
392. Orthoefer, F. T., 1987. Corn starch modification and uses. Pp. 479-499 in S. A. Watson and P. E. Ramstad, eds., (which see).
393. Orthoefer, F. T., Sinram, R. D., 1987. Corn Oil: Composition, processing, and utilization. Pp. 535-551 in S. A. Watson and P. E. Ramstad, eds., (which see).
394. Osman, H. A., Koura, F. H., 1984. Reaction of maize cultivars to the attack of Hoplolaimus aegyptii. Egypt. J. Phytopathol. 16:79-84.
395. Osuma, I. A., 1986. Preservation of genetic resources in plants. Cienc. Cult. 38:1675-1677.
396. Ottoboni, L. M. M., Steffensen, D. M., 1987. Localization of zein genes in maize. Biochem. Genet. 25:123-142. f12 o2 o7
397. Ouattar, S., Jones, R. J., Crookston, R. K., 1987. Effect of water deficit during grain filling on the pattern of maize kernel growth and development. Crop Sci. 27:726-730.
398. Ouattar, S., Jones, R. J., Crookston, R. K., Kajeiou, M., 1987. Effect of drought on water relations of developing maize kernels. Crop Sci. 27:730-735.
399. Pace, G. M., Reed, J. N., Ho, L. C., Fahey, J. W., 1987. Anther culture of maize and the visualization of embryogenic microspores by fluorescent microscopy. Theor. Appl. Genet. 73:863-869.
400. Pandey, S., Diallo, A. O., Islam, T. M. T., Deutsch, J., 1987. Response to full-sib selection in 4 medium maturity maize populations. Crop Sci. 27:617-622.
401. Panwar, V. P. S., 1986. Location of sources for resistance in early maturing maize germplasms to the stalk borer Chilo partellus Swinhoe under artificial infestation. J. Entomol. Res. 10:203-204.
402. Pappelis, A. J., Kunoh, H., Bemiller, J. N., 1986. Enlargement of maize cob parenchyma cells after silking. Cereal Res. Commun. 14:387-390.
403. Pareddy, D. R., Greyson, R. I., Walden, D. B., 1987. Fertilization and seed production with pollen from in-vitro-cultured maize tassels. Planta 170:141-143.
404. Pataky, J. K., 1987. Reaction of sweet corn germ plasm to common rust and an evaluation of Rp resistance in Illinois. Plant Dis. 71:824-827.
405. Pataky, J. K., Perkins, J. M., Leath, S., 1986. Effects of qualitative and quantitative resistance on the development and spread of northern leaf blight of maize caused by Exserohilum turcicum races 1 and 2. Phytopathology 76:1349-1352.
406. Paul, A.-L., Vasil, V., Vasil, I. K., Ferl, R. J., 1987. Constitutive and anaerobically induced DNase-I-hypersensitive sites in the 5' region of the maize Adh1 gene. Proc. Nat. Acad. Sci. USA 84:799-803.
407. Pavlikova, E., Rood, S. B., 1987. Cellular basis of heterosis for leaf area in maize. Can. J. Plant Sci. 67:99-104.
408. Paz-Ares, J., Ghosal, D., Wienand, U., Peterson, P. A., Saedler, H., 1987. The regulatory c1 locus of Zea mays encodes a protein with homology to myb proto-oncogene products and with structural similarities to transcriptional activators. EMBO J. 6:3553-3558.
409. Peacock, W. J., Wolstenholme, D., Walker, J. C., Singh, K., Llewellyn, D. J., Ellis, J. G., Dennis, E. S., 1987. Developmental and environmental regulation of the maize alcohol dehydrogenase-1 (Adh1) gene promoter-enhancer interactions. Pp. 263-278 in Plant Gene Systems and Their Biology, J. L. Key and L. McIntosh, eds., Liss, New York.
410. Pekic, S., Quarrie, S. A., 1987. Abscisic acid accumulation in lines of maize differing in drought resistance: a comparison of intact and detached leaves. J. Plant Physiol. 127:203-217.
411. Penner, D., Roggenbuck, F. C., Rossman, E. C., 1986. Tolerance of corn inbreds and hybrids to the herbicide trifluralin. Proc. Annu. Corn Sorghum Res. Conf. 41:155-159.
412. Perez, L., Aguilar, R., Sanchez de Jimenez, E., 1987. Effect of an exogenous auxin on maize tissues--alteration of protein synthesis and phosphorylation. Physiol. Plant. 69:517-530.
413. Perkins, J. M., Pedersen, W. L., 1987. Disease development and yield losses associated with northern leaf blight on corn. Plant Dis. 71:940-943.
414. Pernes, J., 1986. Allogamy and cereal domestication: The examples of maize (Zea mays) and pearl millet (Pennisetum americanum). Bull. Soc. Bot. Fr.-Actual. Bot. 133:27-34.
415. Peschke, V. M., Phillips, R. L., Gengenbach, B. G., 1987. Discovery of transposable element activity among progeny of tissue culture-derived maize plants. Science 238:804-807.
416. Petolino, J. F., Thompson, S. A., 1987. Genetic analysis of anther culture response in maize. Theor. Appl. Genet. 74:284-286.
417. Pierozzi, N. I., Cruz, N. D. D., 1985. Chromosomal translocation in a hybrid between Zea mays L. and Zea mexicana Schrad. Kuntze. Bragantia 44:619-628.
418. Pinter, L., Szabo, J., Horompoli, E., 1987. Effect of metaxenia on the grain weight of the corn (Zea mays L.). Maydica 32:81-88.
419. Pintor-Toro, J. A., 1987. Adenine methylation in zein genes. Biochem. Biophys. Res. Commun. 147:1082-1087.
420. Poethig, R. S., 1987. Clonal analysis of cell lineage patterns in plant development. Am. J. Bot. 74:581-594.
421. Polacco, M. L., Walden, D. B., 1987. Genetic, developmental, and environmental influences on Oy-700 expression in maize. J. Hered. 78:81-86.
422. Porter, G. A., Knievel, D. P., Shannon, J. C., 1987. Assimilate unloading from maize (Zea mays L.) pedicel tissues. I. Evidence for regulation of unloading by cell turgor. Plant Physiol. 83:131-136.
423. Prasad, S. K., Singh, T. P., 1986. Heterosis in relation to genetic divergence in maize (Zea mays L.). Euphytica 35:919-924.
424. Prat, S., Perez-Grau, L., Puigdomenech, P., 1987. Multiple variability in the sequence of a family of maize endosperm proteins. Gene 52:41-49.
425. Praznik, W., Schillinger, H., Beck, R. H. F., 1987. Changes in the molecular composition of maize starch during kernel development. Starch 39:183-187.
426. Preiss, J., Bloom, M., Morell, M., Knowles, V. L., Plaxton, W. C., Okita, T. W., Larsen, R., Harmon, A. C., Putnamevans, C., 1987. Regulation of starch synthesis--enzymological and genetic studies. Pp. 133-152 in Tailoring Genes for Crop Improvement: An Agricultural Perspective, G. Bruening et al., eds., Plenum, New York.
427. Pring, D. R., Lonsdale, D. M., Gracen, V. E., Smith, A. G., 1987. Mitochondrial DNA duplication/deletion events and polymorphism of the C group of male sterile maize cytoplasms. Theor. Appl. Genet. 73:646-653.
428. Radojevic, L., 1986. Ultrastructure of callus tissue and somatic embryos of maize Zea mays "Cudu". Period Biol. 88:191-193.
429. Ralston, E. J., English, J. J., Dooner, H. K., 1987. Stability of deletion, insertion and point mutations at the bronze locus in maize. Theor. Appl. Genet. 74:471-475.

430. Ramagopal, S., 1986. Protein synthesis in a maize callus exposed to NaCl and mannitol. Plant Cell Rep. 5:430-434.
431. Ramagopal, S., 1987. Messenger RNA changes during drought stress in maize leaves. J. Plant Physiol. 129:311-318.
432. Ratkovic, S., 1987. Proton NMR of maize seed water: the relationship between spin-lattice relaxation time and water content. Seed Sci. Technol. 15:147-154.
433. Reddy, A. R., Britsch, L., Salamini, F., Saedler, H., Rohde, W., 1987. The A1 (anthocyanin-1) locus in Zea mays encodes dihydroquercetin reductase. Plant Sci. 52:7-14.
434. Reddy, L. V., Peterson, P. A., 1987. Effect of age and genetic background on the in vitro maize endosperm callus initiation. Maydica 32:151-161.
435. Reddy, V. K., Reddy, S. M., 1987. Synthesis of IAA by some seed-borne fungi of maize (Zea mays L.). Natl. Acad. Sci. Lett. 10:267-270.
436. Reeder, L. R. Jr., Hallauer, A. R., Lamkey, K. R., 1987. Estimation of genetic variability in two maize populations. J. Hered. 78:372-376.
437. Rees, C. A. B., Hogan, N. C., Walden, D. B., Atkinson, B. G., 1986. Identification of mRNAs encoding low molecular mass heat-shock proteins in maize (Zea mays L.). Can. J. Genet. Cytol. 28:1106-1114.
438. Reid, J. B., 1986. Gibberellin mutants. Pp. 1-34 in Genetic Approach to Plant Biochemistry, A. D. Blonstein and P. J. King, eds., Springer-Verlag, Vienna.
439. Rengel, Z., Kordan, H. A., 1987. Effects of growth regulators on light-dependent anthocyanin production in Zea mays seedlings. Physiol. Plant. 69:511-516.
440. Reymond, P., Saugy, M., Pilet, P.-E., 1987. Quantification of abscisic acid in a single maize root. Plant Physiol. 85:8-9.
441. Rhodes, C. A., Green, C. E., Phillips, R. L., 1986. Factors affecting tissue culture initiation from maize tassels. Plant Sci. 46:225-232.
442. Rhodes, D., Rich, P. J., Myers, A. C., Reuter, C. C., Jamieson, G. C., 1987. Determination of betaines by fast atom bombardment mass spectrometry--identification of glycine betaine deficient genotypes of Zea mays. Plant Physiol. 84:781-788.
443. Ritland, K., Clegg, M. T., 1987. Evolutionary analysis of plant DNA sequences. Am. Nat. 130:S74-S100.
444. Roberts, J. A., 1987. Mutants and gravitropism. Pp. 135-153 in Developmental Mutants in Higher Plants, H. Thomas and D. Grierson, eds., Cambridge U. Press, Cambridge.
445. Robertson, D. S., Stinard, P. S., 1987. Genetic evidence of Mutator-induced deletions in the short arm of chromosome 9 of maize. Genetics 115:353-361.
446. Robichaud, C., Sussex, I. M., 1986. The response of viviparous-1 and wild type embryos of Zea mays to culture in the presence of abscisic acid. J. Plant Physiol. 126:235-242. vp1
447. Robichaud, C., Sussex, I. M., 1987. The uptake and metabolism of [2-^{14}C]-ABA by excised wild type and viviparous-1 embryos of Zea mays L. J. Plant Physiol. 130:181-189.
448. Rock, C. D., Barkan, A., Taylor, W. C., 1987. The maize plastid psbB-psbF-petB-petD gene cluster: spliced and unspliced petB and petD RNAs encode alternative products. Curr. Genet. 12:69-77.
449. Rodermel, S. R., Bogorad, L., 1985. Photoregulated maize plastid genes. Pp. 285-295 in Molecular Biology of the Photosynthetic Apparatus, K. E. Steinback et al., eds., Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.
450. Rodermel, S. R., Bogorad, L., 1987. Molecular evolution and nucleotide sequences of the maize plastid genes for the alpha subunit of CF_1 (atpA) and the proteolipid subunit of CF_0(atpH). Genetics 116:127-139.
451. Rodermel, S. R., Orlin, P., Bogorad, L., 1987. The transcription termination region between two convergently-transcribed photoregulated operons in the maize plastid chromosome contains rps14, trnR (UCU) and a putative trnfM pseudogene. Nucl. Acid. Res. 15:5493.
452. Rogers, S. O., Bendich, A. J., 1987. Ribosomal RNA genes in plants: variability in copy number and in the intergenic spacer. Plant Mol. Biol. 9:509-520.
453. Roggenbuck, F. C., Penner, D., 1987. Factors influencing corn (Zea mays) tolerance to trifluralin. Weed Sci. 35:89-94.
454. Rollo, F., LaMarca, A., Amici, A., 1987. Nucleic acids in mummified plant seeds: screening of twelve specimens by gel-electrophoresis, molecular hybridization and DNA cloning. Theor. Appl. Genet. 73:501-505.
455. Rood, S. B., Kaufman, P. B., Abe, H., Pharis, R. P., 1987. Gibberellins and gravitropism in maize shoots. Endogenous gibberellin-like substances and movement and metabolism of ^{3}H gibberellin-A_{20}. Plant Physiol. 83:645-651.
456. Rood, S. B., Pharis, R. P., 1987. Hormones and heterosis in plants. Pp. 463-473 in Plant Hormones and Their Role in Plant Growth and Development, P. J. Davies, ed., Martinus Nijhoff, Dordrecht.
457. Rooney, L. W., Serna-Saldivar, S. O., 1987. Food uses of whole corn and dry-milled fractions. Pp. 399-429 in S. A. Watson and P. E. Ramstad, eds., (which see).
458. Rosenkranz, E., Scott, G. E., 1987. Comparison of inoculation with maize dwarf mosaic virus on the abaxial and adaxial leaf surfaces in corn. Phytopathology 77:1243-1246.
459. Rosenkranz, E., Scott, G. E., 1987. Type of gene action in the resistance to maize chlorotic dwarf virus in corn. Phytopathology 77:1293-1296.
460. Rottmann, W. H., Brears, T., Hodge, T. P., Lonsdale, D. M., 1987. A mitochondrial gene is lost via homologous recombination during reversion of cms-T maize to fertility. EMBO J. 6:1541-1546.
461. Rubenstein, I., Geraghty, D. E., 1986. The genetic organization of zein. Adv. Cereal Sci. Technol. 8:297-315. f12 o2 o7
462. Ruget, F., 1987. Estimation of potential grain number of maize, by means of inflorescence observations earlier than flowering. C. R. Acad. Sci. Ser. III-Vie. 304:185-
463. Runeberg-Roos, P., Grienenberger, J.-M., Guillemaut, P., Marechal, L., Gruber, V., Weil, J.-H., 1987. Localization sequence and expression of the gene coding for tRNApro(UGG) in plant mitochondria. Plant Mol. Biol. 9:237-246.
464. Russell, D., Bogorad, L., 1987. Transcription analysis of the maize chloroplast gene for the ribosomal protein S4. Nucl. Acid Res. 15:1853-1867.
465. Russell, D., Muskavitch, K. M. T., Bogorad, L., 1987. Location and sequence of the maize chloroplast gene for $tRNA^{ser}$ (GCU); a third serine isoaccepting tRNA. Nucl. Acid Res. 15:370.
466. Russell, W. A., 1987. Registration of B89 parental inbred line of maize. Crop Sci. 17:1324.
467. Sachan, J. K. S., Sarkar, K. R., 1985. The present status of Maydeae. Indian J. Genet. Plant Breed. 45:480-491.
468. Sachan, J. K. S., Sarkar, K. R., 1986. Sikkim primitive maize: An overview. Indian J. Genet. Plant Breed. 46:153-161.
469. Saha, B. C., Mukherjee, B. K., 1985. Analysis of heterosis for number of grains in maize (Zea mays L.). Indian J. Genet. Plant Breed 45:240-246.
470. Saleem, M., Cutler, A. J., 1987. Stabilizing corn leaf protoplasts with n-propyl gallate. J. Plant Physiol. 128:479-484.
471. Santos, M. X. dos, Naspolini-Filho, V., 1986. Genetic parameters estimates for ear weight in "Flint Composto Nordeste" maize population. Pesq. Agrop. Brasil. 21:739-746.
472. Sapre, A. B., Deshpande, D. S., 1987. Spontaneous emergence of parents from the F1 interspecific hybrids of Coix L. J. Hered. 78:357-360.
473. Saraiva, L. S., 1986. Influence of a new 9B translocation on recombination in maize (Zea mays). Rev. Ceres 33:93-98.
474. Saraiva, L. S., 1986. Preferential segregation involving a knobless chromosome in maize. Rev. Ceres 33:165-172.
475. Saravitz, C. H., Boyer, C. D., 1987. Starch characteristics in cultures of normal and mutant maize endosperm. Theor. Appl. Genet. 73:489-495.
476. Saric, M. R., Saric, Z., Govedarica, M., 1987. Specific relations between some strains of diazotrophs and corn hybrids. Plant Soil 99:147-162.
477. Sarma, J. S. P., Sharma, A. K., 1986. Karyomorphology of different strains of Maydeae. Cytologia 51:527-547.
478. Sarvari, M., 1986. Yielding ability and nutrient utilization of maize hibridy. Novenytermeles 35:547-552.
479. Sato, K., Sasaki, S., 1987. Development of axillary shoot in tillering and no-tillering maize plants. Jpn. J. Crop Sci. 56:345-350.
480. Sato, K., Sasaki, S., 1987. Effects of shoot apex excision and leaf cutting on development of axillary shoot in maize plant. Jpn. J. Crop Sci. 56:351-355.
481. Satyanarayana, V., Reddy, G. M., 1987. Isolation and tentative characterization of certain gene-controlled substances in anthocyanin biosynthesis in maize. Curr. Sci. 56:364-366.
482. Saxena, V. K., Singh, K., Malhi, N. S., 1986. Intra-population variability in a maize cultivar. Crop Improv. 13:144-148.
483. Scandalios, J. G., 1987. Citation classic, Current Contents 18:11. Genetic control of multiple molecular forms of enzymes in plants: a review. Biochem. Genet. 3:37-79.
484. Scandalios, J. G., 1987. The antioxidant enzyme genes Cat and Sod of maize--regulation, functional significance, and molecular biology. Pp. 19-44 in Isozymes: Current Topics in Biological and Medical Research, Vol. 14, M. C. Rattazzi et al., eds., Alan R. Liss, New York.
485. Schmidt, R. J., Burr, F. A., Burr, B., 1987. Transposon tagging and molecular analysis of the maize regulatory locus opaque-2. Science 238:960-963.
486. Schneider, G., Schmidt, J., Phinney, B. O., 1987. GC-MS identification of GA_{20}-13-O-glucoside formed from GA_{20} in normal plants and dwarf-1 mutants of Zea mays L. J. Plant Growth Reg. 5:217-224.
487. Schoper, J. B., Lambert, R. J., Vasilas, B. L., 1987. Pollen viability, pollen shedding, and combining ability for tassel heat tolerance in maize. Crop Sci. 27:27-31.
488. Schoper, J. B., Lambert, R. J., Vasilas, B. L., Westgate, M. E., 1987. Plant factors controlling seed set in maize. The influence of silk, pollen, and ear-leaf water status and tassel heat treatment at pollination. Plant Physiol. 83:121-125.
489. Schwartz, D., Dennis, E. S., 1986. Transposase activity of the Ac controlling element in maize is regulated by its degree of methylation. Molec. Gen. Genet. 476-482.
490. Schwarz, H., Schneider, H. A. W., 1987. Immunological assay of phytochrome in small sections of roots and other organs of maize (Zea mays L.) seedlings. Planta 170:152-160.
491. Schwarz-Sommer, Z., Saedler, H., 1987. Can plant transposable elements generate novel regulatory systems? Mol. Gen. Genet. 209:207-209.
492. Schwarz-Sommer, Z., Shepherd, N., Tacke, E., Gierl, A., Rohde, W., Leclercq, L., Mattes, M., Berndtgen, R., Peterson, P. A., Saedler, H., 1987. Influence of transposable elements on the structure and function of the A1 gene of Zea mays. EMBO J. 6:287-294.
493. Scott, G. E., Rosenkranz, E., 1987. Variable reaction within corn inbreds to maize dwarf mosaic virus not genetically controlled. Crop Sci. 27:78-79.

494. Scowcroft, W. R., Brettell, R. I. S., Ryan, S. A., Davies, P. A., Pallotta, M. A., 1987. Somaclonal variation and genomic flux. Pp. 275-286 in Plant Tissue and Cell Culture, C. E. Green et al., eds., Alan R. Liss, New York.
495. Serratos, A., Arnason, J. T., Nozzolillo, C., Lambert, J. D. H., Philogene, B. J. R., Fulcher, G., Davidson, K., Peacock, L., Atkinson, J., Morand, P., 1987. Factors contributing to resistance of exotic maize populations to maize weevil, Sitophilus zeamais. J. Chem. Ecol. 13:751-762.
496. Shahi, J. P., Singh, I. S., 1986. Analysis of genetic variation in Navin population of maize (Zea mays L.). Crop Improv. 13:181-185.
497. Shamina, N. V., Gruzdev, A. D., 1987. A study of birefringence of cell division spindle in the meiotic mutants of Zea mays. Tsitologiya 29:104-107.
498. Sharma, A. K., Sopory, S. K., 1987. Effect of phytochrome and kinetin on nitrite reductase activity in Zea mays. Plant Cell Physiol. 28:447-454.
499. Sharma, R. K., Dhillon, B. S., Brar, H. S., Khehra, A. S., 1987. Effect of low temperature induced chlorosis in maize. Natl. Acad. Sci. Lett. 10:229-232.
500. Sheen, J.-Y., Bogorad, L., 1986. Expression of the ribulose-1,5-bisphosphate carboxylase large subunit gene and three small subunit genes in two cell types of maize leaves. EMBO J. 5:3417-3422.
501. Sheen, J.-Y., Bogorad, L., 1987. Differential expression of C_4 pathway genes in mesophyll and bundle sheath cells of greening maize leaves. J. Biol. Chem. 262:11726-11730.
502. Sheen, J.-Y., Bogorad, L., 1987. Regulation of levels of nuclear transcripts for C_4 photosynthesis in bundle sheath and mesophyll cells of maize leaves. Plant Molec. Biol. 8:227-238.
503. Sheen, J.-Y., Sayre, R. T., Bogorad, L., 1987. Differential expression of oxygen-evolving polypeptide genes in maize leaf cell types. Plant Mol. Biol. 9:217-226.
504. Shen, D., Wang, Z., Wu, M., 1987. Gene mapping on maize pachytene chromosomes by in situ hybridization. Chromosoma 95:311-314.
505. Sheridan, W. F., Clark, J. K., 1987. Allelism testing by double pollination of lethal maize dek mutants. J. Hered. 78:49-50.
506. Sheridan, W. F., Clark, J. K., 1987. Maize embryogeny: a promising experimental system. Trends Genet. 3:3-6.
507. Shopova, K., 1986. Nucleic acid content in relation with maize heterosis. Rast. Nauki 23:59-65.
508. Shopova, K., 1986. Study of the protein content in normal and amylose maize inbred lines. Rast. Nauki 23:46-50.
509. Silva, H. P., Pereira, O. A. P., Miranda-Filho, J. B., Balmer, E., 1986. Inheritance of resistance to foliar anthracnose (Colletotrichum graminicola) in corn. Fitopatol. Bras. 11:617-626.
510. Silva, P. S. L. e, Paterniani, E., Cesar, M. A. A., 1986. Effects of the suppression partial and total of the frutification on stalks traits of maize cultivars. Pesq. Agrop. Brasil. 21:1273-1282.
511. Sim, W.-S., Kim, H.-R., 1987. Effect of GA_3 on the cyclic AMP biosynthesis in maize seedling. Plant Cell Physiol. 28:415-420.
512. Simon, R., Starlinger, P., 1987. Transposable element Ds2 of Zea mays influences polyadenylation and splice site selection. Mol. Gen. Genet. 209:198-199.
513. Singh, M., Khehra, A. S., Dhillon, B. S., 1987. Winter and rainy season as selection environments for maize. Indian J. Agr. Sci. 57:75-77.
514. Skadsen, R. W., Scandalios, J. G., 1987. Translational control of photo-induced expression of the Cat2 catalase gene during leaf development in maize. Proc. Nat. Acad. Sci. USA 84:2785-2789.
515. Small, I. D., Isaac, P. G., Leaver, C. J., 1987. Stoichiometric differences in DNA molecules containing the atpA gene suggest mechanisms for the generation of mitochondrial genome diversity in maize. EMBO J. 6:865-869.
516. Smith, J. S. C., 1986. Biochemical fingerprints of cultivars using reversed-phase high performance liquid chromatography and isozyme electrophoresis: A review. Seed Sci. Technol. 14:753-768.
517. Smith, J. S. C., 1986. Genetic diversity within the corn belt dent racial complex of maize (Zea mays L.). Maydica 31:349-367.
518. Smith, J. S. C., Smith, O. S., 1987. Associations among inbred lines of maize using electrophoretic, chromatographic, and pedigree data. 1. Multivariate and cluster analysis of data from 'Lancaster Sure Crop' derived lines. Theor. Appl. Genet. 73:654-664.
519. Sobrado, M. A., 1987. Leaf rolling--a visual indication of water deficit in corn (Zea mays L.). Maydica 32:9-18.
520. Sondahl, M. R., Evans, D. A., 1987. Controlled regeneration of corn plants and breeding strategies therewith. U.S. Patent 4659668, April 21, 1987. Off. Gaz. U.S. Pat. Trademark Off. Pat. 1077:1653.
521. Sorenson, J. C., 1986. Plant biotechnology/time for application? Proc. Annu. Corn Sorghum Res. Conf. 41:85-91.
522. Souza, C. L., 1987. Reciprocal recurrent selection with half-sib progenies obtained alternately from non-inbred (S0) and inbred (S1) plants in maize (Zea mays L.). Maydica 32:19-31.
523. Spilatro, S. R., Preiss, J., 1987. Regulation of starch synthesis in the bundle sheath and mesophyll of Zea mays L. Intercellular compartmentalization of enzymes of starch metabolism and the properties of the ADP-glucose pyrophosphorylases. Plant Physiol. 83:621-627.
524. Sponsel, V. M., 1987. Gibberellin biosynthesis and metabolism. Pp. 43-75 in Plant Hormones and Their Role in Plant Growth and Development, P. J. Davies, ed., Martinus Nijhoff, Dordrecht.
525. Springer, B., Werr, W., Starlinger, P., Bennett, D. C., Zokolica, M., Freeling, M., 1986. The Shrunken gene on chromosome 9 of Zea mays L. is expressed in various plant tissues and encodes an anaerobic protein. Molec. Gen. Genet. 205:461-468. sh1 ANP87 sh1-bz1-m4
526. Srinivasa, B. R., Subramanian, A. R., 1987. Nucleotide sequence and linkage map position of the gene for maize chloroplast ribosomal protein S14. Biochemistry-USA 26:3188-3192.
527. Srivastava, H. K., 1985. Mobile genes of mitochondria and cytoplasmic male sterility. Indian J. Genet Plant Breed. 45:447-479.
528. St.Martin, S. K., Loesch, P. J., Hallauer, A. R., 1986. Heritabilities, correlations, and index selection in BSBBo2 maize synthetic. Maydica 31:325-333.
529. Stack, S., Anderson, L., 1987. Hypotonic bursting method for spreading synaptonemal complexes of Zea mays. J. Hered. 78:178-182.
530. Stamp, P., 1987. Growth patterns of early maturing maize cultivars. J. Agron. Crop Sci. 159:101-107.
531. Stamp, P., 1987. Seedling development of adapted and exotic maize genotypes at severe chilling stress. J. Exp. Bot. 38:1336-1342.
532. Stamp, P., 1987. Seedling development of maize genotypes at constant and at fluctuating temperatures. J. Agron. Crop Sci. 158:289-293.
533. Stamp, P., 1987. The expression of photosynthetic traits during and following severe chilling stress of European and tropical maize genotypes. Physiol. Plant. 71:73-76.
534. Stamp. P., 1987. Seedling growth and photosynthetic traits of a maize cross and its parental lines at constant and fluctuating temperatures. Angew. Bot. 61:193-202.
535. Stamper, S. E., Dewey, R. E., Bland, M. M., Levings, C. S. III, 1987. Characterization of the gene URF13-T and an unidentified reading frame, ORF25, in maize and tobacco mitochondria. Curr. Genet. 12:457-463.
536. Stanfield, Z. A., 1986. One-step husking system for seed corn. Proc. Annu. Corn Sorghum Res. Conf. 41:121-133.
537. Staub, R. W., 1987. Leaf striping correlated with the presence of B-chromosomes in maize. J. Hered. 78:71-74.
538. Stinemetz, C. L., Kuzmanoff, K. M., Evans, M. L., Jarrett, H. W., 1987. Correlation between calmodulin activity and gravitropic sensitivity in primary roots of maize. Plant Physiol. 84:1337-1342.
539. Stinson, J. R., Eisenberg, A. J., Willing, R. P., Pe, M. E., Hanson, D. D., Mascarenhas, J. P., 1987. Genes expressed in the male gametophyte of flowering plants and their isolation. Plant Physiol. 83:442-447.
540. Stockel, J., Bar, M., Boidron, J. N., Bourgeois, G., 1987. Methodological approach to identify chemical oviposition stimulants from maize for European corn borer. J. Chem. Ecol. 13:557-567.
541. Storey, C. L., 1987. Effect and control of insects affecting corn quality. Pp. 185-199 in S. A. Watson and P. E. Ramstad, eds., (which see).
542. Stromberg, E. L., Donahue, P. J., 1986. Hybrid performance and yield losses associated with gray leaf spot disease. Proc. Annu. Corn Sorghum Res. Conf. 41:92-105.
543. Struik, P. C., Doorgeest, M., Boonman, J. G., 1986. Environmental effects on flowering characteristics and kernel set of maize (Zea mays L.). Neth. J. Agr. Sci. 34:469-484.
544. Stuber, C. W., Edwards, M. D., 1986. Genotypic selection for improvement of quantitative traits in corn using molecular marker loci. Proc. Annu. Corn Sorghum Res. Conf. 41:70-83.
545. Stuber, C. W., Edwards, M. D., Wendel, J. F., 1987. Molecular marker-facilitated investigations of quantitative trait loci in maize. 2. Factors influencing yield and its component traits. Crop Sci. 27:639-648.
546. Styles, E. D., Singla, C., 1987. Pollen tube growth in maize. Maydica 32:139-150.
547. Sugawara, F., Strobel, G., Strange, R. N., Siedow, J. N., Van Duyne, G. D., Clardy, J., 1987. Phytotoxins from the pathogenic fungi Drechslera maydis and Drechslera sorghicola. Proc. Natl. Acad. Sci. U.S.A. 84:3081-3085.
548. Sullivan, J. A., Kannenberg, L. W., 1987. Comparison of S1 and modified ear-to-row recurrent selection in 4 maize populations. 1. Effects on population means and variances. Crop Sci. 27:1161-1166.
549. Sundaresan, V., Freeling, M., 1987. An extrachromosomal form of the Mu transposons of maize. Proc. Nat. Acad. Sci. USA 84:4924-4928.
550. Sundberg, M. D., 1987. Development of the mixed inflorescence in Zea diploperennis Iltis, Doebley & Guzman (Poaceae). Bot. J. Linn. Soc. 95:207-216
551. Sutton, A., Sieburth, L. E., Bennett, J., 1987. Light-dependent accumulation and localization of photosystem II proteins in maize. Eur. J. Biochem. 164:571-578.
552. Swaminathan, M. S., 1986. Today's research and tomorrow's food production prospects. Impact Sci. Soc. 36:105-116.
553. Szaniel, I., Paplogo, Z. P., Gossmann, F., 1987. The evaluation of various genotypes of maize (Zea mays L.) hybrids from point of view of the milling industry. Novenytermeles 36:183-190.
554. Szatmari, M., Kovacs, G., Abranyi, A., Szundy, T., 1986. Study of the heredity of ear characteristics on systematic genetic sets in maize. Novenytermeles 35:391-396.
555. Tanner, A. H., Smith, O. S., 1987. Comparison of half-sib and S1 recurrent selection in the Krug yellow dent maize populations. Crop Sci. 27:509-513.

556. Taylor, A. G., Chirco, E. M., 1986. Endogenous inhibitors in corn seed genotypes: Inhibition of root growth on roll towel germination tests. J. Seed Technol. 10:111-120.
557. Taylor, L. P., Walbot, V., 1987. Isolation and characterization of a 1.7 kb transposable element from a mutator line of maize. Genetics 117:297-307.
558. Theres, N., Scheele, T., Starlinger, P., 1987. Cloning of the Bz2 locus of Zea mays using the transposable element Ds as a gene tag. Mol. Gen. Genet. 209:193-197.
559. Thiraporn, R., Geisler, G., Stamp, P., 1987. Effects of nitrogen fertilization on yield and yield components of tropical maize cultivars. J. Agron. Crop Sci. 159:9-14.
560. Thompson, D. L., Bergquist, R. R., Payne, G. A., Bowman, D. T., Goodman, M. M., 1987. Inheritance of resistance to gray leaf spot in maize. Crop Sci. 27:243-246.
561. Todorova, L., Dorosiev, L., 1986. The possibilities for obtaining haploids in maize. Genet. Sel. 19:436-438.
562. Torigoe, Y., 1986. A conceptual model of developmental phases of maize on the basis of the relationship between differentiation and growth of vegetative and reproductive organs. Jpn. J. Crop Sci. 55:465-473.
563. Torigoe, Y., Manabe, I., Minami, M., Kurihara, H., 1987. Differences between inbreds and hybrids in morphological and physiological characters of maize. Jpn. J. Crop Sci. 56:395-403.
564. Torigoe, Y., Watanabe, H., Kurihara, H., 1986. Varietal differences in morphological and physiological characters of the developmental phases of maize. Jpn. J. Crop Sci. 55:474-482.
565. Tracy, W. F., Galinat, W. C., 1987. Thickness and cell layer number of the pericarp of sweet corn and some of its relatives. Hortscience 22:645-647.
566. Tracy, W. F., Schmidt, D. H., 1987. Effect of endosperm type on pericarp thickness in sweet corn inbreds. Crop Sci. 27:692-694.
567. Tsaftaris, A. S., 1987. Isozymes in plant breeding. Pp. 103-125 in Isozymes: Current Topics in Biological and Medical Research, Vol. 13, M. C. Rattazzi et al., eds., Alan R. Liss, New York.
568. Tsaftaris, A. S., Efthimiadis, P., 1987. F1 heterosis and heterozygosity for isozymic structural loci in maize. Pp. 157-174 in Isozymes: Current Topics in Biological and Medical Research, Vol. 16, M. C. Rattazzi et al., eds., Alan R. Liss, New York.
569. Tsaftaris, A. S., Scandalios, J. G., 1987. Expression of the Cat2 catalase gene in different cell types of maize. Pp. 63-76 in Isozymes: Current Topics in Biological and Medical Research, Vol. 15, M. C. Rattazzi et al., eds., Alan R. Liss, New York.
570. Tsay, H. S., Miao, S. H., Widholm, J. M., 1986. Factors affecting haploid plant regeneration from maize anther culture. J. Plant Physiol. 126:33-40.
571. Turhollow, A. F., 1986. Large-scale ethanol production from corn and grain sorghum and improving conversion technology. Energ. Agr. 5:309-316.
572. Ulger, A. C., Kahnt, G., Becker, H. C., 1987. Root characteristics of maize inbred lines and single crosses in controlled environments and in field trials. J. Agron. Crop Sci. 158:333-339.
573. Undersander, D. J., 1987. Yield and yield component response of maize to water stress in hybrids with different sources of stress tolerance. Maydica 32:49-60.
574. Upham, S., MacNeish, R., Galinat, W. C., Stevenson, C. M., 1987. Evidence concerning the origin of Maiz de Ocho. Amer. Anthropol. 89:410-419.
575. Valdes, E. V., Hunter, R. B., Pinter, L., 1987. Determination of quality parameters by near infrared reflectance spectroscopy in whole-plant corn silage. Can. J. Plant Sci. 67:747-754.
576. Van Lammeren, A. A. M., 1986. A comparative ultrastructural study of the megagametophytes in two strains of Zea mays before and after fertilization. Agric. Univ. Wageningen Pap. 86:1-37.
577. Vance, V. B., Huang, A. H. C., 1987. The major protein from lipid bodies of maize. Characterization and structure based on cDNA cloning. J. Biol. Chem. 262:11275-11279.
578. Vantoai, T. T., Fausey, N. R., McDonald, M. B., Jr., 1987. Anaerobic metabolism enzymes as markers of flooding stress in maize seeds. Plant Soil 102:33-40.
579. Vasil, V., Vasil, I. K., 1987. Formation of callus and somatic embryos from protoplasts of a commercial hybrid of maize (Zea mays L.). Theor. Appl. Genet. 73:793-798.
580. Vasilas, B. L., Seif, R. D., 1986. Effect of defoliating corn inbreds on seed quality. J. Seed Technol. 10:121-128.
581. Vaughan, M. A., Vaughn, K. C., 1987. Effects of microfilament disrupters on microfilament distribution and morphology in maize root cells. Histochemistry 87:129-138.
582. Vedel, F., 1986. Molecular basis of cytoplasmic genomes diversity in higher plants. Bull. Soc. Bot. Fr.-Actual. Bot. 133:53-67.
583. Vedeneev, G. I., 1987. Genetic control of quantitative characters in maize. III. The number of kernel rows of the ear and of kernels per ear. Genetika 23:123-135.
584. Venis, M. A., 1987. Auxin-binding proteins in maize--purification and receptor function. Pp. 219-228 in Molecular Biology of Plant Growth Control, J. E. Fox and M. Jacobs, eds., Alan R. Liss, New York.
585. Vergne, P., Delvallee, I., Dumas, C., 1987. Rapid assessment of microspore and pollen development stage in wheat and maize using DAPI and membrane permeabilization. Stain Technol. 62:299-304.
586. Vershinin, A. V., Salina, E. A., Svitashev, S. K., Shumny, V. K., 1987. The occurrence of Ds-like sequences in cereal genomes. Theor. Appl. Genet. 73:428-432.
587. Vig, J. C., Limberg, P., 1986. Maize ear formation as affected by photoperiod. Landwirt. Forsch. 39:179-188.
588. Villegas de los, R. E., Aragon-Correa, J. J., Munoz, S., 1985. Tolerance of maize genotypes for drought in El Cerrito Valle, Colombia. Acta Agron. 35:7-22.
589. Vincent, J. R., Nicholson, R. L., 1987. Evidence for isoenzymes of 4-hydroxy-cinnamic acid: CoA ligase in maize mesocotyls and their response to infection by Helminthosporium maydis race O. Physiol. Molec. Plant Pathol. 30:121-129.
590. Vlchinkov, V., Blchinkov, S., 1986. Combining ability and productivity of inbred maize lines. Rast. Nauki 23:5-13.
591. Walbot, V., Chandler, V. L., Taylor, L. P., McLaughlin, P., 1987. Regulation of transposable element activities during the development and evolution of Zea mays L. Pp. 265-284 in R. A. Raff and E. C. Raff, eds., Development as an Evolutionary Process, Alan R. Liss, Inc.
592. Walbot, V., Dewet, J. R., Fromm, M., 1987. Visual assays of transformation in plant cells. Pp. 183-188 in Tailoring Genes for Crop Improvement: An Agricultural Perspective, G. Bruening et al., eds., Plenum, New York.
593. Walker, J. C., Howard, E. A., Dennis, E. S., Peacock, W. J., 1987. DNA sequences required for anaerobic expression of the maize alcohol dehydrogenase 1 gene. Proc. Nat. Acad. Sci. USA 84:6624-6628.
594. Walker, N. H., Qin, J., Abbott, A. G., 1987. Northern hybridization analysis of mitochondrial gene expression in maize cytoplasm with varied nuclear backgrounds. Theor. Appl. Genet. 74:531-537.
595. Wall, J. S., Bietz, J. A., 1987. Differences in corn endosperm proteins in developing seeds of normal and opaque-2 corn. Cereal Chem. 64:275-280.
596. Wang, A. S., 1987. Callus induction and plant regeneration from maize mature embryos. Plant Cell Rep. 6:360-362.
597. Wann, E. V., 1987. Registration of M6388, M6411, M6415, and M6421 sweet corn germplasm lines with high-sugar endosperm genotype. Crop Sci. 27:821-822.
598. Warmbrodt, R. D., 1987. Solute concentrations in the phloem and apex of the root of Zea mays. Am. J. Bot. 74:394-402.
599. Watson, S. A., 1987. Measurement and maintenance of quality. Pp. 125-183 in S. A. Watson and P. E. Ramstad, eds., (which see).
600. Watson, S. A., 1987. Structure and composition. Pp. 53-82 in S. A. Watson and P. E. Ramstad, eds., (which see).
601. Watson, S. A., Ramstad, P. E., 1987. Corn: Chemistry and Technology. Amer. Assn. of Cereal Chemists, Inc., St. Paul.
602. Weber, E. J., 1987. Carotenoids and tocols of corn grain determined by HPLC. J. Amer. Oil Chem. Soc. 64:1129-1134.
603. Weber, E. J., 1987. Lipids of the kernel. Pp. 311-349 in S. A. Watson and P. E. Ramstad, eds., (which see).
604. Wendel, J. F., Edwards, M. D., Stuber, C. W., 1987. Evidence for multilocus genetic control of preferential fertilisation in maize. Heredity 58:297-301.
605. Wenzler, H., Meins, F., Jr., 1987. Persistent changes in the proliferative capacity of maize leaf tissues induced by Ustilago infection. Physiol. Molec. Plant Pathol. 30:309-319.
606. Wermke, M., Vonhoyningenhuene, J., 1987. Influence of genotype and density on chemical composition and nutritive value of silage maize. J. Agron. Crop Sci. 158:73-83.
607. Wernicke, W., Gorst, J., 1987. Fucose-specific lectins disclose perpetuation of root primordia in callus cultures of maize (Zea mays L.). Develop. Biol. 120:85-91.
608. Wessler, S. R., Baran, G., Varagona, M., 1987. The maize transposable element Ds is spliced from RNA. Science 237:916-918.
609. West, D. R., Kincer, D. R., Kincer, H. C., Josephson, L. M., 1987. Registration of T62S and T64S germplasm lines of maize. Crop Sci. 17:1317-1318.
610. West, D. R., Kincer, H. C., 1985. A comparison of near-isogenic high lysine and normal endosperm corn hybrids. Tenn. Farm Home Sci. (136):3-5.
611. Westcott, B., 1987. A method of assessing the yield stability of crop genotypes. J. Agric. Sci. 108:267-274.
612. White, D. G., Yanney, J., Anderson, B., 1987. Variation in pathogenicity, virulence and aggressiveness of Colletotrichum graminicola on corn. Phytopathology 77:999-1001.
613. White, J. A., Scandalios, J. G., 1987. In vitro synthesis, importation and processing of Mn-superoxide dismutase (SOD-3) into maize mitochondria. Biochim. Biophys. Acta 926:16-25.
614. Wicks, Z. W., Carson, M. L., 1987. Line source irrigation to assess maize inbred line stability. Crop Sci. 27:354-356.
615. Wicks, Z. W., Carson, M. L., Scholten, G. G., 1987. Registration of SD42 parental line of maize. Crop Sci. 27:1324.
616. Widstrom, N. W., Carr, M. E., Bagby, M. O., Black, L. T., 1987. Harvest methods for estimated ethanol yields from relative fermentable carbohydrate accumulation in maize hybrids. Agron. J. 79:758-
617. Widstrom, N. W., McMillian, W. W., Wilson, D. M., 1987. Segregation for resistance to aflatoxin contamination among seeds on an ear of hybrid maize. Crop Sci. 27:961-963.

618. Wilkinson, T. C., Thompson, S. A., 1987. Genotype, medium, and genotype x medium effects on the establishment of regenerable maize callus. Maydica 32:89-105.
619. Williams, W. P., Buckley, P. M., Davis, F. M., 1987. Feeding response of corn earworm (Lepidoptera: Noctuidae) to callus and extracts of corn in the laboratory. Environ. Entomol. 16:532-534.
620. Williams, W. P., Buckley, P. M., Davis, F. M., 1987. Tissue culture and its use in investigations of insect resistance of maize. Agric. Ecosyst. Environ. 18:185-190.
621. Willman, M. R., Below, F. E., Hageman, R. H., Lambert, R. J., 1987. Relationship between ear-removal-induced leaf senescence and green score in maize. Crop Sci. 27:898-902.
622. Willman, M. R., Below, F. E., Lambert, R. J., Howey, A. E., Mies, D. W., 1987. Plant traits related to productivity of maize. 1. Genetic variability, environmental variation, and correlation with grain yield and stalk lodging. Crop Sci. 27:1116-1121.
623. Willman, M. R., Below, F. E., Lambert, R. J., Howey, A. E., Mies, D. W., 1987. Plant traits related to productivity of maize. 2. Development of multiple trait models. Crop Sci. 27:1122-1126.
624. Wilson, C. M., 1987. Proteins of the kernel. Pp. 273-310 in S. A. Watson and P. E. Ramstad, eds., (which see).
625. Wise, R. P., Fliss, A. E., Pring, D. R., Gengenbach, B. G., 1987. urf13-T of T cytoplasm maize mitochondria encodes a 13 kD polypeptide. Plant Molec. Biol. 9:121-126.
626. Wise, R. P., Pring, D. R., Gengenbach, B. G., 1987. A mitochondrial open reading frame associated with mutation to male fertility and toxin insensitivity in T-cytoplasm maize. Pp. 107-114 in Molecular Strategies for Crop Protection, C. J. Arntzen and C. Ryan, eds., Alan R. Liss, New York.
627. Wise, R. P., Pring, D. R., Gengenbach, B. G., 1987. Mutation to male fertility and toxin insensitivity in Texas (T)-cytoplasm maize is associated with a frameshift in a mitochondrial open reading frame. Proc. Nat. Acad. Sci. USA 84:2858-2862.
628. Wiseman, B. R., Wilson, R. L., 1987. Corn earworm development on meridic diets containing pollinated and unpollinated corn silks from 2 planting dates at 2 locations. Maydica 32:201-220.
629. Wright, K. N., 1987. Nutritional properties and feeding values of corn and its by-products. Pp. 447-478 in S. A. Watson and P. E. Ramstad, eds., (which see).
630. Wu, S.-M., Rebeiz, C. A., 1985. Chloroplast biogenesis: Molecular structure of chlorophyll b (E489 F666). J. Biol. Chem. 260:3632-3634. nec2
631. Wu, W.-S., Wang, B.-C., 1987. Identification of race, mating type and pathogenicity of and response of different corn cultivars to Bipolaris maydis. Plant Prot. Bull. 29:13-24.
632. Wurtzel, E. T., Burr, F. A., Burr, B., 1987. DNase I hypersensitivity and expression of the Shrunken-1 gene of maize. Plant Mol. Biol. 8:251-264.
633. Yacoob, R. K., Filion, W. G., 1986. Temperature-stress response in maize: a comparison of several cultivars. Can. J. Genet. Cytol. 28:1125-1131.
634. Yacoob, R. K., Filion, W. G., 1987. The effects of cold-temperature stress on gene expression in maize. Biochem. Cell Biol. 65:112-119.
635. Yang, T., Zeng, M., 1986. Analyses of Px-4 Px-5 gene and their biochemical characters of translation products in leaves of maize. Acta Genet. Sin. 13:346-352.
636. Yoder, J., Belzile, F., Alpert, K., Palys, J., Michelmore, R., 1987. Mobilization of the maize transposable element Ac in tomato. Pp. 189-198 in Tomato Biotechnology: Plant Biology, Vol. 4, D. J. Nevins and R. A. Jones, eds., Alan R. Liss, New York.
637. Yordanov, Y. G., Pok, L. H., 1987. Investigation of maize photosynthetic rate in relation with heterosis. Fiziol. Rast. (Sofia) 13:29-33.
638. Yurankova, S. I., Razumovich, A. N., 1986. Kinetic characteristics of glucose-6-phosphate dehydrogenase in maize lines and hybrids. Vyestsi Akad. Navuk BSSR Syer. Biyal Nauk (4):63-65.
639. Zabala, G., O'Brien-Vedder, C., Walbot, V., 1987. S2 episome of maize mitochondria encodes a 130-kilodalton protein found in male sterile and fertile plants. Proc. Nat. Acad. Sci. USA 84:7861-7865.
640. Zehr, B. E., Williams, M. E., Duncan, D. R., Widholm, J. M., 1987. Somaclonal variation in the progeny of plants regenerated from callus cultures of seven inbred lines of maize. Can. J. Bot. 65:491-499.
641. Zeng, M. Q., Yang, T. X., Wei, J. K., Cui, R. J., 1987. Determinations of the biochemistry for cytoplasmic male-sterility and the genetics for their restoration in maize (Zea mays L.). Sci. Sinica Ser. B 30:283-296.
642. Zhang, H., Somerville, C. R., 1987. Transfer of the maize transposable element Mu1 into Arabidopsis thaliana. Plant Sci. 48:165-173.
643. Zhelev, R., 1986. The vigor of maize root system and lodging resistance. Rastenlev'd Nauki 23:8-12.
644. Ziegler, K. E., Guthrie, W. D., Foley, D. C., 1987. Registration of BSP1C1 and BSPW1C1 popcorn (maize) germplasms. Crop Sci. 27:1318-1319.
645. Zuber, M. S., Darrah, L. L., 1987. Breeding, genetics and seed corn production. Pp. 31-51 in Corn: Chemistry and Technology, S. A. Watson and P. E. Ramsted, eds., AACC Books, St. Paul.
646. Zurawski, G., Clegg, M. T., 1987. Evolution of higher-plant chloroplast DNA-encoded genes: implications for structure-function and phylogenetic studies. Ann. Rev. Plant Physiol. 38:391-418.

To All Flavonoid Lovers

Giuseppe Gavazzi and I are attempting to organize a 'Post International Congress of Genetics' meeting in Victoria, British Columbia, on The Genetics of Flavonoids. Victoria is the ideal place for a relaxed discussion following a busy meeting, and it would be nice to have an informal get-together similar to that of the Maize Genetics Meetings. However, the reality of financing such a meeting requires that there is a registration fee and a more formal type of meeting with invited speakers, submitted papers, posters, etc. Of course there will also be the usual benefits of a more formal meeting -- banquets, reasonable housing, pleasurable field-trips, etc., etc. For those who have never been to Victoria, I should mention that it is a beautiful city, and it should not be confused with that other place over on the mainland. For anyone who is interested, write to me and I will keep you updated on the meeting and make sure you receive the usual Chamber of Commerce material on Victoria.

E. D. Styles

Announcement for the Second International Symposium on Genetic Manipulation in Crops

---

Second International Symposium on
Genetic Manipulation in Crops
29 to 31 August 1988
International Maize and Wheat
Improvement Center, Mexico

Scheduled sessions on anther culture and haploid breeding, protoplast culture and plant regeneration, protoplast fusion and somatic hybrids, distant hybridization, somaclonal variation, endosperm culture, international collaboration in genetic manipulation of crop plants.

Organizing committee: Prof. Li Zhensheng, Prof. Hu Han, Prof. Shao Qiquan, Institute of Genetics, Academia Sinica, China; Dr. A. Mujeeb-Kazi, CIMMYT, Mexico; Dr. G. S. Khush, Dr. L. A. Sitch, IRRI, Philippines.

For information and forms write:
Dr. L. A. Sitch
IRRI, P. O. 933
Manila, Philippines

The XVI International Genetics Congress will be held in Toronto August 20-27. Some highlights are reproduced here.

2.2.9 SOMATIC VARIATION
VARIATIONS SOMATIQUES

Organizers/Organisateurs :
S. Dellaporta, Yale University, New Haven
W.R. Scowcroft, Biotechnica Canada Ltd., Calgary

Speakers/Conférenciers :
J.L. Kermicle, University of Wisconsin, Madison
P. Larkin, CSIRO, Canberra

2.2.12 ORGANELLES I
ORGANITES I

Organizers/Organisateurs :
A.J.F. Griffiths, University of British Columbia, Vancouver
P.R. Slonimski, Centre de génétique moléculaire, Gif-sur-Yvette

Speakers/Conférenciers :
H. Bertrand, University of Regina
C.S. Levings, North Carolina State University, Raleigh
H. Ozeki, Kyoto University
P.R. Slonimski, Centre de génétique moléculaire, Gif-sur-Yvette

2.2.13 ORGANELLES II
ORGANITES II

Organizers/Organisateurs
H. Bertrand, University of Regina
L. Bogorad, Harvard University Cambridge

Speakers/Conferenciers
L. Bogorad, Harvard University, Cambridge
R.A. Butow, University of Texas Health Science Center, Dallas
A.M. Lambowitz, Ohio State University, Columbus

2.5.3 TRANSPOSABLE ELEMENTS/
ÉLÉMENTS TRANSPOSABLES

Organizers/Organisateurs :
G. Chaconas, University of Western Ontario, London
N.A. Jenkins, Frederick Cancer Research Facility, MD

Speakers/Conférenciers :
P. Bingham, State University of New York, Stony Brook
G. Chaconas, University of Western Ontario, London
D. Garfinkel, Frederick Cancer Research Facility
G. P. Georgiev, Institute for Molecular Biology, Moscow
H. Saedler, Max Planck Institute, Cologne

2.5.6 PLANT MOLECULAR GENETICS/
GÉNÉTIQUE MOLÉCULAIRE DES VÉGÉTAUX

Organizers/Organisateurs
R.B. Flavell, Plant Breeding Institute, Cambridge
O.E. Nelson, University of Wisconsin, Madison

Speakers/Conférenciers
N. Fedoroff, Carnegie Institute of Washington, Baltimore
R.B. Flavell, Plant Breeding Institute, Cambridge
J. Nasrallah, Cornell University, Ithaca

2.5.7 PLANT BIOTECHNOLOGY/
BIOTECHNOLOGIE VÉGÉTALE

Organizers/Organisateurs :
W.D. Beversdorf, Allelix Inc., Mississauga
R.L. Phillips, University of Minnesota, St. Paul

Speakers/Conférenciers :
P.C. Anderson, Molecular Genetics, Inc., Minnetonka, MN
B. Hohn, Friedrich Miescher Institute, Basel
W. Keller, Agriculture Canada, Ottawa
M. Van Montagu, University of Ghent

2.5.8 NEW DEVELOPMENTS IN PLANT BREEDING/
DÉVELOPPEMENTS RÉCENTS EN AMÉLIORATION DES VÉGÉTAUX

Organizers/Organisateurs :
B.L. Harvey, University of Saskatchewan, Saskatoon
G. Wenzel, Federal Biological Research Centre for Agriculture and Forestry, Grünbach, FRG

Speakers/Conférenciers :
R. Horst, Monsanto Co., St. Louis
S. Peloquin, University of Wisconsin, Madison
E. Picard, C.N.R.S., Gif-sur-Yvette
F. Salamini, Max Planck Institute, Cologne

Subscription Form: MAIZE GENETICS COOPERATION NEWS LETTER

Effective with the 1988 issue, an annual subscription fee will be charged for the News Letter, to cover production and distribution costs. In addition, an endowment fund is being initiated with gifts from individuals, corporations and other institutions, eventually to underwrite the annual costs or to reduce subscription fees to the extent possible. Our goal is $100,000. Please consider making a gift toward the endowment; a gift of $150 or more will confer a lifetime subscription.

Please mark your choice below and return with your check, payable to Maize Genetics:

/ / 1988 Subscription	$ 10.00
/ / 1989 Subscription	10.00
/ / Student Subscription 1988	5.00
/ / Student Subscription 1989	5.00
(Advisor's Signature: _____)	
/ / Gift to Endowment, Lifetime Subscription	
(Donors will be acknowledged in the News Letter)	
/ / Kernel Endowment	$ 150.00
/ / Ear Endowment	250.00
/ / Whole Plant Endowment	1000.00
/ / Other	$ _ _ _ _

/ / Request Relief from Subscription Fee as Follows:
- / / Financial or Exchange Limitations
- / / Public Library, Cannot Afford
- / / Other (Please State Basis)

/ / Retain My Name on the Address List (no subscription)

/ / Remove My Name from the Address List

Back issues will be available at $5.00 per issue.

Please return this form with check payable to "Maize Genetics" to:

Maize Genetics Cooperation News Letter
Curtis Hall - Agronomy Dept.
University of Missouri
Columbia, MO 65211 U.S.A.

Amount enclosed:

$ _____

Mailing address:

Telephone number (if you wish to have it given in the Address List):

Are you a new subscriber? / / yes / / no
If the address above is a change, please mark here. / /