MAIZE GENETICS COOPERATION

NEWSLETTER

65

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I. FOREWORD

The working research information here is shared with the understanding that each item is unpublished and is not to be cited in publications without specific consent of the authors. By sharing our research information here, we contribute to the advancement of biology and to the power of shared technical knowledge.

Information here is in the form of "notes" and is not "published" in the sense of a refereed journal. In event a policy statement should be needed, the following perspectives may ensure against misunderstanding of our Newsletter:

1) In publications, refer to MNL notes in the text, never in the bibliography. Specify "unpublished data", or "personal communication" (i.e., with the colleague's consent). The volume and page numbers might be given, as an aid to the reader.

2) Emphasize, in preparing your MNL notes, brief technical notes, updates, mutants, mapping data, and the like. Avoid presenting comprehensive material and analyses that are better directed to formal publication.

3) Never refer to MNL notes as "published".

4) If challenged, forward these comments as a statement of the purpose, intent, and policy of the cooperators who contribute to this Newsletter.

More and more cooperators supplied notes, tables and figures in electronic form, and this greatly facilitated the editing and compiling.

Gifts to the Endowment Fund for support of the Newsletter total over $68,000. Please see the listing, in the front of this issue, of donors whose generosity has made this total. We are all grateful for the support of our colleagues and of organizations with which we have common interests. Part of our support also comes from the National Science Foundation, from the Integrated Mapping Project.

A warm acknowledgement for advice and ideas is given to my colleagues, Shiaoman Chao, Gerry Neuffer, and Dave Hoisington. Their advice and encouragement is greatly appreciated.

Shirley Kowalewski again cajoled the word-processor, edited the copy, and screened the year's literature, and gave special creative advice at critical moments. Mary Ann Steyaert booked addresses and subscriptions through the year, and artfully prepared the mockup. Denis Hancock lashed our computers when needed, aided by programming help of Lloyd McKenzie. At University Printing Services, Yvonne Ball and the printshop staff again efficiently and carefully made sure that the job was done promptly and well.

Included in this issue is a new chloroplast map, once again generously volunteered and provided by Steven Rodermel. This contribution is gratefully acknowledged. The mitochondrial maps in MNL64, according to Christiane Fauron, are reasonably current.

For submission of notes for the next issue (Number 66, 1992), please see details inside the back cover.

If you wish to subscribe to this Newsletter please use the form in the back of this issue. Gifts to the Endowment Fund, toward our goal of $100,000, will be very much appreciated.

Ed Coe

ALBANY, CALIFORNIA
Plant Gene Expression Center

Terminal ear mutants isolated from Mutator lines

--Edward Robbie, Bruce Veit, Paul Chomet and Sarah Hake

Two independent terminal ear mutants were found in lines carrying Robertson's Mutator elements. The phenotype of the first isolate, found in a transposon tagging experiment, is pleiotropic, with the most striking effect on the internodes. Severely affected plants are extremely stunted and two-dimensional, with the leaves emerging strictly 180 degrees apart. Less severe plants vary in their internode length, often alternating between short and long internodes, to produce plants that appear to have opposite phyllotaxy. Variability in phyllotaxy sometimes gives plants with successive leaves that arise from one side of the plant. The tassels are often feminized. Either the lower tassel branches are somewhat feminized or, alternatively, they may be transformed into entire ears enveloped in husks. Usually the entire tassel is enclosed in husks and rarely it is a completely feminized, unbranched inflorescence. The second mutant with a similar phenotype was discovered by Paul Chomet in his Mutator lines. Crossing of the two mutants showed that they were allelic.

Morphological analysis suggested that the new mutants could be alleles of terminal ear (*te1*). We determined that our allele, *te1-sn*, was allelic to the reference allele, *te1-R* by crossing the two together. We also uncovered the phenotype by crossing *te1-sn* by TB-3La. The reference allele is also uncovered by TB-3La according to J. Beckett. We are in the process of mapping terminal ear more closely.

We investigated the possibility of linkage of the *te1-sn* phenotype with a Mutator element. Genomic DNA from families segregating for *te1-sn* were digested with *Bcl*I, blotted and hybridized to the internal portions of *Mu8* and *Mu1*. Upon analysis of over sixty chromosomes, tight cosegregation of a *Mu8* element with the mutant phenotype was demonstrated. We determined that a fragment of the same size is segregating with the mutant phenotype in the line of Paul Chomet's. Because these two mutants do not share progenitors, it suggests that two different *Mu8* elements may have inserted in the same *Bcl*I fragment. We are in the process of cloning this *Bcl*I fragment in order to determine if it is part of the terminal ear locus.

ALBANY, NEW YORK
State University of New York

Codon usage tables for zein and non-zein genes: an update

--Douglas A. Hamilton and Joseph P. Mascarenhas

In an earlier codon usage table for maize based on 25 nuclear genes (D. M. Bashe and J. P. Mascarenhas, MNL 63: 4-5, 1989) it was found that at least one of the zeins (22 kD family) was not typical of the codon usage profile of other maize genes. With the additional sequences now available we have updated the codon usage table. A total of 56 maize nuclear genes have now been analyzed. This analysis shows that zeins of the 19 kD and 22 kD families exhibit a codon usage pattern that is different from that of the bulk of other nuclear encoded genes. Accordingly we have created two tables, one for the 19 and 22 kD zeins and the second for nuclear genes other than those of the 19 and 22 kD zeins. Codon usage in maize has also been discussed by W. H. Campbell and G. Gowri (Plant Physiol. 92: 1-11, 1990).

Genes were selected from the GenBank database (Release 64, 6/90) on the basis of the presence of a complete coding sequence, either as mRNA or as combined exons from a genomic sequence. Codon usage tables were created by the repetitive addition of tables generated for individual genes by utilizing the Genetics Computer Group (GCG) Sequence Analysis Software (Version 6.2) program "CodonFrequency" (J. Devereux, P. Haeberli and O. Smithies, Nucleic Acids Res. 12: 387-395, 1984). Fourteen sequences for the 19 and 22 kD zein genes and forty two sequences of other nuclear genes were used (Table 1). The

Table 1. GenBank sequences used in the preparation of the codon usage tables.

GenBank locus	Description
"OTHER NUCLEAR GENES"	
MZEA1G	A1 gene for NADPH-dependent reductase
MZEACT1G	Actin 1 gene
MZEADH1F	Alcohol dehydrogenase (ADH1-F) mRNA
MZEADH1FA	Alcohol dehydrogenase (ADH1-1F) gene
MZEADH2NR	Alcohol dehydrogenase (ADH2-N) mRNA
MZEALBB32	Albumin b-32 mRNA
MZEALD	Aldolase mRNA
MZEANT	ATP/ADP translocator mRNA
MZEBRN2W	UDP glucose flavonoid glycosyl transferase (Bz-W22)
MZEBRN2ZA	UDP glucose flavonoid glycosyl transferase (Bz-McC)
MZECAT1I	Catalase-1 isoenzyme (cat-1) mRNA
MZECAT3I	Catalase-3 isoenzyme (cat-1) mRNA
MZEEG2R	Endosperm glutelin-2 protein mRNA
MZEGAPDH	Glyceraldehyde-3-phosphate dehydrogenase (GAPDH) mRNA
MZEGGST3B	Glutathione-S-transferase (GSTIII) mRNA
MZEGLB1SA	Embryo globulin S allele mRNA
MZEGLUT2E	Endosperm glutelin-2 gene
MZEGSTI	Glutathione-S-transferase I mRNA
MZEGSTIB	Glutathione-S-transferase (GST-I) mRNA
MZEH3C2	Histone 3 gene
MZEH3C4	Histone 3 gene
MZEH4C14	Histone 4 gene
MZEH4C7	Histone 4 gene
MZEHSP701,2	Heat shock protein 70, exons 1+2
MZELHCP	Light-harvesting chlorophyll a/b binding protein mRNA
MZEMPLJ	Major lipid body protein LJ mRNA
MZENAR,1	NADH:nitrate reductase (NR) mRNA (5'+3'ends)
MZENDMEX	NADP-dependent malic enzyme (Me1) mRNA
MZEPCSSU	RuBisCo small subunit mRNA
MZEPEPCR	Phosphoenolpyruvate carboxylase (PEPCase) mRNA
MZEPLTP	Phospholipid transfer protein mRNA
MZEPOD	Pyruvate, orthophosphate dikinase mRNA
MZERBCS	rbcS gene for RuBisCo small subunit
MZEREGG	Lc regulatory protein mRNA
MZESOD2A	Superoxide dismutase 2 (SOD2) mRNA
MZESOD3I	Superoxide dismutase-3 isoenzyme mRNA
MZESUSYSG	Sucrose synthase gene (shrunken)
MZETPI1	Triosephosphate isomerase 1, exon 1
MZEWAXY	Amyloplast-specific transit protein (waxy locus)
MZEZE15A3	15kD zein
MZEZE15G	15kD zein
MZEZE16	16kD zein
"19 & 22 kD ZEIN GENES"	
MZEI19	19kD zein
MZEZE19A	19kD zein
MZEZE19B1	19kD zein
MZEZE19C1	19kD zein
MZEZE19C2	19kD zein
MZEZE19D1	19kD zein
MZEZE22A	22kD zein
MZEZE22B	22kD zein
MZEZEA20M	19kD zein
MZEZEA30M	19kD zein
MZEZEAZ124	19kD zein
MZEZEPCM1	22kD zein
MZEZEZ4G	22kD zein
MZEZEZG3A	22kD zein

Table 2. Table of codon usage with the number of occurrences of each codon, and the occurrences per 100 codons for the same amino acid.

		OTHER NUCLEAR GENES		19 AND 22 kD ZEIN GENES	
Amino Acid	Codon Used	Occur-rences	% Usage	Occur-rences	% Usage
Arg	CGA	34	4	3	8
	CGC	332	39	0	0
	CGG	134	16	4	11
	CGT	80	9	3	8
	AGA	48	6	2	5
	AGG	231	27	25	68
Leu	CTA	39	3	130	20
	CTC	466	36	70	11
	CTG	515	40	79	12
	CTT	165	13	141	22
	TTA	9	1	70	11
	TTG	104	8	157	24
Ser	TCA	59	7	60	26
	TCC	272	30	30	13
	TCG	174	19	18	8
	TCT	88	10	62	27
	AGC	269	30	49	21
	AGT	45	5	13	6
Thr	ACA	77	10	34	33
	ACC	361	47	39	38
	ACG	217	28	14	13
	ACT	117	15	17	16
Pro	CCA	159	17	148	48
	CCC	268	29	56	18
	CCG	347	38	23	7
	CCT	144	16	80	26
Ala	GCA	147	10	137	29
	GCC	598	39	99	21
	GCG	459	30	61	13
	GCT	311	21	179	38
Gly	GGA	151	12	4	7
	GGC	610	50	14	24
	GGG	254	21	3	5
	GGT	211	17	37	64
Val	GTA	41	4	24	19
	GTC	411	37	19	15
	GTG	500	44	64	50
	GTT	172	15	20	16
Lys	AAA	97	13	5	38
	AAG	673	87	8	62
Asn	AAC	392	81	132	89
	AAT	90	19	17	11
Gln	CAA	67	12	407	69
	CAG	496	88	185	31
His	CAC	265	73	15	45
	CAT	96	27	18	55
Glu	GAA	165	17	15	100
	GAG	780	83	0	0
Asp	GAC	552	76	6	67
	GAT	176	24	3	33
Tyr	TAC	381	89	82	77
	TAT	48	11	24	23
Cys	TGC	242	86	21	58
	TGT	39	14	15	42
Phe	TTC	493	85	106	63
	TTT	87	15	63	37
Ile	ATA	42	6	35	24
	ATC	476	73	56	39
	ATT	134	21	53	37
Met	ATG	391	100	69	100
Trp	TGG	187	100	1	100
Term.	TAA	7	17	0	0
	TAG	10	24	14	100
	TGA	24	59	0	0

results in Table 2 show the number of occurrences of each codon for an amino acid, and the number per 100 codons for that amino acid. It is interesting that the most frequently used codon for the majority of nuclear genes, GAG, is entirely absent in the table for the 19 and 22 kD zein genes.

To test the ability of the tables in distinguishing the correct reading frame of a sequence, and whether the sequence utilized a "19 & 22 kD zein" or "other nuclear gene" codon preference, the tables were used to analyze several maize sequences whose reading frames were known. The results are shown in Table 3. As in our earlier report, the scoring was calculated as follows: for each codon in the sequence a score was assigned as the percent occurrence of that codon divided by the highest possible percent occurrence of any codon for that amino acid, as determined by the table. The scores for all codons in the sequence to be scored were summed and divided by the total number of codons to give a percent similarity to the table. The genes in Table 3 were scored in each of the three reading frames,

Table 3. Scoring of some maize coding regions on the basis of the codon usage tables (highest score in bold print).

	Score for reading frame based on codon usage for:					
Gene tested	(other nuclear genes) 1	2	3	(19 + 22 kD zeins) 1	2	3
MZEA1G	**77**	55	50	**46**	50	49
MZEGGST3	**77**	60	56	**48**	49	46
MZEGLB1SA	**74**	62	45	**44**	49	41
MZEGLUT2E	**74**	72	57	46	46	66
MZEH3C4	**79**	58	55	**42**	54	46
MZELHCP	**84**	57	48	**48**	49	46
MZEPEPCR	**73**	58	46	**49**	54	49
MZERBCS	**89**	53	51	**54**	56	48
MZESUSYSG	**81**	57	47	**52**	48	43
MZEZE15G	**83**	68	50	**48**	52	50
MZEZE16	**79**	68	54	**43**	46	60
MZEI19	**47**	60	49	**76**	64	70
MZEZE19A	**46**	62	48	**76**	63	67
MZEZE22A	**51**	65	46	**73**	68	65
MZEZE22B	**49**	63	45	**71**	67	65

over the same region, using both of the tables. The first frame is that which has been identified as the coding frame. All nuclear genes tested, as well as zeins of the 15-16 kD classes, are correctly distinguished by the "other nuclear gene" table. In contrast, however, the coding frames of zeins of the 19 and 22 kD classes are not correctly distinguished by a codon usage table made from other nuclear genes, but are distinguished by the "19 & 22 kD zein" table. Note that correct reading frames of two other seed storage proteins tested (MZEGLUT2E and MZEGLB1SA) are also correctly distinguished by the "other nuclear gene" table. The alternate coding preference raises important questions about the evolutionary origin of the 19 and 22 kD families of zein genes.

We thank David Bashe for his program to calculate reading frame scores in Table 3.

Permission of the authors is not required for citing the codon usage tables.

AMES, IOWA
Iowa State University

Another recording of excision to a closely linked site

--Peter A. Peterson

There have been numerous reports and authentications of transposons transposing to linked sites. Notably, the case with *P-VV*, *a-m(Au)*, *a-m(papu)*, *bz-m* etc. This is another case. This one from the *a2-m55064* allele that reverted to *A2-Rev*. This *A2-Rev* was crossed to *a2-m(r)* and back crossed by *a2 bt*. The following progeny types were observed:

Cld.	cls.	spt.	Total	% spt.	Exchange
106	86	12	204	5.8	± 12%

In this one case, the *En* transposed 12cM from *A2*.

En2 - how much transposase?

--Peter A. Peterson

En2 (*wx-m86246x*) is a derivative of the original *wx-m844* allele. It is described as a large deletion that includes ORF2 and part of ORF1 of the *En* transposon. In an examination of progeny of the cross *a-o Sh2 wx* x *a-m-1 sh2/a-m(Au)-1a, wx-m86246x/wx* the progeny kernels are expected to have very low *wx* mutability and an occasional aleurone spot. This is what is found except in one kernel there was an early occurring *Wx* sector. This is not surprising but what was surprising was that within the *Wx* sector, $a \rightarrow A$ mutability was very high. This sector of high mutability is a rare occurrence. What is striking is that the *a* to *A* sectoring overlies the *Wx* sector. What this implies is that what triggered the early *Wx* sector retained its activity and thereby acted on the *a* allele. Whether it was the *En2* allele that had this burst of activity or some quiescent *En* that was activated cannot be determined because the sector was not heritable.

a-m(Au)-pale-m(r) derivatives

--Peter A. Peterson

A number of derivatives of the *a-m(Au)* allele are non-responsive to *En* colorless or pale types. Some, however, are responsive to *En*. They are similar to the *a-m-15719* that was described by McClintock that is colored in the absence of *En/Spm* but is suppressed with *En/Spm* and shows mutability. The *a-m-15719* allele is in the second exon of the *A1* gene and the explanation for the coloration of the *A1* gene with the *I* insert was based on the position of the insert--namely in the last and next to the last nucleotide (3') of the second exon. Appropriately located splice sites splice out the insert. The *a-m(Au)pale-m(r)* derivatives are at position 336 into the second exon with an orientation the same as that of the *A1* gene promoter. Thus, there must exist similar splice sites in this region of the second exon.

c-m897210 - high frequency of pale sectoring

--Peter A. Peterson

The position of an element in a gene should be significant as to the products that are derived following excision events. This has already been determined for the *c* locus using only a few inserts and determining the phenotypic consequences when related to the putative protein (Franken, Phillip, Köln, unpublished). Such is the case with *c-m897210*. First, this allele gives rise to a high frequency of colored derivatives (see table). Secondly, many of the sectors are pale. This implies that many of the excision events modify the protein, making it less efficient in its control of the anthocyanin pathway.

c-m/c-s or *c-m/c sh wx* x *c sh wx/c sh wx*

	Colored	Spotted	Colorless	Total
c-m/c-s	30	59	82	171
*c-m/c sh wx**	17	75	33	125

**sh* progeny not presented.

a-m(Au)866371 - lacking the *S* function of *En*

--Peter A. Peterson

This derivative arose from the *a-m(Au)* allele (at the *A* locus with a fully functional and excising *En*). The progeny of the cross (3aa) of this allele to *a-m-15719* was colored (pale) with superimposed very late spotting. A backcross x *a-o/a-o* confirmed the *a-m-15719* allele and the late spotting allele. Further test of this *a-m-15719* allele with *En* confirmed that it was valid in that it was suppressible. The progeny tests of this 3aa spotting indicate that it is autonomously mutable (3aa) at the *A* locus.

These tests (genetic) indicate that a fully functional *En* has changed its state to one that is 3aa (mutating) and *S* in lacking the capacity to suppress the *a-m-15719* allele. Thus in this instance, the *M* function was retained with modification (3aa) but the *S* function was modified or eliminated (modified for *S* is not certain since the degree of paleness of *a-m-15719* cannot readily be objectively judged).

tnpR - one form can be *En*; is this a possible reverse dosage

--Peter A. Peterson

tnpR is an *En*-related product that reduces the excision frequency of autonomously functioning alleles such as *wx-844* (Cuypers et al.) or *a-m(Au)*. Cuypers et al. identified the *a-m(r)* allele with a 1.8 kb transcript that reduces excision frequency. The phenotype of *a-m(Au)/a-m1 sh2* equals a low near colorless kernel with low frequency (2b) spots. This indicates that an *En* was suppressing the *a-m1* expression. When selfed many of the *sh2* kernels were colorless with spots. When backcrossed to the *a sh2* tester, *a-m(Au)* reappears. Taken together, this would indicate that an *En* could act as *tnpR* or that a functioning, though modified, *En* could act as *tnpR*.

Tunicate is cell autonomous

--Peter A. Peterson

Tunicate (*Tu*) on chromosome four is a dominant gene that is typified by the growth of long glumes completely enclosing the kernels. In a survey of putative *Tu* mutants, a number of ears were observed with the partial loss of the *Tu* phenotype. This partial loss varied from a single ear row to one that covers 1/3 to 1/2 of the ear. The sharp boundaries of these sectors indicates that the genetic control of this phenotype is cell autonomous much like pericarp variegation of *P-VV* and unlike the non-cell autonomous expression of *Tp1* (Poethig, Nature 336:82, 1988).

c-m888104 - Determination of autonomous-mutability

--Peter A. Peterson

When mutable alleles give a high frequency of colorless derivatives in testcrosses, there is a suspicion that the control is by many functional elements on a reporter allele. When a receptive allele is isolated, the suspicion is confirmed. Another tack to test the distribution-of-many-ele-

ments concept is to test the residual progeny for the element.

Since *c-m888104* is *En*-controlled, the colored progeny from the cross of *C/c-m* x *c sh wx* were crossed by the *En* reporter *c-m(r)*. In 11 tests in two families no *En* were detected. This would support the contention that *c-m888104* is autonomously mutable and the high frequency of colorless derivatives are derivative products from this allele.

Tp-889703-1: Environmental effect on expression

--Peter A. Peterson

Tp-889703-1 has an ear type that is similar to *Tp1*. It arose as a single event in a genetics nursery. It is a dominant mutant. In the greenhouse, either in pots or in a ground bed, it does not have an unusual growth habit, yet is distinguishable from normal non-mutant plants because of thin leaves and a distinguishable tassel. In the field, however, these mutants have a distinguishable corn-grass appearance with heavy tillering (in some cases, upwards of 15). Though it appeared in an *En* plot, no system has been assigned to it yet.

c2-m884259Y - complementation between two non-autonomous elements yields a functional *En*

--Michael G. Muszynski and Peter A. Peterson

The *c2-m884259Y* allele was thought to carry a receptor element that responded to an independent regulator (MNL 64:9). Results from recent crosses have led to a modification of this hypothesis. Spotted kernels of genotype *c2-m/c2, Wx/wx-m8*, were crossed by an *a-o wx* line and a *C sh bz wx* line to generate F1's for system tests. The expected genotypes from those crosses are: 1/4 *C2/c2 Wx/wx*, 1/4 *C2/c2 wx-m8/wx*, 1/4 *C2/c2-m Wx/wx*, and 1/4 *C2/c2-m wx-m8/wx*. Kernels on the progeny ears were colored due to the *C2* from the tester lines. Because of the *c2* dosage effect, two classes of colored kernels could be distinguished: kernels which were only colored (*C2/c2*) and kernels which were colored plus spots (*C2/c2-m*). Because the tester lines were both *wx* and the reporter allele *wx-m8* was segregating, half of the kernels could be scored for the presence of *En* by assaying for *wx* mutability. If *En* is unrelated to the *c2-m* mutability, there will be no correlation between spots and *wx* mutability. If one *En* is segregating and controls the *c2-m* mutability, the following ratios would be expected:

	C2/c2 *Wx/wx*	*C2/c2* *wx-m8/wx*	*C2/c2-m* *Wx/wx*	*C2/c2-m* *wx-m8/wx*
+En	1/8 Cl Wx	1/8 Cl wx-m	1/8 Cl+sp Wx	1/8 Cl+sp wx-m
-En	1/8 Cl Wx	1/8 Cl wx	1/8 Cl Wx	1/8 Cl wx

The following ratios were observed:

Cl Wx	Cl wx	Cl wx-m	Cl+sp Wx	Cl+sp wx	Cl+sp wx-m
3/8	3/8	0	1/8	0	1/8

All Cl+sp kernels are also *wx* mutable; there are no Cl+sp *wx* kernels, thus *En* is implicated in controlling mutability.

The *En* may be independent of the *c2-m* allele or linked. If an independently segregating *En* is controlling mutability, then a Cl *wx-m* class should also be segregating (*C2/c2 wx-m8/wx En/-*). Clearly this class is missing and the amount expected for this class has been incorporated into the Cl *wx* class. If *En* was closely linked to the *c2-m*, then the expected ratio for the Cl+sp *wx-m* class would be closer to 1/4. Any crossovers would be classified as Cl *wx-m* and they are not present. Neither of these hypotheses fits the observed data.

The data do indicate that an independent factor controls spotting at *c2-m* and triggers *wx-m8*, implicating *En*. But this factor does not elicit a response from *wx-m8* when it is separate from the *c2-m* allele. Therefore, this factor only has *En* function when it is together with the *c2-m* allele. Since neither the factor nor the *c2-m* provide an *En* function by themselves, they must be classified as non-autonomous elements. Because *En* is implicated, the element at *c2-m884259Y* and the independent factor are defined as *I(dSpm)* elements. To date, all *I*'s have been found to be deletion derivatives of *En* that have lost sequences which encode *En* specific functions. These two *I* elements may possess complementary deletions that allow them to function together as an intact *En*. Frey et al. (EMBO J. 9:4037, 1990) have demonstrated that two components (TNPA and TNPD) are needed for complete *En* function in transgenic tobacco. This mutant may be an "in maize" demonstration of this aspect of *En* function. Also either the *c2-m* allele or the factor needs to be present in at least two doses, since neither spots nor *wx* mutability is male transmitted. Tests of the dosage effect and the modified hypothesis are in progress.

c2-m881058Y - an example of three factor control

--Michael G. Muszynski and Peter A. Peterson

The *c2-m881058Y* allele (MNL 64:9) was previously shown to be non-autonomously controlled. To investigate the relationship to *En(Spm)*, this mutant was crossed to a colored *wx* line. The F1 (*C2/c2-m Wx/wx*) was backcrossed to a *c2/c2 wx-m8/wx-m8* tester . The results from these crosses indicate that *En* was present. If *En* is responsible for the *c2-m* mutability, then all spotted kernels should also be *wx*-mutable (*wx-m*) and all colorless kernels should be *wx*-stable (*wx-st*). If the *En* is unrelated to the *c2-m* and another factor is controlling spotting, there should then be no correlation between spots and *wx* mutability. The expected results if *En* is unrelated and the observed results are summarized as follows:

		Expected		Observed	
En	Factor	*c2-m*	*wx-m8*	*c2-m*	*wx-m8*
+	+	sp	wx-m	sp	wx-m
+	-	cl	wx-m	cl	wx-m
-	+	sp	wx-st	cl	wx-st
-	-	cl	wx-st	cl	wx-st

There are several interesting features here. All spotted kernels are *wx*-mutable, but the presence of the colorless *wx*-mutable class indicates that *En* alone is unable to trigger spotting. Further, the lack of a spotted *wx*-stable class indicates that a separate factor alone is also not able to trigger spotting. Therefore, this unstable allele requires *En* and an independent factor. It may be jointly controlled. This would be the first instance of three factor control (the

locus, *En* and a *non-En* factor) of mutability. Tests to confirm this hypothesis are underway.

A *c2-m::En-low* and two modifiers of mutability

--Michael G. Muszynski and Peter A. Peterson

The original phenotype of the *c2-m881058P* allele (MNL 64:9) was medium spotted (2-6 aa-b, Reddy and Peterson MGG 194:124, 1984), but upon outcrossing three mutable phenotypes segregated: high (6-8 aa-d), medium (2-6 aa-b) and low (1-3 aa-a) in approximately equal ratios. Kernels (*c2-m/c2*) were selected from each class and crossed by and on *c2/c2*. Each class segregated as follows:

Phenotype	Segregation on Outcross Ear			
Planted	High	Medium	Low	cl
High	1/4	-	1/4	1/2
Medium	1/8	1/8	1/8	5/8
Low	-	-	1/2	1/2
colorless	-	-	1/4	3/4

The only class which bred true was the low spotted class and also segregated 50% on an outcross, thus the *c2-m* allele contains a regulatory element which conditions low mutability. Two factors which modify this low spotting are independently segregating in this mutant. One factor (*Stimulator*) stimulates the *c2-m* low spotting to high spotting and segregates upon outcrossing plants from high spotted kernels. Another factor (*Restrainer*), which restrains the high spotting to medium and the low spotting to colorless, is also segregating. Both factors are unlinked to the *c2-m* allele and to each other.

F1 kernels (*C2/c2-m A1/a1-o Wx/wx ± Stim.*) were backcrossed to *c2/c2* and *a1-m(r)/a1-m(r)* tester lines to test for *En(Spm)* relation. The results, on a per plant basis, are summarized as follows:

Cross to *c2* Indicates	Cross to *a1-m(r)* Indicates
1. *c2-m*	1 *En-low*
2. *c2-m* + 1 *Stim.*	1 *En-low* and 1 *En-std*
3. *c2-m* + 2 *Stim.*	1 *En-low* and 2 *En-std*
4. *C2* + 1 *Stim.*	1 *En-std*

These data indicate that the element at *c2-m881058P* is a weak or low *En*. The *Stimulator* is a standard *En*, which can compensate for the *En-low* and boost the mutability to a high spotting pattern. The *Restrainer* is probably a suppressor of mutability similar to the *I* element of *a1-m(r)102* (Cuypers et al., EMBO J. 7:2953, 1988), which restrains the high mutability to a medium pattern and the low *c2-m::En-low* spotting to colorless. System tests to other element systems were negative.

V*-m894905-11 - a dominant virescent mutable

--Michael G. Muszynski and Peter A. Peterson

In a row planted with sectored kernels (*C2-b857246/c2*) from the previously reported *C2* breaker (MNL 64:9), a variegated plant appeared among 14 normal green plants. The youngest leaves inside the whorl are a pale yellow-green with dark green clonal sectors. This variegation was lost as the leaves matured and turned a normal dark green. The variegation in the leaves of the tiller was more extreme.

This exceptional plant was selfed and outcrossed onto *c2/c2*. Progeny plants from the selfed and outcrossed ears also showed this same variegation. Since the original kernel came from an outcross and the mutant phenotype is heritable by outcrossing, this mutant has been designated *V*-m* for a dominant, virescent mutable.

The progeny plants from the original outcross and self are segregating for a stable dominant virescent phenotype as well as the green and *V*-m* phenotypes.

Cross	green	virescent	virescent mutable
C2-b/c2 V-m/+* self	9	1	9
C2-b/c2 V-m/+* x *c2/c2*	13	4	4

Because of the limited sample size no definite conclusions can be made, but the presence of the three phenotypes indicates two-factor control of mutability. There was no correlation between kernel phenotype (Cl, sect or cl) and plant phenotype. It would therefore appear that the factor controlling *C2* breakage is unrelated to the virescent mutability. Studies on the heritability of plant phenotypes and system tests are in progress.

Rp Tagging with *Uq* transposable element

--Ru-Ying Chang and Peter A. Peterson

Transposable elements insert into random sites in the genome. The frequency that a transposable element inserts into a particular locus usually ranges between 10^{-5} to 10^{-6}. In order to tag a disease resistance gene such as *Rp*, a functioning element is introduced into a homozygous dominant line for the particular gene, e.g., *Rp-Rp* (in this case, the *Rp-D* allele). This is then made homozygous by selfing and selecting for both the dominant trait and element activity. Once the homozygous dominant line with an element is obtained, it can be crossed by a recessive tester as shown below. The progeny seedlings (*Rp rp*) are then tested for resistance and screened for a mutant type which is expected to be susceptible.

In this experiment, the *Uq* (along with others) element was introduced into the *Rp/Rp* line. Following the procedure described in the preceding paragraph, the resulting *Rp/Rp Uq/Uq* line was then crossed by *rp-o/rp-o* to produce seeds for screening.

The resulting offspring are resistant. However, if the element has inserted into the *Rp* gene (designated *rp-m*), a susceptible individual will be produced. This seedling test uncovers any susceptible individuals which will be candidates for insertional mutations. The screening results are shown in Table 1.

Table 1. Screening results for *Rp* tagging

1) Total seedlings screened	600,826
2) Seedlings from contaminated suscept. lines (discarded)	142,738
1)-2)	458,088
Mutants found	25
Mutation rate	5.5×10^{-5}

The susceptible lines (2) are probably due to contamination of pollen or to misclassification in making the *Rp/Rp UqUq* line. These seedlings are subtracted from the total because they do not contribute to the population. The mutants found are designated *rp-m*-1 through *rp-m*-25.

After the mutants are obtained they are verified by a

co-segregation test. Two types of mutants are expected as to the status of *Uq*. One is co-segregating with *Uq*, i.e., *Uq* inserted into the *Rp* gene; the other is with a receptor element controlled by an independent *Uq*, i.e., *Uq* is outside the *Rp* locus. In the *Uq* insert case, a strategy is used to test co-segregation. Figure 1 shows the strategy and the expected results for the co-segregation test.

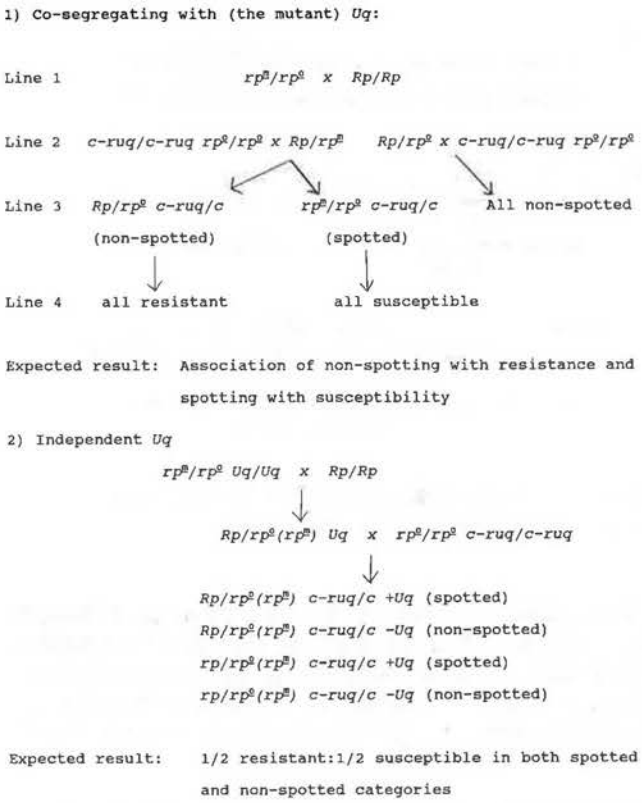


Figure 1. Tests of co-segregation.

Table 2. Results of co-segregation test for *Uq**

Mutant	Spotting	# of F1 or S1 ears tested	Uq status
RpM-2	-	1	
-3	-	7	
-4	+	10	Independent
-5	-	10	
-6	+	7	Independent
-7			Died
-8	+	2	Independent
-9	+	7	Independent
-10	-	3	
-11	+	5	Being tested
-12	+	6	Independent
-13	+	3	Independent
-14	+	1	Independent

* *RpM*-15 to *RpM*-25 are still being tested.

The two cases can be distinguished readily. In the former, rows from spotted kernels are susceptible and rows from non-spotted kernels are resistant. In the latter, all rows will be mixtures of susceptible and resistant seedlings. In the latter element relation can not be determined. The results of co-segregation tests are shown in Table 2.

Of the 25 mutants identified in the screening test, 2 died, and 12 have been tested for co-segregation. Four out of the 12 tested had no element activity at all. The other 8 had element activity. But element activity was independent of the mutant phenotype. The remaining mutants are currently being tested.

There are a few possible reasons for the mutants with independent *Uq*. First, the mutants might be due to spontaneous mutation of *Rp* to *rp*. Secondly, the element in *Rp* is a defective derivative of the *Uq* element.

The identity of the alleles in the mutants is being verified by RFLP analysis in Köln,, i.e *rp-m* vs *rp-o*-line 2 in Figure 1. This analysis is to distinguish the *rp-m* vs. *rp-o* types following outcrosses to *Rp-Rp*.

Chromosome labelling with transposable elements

--Ru-Ying Chang and Peter A. Peterson

Transposable elements randomly insert into a specific locus at a frequency of 10^{-6} to 10^{-5}. In previous studies the *En* element has been shown to insert more frequently into closely linked sites than to other chromosome sites (P.A. Peterson, TAG 40:367-377,1970; E.M. Nowick and P.A. Peterson, MGG 183:440-448, 1981). Thus it is appropriate to place an *En* element onto each of the 20 chromosome arms in maize. This is called chromosome labelling. After an arm has been labeled, genes existing on the arm and targeted for isolation will have a greater chance of becoming the recipient of the element insertion.

Five *En*-containing alleles [*a-m-(papu)*, arm 3L; *a2-m-655064*, 5S; *c2-m-1*, 4L; *c2-m-826019*, 4L *and wx-84-4*, 9S] are used to label all the 20 chromosome arms of maize. They are linked to the arms with the assistance of translocations. Homologous pairing, followed by the appropriate cross-over between a translocated segment and its normal counterpart, will enable us to relocate an En allele in the genome (R-Y. Chang and P.A. Peterson, Abstracts of Life Sciences Symposium, Iowa State University, 1989; S. Dash and P.A. Peterson, Maydica 34:247-261, 1989). The strategy used is shown in Figure 1.

The *a-m(papu)* line is first crossed with the translocation line, which has the *A* allele corresponding to *a-m(papu)*. At meiosis, four types of gametes are formed. Testing those gametes by an *a*/*a* tester enables us to select the genotype T *a-m*/N *a* (spotted and pollen-semi-sterile). This genotype is selfed. The resulting spotted and pollen-normal offspring are selfed again and tested to prove the homozygous translocation status (see Figure 1).

The labeling results are shown in Table 1. Seven or eight arms (1S, 2L, 6S, 6L or 6S, 7L, 8L and 10L) are labeled and achieved homozygosity [T6-9(5454) has the break point at the centromere of chromosome 6, but it is not known which side of the centromere it is on]. Those lines have been crossed to normal individuals and all give

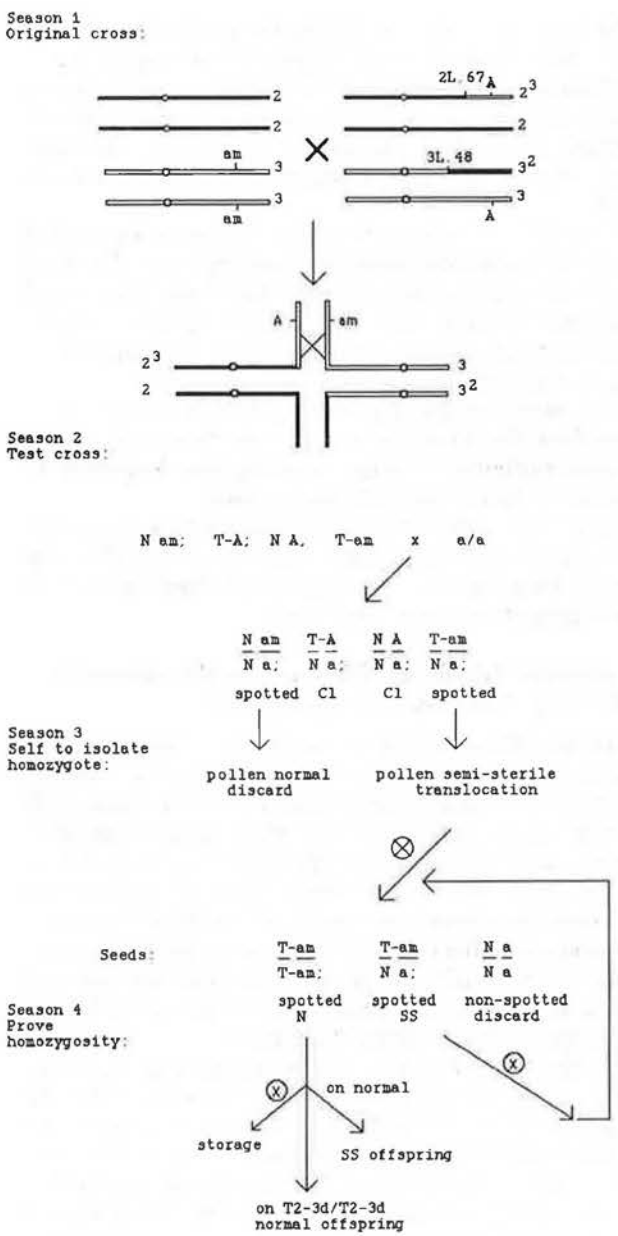


Figure 1. Strategy for chromosome labelling.

Table 1. Results of chromosome labelling

Arm	Mutable allele	Translocation	Labelling status
1S	*a-m(papu)*	T1-3(5597)	homozygous
2L	*a-m(papu)*	T2-3d	"
	a2-m665064	T2-5(032-9)	"
6S	*c2-m-1*	T4-6(033-16)	"
6L or 6S	*wx-84-4*	T6-9(5454)	"
7L	*a-m(papu)*	T3-7(6466)	"
	wx-84-4	T7-9(027-9)	"
8L	*a-m(papu)*	T3-8(4874)	"
8S	*a-m(papu)*	T3-8(043-14)	"
10L	*a-m(papu)*	T3-10(036-15)	"
1L	*a-m(papu)*	T1-3e	heterozygous
2S	*a-m(papu)*	T2-3e	"
4S	*a2-m655064*	T4-5e	"
7S	*a-m(papu)*	T3-7e	"

semi-sterile offspring. A few have also been crossed to the original homozygous translocations. In this case normal offspring are achieved (see Figure 1). Four arms (1L, 2S, 4S and 7S) are labeled in heterozygous condition. Achieving homozygosity is needed. Four arms already contain an element (3L, *a-m(papu)*; 4L, *c2-m*; 5S, *a2-m* and 9S, *wx-844*). Four arms are yet to be labeled (3S, 5L, 9L and 10S).

As an example the labelling of chromosome arm 2L with *a-m(papu)* and *a2-m655064* through T2-3d and T2-5(032-9) is shown in Figure 2.

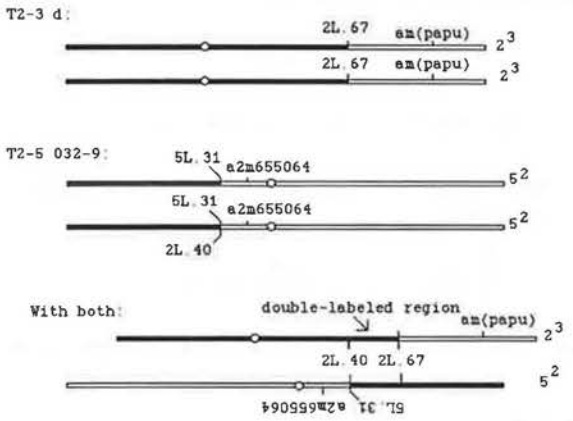


Figure 2. An example of labeled chromosome arms, labelling of 2L with *am(papu)* and *a2m655064* through T2-3 d and T2-5 032-9

With T2-3d, the proximal 0.67 portion of 2L is labeled with *a-m(papu)*. With T2-5(032-9), the distal 0.60 portion of 2L is labeled with *a2-m655064*. With the two translocations, the arm 2L is completely labeled with either *a-m(papu)* or *a2-m*. In fact the portion between 0.40 and 0.67 is labeled twice as shown in Figure 2.

Comparison of the dosage effects and dominance of 10 *C1* alleles

--Elizabeth E. O. Caldwell and Peter A. Peterson

Ten alleles of the *C1* locus (Table 1) were assembled to represent the diversity of expressivity of this anthocyanin pathway gene. The *C-I* alleles (dominant inhibitors) produce a colorless kernel when homozygous and suppress

Table 1. Ten alleles of the C1 locus are listed with their source, type of allele and homozygous phenotype.

Allele	Source	Allele Type	Phenotype
C-I(Cornell)		dominant color inhibitor	colorless
C-I(K55)	inbred K55	same	colorless
C-I(A69)	inbred A69	same	colorless
C-S	B. McClintock*	dominant color producer	colored
C-(hiloss)	Hi Loss B line**	same	colored
C-(Cshbz)	*C sh bz wx* line	same	colored
C-(lineC)	color converted W22	same	colored
c-ruq67	*c-m816667*	mutable (+Uq), colorless (no Uq)	spotted, colorless
c-ruq3	*c-m804531*	same	spotted, colorless
c	*c sh Bz wx* line	recessive colorless	colorless

* via K. Cone
**from M. M. Rhoades

the pigment expression of the *C* alleles. The *C* alleles produce colored kernels both when homozygous and in combination with the recessive colorless alleles. These recessive colorless alleles can be subdivided into mutable and stable classes. The mutable (*c-ruq*) alleles were created by the insertions of the transposable element *Ruq* into a *C* allele. A spotted phenotype is expressed in the presence of either *Uq* or *Ac* and is dominant over the stable recessive *c* allele. In the absence of *Uq* and *Ac*, the *c-ruq* alleles are colorless.

To further describe the expressivity of these alleles, the 10 were intermated (Crosses 1 and 2) to establish their relative dominance and dosage effects. This evaluation of the alleles' relative potency was made using the original lines. All were homozygous dominant for *A1*, *A2*, *C2*, and *R*, but no attempt to control allelic variation at loci other than *C1* was made.

	Female		Male	Embryo	Aleurone
Cross 1	colorless		colored	colorless	colorless
	colorless	x	colored	colored	colorless
					colored
Cross 2	colored		colorless	colorless	colorless
	colored	x	colorless	colored	colored
					colored

Crosses 1 and 2 reveal differences among both the dominant color inhibitors and the dominant color producing alleles. The dosage was controlled by the mating scheme. When *C-S* is present in one dose in the aleurone (Cross 1), only the suppression by *C-I(Cornell)* and *C-I(K55)* is complete. *C-I(A69)* allows limited pigment formation (light pale). When *C-S* is present in two doses (Cross 2), *C-I(Cornell)*, and *C-I(K55)* only partially suppress *C-S* expression (dark pale). *C-I(A69)* allows the fully colored *C-S* phenotype. None of the other colorless alleles tested affect the *C-S* colored phenotype.

Except for the interaction with *C-S*, the *C-I* alleles are indistinguishable when examining progeny with two doses of *C-I* (Cross 1). With one dose of *C-I* (Cross 2), allelic differences emerge. *C-I(A69)* is not able to suppress any of the *C* alleles tested. *C-I(Cornell)* allows a weak pigmentation with *C-(lineC)*. These observations allow the ordering of the *C-I* alleles based on their suppression ability with the strongest listed first: *C-I(K55)*, *C-I(Cornell)*, and *C-I(A69)*. These observations also allow the ordering of the *C-S*, *C-(hiloss)*, *C-(lineC)*, and *C-(Cshbz)* in decreasing order of dominance after the *C-I* alleles.

For the recessive *c* and *c-ruq* alleles, one dose of the allele does not affect the expression of the *C* alleles. The one exception may be *c* in combination with *C-(Cshbz)* or *C-(lineC)*. The progeny of these two crosses appear paler than the other crosses of this group. When the *c-ruq* and *c* alleles are present in two doses the expression of the *C-(Cshbz)* and *C-(lineC)* alleles is reduced. This reduction in color may be slightly less for *c-ruq31* when compared with *c-ruq67*. Therefore, the *c-ruq31* allele is either slightly more dominant than *c-ruq67* or equally dominant. The *c* allele has the least dominance (most recessive) of the tested alleles (note: the *c x C-(hiloss)* progeny are not available.) The order of this allelic series is: *C-I(K55)*, *C-I(Cornell)*, *C-I(A69)*, *C-S*, *C-(hiloss)*, *C-(lineC)*, *C-(Cshbz)*, *c-ruq31*, *c-ruq67*, and *c*.

Uq transposable elements in maize breeding populations

--Thomas Horejsi, Elizabeth E.O. Caldwell, and Peter A. Peterson

Active *Uq* transposable elements have been found in numerous maize breeding populations (Cormack, Crop Sci. 28:941-944). This observation could be explained in two ways. First, one could argue that the *Uq* elements are inadvertently grouped into the populations because they are linked to favorable alleles. If this is the case, one would expect the *Uq* elements to be limited to a few linkage groups. A second probable explanation is that they are specifically incorporated into the populations because they provide a useful function to the maize breeder. This would predict widely dispersed elements.

To address this problem, a diallel cross between 13 breeding populations was used to construct stocks that contained a *Uq* element from two different breeding populations. These stocks were crossed to a *c-ruq* tester as follows:

c-rcq/c-ruq Uq(pop. #1)/+ *Uq*(pop. #2)/+ X *c-ruq/c-ruq*

The results indicate that there is independent segregation in all but two cases. In these two cases there is only a weak linkage of about 40 centimorgans in one and about 30 centimorgans in the other.

The disperse nature of the *Uq* elements adds support to the argument that they are specifically selected and must be advantageous in maize breeding populations.

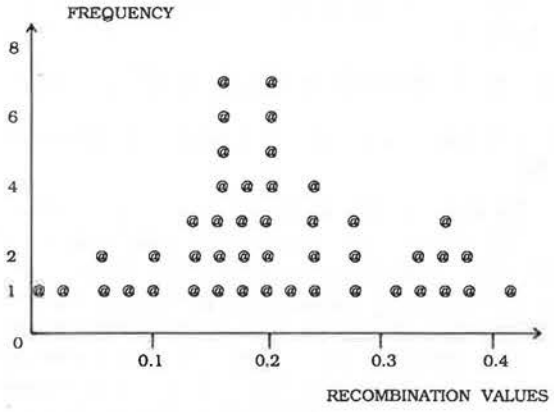
En transposition from the *A1* locus on chromosome 3

--Etienne Kaszas and Peter A. Peterson

This is an extension and amplification of the Nowick and Peterson study (1981). In this study, 190 germinal derivatives from the autonomous allele *a-m(papu)*--see cross below--were isolated to study the transposition of *En* from its initial site at the *A1* locus. The transposition of *En* at the new location was confirmed by crosses of the stable colorless or pale derivatives to responsive tester lines (*a-m(r)* and *a-m1*). Our study, although based only on 190 derivatives, is in agreement with Nowick and Peterson's data (MGG 183:440-448, 1981): 24% of the derivatives do contain a linked *En*, as it appears in the table of percentages below. Around 10% of the lines do contain an independent *En*. The other classes indicate either an *En* loss, or a changed *En* that gave incomplete signals, or two or more *En*s.

Study*	no *En*	*En* indep.	*En* linked	Two or more *En*s	Low freq. of *En*
1	28	12	29	21	10
2	16	9	24	25	26

*1, Nowick and Peterson, 1981: 1627 derivatives analyzed
2, This study, 1988 and 1989: 190 derivatives analyzed



DISTANCE A1 — En

Cross: *a-m(papu)/a sh2* x *a sh2*; progeny expected are 1/2 spotted round and 1/2 colorless shrunken; exceptions are colorless round or pale round.

An estimate of *A1 - En* distance was calculated (direction undetermined), as shown in the figure. This distribution is not random. 60% of the values are in the range of 5 to 20 map units. These results are supportive of Nowick and Peterson (1981), where *En* transposed preferentially between 5 and 15 map units from *A1*. Tests for precise location of *En* are underway.

Cloning of three putative defective kernel loci by transposon tagging

--M. G. James, M. S. Scanlon, D. S. Robertson and A. M. Myers

Approximately 75 defective kernel mutants were generated from crosses with Robertson's Mutator stock. Using *Mu* as a transposon tag, genomic DNA that cosegregates with the mutant phenotype was cloned from three of these mutants (*dek*-1047, dek*-NS807,* and *dek*-NS326*) into phage lambda vectors. Mutant *dek*-1047* (which has a flattened, nearly empty pericarp) was produced by the cross of standard and Mutator plants, and mutants *dek*-NS807* (which has a shrunken, slightly opaque kernel) and *dek*-NS326* (which has a nearly empty seed) were gifts from Nancy Shepherd, E. I. DuPont de Nemours & Co. Another mutant received from Nancy Shepherd, *dek*-NS95*, was found to be allelic to *dek*-NS326*.

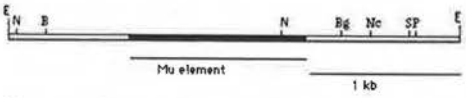


Figure 1. Restriction map of pMS1, containing cloned fragment from *dek*-1047*. E = *Eco*RI; N = *Not* I; B = *Bam*HI; S = *Stu*I; P = *Pst* I; Bg = *Bgl*II; Nc = *Nco*I.

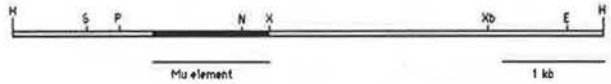


Figure 2. Restriction map of pMJ1 and pMJ2, containing cloned fragment from *dek*-807*. H = *Hin*dIII; S = *Sst*I; P = *Pst*I; N = *Not* I; X = *Xho* I; Xb = *Xba*I; E = *Eco*RI.

Figure 3. Restriction map of pMJ26 and pMJ27, containing cloned fragments of *dek*-326*.. H = *Hin*dIII; P = *Pst*I; S = *Sst*I; N = *Not* I.

All three putative defective kernel loci contain *Mu1*-homologous insertions of approximately 1.4kb, most likely *Mu1*. In at least two instances, sequence analysis of the *Mu*-element junctions shows 9bp genomic DNA repeats. Genomic fragments flanking the *Mu* elements of all three clones were subcloned into plasmid vectors and used as hybridization probes. In each instance, this DNA is single-copy and shows cosegregation with the defective kernel phenotype. Restriction maps for each of the cloned fragments are presented in Figures 1-3.

Positive allele tests among defective kernel mutants

--M. G. James, M. S. Scanlon, P. S. Stinard and D. S. Robertson

Using Robertson's Mutator, a large number of recessive defective-kernel mutants have been produced for the purpose of transposon tagging. As part of their genetic characterization, we have allele-tested many of the mutants with each other and with previously described *dek* mutants (Neuffer and Sheridan, Genetics 95:929, 1980). Tests were conducted on the basis of similarity of phenotype and/or assignment to chromosome arm. Table 1 shows

Table 1. Allelic defective kernel mutants.

Mutant designation	Allelic to:	Chromosome arm	Kernel phenotype	New designation
dek-2608*	*dek7*	4S	shrunken, sugary endosperm	*dek7-2608*
dek-2410*	*dek25*	4S	shrunken endosperm	*dek25-2410*
dek-1566*	*dek25*	4S	shrunken endosperm	*dek25-1566*
dek-2689*	*dek31*	4L	pitted endosperm, germless	*dek31-2689*
dek-25*	*dek5*	3S	shrunken endosperm	*dek5-25*
dek-33*	*dek5*	3S	shrunken endosperm	*dek5-33*
dek-2221*	*dek21, w2*	10L	aleurone mosaic	*dek21-2221*
dek-NS326*	*dek*-NS95*	7L	shrunken endosperm	*dek*-NS326/95*
dek-2162*	*et1*	3L	etched kernel	*et1-2162*
dek-3328*	*et1*	3L	etched kernel	*et1-3328*
dek-5079*	*et1*	3L	etched kernel	*et1-5079*
dek-24*	*et1*	3L	etched kernel	*et1-24*
dek-27*	*et1*	3L	etched kernel	*et1-27*
dek-34*	*et1*	3L	etched kernel	*et1-34*
dek-2320*	*et1*	3L	etched kernel	*et1-2320*
dek-2424*	*et1*	3L	etched kernel	*et1-2424*
dek-2457*	*et1*	3L	etched kernel	*et1-2457*

Mu-induced defective kernel mutants that were found to be allelic to previously described mutants and/or to each other.

Mutator-tagged defective kernel mutants as putative QTL for kernel size

--M.Lee, L.Veldboom, M.Scanlon, A.Myers, M.James and D.Robertson

Mutator-induced defective kernel mutants are being pursued in order to investigate their role in kernel growth and development and to test an hypothesis regarding the relationship between qualitative and quantitative genetic

variation (see D. Robertson, J. Theor. Biol. 117:1-10, 1985; T. Helentjaris and D. Shattuck-Eidens, MNL. 61:88-89, 1987). In this note, we describe initial results of the test of the possible contribution of *dek* mutants to quantitative genetic variation for kernel size.

Briefly, Robertson's hypothesis states that a locus may contribute to quantitative or qualitative variation for a trait depending upon the allelic forms at the locus. For example, null alleles would presumably represent qualitative Mendelian variation while functional alleles of the same locus, perhaps coding for more or less effective or efficient gene products, contribute to quantitative genetic variation (quantitative trait loci, QTL) by differences in degree of trait expression. If true, the hypothesis further suggests the swiftest, surest routes to understanding the genetic basis of many polygenic traits, identifying and characterizing key genetic factors, may be created through studies of genetically well-defined, easily characterized mutants. While the basis of the hypothesis may seem obvious, and perhaps trivial, obtaining physical and genetic evidence has been difficult. With the advent of maize RFLP mapping and more routine transposon tagging coupled with decades of maize genetic research, such evidence may be within reach.

To test the hypothesis, Mutator-induced *dek* mutants will be tagged and cloned. The clones will be used as probes, mapped relative to known, previously placed RFLP probes and evaluated for their contribution to kernel size in RFLP analysis of an F2 population created by crossing inbreds with relatively large differences for kernel size (Mo17 and H99; 95 and 50 grams per 300 kernels, respectively). To date, two *dek* mutants have been tagged, *dek*-807* and *dek*-1047*, and co-segregating genomic clones have been obtained.

The *deks* have been mapped genetically through RFLP mapping and physically using B-A translocations. RFLP mapping with MAPMAKER located *dek*-807* to chromosome 5, 8 cM distal to *UMC51* and 12 cM proximal to *UMC68*. *dek*-1047* was placed to chromosome 2, 9 cM distal to *PIO1012* and 14 cM proximal to *UMC131*. Chromosome placement of *dek*-1047* was in agreement with the physical location revealed by the translocations (see note by M. Scanlon et al. - this issue). Molecular and biological characterizations of *dek*-807* and *dek*-1047* are in progress.

The chromosome regions defined by the *dek* clones and other adjacent RFLP loci will be evaluated for their contribution to genetic variation for kernel size among 150 F3 families derived from the Mo17/H99 F2 population. Initial analysis indicated the *dek* clones may be identifying loci or chromosome regions affecting kernel size. Comparison of average kernel weight (gms/300k) for the three genotypic classes (H99/H99, Mo17/H99, Mo17/Mo17) revealed the following: 76, 77, and 79gms/300k for *dek*-807* and 75, 78, and 79 gms/300k for the *dek*-1047* probe. While these differences are clearly very small, the trend is in agreement with the hypothesis. Additional statistical analyses of traits possibly affecting kernel size and of RFLP probes in regions linked and unlinked to *dek*-807* and *dek*-1047* are in progress. The *dek* clones will be evaluated in other populations. Also, other *dek* mutants are being tagged and cloned. The availability of Mutator-induced mutants for other traits, such as plant height, may allow us to extend our investigation of the molecular and biological basis of the relationship between qualitative and quantitative genetic variation.

Genetic placement and mapping of Mutator-induced defective kernel mutants

--M. J. Scanlon, M. James, P. S. Stinard and D. S. Robertson

For use in transposon tagging experiments of seed developmental loci we have generated a large number of defective kernel mutants (*dek*'s) of these putative *Mu*-induced *dek*s. Towards this end we have utilized B-A translocations, the waxy series of reciprocal translocations, linkage analyses and tests of allelism to previously described mutants to map or place a number of these *dek*s. In the accompanying columns are listed those *dek*s which have been placed to chromosome arm. Those *dek*s which have tested positive for allelism to a known endosperm mutant are listed according to our original designations, followed by the name of the previously described mutant in parentheses. More detailed map locations are indicated in the footnotes.

1S	1L	2L	3S
dek-2115* *dek*-2045*[a] *dek*-8319*	*dek*-6214* *dek*-1563*[b]	*dek*-4160* *dek*-2444* *dek*-1365-6* *dek*-1047*[c] *dek*-2159*[d]	*dek*-1364* *dek*-1185*[e] *dek*-33 (dek5)* *dek*-2525 (dek5)*
3L	**4S**	**4L**	**5L**
dek-5079 (et1)* *dek*-3328 (et1)* *dek*-2424 (et1)* *dek*-24 (et1)* *dek*-2320 (et1)* *dek*-2352 (et1)* *dek*-2457 (et1)* *dek*-2162 (et1)* *dek*-27 (et1)* *dek*-34 (et1)*	*dek*-2058* *dek*-2410 (dek25)* *dek*-1566 (dek25)* *dek*-2608 (dek7)*	*dek*-2689 (dek31)*	*dek*-2146* *dek*-1182* *dek*-5133* *dek*-8186*[f] *Dap1*[g]
6L	**7 ctr.**	**7L**	**8L**
dek-1104* *dek*-1184*[h]	*dek*-2082*[i]	*dek*-3193* *dek*-95*[k] *dek*-326*[k] *dek*-5153*	*dek*-5132*
10S	**10L**		
dek-2424-9* *dek*-2181*	*dek*-1339*[j] *dek*-8627* *(dek21/w2)*		

[a] *dek** - *wx* T1-9(8918)=31.57±3.04
[b] *dek**- *bz2*=10.06±1.77; *dek** - *Kn*=17.05±1.89; *dek** - *bm2*=25.37±2.01
[c] *dek** - *wx* T2-9d=18.46±1.55
[d] *dek** - *wx* T2-9d=18.46±1.55
[e] *dek** - *cl1*=29.67±2.78
[f] *dek** - *ae*=12.50±1.37; *dek** - *wx* T5-9a=20.83±1.37
[g] *dek** - *wx* T5-9a=3.57±0.27
[h] *dek** is tightly linked to *y1*
[i] *dek** - *wx* T7-9(4363)=34.72±2.89
[j] *dek** - *o7*=32.03±3.58; *dek** - *wx* T9-10b=31.37±2.16
[k] *dek**-95 and *dek**-*326* are allelic and were both kindly provided by Nancy S. Shepherd of E. I. Du Pont De Nemours & Co.

Mapping regulators of somatic mutability of Mutator-induced *a1* alleles

--Donald S. Robertson and Philip S. Stinard

In last year's Newsletter (MNL 64:13, 1990) we pre-

sented preliminary data indicating that a regulator of the somatic mutability of the Mutator-induced *a1-Mum3* allele was located on chromosome two. These data were based on only a few crosses involving *wx* T2-9d (2L.83, 9L.27). Additional kernels from the F1 ears (*wx* T2-9d/*a1-Mum3*) that had indicated linkage were planted in 1990 and the resulting plants crossed to *a1 wx* stocks. In 1989, a new set of crosses was made to the waxy marked translocation series using plants grown from mutable kernels of outcross ears segregating for one regulator. These involved *a1-Mum2* and *a1-Mum3* stocks, for which we had not previously obtained any positive tests for linkage. In the winter nursery of 1989-90, the F1's of this new set of crosses were testcrossed to *a1 wx* stocks. The resulting ears were screened for indications of linkage of a regulator with *wx*. Kernels from ears yielding F1 plants that showed linkage in the winter nursery were planted in large numbers (30 to 100 kernels, depending upon the F1) in the summer of 1990 and the resulting plants were testcrossed to *a1 wx* testers.

The results are summarized in Table 1. Although all the plants used for crossing to the translocation series grew from mutable kernels of ears segregating in a 1:1 ratio for mutable and stable kernels, some of the progeny, when tested, gave ratios that indicated there had been replication of the regulator resulting in plants with two or three regulators. The outcrosses of these plants to the translocation series were also grown to determine if linkage of a regulator with more than one translocation could be demonstrated.

The crosses of *a-Mum2* with only one element linked to waxy (Table 2) showed some variation in the amount of crossing over; from a low of 5.43% to a high of 19.40%. There seem to be three levels of recombination (i.e., 5-10%, 10-15%, 15-20%) based on a statistical analysis of the differences between the different recombination values. If these are real differences in recombination, as the statistics indicate, these data would suggest that the regulator has transposed from one location on chromosome two to another. Based on the raw data, the average crossing over for all seven crosses is 12.89%. However, in most crosses there is a deficiency of waxy kernels. The reduced transmission of the *wx* allele by heterozygous *Wx wx* plants has been reported in the literature. The "corrected totals" in this and other tables have been calculated by increasing the waxy classes to make them equal to the starchy classes. The added waxy values were distributed among the mutable and stable classes in the same proportion they were observed in the raw data. With this correction in Table 2, the average percentage crossing over is increased

Table 1. Summary the testcrosses al al wx wx x wx T/al-Mum, which segregated for mutability.

al-Mum allele and translocation	No. of ears with less than 50% mutable kernels	No. of ears with wx and mutability showing independence	No. of ears with linkage of wx with one regulator	No. of ears with linkage of wx with one regulator plus one independent regulator	No. of ears with linkage of wx with one regulator plus two independent regulators	No. of ears with insufficient data	No. of ears with non-conforming ratios	Total
T2-9d/al-Mum2	5	7	7	5	4	1	2	
T4-9e/al-Mum2		9		1	2			
T4-9b/al-Mum2		11		1	1			
T2-9d/al-Mum3	7		5		1			
Totals	12	27	12	7	8	1	2	69
Percent	17.39	39.13	17.39	10.14	11.59	1.45	2.90	

Table 2. Testcrosses of wx T2-9d/al-Mum2 with al al wx wx showing linkage of wx with one regulator element.

Wx		Parental Classes: Mutable Wx	Parental Classes: Stable wx	Crossovers Classes: Stable Wx	Crossovers Classes: Mutable wx	Total	% c.o.	Wx/wx	S.E.	Plant crossed to translocation series	No. of regulators in plant crossed to translocation series
89-90-4508-9											
3509-2	obs.*	91	62	25	13	191	19.90	1.5467		89-4001-2	1
	corr.*	91	96	25	20	232	19.40[a]		±2.60		
90-5001-5											
4001-1	obs.	51	52	10	8	121	14.88	1.0167		89-4001-2	1
	corr.	51	53	10	8	122	14.75[ab]		±3.21		
90-5001-6											
4003-9	obs.	43	27	3	1	74	5.41	1.6429		89-4001-2	1
	corr.	43	44	3	2	92	5.43[c]		±2.36		
90-5007-4											
4003-10	obs.	76	37	5	8	126	10.32	1.8000		89-4001-2	1
	corr.	76	67	5	14	162	11.73[b]		±2.53		
90-5004-8											
4004-9	obs.	121	79	11	16	227	11.89	1.3894		89-4001-2	1
	corr.	121	110	11	22	264	12.50[b]		±2.03		
90-5012-3											
4005-9	obs.	47	37	3	6	93	9.68	1.1628		89-4001-2	1
	corr.	47	43	3	7	100	10.00[bc]		±3.00		
90-5010-9											
4007-7	obs.	75	67	9	10	161	11.80	1.0909		89-3001-9	2
	corr.	75	73	9	11	168	11.90[b]		±2.50		
TOTALS	obs.	504	361	66	62	993	12.89	1.3475			
	corr.	504	487	66	84	1,141	13.15		±0.96		

*obs. = observed values, corr. = values corrected for the deficiency of waxy kernals

a,b,c = % c.o. values that share the same letter do not differ at the 1% level of significance. Those that do not have a letter in common do differ at the 1% level of significance.

from 12.89% to 13.15%. Plants grown from kernels of two different 1:1 ears (i.e., 89-4001-2, 89-3001-9) were crossed to *wx* T2-9d. The crossover value for the one regulator outcross of 89-3001-9 is very close to some of the crossover values observed for the outcrosses of 89-4001-2. These 1:1 ears may have carried the same regulator at the identical locus as the result of descent from a common progenitor (i.e., the original *a1-Mum2* mutation event). Alternatively, their common values may be just coincidental.

Plant number 89-3001-9 showed evidence of carrying two regulators. The testcross with *wx* T2-9d (Table 2) indicated the presence of only one regulator. Outcrosses of this same plant to other translocations gave results consistent with the presence of 2 or 3 regulators in different F1's (Tables 3 and 4), thus demonstrating that the number of regulators can either increase or decrease with a single generation of outcrossing.

Table 3 summarizes the data from crosses with a regulator linked to *wx* T2-9d plus a second independent regulator. The observed and expected results are in good agreement. The expected results were calculated by using the corrected linkage value of the last cross of Table 2, because this cross and all the crosses in Table 3 shared the same 1:1 parent plant (89-3001-9). Thus, it is likely that the regulator linked to *wx* T2-9d is at the same position in both the F1 with one linked regulator and the F1's with a second independent regulator.

Table 4 summarizes the data from crosses with a regulator linked to *wx* T2-9d and two additional independent regulators. Again the corrected linkage value for the last

Table 3. Testcrosses of wx T2-9d/al-Mum2 with al al wx wx showing linkage of wx with one regulator element plus a second independent regulator.

	Parental Classes		Crossovers Classes				Plant crossed to trans-location series	No. of regulators in plant crossed to translocation series
	Mutable Wx	Stable wx	Stable Wx	Mutable wx	Total	Wx/wx		
89-90-4534-6 3537-3	24	11	2	23	60		89-3001-9	2
90-5006-5 4006-3	102	30	9	49	190		89-3001-9	2
90-5006-4 4009-8	97	44	5	39	185		89-3001-9	2
90-5007-2 4011-1	136	73	5	43	257		89-3001-9	2
90-5010-7 4011-4	34	17	2	19	72		89-3001-9	2
TOTALS	393	175	23	173	764	1.1954		2
Corrected Totals*	393	209	23	207	832			
Expected Totals**	391.25	183.25	24.75	232.75				

x^2 observed vs. expected = 6.5986 p = < 10% and > 5%.

* Corrected for the deficiency of waxy kernels.

** Expected totals calculated using the crossover values from the corrected data of cross 90-5010-9/4007-7 from Table 2 and assuming independence for the second regulator.

Table 4. Testcrosses of wx T2-9d/alMum2 with al al wx wx showing linkage of wx with one regulator element plus 2 independent regulators.

	Parental Classes		Crossovers Classes				Plant crossed to trans-location series	No. of regulator in plant crossed to translocation series
	Mutable Wx	Stable wx	Stable Wx	Mutable wx	Total	Wx/wx		
90-5008-3 4002-7	123	28	4	88	243		89-4001-2	1
90-5006-7 4007-8	154	20	11	79	264		89-3001-9	2
90-5005-4 4008-2	107	30	5	72	214		89-3001-9	2
90-5010-2 4010-2	105	23	2	74	204		89-3001-9	2
Totals	489	101	22	313	925	1.2343		
Corrected Totals*	489	125	22	386	1,022			
Expected Totals**	495.77	112.52	15.23	398.48				

x^2 - observed vs. expected = 4.8769 p < 20% and > 10%

* Corrected for the deficiency of waxy kernels.

** Expected totals calculated using the crossover values from the corrected data of cross 90-5010-9/4007-7 from Table 2 and assuming two additional independent regulators.

cross in Table 2 was used to calculate expected values, because all but one cross in Table 4 involved plant 89-3001-9. The data do not differ significantly from the expected values. The first cross of Table 4 came from an *a1-Mum2* plant (89-4001-2) that had only one regulator and yet now three are observed, one linked to *wx* T2-9d and two others at independent locations. Plant 89-3001-9 contributed to testcrosses in Tables 2, 3 and 4. This plant carried two independent regulators, one of which is shown to be linked to *wx* T2-9d (Table 2). Because the second regulator is not linked to *wx* one third of the plants segregating for somatic mutability would be expected to receive only the linked regulator, one third only the nonlinked regulator and one third both regulators. One of the outcrosses carried only the linked regulator (Table 2), and seven carried the unlinked regulator (the 7 crosses shown in line one column three of Table 1), and eight had both regulators (Tables 3 and 4).

Three crosses involving *wx* T4-9e (4S.53, 9L.26) and *a1-Mum2* gave plants with one element linked to this translocation plus one independent regulator (one plant) or two independent regulators (two plants, Table 5). For both of these situations, the observed values do not differ significantly from the predicted values estimated by using the frequency of the starchy stable classes (0.0741 and 0.0446 respectively) to calculate the expected frequencies. Using these values gives estimates of *wx* to regulator distance of 29.64 and 35.68 respectively. All of the crosses of Table 5 are derived from the same *a1-Mum2* plant, which had 3 regulators. Thus, it is possible that the same regulator located at the same position relative to the translocation breakpoint is present in the plants with one and two independent regulators. The difference between the two crossover values (6.04%), is not statistically significant.

The *a1-Mum2* plant crossed to *wx* T4-9e (89-3001-4, Table 5) was a sibling plant to the one that yielded one of

Table 5. Testcrosses of wx T4-9e/a1-Mum2 with a1 a1 wx wx showing linkage of wx with one regulator element plus one or two independent regulators

	Parental Classes		Crossovers Classes				Plant crossed to translocation series	No. of regulators in plant crossed to translocation series
	Mutable Wx	Stable wx	Stable Wx	Mutable wx	Total	Wx/wx		
Cross with one independent regulator.								
90-5020-8 4015-1	60	29	9	33	131	1.1129	89-3001-4	3
Corrected*	60	32	10	33	135			
Expected	57.50	23.75	10.00	43.75	(Assuming 29.63% c.o.)			
x^2-observed vs. expected = 5.6159 p = < .20 and > .10								
Crosses with two independent regulators.								
89-90-4528-1 3528-2	45	10	6	39	100		89-3001-4	3
89-90-4528-2 3528-4	34	11	2	41	88		89-3001-4	3
Totals	79	21	8	80	188	0.8614		
Corrected Totals**	92	21	9	80	202			
Expected	91.99	16.24	9.01	84.76	(assuming 35.68% c.o.)			
x^2 - observed vs. expected = 1.6625 p = < .99 and > .98								

* Corrected for the deficiency of waxy kernels.
** Corrected for the deficiency of starchy kernels.

Table 6. Testcrosses of wx T4-9b/a1-Mum2 with a1 a1 wx wx showing linkage of wx with one regulator element plus one or two independent regulators.

	Parental Classes		Crossovers Classes			Plant crossed to translocation series	No. of regulator in plant crossed to translocation series
	Mutable Wx	Stable wx	Stable Wx	Mutable wx	Total		
Cross with one independent regulator							
90-5011-8 4013-4	81	49	19	53	202	89-3001-1T	2
Corrected*	92	36	14	60	202		
Expected	87.00	36.5	14.00	64.50	(assuming = 27.72% c.o.)		
x^2 - observed vs. expected = .6002 p = < .90 and > .80							
Cross with two independent regulators							
89-90-4533-5 3540-6	89	25	1	66	181	89-3001-1T	2
Expected	89.50	21.63	1.00	68.87	(assuming 4.42% c.o.)		
x^2 = 0.6475 p = < .90 and > .80							

*Corrected for the surplus of stable kernels by reducing the stables proportionately (to 25% of the total) and distributing the excess of stables above 25% proportionately to the mutable classes.

the crosses that had one regulator linked to *wx* T2-9d (89-3001-9, Table 2), all of the crosses with one regulator linked to *wx* T2-9d plus an independent regulator (Table 3), and three out of four crosses with one regulator linked to *wx* T2-9d plus two independent regulators (Table 4). It is conceivable that one of the independent regulators of Table 5 is the regulator linked to *wx* T2-9d of Tables 2, 3 and 4, although this can not be proved from the existing data.

Two crosses of *wx* T4-9b/*a1-Mum2* gave results consistent with a regulator linked to *wx* T4-9b (4L.90, 9L.29) and one or two additional independent regulators (Table 6). The crossover value and the chi-square test for the cross with two regulators is not reliable because one class (starchy, stable) had only one kernel and because this was the class used to estimate the amount of crossing over. However, all classes show close agreement between observed and expected values. Thus there is a strong indication of linkage of a regulator with the *wx* T4-9b break point, with a crossover value that probably is not greatly different from the estimated one. There is little likelihood that the linked regulators in the one independent and the two independent regulator crosses are at the same positions because of the large difference in recombination values (Table 6). The *a1-Mum2* plant outcrossed to *wx* T4-9b (89-3001-1T, Table 6) was a sibling plant to the one crossed to *wx* T4-9e (89-3001-4, Table 5) and also a sibling to one of those crossed to *wx* T2-9b (89-3001-9, Tables 2, 3 and 4). Again it is possible, but by no means certain, that an independent regulator in the *wx* T4-9b multiple regulator crosses could be the one mapped in the *wx* T2-9d crosses.

Evidence for linkage of a regulator to *wx* T2-9d was also found in a 1:1 *a1-Mum3* test (Table 7). The crossing over for the total population was 19.95 percent (corrected 20.04 percent), which is significantly higher than that observed for the *a1-Mum2* linkage with the same translocation (13.15%, Table 2). Some of the individual *a1-Mum3* crossover frequencies, however, do not differ significantly from those observed in the *a1-Mum2* crosses. Values of between 12 and 20 percent are found in both sets of testcrosses. (The Table 2 crossover percentages of 14.75, 12.50, 11.73, 11.90 and 10.00 do not differ significantly at the one percent level from those of 19.51, 18.52, 16.10 and 12.79 of Table 7. The crossover percentage of 19.40 in Table 2 is not significantly different from those of 19.51, 18.52 and 16.10 of Table 7.) One *a1-Mum2* cross has a crossover value considerably less (5.43 percent) than any found for *a1-Mum3* crosses and one *a1-Mum3* cross has a

Table 7. Testcrosses of wx T2-9d/a1-Mum3 with al al wx wx showing linkage of wx with one regulator elements

		Parental Classes		Crossover Classes						Plant crossed to translocation series	No. of regulatory in plant crossed to translocation series
		Mutable Wx	Stable wx	Stable Wx	Mutable wx	Total	% c.o.	S.E.	Wx/wx		
89-6255-6 5254-4	obs.*	78	83	8	16	185	12.97		0.8687	85-9009-4	1
	corr.*	78	72	8	14	172	12.79[b]	±2.55			
90-845-4 745-3	obs.	55	24	4	8	91	13.19		1.8438	88-9009-4	1
	corr.	55	44	4	15	118	16.10[ab]	±3.38			
90-847-1 747-6	obs.	105	74	32	37	248	23.79		1.2342	88-9009-4	1
	corr.	105	91	32	46	274	28.47	±2.73			
90-847-3 747-7	obs.	93	97	30	17	237	19.83		1.0789	88-9009-4	1
	corr.	93	105	30	18	246	19.51[a]	±2.53			
90-842-5 748-7	obs.	40	33	14	4	91	19.78		1.4595	88-9009-4	1
	corr.	40	48	14	6	108	18.52[ab]	±3.74			
Totals	obs.	371	311	88	82	852	19.95		1.1679		
	corr.	371	363	88	96	918	20.04	±1.29			

* obs. = observed values, corr. - values corrected for the deficiency or surplus of waxy kernels.
a,b - % c.o. values that share the same letter do not differ at the 1% level of significance. Those that do not have a letter in common do differ at the 1% level of significance.

Table 8. Results of a testcross of wx T2-9d/a1-Mum3 with al al wx wx showing linkage of wx with one regulator plus two independent regulators.

	Parental Classes		Crossovers Classes				Plant crossed to translocation series	No. of regulators in plant crossed to translocation series
	Mutable Wx	Stable wx	Stable Wx	Mutable wx	Total	Wx/wx		
90-748-77 745-3	40	5	1	16	62	1.95	88-9009-4	1
Corrected*	40	10	1	31	82	(Assuming 9.76% c.o.)		
Expected	40.00	9.25	1.00	31.75				
x^2 - observed vs. expected 0.9494			p =		> 99%			

*Corrected for the deficiency of waxy kernels.

value of 28.47 percent, which is considerably higher than any found for *a1-Mum2*. The *a1-Mum2* and *a1-Mum3* alleles were produced in the same isolation plot, in which purple aleurone Mutator plants were used as male parents. It is possible that these two mutant alleles arose from a sector in the tassel of a purple Mutator plant and thus might have had some regulators in common. Analyses of the original isolates of these mutants in their immediate progenies and in later generations indicated that multiple regulators of somatic mutability were present in the genome of these mutants when they were first isolated. Even if these are not alleles derived from a single mutation event but arose independently they nonetheless were induced in a common purple aleurone Mutator population, and thus progeny plants carrying the independent mutations could have many regulators in common. Therefore, it is possible that lines carrying the different *a1-Mum* alleles have a common regulator on the long arm of chromosome two.

One test cross of a *wx* T2-9d *a1-Mum3* F1 segregated for a regulator linked to the translocation plus two independent regulators (Table 8). The chi-square in this instance is not reliable because only one starchy stable kernel was observed. However, the expected frequencies of all classes are in close agreement with the observed values, suggesting that this cross indeed involves two independent regulators.

These studies are illustrative of how readily the number of regulators can change. Plant 89-3001-9 (*a1-Mum2*), with two regulators, was outcrossed to the translocation series, followed by testcrossing the next generation. In three of the progeny of the crosses of this plant to *wx* T2-9d that showed linkage, the number of regulators had increased from the two found in the parent to 3 in the testcrosses (Table 4). One sibling plant of 89-3001-9, 89-3001-1T, also went from two regulators in the parent to three in the testcross (Table 6). The *a1-Mum3* plant which was crossed to *wx* T2-9d, 88-9009-4, had only one regulator, and in most testcrosses of the F1 only one was retained. However, in one cross two additional regulators were found (Table 8).

Conclusions. 1) Ears that have 1:1 or other ratios of mutable:stable kernels, indicative of the presence of one or more regulators of somatic mutability, have been shown to be carrying regulators that can be mapped to chromosome locations using standard linkage tests. In the stocks tested, regulators have been located to the long arm of chromosome 2 and to the long and short arms of chromosome 4.

2) In different sibling plants from an outcross of a single plant to *wx* T2-9d, several different crossover values are observed, suggesting that the regulator on chromosome two may be transposing to different positions on the chromosome.

3) Regulators of somatic activity of Mutator-induced mutants can both increase and decrease in number from one generation to the next. In these studies, the increase in number is probably the result of the transposition of the regulator to a locus independent of its original site. The decrease in number is undoubtedly the result of the independent assortment of unlinked regulators.

Miniature-3 (*mn3*): a viable miniature kernel mutant on chromosome 6

Philip S. Stinard

A small kernel mutant tightly linked to *y1* was found segregating on the selfed ear of a plant (86-1184-8) grown from the cross *Y1 Y1* X *y1 y1-pastel(Mu)*. Seedling tests of white kernels from the segregating ear indicated that the mutant, originally designated *de*-1184*, was in coupling with the standard *y1* allele, and not the putative *Mu*-induced *y1* pastel allele. Mutant kernels of *de*-1184* are smaller than wildtype, ranging in size from near normal to nearly empty pericarp, and have etched/pitted endosperm. There is a tendency for mutant kernels to lack germs, but many mutant kernels have healthy germs, and give rise to green, normal-appearing plants. Upon self-pollination, these plants produce all mutant kernels. Because there are no mutants with similar phenotype on chromosome 6, we have given *de*-1184* the name miniature-3 (*mn3*).

Crosses of known heterozygotes of *mn3* by TB-6Lc and TB-6Sa produce only normal sized kernels, indicating that *mn3* is proximal to the TB breakpoints on chromosome 6. Three-point linkage tests of *mn3* with the chromosome 6 linkage stocks *w15 y1* and *y1 l15* were made as indicated in Tables 1 and 2. Plants grown from the testcrosses were

Table 1. Three-point linkage data for mn3--w15--y1.

Testcross: Mn3 Mn3 W15 W15 Y1 Y1 X (Mn3 w15 y1 / mn3 W15 Y1)

Reg.	Phenotype	No.	Totals
0	mn + +	177	
	+ w y	161	338
1	mn w y	8	
	+ + +	7	15
2	mn + y	0	
	+ w +	8	8
1+2	mn w +	1	
	+ + y	0	1

% recombination mn3 -- w15 = 4.4 ± 1.1

% recombination w15 -- y1 = 2.5 ± 0.8

Table 2. Three-point linkage data for mn3--y1--l15.

Testcross: Mn3 Mn3 Y1 Y1 L15 L15 X (Mn3 y1 l15 / mn3 Y1 L15)

Reg.	Phenotype	No.	Totals
0	mn + +	173	
	+ y l	168	341
1	mn y l	2	
	+ + +	5	7
2	mn + l	14	
	+ y +	14	28
1+2	mn y +	0	
	+ + l	1	1

% recombination mn3 -- y1 = 2.1 ± 0.7

% recombination y1 -- l15 = 7.7 ± 1.4

self-pollinated, and the resultant ears scored for *y1* and *mn3*. Kernel samples from the selfed ears were planted in the sandbench, and the resultant seedlings scored for *w15* or *l15*. These tests give the following linkage relationships: *mn3*-4.4-*w15*-2.5-*y1* and *mn3*-2.1-*y1*-7.7-*l15*. The *mn3-y1* distance is greater in the *mn3 w15 y1* cross (6.9cM) than in the *mn3 y1 l15* cross (2.1cM). The *y1-l15* distance

(7.7cM) does not agree with distance for the same interval on the 1990 linkage map (13cM), but the *w15-y1* distance (2.5cM) does. The linkage distances in the *mn3 y1 l15* cross seem to be smaller than they should be. The reason for this is not known, but could be due to the different genetic backgrounds of the two linkage stocks. It is likely, therefore, that the *mn3 w15 y1* data better reflect the true linkage relationships, at least with respect to the established linkage map of chromosome 6. If this is the case, then *mn3* is located at map position 10, in the vicinity of *Mdm1*. Until additional linkage studies are conducted to verify the order of *mn3* and *Mdm1* on the chromosome 6 map, the suggested map revision is:

```
                mn3
rgd1----Mdm1---w15-------y1 -----------l15
  8       11      13       17          30
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Crinkly-4 (*cr4*): a new crinkly-leaf/aleurone mosaic mutant on 10S

--Philip S. Stinard

A new recessive mutant conditioning aleurone mosaicism was found segregating on the selfed ear of a *bz-mum8* plant, 84-6143-8 selfed. When planted in the field, mosaic kernels give rise to crinkly-leafed seedlings, which in turn mature to become short, crinkly-leafed plants. The crinkling of mature mutant plant leaves is much more extreme than that of *cr1*, and the leaves are rough, with a dull luster. The tassels of mutant plants are club-shaped, and are poor shedders. The aleurone mosaicism of mutant kernels shows incomplete penetrance. This mutant, originally designated *dap*-6143* (MNL 61:9), was propagated and placed in our 1989 TB block. Two crosses by TB-10Sc uncovered mosaicism in the aleurone. When wildtype kernels from the TB cross were planted in the sandbench, only wildtype seedlings resulted. However, a close inspection of the wildtype kernels from the TB cross ears revealed that some of them were germless. Perhaps these germless kernels represent the hyperploid endosperm/hypoploid mutant embryo condition. If this is the case, then hemizygosity for the mutant allele has a deleterious effect on embryo development that is not observed in diploid mutant embryos.

In order to confirm the placement of this mutant to chromosome, ten mosaic kernels from the TB-10Sc cross, presumably representing the hypoploid mutant endosperm/hyperploid embryo condition, were planted in our 1990 summer nursery, and the resulting plants selfed and outcrossed to both *oy* and *y9* testers. None of the selfed ears segregated for *y9*, but they did segregate for a low frequency of mosaic kernels. The outcrosses to *y9* all segregated for pale yellow kernels. The crosses to *oy*, when seedling tested, segregated for oil yellow seedlings. Thus, all ten plants grown from mosaic kernels were hyperploids, confirming that this mutant is located on 10S.

Since the expression of the crinkly mutant phenotype is good, but the expression of aleurone mosaicism is not always consistent, we have given this mutant the name crinkly-4 (*cr4*) (mutants given the names *cr2* and *cr3* have been reported, but are lost). Three-point linkage studies of *cr4* utilizing the chromosome 10 linkage stocks *oy y9* and *oy bf2* are in progress.

An alternative method of using B-A translocations to locate duplicate factors

--Philip S. Stinard

A method for using B-A translocations to locate duplicate factors to chromosome arm was presented by Kindiger and Beckett in MNL 60:43, and in modified form by Neuffer and Beckett in MNL 61:49-50. This method involves crossing the B-A translocation set onto plants carrying both duplicate factors, selecting hypoploids, and self-pollinating or testcrossing the hypoploids to detect diagnostic segregation ratios.

An alternative method can be used to locate duplicate factors to chromosome arm directly, in a manner similar to that of using B-A translocations to locate single factors. This method relies on the idea that the second factor of a duplicate factor pair behaves as a single factor when the first factor is homozygous. If a B-A translocation stock carrying the first factor is crossed onto a stock carrying both factors, the possibility arises that progeny kernels homozygous for the first factor, and hyperploid/hypoploid for the chromosome arm that uncovers the second factor will be produced, yielding a mutant genotype in the endosperm or embryo.

The crosses can be set up as follows: a stock homozygous for the mutant allele of the first factor (*fac1*), but not carrying the mutant allele of the second factor (*fac2*), can be obtained from the self of a stock homozygous for *fac1* and heterozygous for *fac2*. The homozygous *fac1* stock (*fac1 fac1 Fac2 Fac2*) is crossed onto each of the different B-A translocation stocks. F1 plants heterozygous for *fac1* and carrying the B-A translocations are crossed onto plants carrying both *fac1* and *fac2* (either *fac1 fac1 fac2 fac2*, *fac1 fac1 Fac2 fac2*, *Fac1 fac1 fac2 fac2*, or *Fac1 fac1 Fac2 fac2*, depending on the availability of stocks, and whether the double homozygote is lethal). If a cross uncovers the mutant phenotype (i.e., produces kernels with hyperploid endosperms and hypoploid mutant germs, or vice versa), then *fac2* is located on the A chromosome arm involved in the B-A translocation used in the cross. The same procedure can be done starting with a homozygous *fac2* line to locate *fac1* to chromosome arm.

This method has the advantage that it is not necessary to grow hypoploids to maturity unless it is for the observation of mature plant traits, or for the purpose of self-pollinating hypoploids to locate mutants that are proximal to the B-A breakpoints.

An accompanying article describes how this method was applied to the *lw3 lw4* duplicate factor pair to isolate a homozygous *lw4* line and demonstrate that *lw3* is uncovered by TB-5La.

Linkage studies of the duplicate factor pair *lw3* and *lw4*

--Philip S. Stinard

The duplicate factor pair *lw3* and *lw4* conditions lemon (pale yellow) kernels that give rise to white seedlings. The two factors are located on 5L, and on chromosome 4 near

the centromere, respectively. Since a *lw3 lw4* stock was available in our cold storage facilities, but not at the Stock Center, we undertook to characterize our stock for the purpose of donating it to the Stock Center. We thought it most appropriate to isolate lines homozygous for each factor individually. As this had not been done before, we developed the theoretical approach for using B-A translocations to locate duplicate factors outlined in an accompanying article, and applied it to the *lw3 lw4* system.

A stock segregating 3:1 for yellow:lemon-white kernels was obtained by self-pollination of plants grown from yellow kernels of a selfed ear segregating 15:1. The 3:1 stock was maintained for several generations by self-pollination of plants grown from yellow kernels of selfed 3:1 ears. As it was not known which duplicate factor was homozygous in the 3:1 stock, plants producing 3:1 ears were assigned the genotype symbol *lwx lwx Lwy lwy*, where x=3 and y=4, or vice versa. A homozygous *lwx lwx Lwy Lwy* stock was in turn derived by self-pollination of plants grown from yellow kernels of 3:1 ears, and selection of ears producing only yellow kernels. The *lwx lwx Lwy Lwy* stock was crossed onto the B-A translocation stocks TB-4Lf and TB-5La, and the F1's crossed onto independently derived 3:1 *lw3 lw4* stocks as well as the appropriate TB testers (*c2* and *pr gl8*, respectively).

Of four crosses involving TB-5La, two segregated for lemon kernels, suggesting that the duplicate factor uncovered in the cross is on 5L (and is therefore *lw3*). Yellow kernels from the two segregating ears were sown in the sandbench, and gave rise to seedlings, some of which were white. If *lw3* is indeed uncovered by TB-5La, then the lemon kernels with putative hypoploid mutant endosperms should have hyperploid germs that carry TB-5La as well as *lw3*. To test this, three lemon kernels from each ear were sown in the field, and the resulting plants selfed and outcrossed to the 5L tester *pr gl8*. The selfed ears segregated for a low frequency of lemon-white kernels (expected of the selfs of segmental trisomics involving duplicate factors; see Neuffer and Beckett, MNL 61:49-50). The outcrosses to *pr gl8* all segregated for *pr* kernels giving rise to wildtype seedlings, and *Pr* kernels giving rise to seedlings segregating for glossy, thus confirming the hyperploid status of the male parent and the location of *lw3* to 5L.

Since the factor uncovered by TB-5La was *lw3*, the stock crossed onto TB-5La must have been homozygous for *lw4*. Thus, in the stock *lwx lwx Lwy lwy*, x=4 and y=3.

Linkage studies were conducted on the homozygous *lw4* stock in order to confirm the location of *lw4* on chromosome 4. The *lw4 lw4 Lw3 Lw3* stock was crossed onto a homozygous *su1 gl4* tester. The F1 was pollinated by *lw4 lw4 Lw3 lw3* plants, and the progeny grown in our selfing block and self-pollinated. From the manner in which the cross was set up, only half of the selfed ears should carry *lw3* and thus segregate for lemon kernels. It is only such ears that provide meaningful linkage data. Ears segregating for lemon kernels were scored for *su1* and for the ratio of yellow:lemon kernels. Kernel subsamples from these same ears were planted in the sandbench, and the resulting seedlings scored for *gl4*. The data from

Table 1. Three-point linkage data for su1--lw4--gl4.

Testcross: (su1 Lw4 gl4 Lw3 / Su1 lw4 Gl4 Lw3)
X Su1 Su1 lw4 lw4 Gl4 Gl4 Lw3 lw3

Reg.	Phenotype			No.	Totals
0	su	+	gl	30	
	+	lw	+	40	70
1	su	lw	+	4	
	+	+	gl	3	7
2	su	+	+	4	
	+	lw	gl	3	7
1+2	su	lw	gl	1	
	+	+	+	1	2

% recombination su1 -- lw4 = 10.5 ± 3.3

% recombination lw4 -- gl4 = 10.5 ± 3.3

this linkage test, summarized in Table 1, yield the following linkage relationship: *su1*-10.5-*lw4*-10.5-*gl4*. Thus, *lw4* is approximately equidistant between *su1* and *gl4* on chromosome 4. These data are in good agreement with the F2 data of Tulpule (Am. J. Bot. 41:294-301) that place *lw4* 13cM distal to *su1*. These data would also increase the distance from *su1* to *gl4* from 15cm (current linkage map distance) to 21cM. Since the number of individuals in our linkage study is rather small (n=86), this experiment will be repeated in order to better place *lw4* on the chromosome 4 linkage map. For now the suggested map revision is:

lw4

---*su1*----------*gl4*--

66 81

ts8 is allelic to *si*

--Philip S. Stinard and Donald S. Robertson

In the list of factors dropped from the 1983 linkage map (MNL 58:216), the factor *ts8* is listed as lost. We have been using a *y1 ts8* stock for some years in chromosome 6 linkage studies. The original source of our stock is unknown, but has been traced back as far as 1959. Because the phenotype of *ts8* is identical to that of *si*, also on chromosome 6 (sterile tassel with a few silk-like appendages, proliferation of silks on the ear), we set up allele tests of these two mutants as follows: a homozygous *y1 si* stock obtained from the Stock Center was pollinated by a *y1 ts8* heterozygote (*Y1 Ts8/y1 ts8*). In addition, a homozygous *y1 ts8* stock was pollinated by a *y1 si* heterozygote (*Y1 Si/y1 si*), also obtained from the Stock Center. Ten white kernels from each cross were planted in our 1989-90 winter nursery. With the exception of one fertile plant (a probable crossover event), all progeny had the *si* phenotype, indicating allelism of *ts8* and *si*.

Some thoughts on nuclear genes affecting plastid development

--Philip S. Stinard

In the course of our studies of the Mutator transposable element system, we have had the occasion to observe many different endosperm and seedling mutants. The mutants conditioning etched/sugary endosperm and seedlings with reduced chlorophyll content make up a class of frequently observed mutants that has received rel-

atively little attention. Mutants fitting into this class include *et, dek5, dek7, w2 (dek21),* and *cp2*. All of these mutants have alleles that produce mosaicism for aleurone color in a colored aleurone background; all have etched or pitted endosperm. The mutants *et, dek5, dek7,* and *cp2* have alleles producing a sugary/shrunken endosperm phenotype. The mutant *et* produces virescent seedlings; the mutants *dek5, dek7,* and *cp2* produce striate seedlings (white or pale green with green stripes), and *w2* produces white seedlings (yellow seedlings with homozygous *l1*; the interaction of *l1* with the other mutants in this class is not known).

Previously reported studies of *et* (Ramesh and Reddy, MNL 59:52) and *w2* (Han and Coe, MNL 64:45-46) have indicated that seedling plastids are affected in both mutants (plastid membrane structure in *et*, amount of plastid DNA in *w2*). If the abnormal seedling phenotypes associated with this class of mutants are due to plastid defects, then perhaps the abnormal endosperm phenotypes are due to plastid (amyloplast) defects as well. If this is the case, then these nuclear gene mutations may be disrupting biochemical pathways involved in general plastid structure or development. Studies of these mutants may aid in the elucidation of these pathways.

dek21 is allelic to *w2*

--Philip S. Stinard and Donald S. Robertson

The mutant *dek21*, located on 10L, is a defective kernel mutant conditioning etched/pitted endosperm kernels that produce white seedlings. In a purple aleurone background, mutant kernels have mosaicism for aleurone color as well. An aleurone mosaic mutant that was found segregating on the selfed ear of a purple Mutator plant, 87-2221-12, proved to be allelic to *dek21*. This mutant was designated *dek21-2221*. In the course of selecting mutants on 10L that could be used in linkage studies of *dek21-2221*, we found that *w2*, also on 10L, produces a pitted endosperm/white seedling phenotype. Since the mutant phenotypes of *dek21* and *w2* are similar, we set up crosses to test allelism of *dek21* with *w2* in our 1989-90 winter nursery. From intercrosses of these two mutants, ears were obtained that segregated for kernels with mosaic aleurone (in purple aleurone background) or pitted endosperm (in colorless background). When sown in the sandbench, mutant kernels gave rise to white seedlings. We conclude that *dek21* is allelic to *w2*, and suggest that the designation with precedence, *w2*, be used in preference to *dek21*.

AMES, IOWA
Iowa State University
STUTTGART, GERMANY
University of Hohenheim

Graphical genotype for inbred line B86 determined by RFLPs

--S. Fähr, M. M. Messmer, A. H. Melchinger, M. Lee, and W. L. Woodman

As part of an extensive RFLP study of maize inbreds, we applied the concept of graphical genotypes (Young and Tanksley, Theor. Appl. Genet. 77:95-101, 1989) to maize inbred line B86. Our primary objective was to investigate the origin of the B86 genome from its parental inbreds B52 and Oh43.

Genomic DNA from all three inbreds (B52, Oh43, and B86) was digested separately with restriction enzymes *Eco*RI, *Eco*RV, and *Hin*dIII. RFLP analyses were performed with a total of 187 maize DNA probes (mainly genomic clones) kindly provided by four labs (B. Burr, Brookhaven National Laboratory; D. Grant, Pioneer Hi-Bred International Inc.; T. Helentjaris, Native Plants Inc.; D. Hoisington, University of Missouri-Columbia).

The DNA probes were positioned on the maize linkage map according to the most recently presented RFLP maps (MNL 64:154-163, 1990). The genotype of B86 for each marker was determined by comparing its RFLP patterns with those of B52 and Oh43.

Three probes yielded deviating RFLP patterns for B86 not present in the parental inbreds (details are given in the following note of this MNL by Messmer et al.). Altogether, 132 (71.7%) out of the remaining 184 DNA probes revealed polymorphic RFLP patterns in B52 and Oh43 and only these are subsequently considered. RFLP variants in B86 at loci mapping to chromosomes 3, 4, 5, 6, and 10 originated mostly from B52 whereas those mapping to chromosomes 1, 2, 7, 8, and 9 originated mostly from Oh43 (Table 1). Averaged across the whole genome, B86 inherited 51.5% of the RFLP variants from B52 and 48.5% from Oh43.

Figure 1 shows a graphical representation of the origin of the RFLP variants detected in B86. Chromosomal regions, in which several adjacent markers had the same

Table 1. Number of DNA probes, yielding monomorphic and polymorphic RFLP variants for maize inbreds B52 and Oh43, associated with each of the 10 maize chromosomes. For polymorphic RFLP variants, the last two columns show the origin of the RFLP variants in inbred B86.

Chromosome	RFLP variants in B52 and Oh43		RFLP variant in B86 from	
	Monomorphic*	Polymorphic*	B52	Oh43
	no.		%	
1	3	25	32.0	68.0
2	2	17	35.3	64.7
3	10	14	78.6	21.4
4	6	12	75.0	25.0
5	7	17	64.7	35.3
6	4	8	87.5	12.5
7	8	7	28.6	71.4
8	5	10	40.0	60.0
9	5	13	23.1	76.9
10	2	9	77.8	22.2
Total	52	132	51.5	48.5

* Monomorphic with the three restriction enzymes analysed (EcoRI, EcoRV, HindIII).

* Polymorphic with at least one restriction enzyme (EcoRI, EcoRV, HindIII).

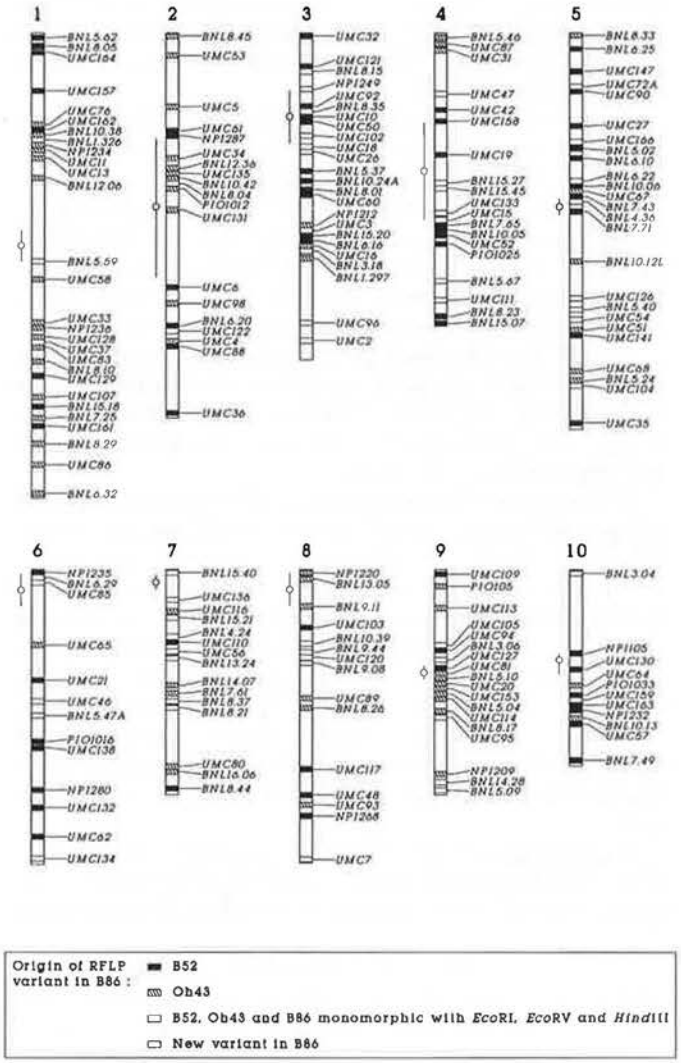


Figure 1.

origin of RFLP variations, were probably transmitted from the respective parent without effective recombination events. Examples include chromosome arms 8S and 9L, probably inherited intact from Oh43, and chromosome arm 3S from B52. In general, the graphical genotype provides information about the number of effective recombination events that accumulated during the selfing process and in the development of B86. However, our results must be interpreted with caution because valid conclusions about recombination events could only be drawn based on an RFLP linkage map especially constructed for cross B52xOh43. Despite this limitation it seems safe to assume that a recombination event occurred on the short arm of chromosome 4, because the RFLP variants at the three distal markers came from Oh43, whereas the remaining part of the chromosome was very likely inherited from B52.

B86 is a selection from a breeding program conducted by W. A. Russell and W. D. Guthrie at Iowa State. The aim of selection was to combine leaf-feeding resistance (1st brood resistance) and sheath and collar-feeding resistance (2nd brood resistance) to the European corn borer (ECB) *Ostrinia nubilalis* Hübner into a single genotype. Leaf-feeding resistance was available in Oh43, which is moderately resistant to 1st brood and has good combining ability for grain yield but is highly susceptible to 2nd brood (Russell et al., Crop Sci. 14:725-727, 1974). B52 is highly resistant to 2nd brood and also shows intermediate resistance to 1st brood but has poor yield performance (Russell and Guthrie, Crop Sci. 19:565, 1979). B86 was the best inbred developed from 200 F3 lines of the cross B52xOh43 by selection and selfing according to the ear-to-row method from the F3 to F7 generations, using artificial infestations with ECB egg masses in each generation (Russell et al., 1974, see above; Guthrie et al., J. Econ. Entomol. 78:93-95, 1985). The main selection criteria were resistance to both generations of ECB and, within highly resistant progenies, recovery of the Oh43 phenotype (W. A. Russell, pers. comm.). B86 proved to be highly resistant to 1st brood and nearly as resistant as B52 to 2nd brood of ECB.

The estimated number of genes conferring resistance to 2nd brood of ECB in B52 is seven (Onukogu et al., J. Econ. Entomol. 71:1-4, 1978). Russell et al. (1974) assumed three loci conditioning 1st brood resistance in Oh43 and a different locus in B52, but absence of genes for 2nd brood resistance in Oh43. These authors also reported that the genes conferring the two types of resistance are not identical. Based on this information, it seems reasonable to assume that approximately eight loci from B52 and three loci from Oh43 were under intense selection pressure during the development of B86. Thus, B86 is expected to have a higher proportion of RFLP variants from B52 than from Oh43, which deviates from the observed ratio of approximately 1:1 (Table 1).

So far, no genes conferring resistance against 1st or 2nd brood of ECB have been reported for chromosomes 7, 9, and 10. With regard to these chromosomes, 41.4% of the polymorphic variants observed in B86 were derived from B52. This suggests that parts of the B86 genome, having no effect on ECB resistance, were mainly inherited from Oh43 due to selection for recovery of the agronomically desirable Oh43 phenotype. B86 probably inherited four genes conditioning 1st brood resistance to ECB (three from Oh43 and one from B52), but not all seven genes from B52 conditioning 2nd brood resistance to ECB, because it is slightly less resistant to 2nd brood than B52. In fact, a higher proportion (54.4%) of RFLP variants in B86 on chromosomes 1, 2, 3, 4, 5, 6 and 8, which are supposed to carry resistance genes to 1st and 2nd brood, were derived from B52.

In addition to selection for genes conditioning the expression of different traits from each parent, genetic drift of genomic regions with neutral effect on the selection criteria would result in random fixation of RFLP variants from each parent. Thus, random genetic drift might be another explanation for the observed 1:1 ratio of RFLP variants of B52 and Oh43 found for the molecular genotype of B86 in this study.

Deviations of RFLP patterns for B86 from its progenitors B52 and Oh43

--M. M. Messmer, A. E. Melchinger, M. Lee and W. L. Woodman

We assayed maize inbred line B86, derived from the single cross B52xOh43, and its progenitors B52 and Oh43 with 187 mapped maize DNA clones in combination with three different restriction enzymes (*Eco*RI, *Eco*RV, and *Hin*dIII). The graphical genotype of B86 revealed by this RFLP analysis is shown in the previous note by Fähr et al.

For clones *UMC111, BNL5.47,* and *BNL6.22*, previously located to chromosomes 4, 6, and 5, respectively, the RFLP banding patterns of B86 differed from those of its progenitors (Fig. 1). Explanations for the occurrence of new RFLP patterns in B86 could be residual heterozygosity or heterogeneity in the parental inbreds, mutation, or recombination during line development and maintenance. Pollen contamination or seed mixture are unlikely, because new RFLP variants in B86 occurred with a frequency of less than 2 percent. Also flanking markers were of parental types.

Based on the results of *UMC111* with *Eco*RI and *Hin*dIII, B86 very likely inherited this genomic region from B52. The larger fragment of 6.3kb for B86 versus 5.0kb for B52 with *Eco*RV could be due to a point mutation that eliminated one restriction site for *Eco*RV in this region. This would agree with the observation of Fähr et al. that all RFLP patterns of B86 found on chromosome arm 4L were identical to those of B52. The RFLP pattern of B86 with *UMC111* could also be explained by a crossover event within this region during line development, if B52 and Oh43 reveal class I RFLPs (R. B. Meagher et al., Genetics 120:809-818, 1988) with *Eco*RI and *Hin*dIII (i.e., the polymorphic fragments differ at only one end), but class II RFLP with *Eco*RV (i.e., the fragments differ at both ends).

The deviating RFLP patterns of B86 from those of B52 and Oh43 found with clone *BNL5.47* and all three restriction enzymes could be caused by chromosomal mutation (deletion, insertion, or inversion) in this region in B86 that affects the bands of all three digests simultaneously. Another explanation for the different RFLP bands observed in B86 could be the presence of two RFLP variants in one of the parental lines. one of these variants was lost in the respective progenitor as a consequence of genetic drift, while the other was fixed in B86 during line development and maintenance.

The RFLP pattern of B86 for clone *BNL6.22* was identical to Oh43 with *Eco*RI, but identical to B52 with *Eco*RV. This indicates a crossover event between the restriction sites of *Eco*RI and *Eco*RV in the early generations of line development, assuming class I RFLPs between B52 and Oh43. This matches with the graphical genotype of B86, where the RFLP pattern of the nearest clones, *BNL10.06* and *UMC67*, originated from B52 and Oh43, respectively.

The deviations in the RFLP patterns of B86 from its progenitors for DNA clones *UMC111* and *BNL5.47* suggest that some instability at these RFLP loci could occur over several generations of line development and maintenance of B86 (1% of all probes examined). Further analyses with a larger set of materials, more DNA clones, and several restriction enzymes are needed for a more detailed investigation of (1) the molecular basis of these deviations in the RFLP patterns and (2) the frequency with which they occur.

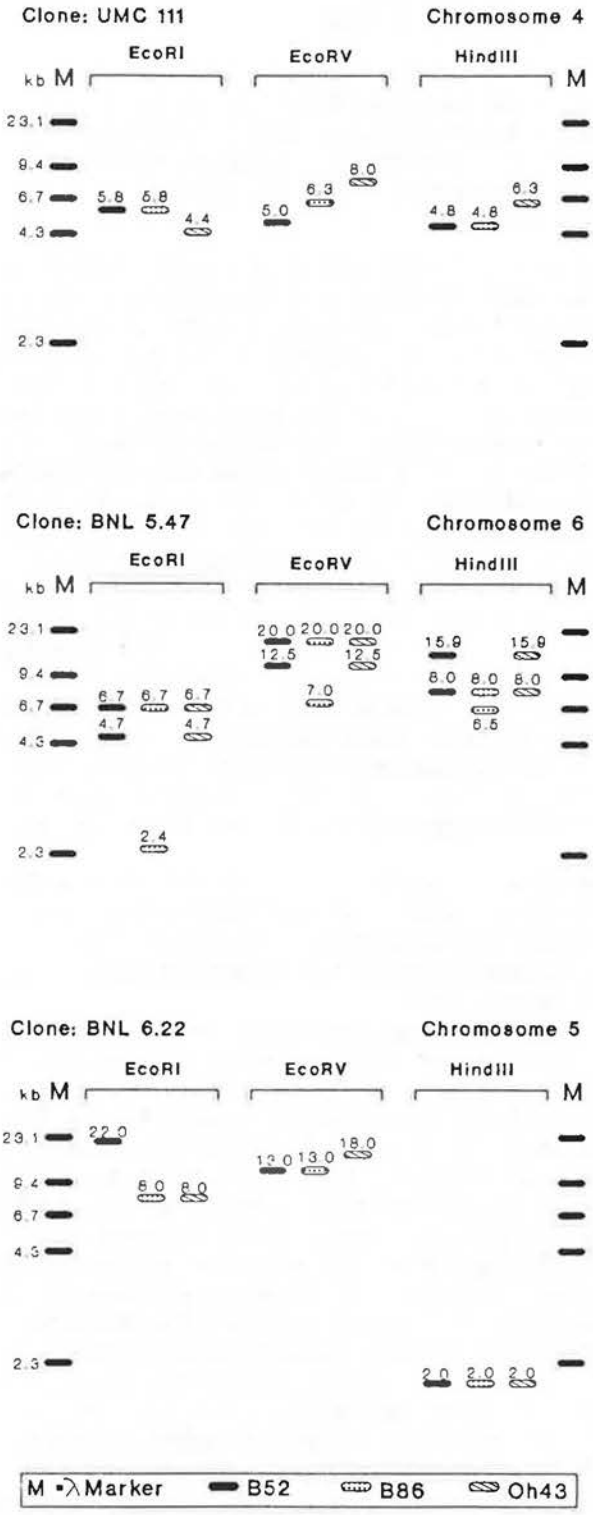


Figure 1. RFLP banding patterns of inbred lines B52, B86, and Oh43 with DNA clones *UMC111, BNL5.47,* and *BNL6.22*.

AUSTIN, TEXAS
University of Texas

A technique for spreading maize microsporocyte pachytene chromosomes for phosphotungstic acid staining to allow simultaneous EM visualization of synaptonemal complex lateral and central elements, recombination nodules, and centromeres

--M. P. Maguire

EM viewing of silver-stained spread preparations allows very clear visualization of synaptonemal complex (SC) lateral elements, either as cores in advance of synapsis or in fully synapsed configurations. However, only the lateral elements of the SC stain appropriately with silver, and central elements, recombination nodules, and centromeres remain invisible. Other stains such as phosphotungstic acid (PTA) or uranyl acetate with lead citrate must be used to stain the additional features. But these other stains also stain chromatin and other nuclear components as well, so as to obscure viewing of the SC structures and recombination nodules (RNs). Procedures must be adapted for making spread preparations which effectively remove the undesired components while keeping the SCs and RNs. Since organisms differ somewhat, differing procedures must be adapted for different organisms, notably maize, where meiotic mutants and altered chromosome complements are available for study. These studies are of special interest if late RNs, for example, can be dependably visualized as indicators of crossover positions.

The following is a procedure which allows visualization of maize SC components and RNs and also displays centromeres as rather large spheres. It represents a modification of a procedure for silver staining which was reported in MNL 63:26-27, 1989.

Fresh anthers at pachytene stage are macerated in a deep depression slide in 5µl of a freshly prepared ice cold medium: 1.5% sucrose, 1% polyvinylpyrrolidone and 2.5mM acid EDTA, adjusted to pH 4.7-4.8 with KOH. The suspension is then transferred by pipette to the surface of a 5µl drop of 0.5% Nonidet P40 in another deep depression slide, where it is left for 2 minutes. Then 60µl of an ice cold fix-detergent mixture is added. The fix-detergent mixture consists of 6% paraformaldehyde solution adjusted to pH 8.6 (as described earlier) to which Lipsol detergent has been added to a final concentration of 1.5%. The depression slide is then covered and placed over an ice bath for at least 30 minutes before the suspension is micropipetted to plastic coated microscope slides. Slides are then thoroughly dried, and fixed as described earlier, and stained with PTA at concentrations from 0.1% to 1.0%.

Future adjustments of the procedure are expected to give even clearer preparations, especially with cautious increases in detergent concentration.

BATON ROUGE, LOUISIANA
Louisiana State University Agric. Ctr.

Absolute resistance to weevils

--Manjit S. Kang

Weevils (*Sitophilus* ssp.) can be devastating to stored maize grain, especially in the southern USA. There can be 100% loss. Several varieties, especially plant introductions, were evaluated, over a four-year period, for resistance to weevils. 'Philippines' (PI91414) showed an absolute resistance to weevils (not a single kernel was damaged), whereas all other varieties were completely destroyed by weevils. Seed of 'Philippines' has been increased and is available, upon request, from this cooperator. Seed of 'Philippines' may also be available from the Plant Introduction Station at Ames, Iowa, whence this variety was originally obtained.

Recently, 'Philippines' has also been reported to have the lowest amount of aflatoxin B_1 (AFB_1) among 12 varieties at Baton Rouge and one of the lowest amounts of AFB_1 at Tifton, Georgia (Kang, Lillehoj, and Widstrom, Euphytica 51:19-23, 1990).

'Philippines' was crossed in 1990 to several Louisiana inbred lines. Future plans are to determine the genetic nature of resistance to weevils and to examine correlation between resistance to weevils and resistance to AFB_1.

BEIJING, CHINA
Beijing Agricultural University

The culture and transformation of protoplasts

--Sai Jiqing, Liu Zhihua, Ding Qunxing and Xie Youju

Protoplasts were isolated and purified from BMS (Black Mexico Sweet) suspension cells by different methods. The results show that the direct filtering method can give the highest yield of 3-4 x 10^6 protoplasts per gram fresh weight and that collecting protoplasts on the interface between KMC solution (Theor. Appl. Genet. 53:57, 1978) and 0.6M sucrose solution can enhance the level of protoplast purification even though the protoplast yield reduces.

The protoplasts were cultured in modified NN67, N6ap, KM media. The culture methods included thin liquid layer, multiple layers, embedding, C. A. Rhodes method (Bio/Technology 6:56, 1988) and so on. The highest plating efficiency of 3.5 x 10^{-3} could be obtained when 0.45M glucose was used as the stabilizer of osmotic pressure and the protoplasts were embedded in modified N6ap or KM medium with 0.6% low melting point agarose.

A special vector was constructed for protoplast transformation: Plasmid-like DNA S1 was purified from S male sterile cytoplasmic mitochondria of maize. Almost the full-length of S1 was cloned to pBR322 and the recombinant plasmid was named pBS. The GUS (glucuronidase) reporter gene with CaMV 35S promoter from pBI221 was inserted into plasmid pBS and two kinds of recombinant plasmids (pBSG1 and pBSG2) were obtained due to reversal direction of integration (Fig. 1). Increasing transformation frequency was hoped for this kind of vector con-

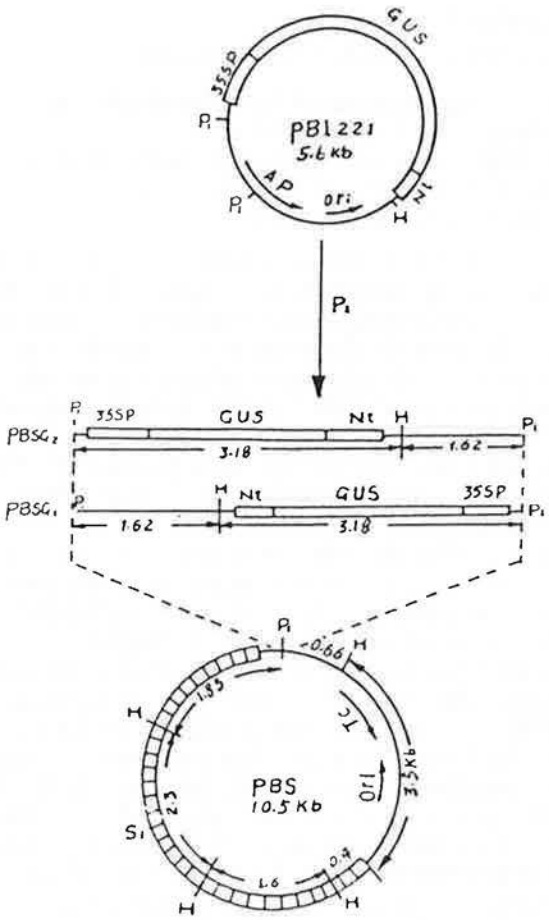


Figure 1. Construction of recombinant plasmids pBSG1 and pBSG2.

struction due to homology between plasmid-like DNA S1 and maize nuclear DNA (Nature 304:744, 1983).

With the success of protoplast culture and vector construction, the Baekon 2000 Advanced Gene Transfer System was used to transfer pBSG or pBI221 (CK) containing 35SP-GUS-Nt chimeric gene into protoplasts. Suitable electroporation parameters were studied before doing transformation. In the experiments, the burst time (T_B), the number of cycles (C_Y) and the distance between the positive electrode and the surface of gene-cell mixture (D) were stabilized. The amplitude (A) and the number of pulses (NP) were changed to observe the effects of the different combinations of these two parameters on the frequency of protoplast division. The results are presented in Table 1.

Table 1. Effects of different combinations of electroporation parameters (A and NP) on the frequency of protoplast division (%).

	A(KV)		
NP	2	6	10
2^2	32.01	33.00	32.02
2^4	30.39	32.69	24.50
2^6	34.96	30.06	22.87

The frequency of protoplast division of control (without electroporation) was 32.67%; T_B=0.4s, C_Y=10, D=2mm; Electroporation: No. protoplasts/ml=2 x 10^6, volume of electroporation mixture=200ul; Culture: No. protoplasts/ml=5 x 10^5; The Nos. in the table are the means of 5 fields of vision of inverted microscope from 3 experiments 10 days after culture.

When the amplitude was increased to 10KV and the number of pulses to 2^4 or 2^6, the frequency of protoplast division reduced obviously. We thought that the parameters A and NP suitable for maize protoplast transformation should be a compromise between increasing transformation frequency dependent on raising these two parameters and reducing the protoplast division frequency. So the parameters A and NP we used were 10KV and 2^4. The stabilized parameters T_B, C_Y and D were 0.4s, 5 and 2mm respectively in each experiment. The clones (colonies) from electroporated protoplasts growing to 1-2mm in diameter were picked onto N6ap medium in relatively low density. When the calli from colonies were approximately 1-2cm in diameter, the GUS gene expression was assayed using the histochemical method (Bio/Technology 6:559, 1988). The number of blue calli were counted (Ed. note: color print has been provided and will be supplied on request). The results are in Table 2.

Table 2. Transformation frequencies by electroporation.

Plasmid	No. calli assayed	No. blue calli	Percentage of blue calli[1]	Absolute transformation frequency[2]
pBSG	201	23	11.44	5.49×10^{-4}
pBI221(CK)	168	17	10.12	1.82×10^{-4}

[1]This is also the relative transformation frequency.
[2]Equal to relative transformation frequency x plating efficiency.

No significant difference of the relative transformation frequency between pBSG and pBI221 (CK) as foreign DNA can be seen from Table 2. The reason for this may be that the size of pBSG is much bigger than that of pBI221. Further studies are underway.

A new kernel mutant with pleiotropic effect

--T. M. Song and X. W. Lu

An opaque endosperm-small germ mutant (*os*) was observed and tested allelically with floury alleles (*fl1, fl2, fl3*) and opaque alleles (*o1, o2, o5, o7, o9, o10, o11, o13*). No allelic relationship was found. The endosperm of *os* kernels showed the opaque characteristic but was not typical. The top part of *os* seeds is completely opaque but the basal part is somewhat flint and translucent. The kernel size is larger but the color is lighter. The germ size is also obviously reduced, about 1/2 of the normal. These two traits always appear simultaneously on the same kernel, indicating the pleiotropic effect of the *os* gene.

os seeds are weaker in germinating ability and seedling vigor. Under field condition, *os* seedlings grow slower and seldom reach flowering stage but under high oil genetic background, *os* seedlings grow better and can shed normal pollen and even set seeds.

Since the *os* gene can express in both endosperm and germ and segregate normally, it is a useful seed marker.

Inheritance and function of the *os* gene

--T. M. Song and X. W. Lu

Crosses were made between *os* stock and 10 inbred lines, including 5 high oil and 5 normal. All the F1's were selfed and backcrossed to *os* plants. Data from F2 and by

a single locus. Three other endosperm mutants, *o2*, *wx*, and *su2*, were crossed with *os*. F2 data showed no phenotypic interaction between the germ traits, but there existed a clear interaction between the endosperm traits. The endosperms of double recessive *os os o2 o2* and *os os wx wx* were still opaque and waxy, but the top parts of these seeds were lighter in color and the basal parts duller, so they can be distinguished from the seeds of pure *o2* and *wx* genotypes. On the endosperm of *os os su2 su2* seeds, however, the *os* gene showed typical epistatic effect. The top part of *os su2* seeds is opaque and light color and the basal part is translucent and dull color.

The oil content of *os* seeds was significantly reduced both in the whole kernel and in the germ (see Table 1). it suggests that *os* is probably one of the major genes controlling the biosynthesis of oil. The interaction of the *os* gene with polygenic oil genes can also be seen clearly in the table.

Table 1. Oil content of the germs and the whole kernels of *Os* and *os* seeds in F2 under high oil and low oil background.

Kernel part	Cross type (F2)	Number of cross	Oil content (%) Os	os
Germ	High oil line x *os*	2	56.8	47.9
	Low oil line x *os*	2	37.7	34.4
Whole kernel	High oil line x *os*	10	10.7	6.7
	Low oil line x *os*	10	6.4	4.1

Localization of the *os* gene on the short arm of chromosome 2

--T. M. Song and X. W. Lu

The chromosome location of a recessive opaque endosperm small germ gene (*os*) was tested by using the B-A translocation technique. The heterozygous mutants (+/*os*) were used as female parents and a set of 16 B-A translocations (kindly provided by Dr. E. B. Patterson from Coop Center, USA) as males. All the F1 ears were carefully checked. Among the 16 crosses made, 15 did not have any *os* seeds. Kernels with the recessive trait were discovered in every pollinated ear of only one cross, that is: +/*os* x TB-3L-2S(6270). Among a total of 1003 kernels, 288 showed the recessive traits. However, their endosperm trait and germ trait did not occur correspondingly. Some kernels have opaque endosperm but normal size germ, while others have small germ but normal endosperm (with a ratio about 6:4), indicating the unique characteristics of nondisjunction and preferential fertilization of B^A chromosomes as well as the pleiotropic effect of the *os* gene. Noncorresponding germ and endosperm phenotype clearly reflected their different genotype. Since no *os* seed was found on the ears of +/*os* x TB-3La crosses, the *os* gene must be located on the short arm of chromosome 2. This conclusion was further confirmed by *wx*-marked translocation testing. A series of 11 reciprocal chromosome 9 translocations carrying the recessive waxy allele with one breakpoint close to the waxy locus was used to cross with the *os* mutant. In their F2 population, a significant chi-square value for independence was found only for *wx* T2-9b at the 1% level. The breakpoint of T2-9b is 2S.18 and 9L.22.

BERGAMO, ITALY
Ist. Sperimentale Cerealicoltura

Transcriptional properties of the DNA binding protein encoded by the opaque-2 locus

--M. Maddaloni, S. Lohmer[1], N. Di Fonzo, H. Hartings, M. Motto, F. Salamini[1] and R. D. Thompson[1]
[1]Max-Planck-Inst. Züchtungsf., Köln

To understand the molecular details of the role played by the trans-acting regulatory gene opaque-2 (*O2*), in promoting the coordinated gene expression of zein and *b-32* during endosperm development, the *O2* and *b-32* loci have been recently isolated and characterized in our laboratory (Hartings et al., EMBO J. 8:2895, 1989; Hartings et al., Plant Mol. Biol. 14:1031, 1990).

The genetic data indicated that the expression of *b-32* gene is under the control of the *O2* gene product. Therefore, DNA binding studies with a purified *O2* fusion protein containing 70% of the wildtype protein sequence were carried out. Band shift assays were performed in the presence of purified glutathione-S-transferase (GST)-*O2* fusion protein and a 250 long *b-32* promoter fragment. Under the binding condition applied two complexes with different electrophoretic mobilities appeared. That means that the GST-*O2* fusion protein was able to bind the *b-32* promoter. To demonstrate that the complexes formed were not the results of a potential DNA binding activity of the GST, the labelled *b-32* promoter fragment was incubated in the presence of GST alone. Under this condition no complex formation was seen. It was concluded that the retarded complexes are produced through the DNA binding activity of the *O2* component of the fusion protein.

To verify experimentally the trans-acting property of the *O2* protein on a *b-32* promoter an assay for transient gene expression in tobacco protoplasts has been employed. This assay was based on cotransfection of mesophyll protoplasts with an expression and reporter plasmid. The expression plasmid consisted of the full length *O2* cDNA under the control of promoter of the 35S gene from Cauliflower mosaic virus. The reporter plasmid was assembled by fusing the *b-32* promoter region to the coding region of the bacterial β-glucoronidase (GUS) gene.

The results of the transient expression experiments demonstrated a strong activation of the *b-32* promoter in the presence of the *O2* gene product. The site of interaction of GST-*O2* fusion protein on the *b-32* promoter fragment was mapped using DNAse I footprinting. The promoter fragment tested contains five regions which were protected by *O2* against DNAse I digestion. These experiments show conclusively that the *O2* gene product binds to the promoter region of a *b-32* gene, a finding supported by the cotransfection experiments and consistent with the available genetic data.

Molecular analysis of the *Bg-rbg* transposable element system

--C. Spilmont, H. Hartings, N. Lazzaroni, V. Rossi, M. Motto, R. D. Thompson and F. Salamini

We have continued our work on the molecular analysis of the *Bg-rbg* transposable element system. An au-

tonomous *Bg* element and a non-autonomous *rbg* element which inactivate, respectively, the *Wx* gene and the *O2* gene of maize have been cloned.

The nucleotide sequence of the autonomous *Bg* element has been determined. This element is 4,869bp long. Moreover, sequence data obtained from the autonomous *Bg* and receptor element (*rbg*) indicate that both generate an 8-bp duplication at the insertion site upon transposition, and carry 5-bp terminal inverted repeats. The inverted repeat starts with the bases CA, like a number of other elements, and exhibits further homologies to them (Table 1).

Table 1. 5' —> 3' termini (inverted repeats) of some transposons of a number of species

Element	Inverted repeat	Target	Species
En/Spm	CACTACAAGAAAA	3	Zea mays
Tam1	CACTACAACAAAA	3	Antirrhinum majus
Tgm1	CACTATTAGAAAA	3	Glycine mays
Ac	CAGGGATGAAA	8	Zea mays
dTph1	CAGGGGCGGA	8	Petunia hybrida
Bg	CAGGG	8	Zea mays

It was also interesting to note that the 5 nucleotides forming the inverted repeats of both the *Bg* and *rbg* elements are the initial part of the 11-bp and 10-bp inverted repeats found, respectively, at the end of the *Ac* and *dTph1* elements. Because of the preserved terminal five bases in all these elements they may be grouped into the CAGGG family; the 5-bp terminal repeats of members of this family display perfect sequence homology, and each element generates an 8-bp duplication of the target site upon integration. Homology of 5bp of the terminal inverted repeats has also been used to group *Tam1, Tgm1* and *En/Spm* into the CACTA family (Gierl and Saedler, Plant Mol. Biol. 13;261, 1989).

At 813bp from the 5' end of the *Bg* element an ATG codon is present. This codon is followed by a 735bp long open reading frame and ends with a TAA stop codon. The remainder of the *Bg* element sequence following this site contains three other potential coding regions. A 76bp long direct repeated sequence is present in the subterminal regions of the *Bg* element. This sequence is characterized by the presence, on both strands, of a number of perfect and imperfect copies of the hexanucleotide sequence $TATCG^{G}_{C}$. Finally, restriction enzyme analysis reveals that, compared to *Bg*, the receptor element is distinguishable by small deletion and insertion events. Sequence data indicate that not more than 75% of homology exists between the *rbg* element and the autonomous element.

Unstable alleles generated at various glossy loci

--M. Maddaloni, M. Albano, M. Motto and F. Salamini

The first 5-6 leaves of maize seedlings are covered by a layer of wax which is characterized by a typical chemical composition (Bianchi et al., Maydica 30:179-198, 1985). Starting from the sixth leaf, the metabolic routes leading to the accumulation of the juvenile wax layer are clonally disactivated and only the adult waxes continue to be present, with a specific morphology and chemical composition. The biosynthetic pathway responsible for the accumulation of these organic compounds has been in part dissected by taking advantage of the glossy (*gl*) mutations

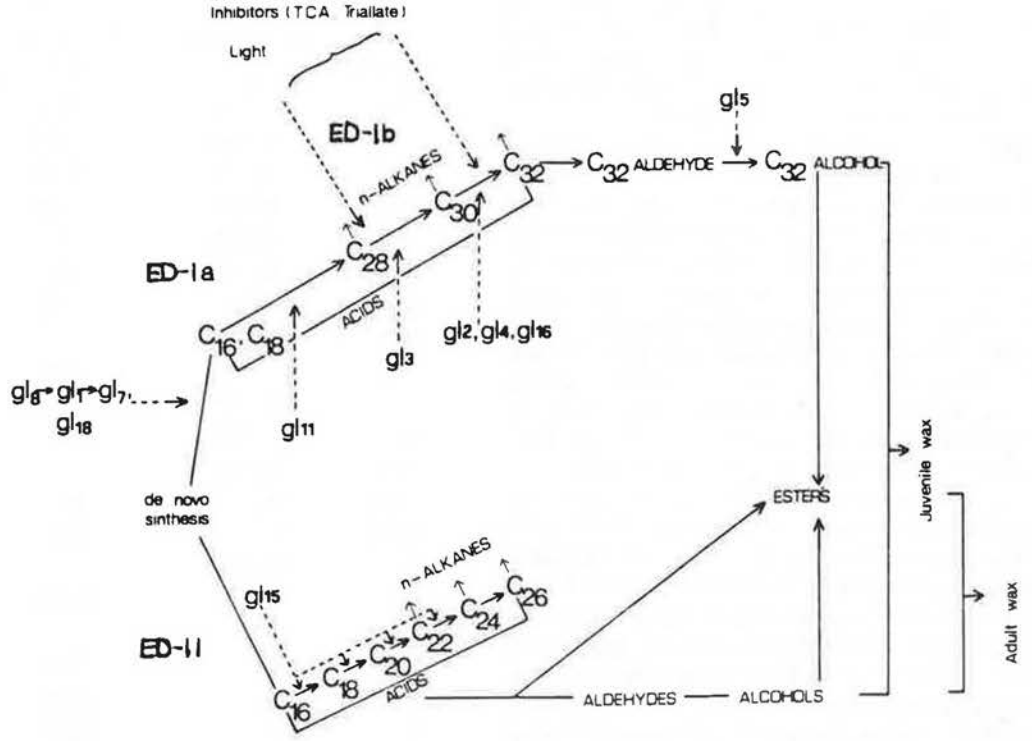


Figure 1. Biosynthetic scheme of maize surface wax assessed by studies on the effect of mutations, light, plant age and inhibitors on its composition.

Table 1 - Summary of tagging experiments at relevant Glossy loci

Female parent	Male parent	No. of F1 seedlings scored	No. of variegated seedlings found	F2 confirmed mutable alleles					
				gl1	gl8	gl12	gl5	gl6	gl3
WF9gl1gl1gl8gl8	Gl1Gl1Gl8Gl8 wx-m7 (Ac)	90,000	9	9	0	-	-	-	-
WF9gl2gl2gl8gl8	Gl2Gl2Gl8Gl8 wx-m7 (Ac)	46,000	21	-	-	6	-	-	-
WF9gl5gl5gl8gl8	Gl5Gl5Gl8Gl8 wx-m7 (Ac)	130,000	0						
WF9gl2gl2gl6gl6	Gl2Gl2Gl6Gl6 wx-m7 (Ac)	10,000	1 (1)						
WF9gl3gl3gl8gl8	Gl3Gl3Gl8Gl8 wx-m7 (Ac)	55,000	6						
WF9gl2gl2gl3gl3	Gl2Gl2Gl3Gl3 wx-m7 (Ac)	42,000	5						

(1) : allelism no yet determined

(Fig. 1) (Bianchi et al.: quoted). However, our present knowledge at the biochemical level, of the mechanism o faction of the genes controlling wax deposition is unknown.

Because the molecular cloning of the glossy loci and the analysis of their structure and expression may shed light on the mechanism affecting waxy deposition, we have begun an extensive tagging program to generate unstable glossy alleles, at the most relevant loci affecting wax accumulation, with the aim to clone these genes. Homozygous plants from variegated *wx-m7* kernels containing the *Ac* transposon at the *Wx* locus were crossed to several versions of the inbred line WF9, each containing stable double mutants at two different glossy loci. Variegated F1 seedlings, showing normal and glossy sectors on the leaves, were selected and selfed to confirm the inheritability of the variegated phenotypes. The results of the tagging experiments are in Table 1. Nine unstable mutations have been generated at the *Gl1* locus. Genetic analyses showed that: 1) seven alleles, *gl1-m1, gl1-m2, gl1-m3, gl1-m5, gl1-m7, gl1-m8* and *gl1-m9* are due to the insertion of an autonomous element, while *gl1-m4* and *gl1-m6* are controlled by the insertion of a receptor element not capable of self-excision; 2) the autonomous elements inserted into the unstable alleles *gl1-m1, 2, 3, 5, 7, 8* and *9* are functionally distinct from *Ac* as demonstrated by crossing to an appropriate *Ac*-tester strain. It is suggested that the autonomous element present in the *gl1-mutable* allele appears to be unrelated to *Ac*. Moreover, the autonomous unstable alleles share in common a rate of somatic reversion dependent on light intensity (Maddaloni et al., Maydica 35:409-420, 1990). It is worth noting that the frequency by which, in our tagging experiments, mutable glossy alleles were obtained is significantly higher in comparison to that reported for other maize genes (cf. Döring et al., Maydica 34:73-88, 1989). Because the pollen of several *wx-m7* (*Ac*) plants was bulked and used to pollinate several ears in the original cross, it cannot be excluded that the variegated phenotypes selected among the F1 seedlings derived from the same transposition event. The genetical analyses of unstable mutants at other glossy loci are currently being addressed in our laboratory.

Relatedness of inbreds based on RFLP data

--P. Ajmone Marsan, C. Livini, A. E. Melchinger[1], M. N. Messmer[1], M. Motto
[1]Univ. Hohenheim, Stuttgart

Genetic relationships among inbreds have always been interesting to maize breeders as a helpful hint to the choice of high yielding crosses. Several morphological and biochemical markers have been used to separate inbred lines into breeding groups and to assign inbreds of unknown or uncertain origin to these heterotic groups (Smith and Smith, Maydica 34:151-161, 1989).

RFLP analysis can be used to assess genetic distance among genotypes at the molecular level. In maize, a theoretically unlimited number of RFLP markers is available

Tab. 1 - Heterotic groups of 41 inbred lines of maize used for RFLP analysis

Inbred	Origin	Inbred	Origin
B14	SSS B14	Mo17	LSC C103
A68	SSS B14	Lo976	LSC C103
AI1	SSS B14	Lo977	LSC C103
AI2	SSS B14	C103	LSC C103
CM109	SSS B14	C123	LSC C103
B37	SSS B37	Lo881	LSC C103
Lo999	SSS B73	Va22	LSC C103
B73	SSS B73	Va35	LSC
AI3	SSS B73	Va59	LSC
AI4	SSS B73	Va85	LSC
AI8	SSS B73	H99	LSC Oh43
B84	SSS B73	Va26	LSC Oh43
Lo950	SSS	AI10	LSC Oh43
Lo951	SSS	AI6	LSC Oh43
AI5	SSS B3	AI7	LSC Oh43
N28	SSS N28	Lo924	LSC Oh43
Pa91	Wf9	AI13	W153R
AI9	Wf9	AI12	W153R
H55	HY	Lo932	W153R
H96	HY	Lo944	W153R
AI11	HY		

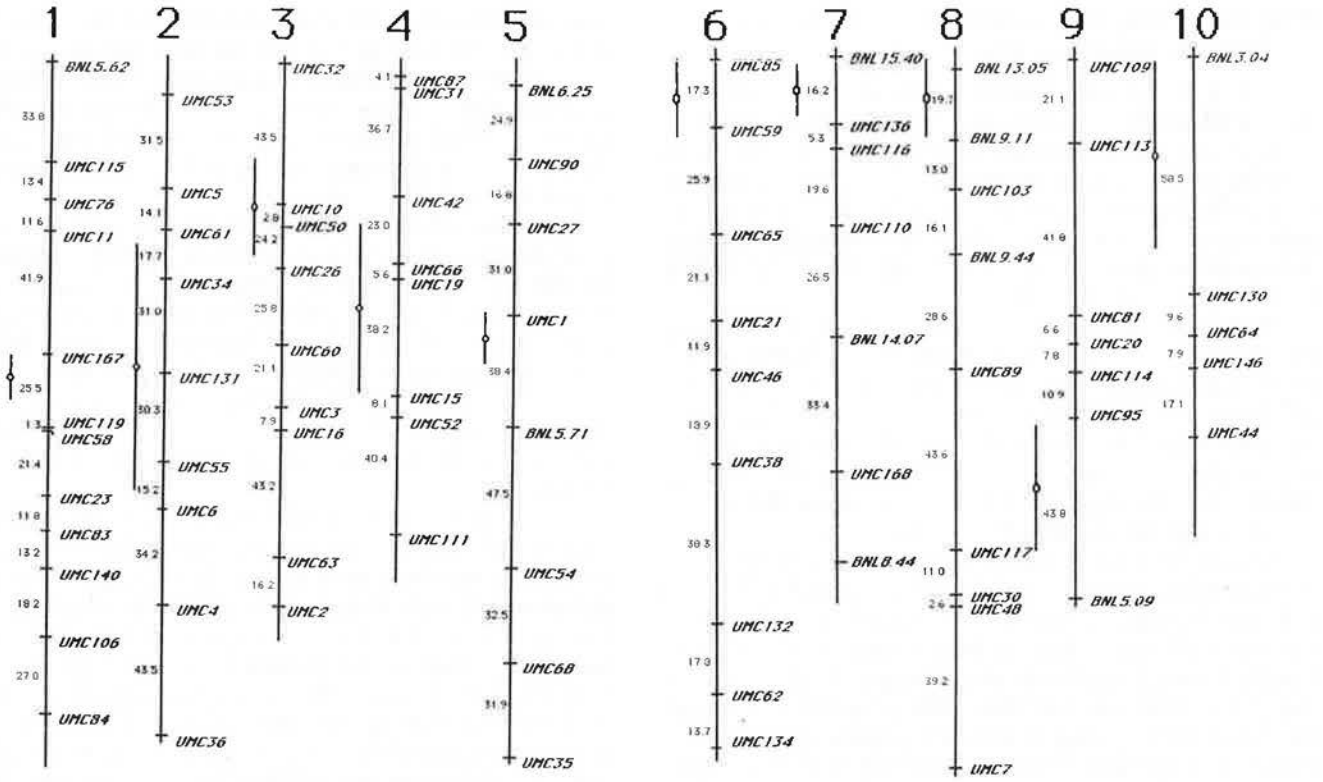


Figure 1. Linkage map of probes used for RFLP analysis of 41 maize inbred lines.

for all chromosomes allowing a very good saturation of the whole genome.

Forty-one inbred lines (Table 1) chosen from the maize germplasm collection present at the Bergamo Cereal Crops Experimental Station, have been submitted to RFLP analysis. DNA from these inbreds was separately restricted with *Eco*RI and *Hin*dIII and hybridized with 83 genomic probes kindly supplied by Dr. Hoisington, University of Missouri, Columbia (Fig. 1). Genetic distances among lines were calculated using Dice (Dice, Ecology 26:297-302, 1945) similarity coefficient. Collected data were subjected to cluster analysis by using the average linkage (UPGMA) method.

The results of this analysis are shown in Fig. 2. The cluster analysis differentiated the inbreds in agreement with known pedigrees. All lines derived from Iowa Stiff Stalk Synthetic (SSS) heterotic group clustered together. Within this group it is possible to distinguish different subgroups of lines related to B73, B37, and B14A. Other clusters are formed by inbreds belonging to the Lancaster Sure Crop breeding groups C103 and Oh43 and by lines related to Hy and WF9.

It was noteworthy to see that closely related inbreds (B14A, B68 and AI1; AI3 and AI4; H55 and H96) have a very high similarity coefficient (S>0.8). Our results show that, using a sufficient number of probes, RFLP analysis was able to classify lines with various origins into different breeding groups and to give information about the real genetic distances among different genotypes.

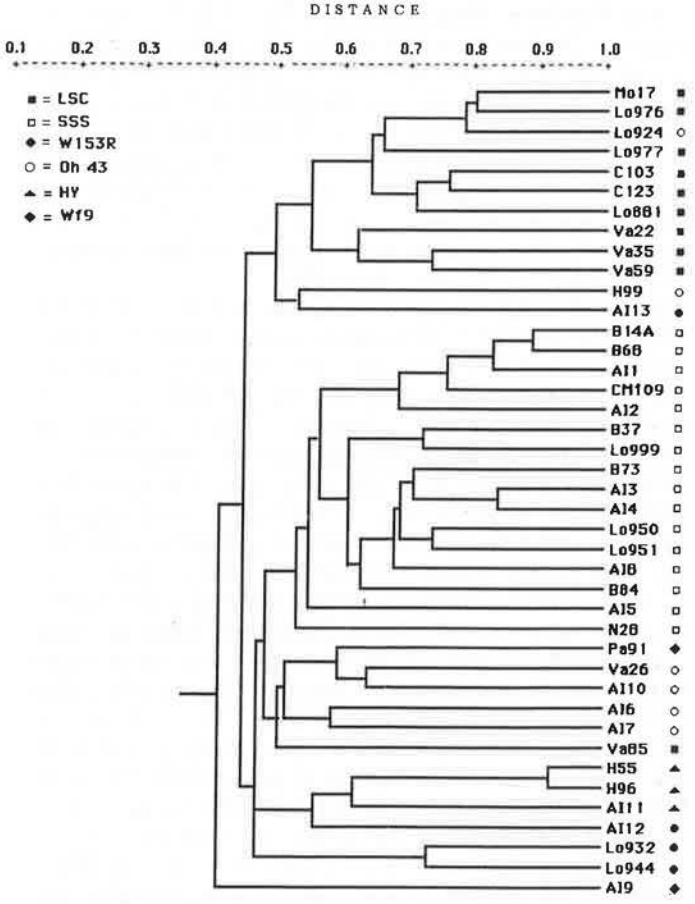


Figure 2. Dendogram revealing associations between lines following cluster analysis of RFLP data. Genetic distance is calculated using Dice similarity coefficient.

Desiccation tolerance in embryogenic cultures

--F. Locatelli and E. Lupotto

During studies on alternative methods for transformation (MNL 64:22-23, 1990), the capacity of embryogenic cultures to sustain rapid and drastic desiccation has been ascertained. This characteristic, peculiar to the zygotic embryo during maturation in planta, has been already detected in other plant species, where dried somatic embryos were proposed as synthetic seeds (Senaratna et al., In Vitro Cell Dev. Biol. 26:85-90, 1990).

Therefore, the system in maize has been more deeply investigated, in order to characterize the conditions needed for an efficient recovery of the tissues and consequent plant regeneration. Because of the strict homology existing among the Gramineae, it is presumable that results may be extrapolated for other monocot cultures, namely cereals. We already have additional evidence that this is true for sorghum and durum wheat cultures.

Embryogenic tissue cultures were derived from genotypes establishing type 2 highly embryogenic, friable calli: B79, LH126 x B79, Lo907 x B79, and Lo976 x B79 F2 selections. Calli were induced from immature embryos in the presence of 2 mg/l 2,4-D and propagated in the dark on 1 mg/l 2,4-D (ref. Locatelli et al., MNL, this issue). For drying treatments, homogeneous embryogenic calli, approximately of equal size, were placed on a layer of filter paper in petri dish lids in constant air stream (0.45 m/sec) in a sterile flow hood. Treatments were from 1 to 6 hours desiccation. Desiccated calli were then tested for growth and development on N6P and MShf, respectively. Performance of treated tissues was compared to that of control calli non-desiccated and grown in the same conditions. Parameters used for evaluation were: callus growth, somatic embryo germination, and plantlet development. When the effect of storage was tested, dried tissues were stored in the dark in 10ml screw cap plastic tubes at room temperature (22±2 C) or at 4 C. Alternatively, storage was also tested at -20 C. Desiccation was tested on 50-70mg FWT calli during 6 hours, allowing us to draw the desiccation curve of these tissues. Two hours treatment reduced the fresh weight of the tissue to 35-40%; at 4 hours desiccation, weight was reduced to 15-20% of the initial fresh weight. Further desiccation did not reduce consistently the callus weight. Calli were induced to recover on N6P medium either directly, or with a previous rehydration in a 100µl drop of liquid medium. Slow rehydration (on agar medium) was more effective than rapid rehydration (on liquid medium). Furthermore, desiccated calli (2 hours treatment) resulted in definitively better growing than control cultures, the highest difference being registered at day 21 of subculture. Fast rehydrated calli partly degenerated, and their growth was far less than control calli. Re-established cultures were consistently similar to the original ones and could be propagated in the same way, thus confirming that desiccation did not interfere either with callus performance or with callus phenotype.

When desiccated calli were placed directly onto MShf medium they proliferated into germinating somatic embryos which subsequently developed into complete plantlets. The whole procedure of regeneration was the three step procedure described in Locatelli et al., MNL, this issue. The number of regenerates was similar or even higher than the total yield of non-desiccated calli. The efficiency of plant regeneration in B79 cultures was 16.21 plants per g FWT in control calli, against 18.47 plants per g FWT in desiccated tissues. Again, the best performing calli were desiccated two hours.

The effect of storage on desiccated calli was investigated; storage up to one year is currently being considered. Results obtained to date indicate a relative resistance of non-treated calli up to 2 weeks either at room temperature or at 4 C in the dark. Growth index was comparable to that of non-stored calli (about 8-9 GI in a 21 day subculture). Conversely, 2 hours-desiccated calli were more efficiently stored at 4 C in the dark. Their GI was about 6-7, compared to the same tissues at room temperature, with GI=3-4.

Furthermore, storage of 2 hours-desiccated tissues stored at 4 C did not affect the recovered callus phenotype, whereas in non-desiccated calli and in 2 hours-desiccated calli stored at room temperature, a change of callus performance, toward a non-embryogenic, rooting type, was registered. Four hours desiccation and storage at 20 C were lethal in all cases. The most interesting observations on the desiccation system were made when desiccated calli (2 and 4 hours) were stored (room temperature, 4 C) and subsequently plated on MShf for direct regeneration. In this case, 2 hours desiccation and storage at 4 C resulted in the best plant regeneration. One hundred percent of the desiccated and stored calli germinated somatic embryos. Among these, a high percentage regenerated into complete plantlets (70-80%). Regenerated plantlets were also more vigorous than control regenerates obtained from non-desiccated calli.

The whole system presents peculiar characteristics which may be particularly interesting as an applicative tool for storage and diffusion of elite maize (or cereal) lines in vitro, and as a model system for studying in vitro response to desiccation.

Evaluation of selectable markers for the identification of transformation events in regenerating cultures

--F. Locatelli, A. Rossini, M. C. Lusardi [1] and E. Lupotto

[1]Inst. Pflanzenwiss., Zürich

Kanamycin resistance, encoded for by the APH(3') (or NPT II) gene, driven by the 35S CaMV or other promoters, is currently used as a selectable marker in many dicot systems for transformation. Kanamycin resistance has also been applied with success to Gramineous species, such as *Zea mays*, *Triticum monococcum* and *Oryza sativa*. However, tissue cultures of cereals, supported by 2,4-D containing medium, are generally hard to select by using kanamycin because of the relative tolerance of the callus at high levels of the aminoglycoside.

Although widely used, little is known about the effect of other antibiotics related to kanamycin:--neomycin, G418, paromomycin and hygromycin B--on the conditions needed for selection in embryogenic systems, such as embryogenic monocot cultures. This information assumes

particular relevance because at least two techniques of transformation, namely microinjection and biolistic method, utilize embryogenic cultures as target tissues. The aim of this study was to screen the effect of these four antibiotics (kanamycin, geneticin, paromomycin and hygromycin B) on the development of embryogenic cultures of maize when selection was applied in regenerative conditions.

Embryogenic tissue cultures of various genotypes were initiated from immature embryos on N6 basic medium, 2% sucrose, 100 mg/l m-inositol; 100 mg/l casein hydrolysate, 2 mg/l 2,4-D (N6I medium), and propagated on the same medium with 1 mg/l 2,4-D (N6P). Regeneration medium was MS basic medium, 2% sucrose, 100 mg/l m-inositol, hormone-free (MShf). The following genotypes, which established type 2, long term, embryogenic cultures were considered: B79 inbred line, LH126 x B79, Lo907 x B79, Lo976 x B79 F2 selections. Regeneration was achieved through a three-step procedure, devised for enhancing the final yield of plantlets. Briefly, 50 mg fresh weight tissue pieces from a 15 day old subculture were transferred from N6P to MShf medium, 16 per 90 mm petri dish, incubated in the light with a 16/8 hours day/night regime. After 5 days, germinating somatic embryos, with enlarged opaque white scutellum arising at the surface, were distinctively detached and transferred into the same conditions. After an additional 7-10 days, regenerated plantlets were separated and secondary somatic embryos, originated from the scutellum of the previous ones, were singularly transferred in the same conditions and induced to regenerate.

The whole procedure yields, on the average, from 15 to 25 plantlets, depending on the genotype, per gram fresh weight tissue of the original inoculum. This procedure is virtually more efficient for the final yield of plant regeneration than a simple transfer of calli into hormone free medium as single step. The efficiency of the system makes reliable the recovery of transformed individuals in regenerative conditions where the interference of 2,4-D on the effect of the selective agent is avoided. Therefore, the influence of the various antibiotics during regeneration was investigated. The interference of antibiotics in regenerative conditions was evaluated by recording: i) somatic embryo germination, ii) coleoptile emergence, iii) greening of the coleoptile, iv) shoot emergence (white/green), v) rooting of the shoot, and vi) plantlet development. Each experiment has a minimum size of 16 calli/genotype/treatment/dosage, and was repeated at least twice. Antibiotics were tested at concentrations of 10, 20, 50 and 100 µg/ml. The results obtained indicated that although kanamycin, geneticin and hygromycin B all act by inhibiting ribosomal protein synthesis, they, however, has a differential effect on the developmental pathway from the early pro-embryogenic structures until complete plantlet formation. Selection applied in regenerative conditions was markedly more stringent than the counterpart applied in propagative conditions in the presence of 2,4-D. For example, a significant inhibition of callus growth was obtained in 21 day subculture in the presence of 100-120 µg/ml kanamycin, whereas 10-15 µg/ml blocked greening of the coleoptile in germinating somatic embryos on MS hormone free medium, in 5-7 days.

Propagation of the embryogenic tissues in a 21 day subculture on 10 µg/ml kanamycin, followed by transfer on 10 µg/ml kan in MShf medium, resulted in somatic embryo germination, swelling of the scutellum and further occasional development of a white coleoptile. No rooting occurred and the general tendency of the tissues was a complete block of development. When occasionally plantlets were produced, they were totally white. Neomycin acted in the same manner. Genotypic differences in response were detected, depending on the genotype of the cultures. Inbred B79 derived cultures were definitively more sensitive than F2 selections from crosses of B79 with other inbred lines; however, no cultures of these genotypes gave escapes when selective agent was applied during regeneration. G418 was less effective than kanamycin. Complete block of the embryo germination occurred at 50 µg/ml G418. Plantlet development and greening occurred at levels as high as 20 µg/ml. Higher selective pressure (50 µg/l) led to embryo swelling and abortive coleoptiles. Among this class of antibiotics, paromomycin was the least effective. Green plantlets could be recovered through germination of the somatic embryos at dosages as high as 50 µg/ml paromomycin. Only at 100 µg/ml a complete block of somatic embryogenesis was recorded. Escapes were detected in the less sensitive genotypes. Hygromycin B was the most effective selection agent in propagative conditions, where 50 µg/ml hygromycin B completely stopped callus growth and no further recovery was achieved upon release of the selection. In regenerative conditions hygromycin B was effective at 10 µg/ml like kanamycin and acted similarly. The sensitivity to hygromycin B was not genotype dependent, selection hampered development in all the genotypes tested, and no escapes were recovered. Selection procedures developed on regenerating embryogenic monocot cultures may help in the recovery of unambiguous events of transformation.

BERKELEY, CALIFORNIA
University of California

Kn2 and *Rs*-1025* are putative alleles of *Rs1-O*

--Philip Becraft and Michael Freeling

Rough sheath 1-O (*Rs1-O*) is a dominant mutation of spontaneous origin, which causes disruption of the ligular region, as well as reducing plant stature and causing the plant to bend over (Khadzhinov, Bull. Appl. Bot. Genet. Plant Breeding. Series 2: 247, 1937). *Rs1-O* leaves contain convoluted, sheath-like tissue around the ligular region and blade base, which appears to result from continued growth of this tissue beyond normal. The ligule can be discontinuous, displaced or missing. The vascular anastomosis which normally occurs in the ligular region as the basipetal veins enter the sheath is greatly disrupted, with veins often crossing each other several times before finally joining. In a B73 background, the *Rs1-O* phenotype is usually apparent from about leaf 9 on.

Knotted 2 (*Kn2*) and Rough sheath-1025 (*Rs*-1025*)

are also dominant mutations which cause phenotypes similar to *Rs1-O*. *Kn2* arose spontaneously in a commercial hybrid, and was originally designated *K-4* (Zuber, MNL 49:135, 1975). *Kn2* is not linked to *Kn1* (Freeling and Hake, Genetics 111:617-637,1985). *Rs*-1025* arose in a Robertson's Mutator family. The *Rs*-1025* phenotype is very similar to *Rs1-O*, although it tends to be less severe, and is more prone to genetic background effects. *Kn2* affects the ligular region much like *Rs1-O*, but the phenotype tends to affect the lower portion of the plant, with normal leaves appearing after about leaf 14, in a B73 background.

To test for linkage, *Kn2* and *Rs*-1025* were crossed to *Rs1-O*, and the double mutants were outcrossed to a wild-type tester. From the *Kn2;Rs1-O* testcross, 23/23 plants displayed a mutant phenotype. In the *Rs*-1025;Rs1-O* testcross, 15/15 progeny were mutant. This demonstrates that *Kn2* and *Rs*1025* are both linked to *Rs1-O*. The genetic linkage and similarity in phenotypes suggest that these mutations are allelic.

Hsf1-O (Hairy sheath frayed): 5L linkage data

--R. Bertrand-Garcia and M. Freeling

Three point backcross linkage data on a population of 258 individuals gave the following: 24.8% crossing over between *pr*1 and *Hsf1-O* and 26.4% crossing over between *zb*3 and *Hsf*1-O. The cross over frequency between *pr*1 and *zb*3 was 42.6% which closely agrees with the unpublished *zb1-pr1* linkage data of M. Demerec. The known linkages between *pr1*, *zb3* and the data generated in this study place the genes in the order: centromere--*pr1*--*Hsf1*-O--*zb3*.

A. + Hsf1 + / pr1 + zb3 x pr1 + zb3

Parental		pr--Hsf		Hsf--zb		Double	
+ Hsf +	pr + zb	pr Hsf +	+ + zb	pr + +	+ Hsf zb	pr Hsf zb	+ + +
69	68	27	26	36	21	0	11
137		53		57		11	
%Recombination		24.8				26.4	

Descriptions of the various phenotypes affected by *Hsf1* and their dependence on generation time are now in press for early 1991(Amer. J. Bot.). *Hsf1* slows the transition of cells from one developmental stage to the next.

The mechanism of GA1 action: is GA1 synthesis separated from its site of action?

--R. G. Winkler and M. Freeling

An understanding of the spatial and temporal relationship between the synthesis of plant hormones and their action (plant growth regulators) is necessary to understand their roles in plant growth and development. Mosaic analysis was employed to test cell and organ autonomy of the plant growth regulator GA1. Maize *d1* is defective in the biosynthesis of the biologically active gibberellin, GA1. Mosaic maize plants were generated that contained sectors of GA-synthesis deficient tissue (*d1*) in otherwise normal plants. Sectors hemizygous for *d1* were identified with the linked carotenoid marker *cl1*. We examined *d1* sectors for variations in cell size and for altered growth. Sector boundaries were examined for distortion of cell shape and size. All smaller sectors (<1 cm wide) indicated that GA1 was diffusible over small distances. However some distortion in cell growth was observed in larger sectors indicting possible undergrowth of GA-deficient tissue.

Liguleless 4, a new dominant mutation that alters the sheath-blade boundary in maize leaves

--John Fowler and Michael Freeling

The ligular region of maize provides an excellent model system for the study of development in plants. This region occurs on the maize leaf at the junction of its two major organ-components: the blade, which bends outward from the stem, and the sheath, which wraps tightly around the stalk. The boundary between these two morphologically distinct tissues is sharply defined by the ligule, a small fringe of tissue angled upward on the inner surface of the leaf, and by the auricle, a yellowish, thickened area which lies just above the ligule. By using mutations which specifically alter the morphology of this region, and therefore define genes which act to control the decisions made during its development, it will be possible to gain insight into the mechanisms by which plants control those decisions. Here we describe a new mutation, Liguleless 4, which appears to affect a gene involved in this process.

Lg4 (originally designated *Lg*-403*) was originally isolated by Dr. Scott Poethig in a cross from a Robertson's Mutator male parent to a W23 standard inbred line. The mutation was crossed into the standard inbreds W23, Mo17Ht, and B73 to observe background effects on expression of the mutant phenotype, and mutant progeny were backcrossed to continue introgression into these lines. *Lg4* has now been backcrossed into these inbred lines four times, and the morphological characteristics described here are seen in all lines in which the mutation shows penetrance. The phenotype of *Lg4* is similar to that of *Lg3*, showing sporadic fingerlike intrusions of transformed tissue from the sheath into the blade, and elimination of the ligule and auricle in the region where these transformations occur. Vestigial fringes of ligule and accompanying auricle can sometimes be seen displaced into the blade along the transformed tissue, and the transformations appear to be associated with the lateral veins in the blade (Figure 1). The "fingers" can appear anywhere along the width of the leaf, although they occur most commonly near the leaf margins and very rarely at the midrib, in direct contrast with *Lg3*, which affects the blade region around the midrib of the leaf. Preliminary evidence from sector analysis of the mutant suggests that *Lg4* acts cell-autonomously.

Hand sections of transformed regions of the blade revealed characteristics which are generally associated with the sheath. These characteristics include few intermediate veins, closely spaced lateral veins, thickened tissue, and fewer chloroplasts. When examined using Scanning Electron Microscopy, the morphology of the epidermal cells of the transformed tissue was found to be similar to that of sheath epidermal cells, and distinct from blade epidermal cell morphology. These results indicate that *Lg4* causes a transformation of blade cells into cells which adopt a

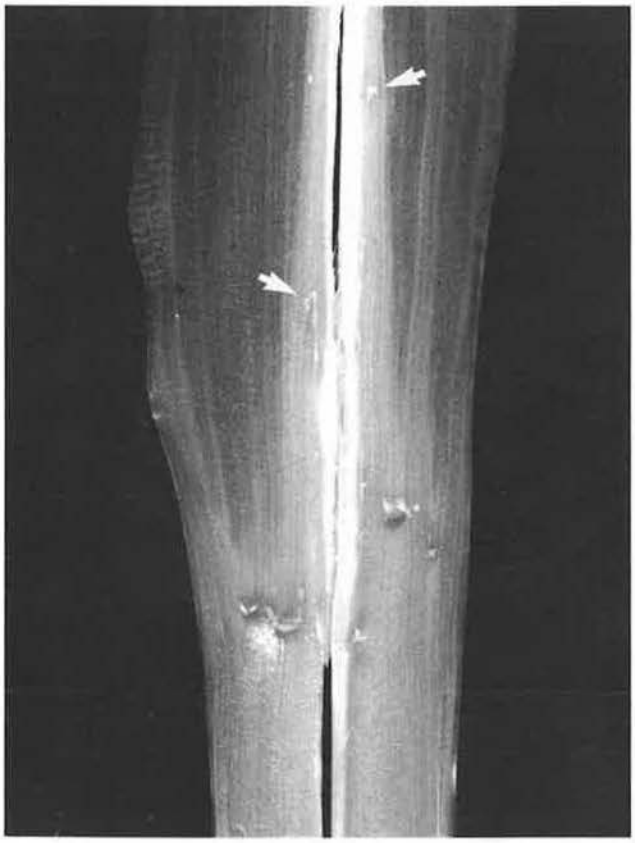


Figure 1. Leaf expressing *Lg4* in a W23 background. Note dispacement of ligule fringes into blade at the boundaries of the transformed tissue.

sheath cell fate, altering the normal blade-sheath boundary in the maize leaf.

Another interesting characteristic of *Lg4* is that its expressivity is highly affected by genetic background. By the second generation of backcrossing into the B73 inbred line *Lg4* is totally suppressed, but continued outcrossing of a B73-suppressed *Lg4* plant to lines which allow penetrance will restore expression of the phenotype. In the Mo17Ht and W23 inbreds *Lg4* shows high penetrance, but the timing of its expression is modified by both lines. In W23, *Lg4* is expressed most severely on the lowest leaves of the plant and is often absent from the topmost leaves. However, in the Mo17Ht background the pattern is reversed and *Lg4* is more severe at the top of the plant (Table 1).

Table 1. Expressivity of *Lg4* heterozygotes in W23 and Mo17Ht inbred lines. Each leaf on plants expressing the phenotype was assayed for appearance of transformed blade tissue. Severity from 0-5 was assigned based on an estimate of % blade tissue transformed to sheath, 0 being none and 5 being ~25%. Families assayed had been backcrossed into the respective lines four times, and segregated 21 *Lg*:26 + in the W23 family and 18 *Lg*:15 + in the Mo17Ht family.

Leaf #	W23		Mo17Ht	
	# Leaves Affected	Avg. Severity	# Leaves Affected	Avg. Severity
2	5/6	nd	0/13	-
3	11/11	nd	0/17	-
4	18/18	nd	0/18	-
5	18/18	nd	1/18	.06
6	18/18	3.0	7/18	.39
7	18/18	2.7	10/18	.61
8	18/18	2.8	14/18	.94
9	18/18	2.5	16/18	1.2
10	17/18	2.3	18/18	1.6
11	18/18	2.1	18/18	2.1
12	17/18	1.6	18/18	2.2
13	15/18	1.3	18/18	2.8
14	14/18	1.1	18/18	2.9
15	10/18	.83	18/18	3.4
16	9/18	.67	18/18	3.6
17	4/10	.70	16/16	3.5
18	1/3	.33	4/4	3.5

Table 2. Mapping *Lg4* to 8L with *wx* translocations. Numbers indicate the total number of progeny of the indicated genotype. Plants 378-3, 379-4, and 379-5 were heterozygous for *Lg* and the translocation indicated; family 358 was a homozygous *wx* tester line.

Cross	Translocation	*Wx Lg*	*Wx +*	*wx Lg*	*wx +*
378-3x358-3	8L.09; 9L.16	53	28	6i	32
379-4x358-L3	8L.35; 9S.31	31	18	nd	nd
379-5x358-L1	8L.35; 9S.31	79	17	9	45

Table 3. Confirming linkage to 8L with RFLPs. Linkage of *Lg4* to 8L was confirmed using the RFLP loci *UMC89* and *UMC120*. 27 *Lg4* individuals from a family segregating for *Lg4* and RFLPs from the W23 and Mo17Ht inbreds were scored for linkage using Southern analysis. Calculated map distances are given in the text.

Progeny Type	#
UMC120-W23 - Lg4 - UMC89-W23	20 (parental)
UMC120-Mo17 - Lg4 - UMC89-W23	6 (single recombinant)
UMC120-W23 - Lg4 - UMC89-Mo17	1 (single recombinant)
UMC120-Mo17 - Lg4 - UMC89-Mo17	0 (double recombinant)

Finally, *Lg4* has been mapped to chromosome 8L. The mutation was placed on 8L by linkage to the waxy translocations T8-9d and T8-9(6673) and confirmed by RFLP analysis. Available data place *Lg4* between *UMC120* and *UMC89*, 22 ± 2.2 map units from *UMC120* and 3.7 ± 0.98 map units from *UMC89* (Tables 2 and 3). Mapping to conventional genetic markers on 8L is in progress.

Twisted 1: a new morphological mutation

--R. Kelly Dawe and Michael Freeling

In its most extreme form, homozygous twisted 1 plants form ten to twenty extra leaves with whorled phyllotaxy (Fig. 1). This recessive mutant was recovered from a Robertson's Mutator background. In the upper two thirds of the plant, at least seven different characters are observed that are collectively responsible for the extreme phenotype shown in Figure 1. In no case have we observed the mutant phenotype in the lower six nodes of the plant. The expressivity of the mutant varies with the environment, being most extreme in our winter greenhouse.

The common traits of the mutant are listed below with respect to the frequency of their occurrence:

1) *multiple midribs*. In the greenhouse every mutant plant shows at least one leaf with more than one midrib (Fig. 2C). There can be as many as four midribs on a single leaf. The wide, lax midribs that are sometimes observed (Fig. 2B) are thought to be the intermediate between one and two midribs. Leaves with no midrib are occasionally observed. The presence of an ectopic midrib clearly inhibits the development of the auricle (see diminished auricle in Fig. 3), and when there is no midrib, the auricle extends from margin to margin. Extra midribs often cause a displacement of the ligule towards the base of the leaf (Fig. 3).

2) *Shortened internodes*. About 70% of greenhouse plants have at least one shortened internode. Some are

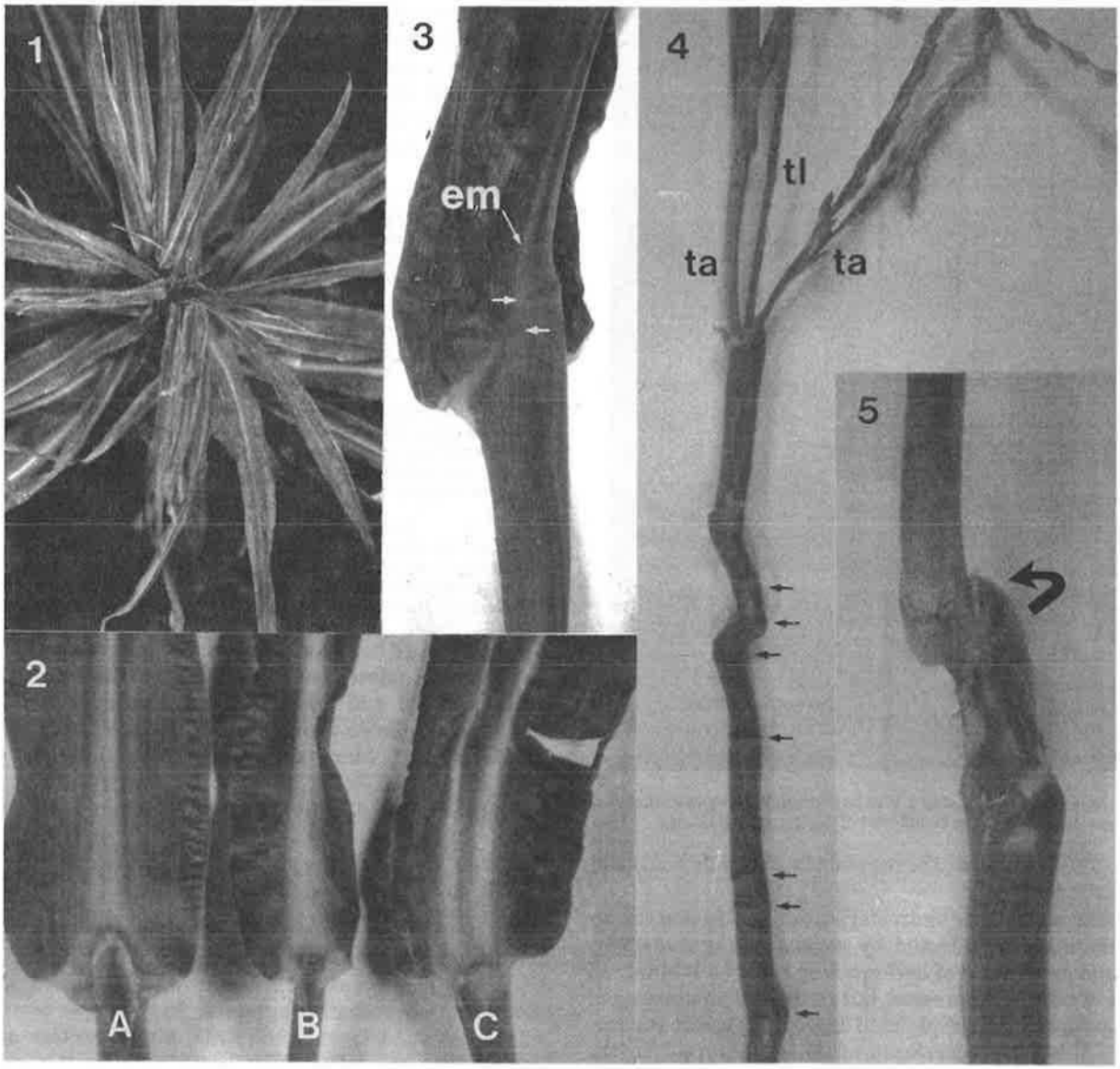


Figure 1. Top view of a twisted 1 plant in its most extreme form.
Figure 2. Multiple midribs. A, wild type leaf; B, leaf with wide, lax midrib; C, leaf with two midribs.
Figure 3. Auricle inhibition and ligule displacement. em, extra midrib. The upper and lower arrows delimit the distance of basipetal ligule displacement at the place where the extra midrib intersects the ligular region.
Figure 4. Shortened nodes, barren nodes, split plant, and tube leaf. ta, tassel; tl, tube leaf. Arrows indicate the positions of barren nodes (leaves were removed from upper two nodes). Note some nodes are shorter than others.
Figure 5. Twisted internode. Arrow shows direction of twist.

shown in Figure 4.

3) *Opposite leaves*. About 40% of greenhouse plants have opposite leaves. It is unclear whether these are the result of an extremely short internode or a legitimate switch in phyllotaxy. Opposite leaves are generally followed by series of additional leaves with an opposite decussate arrangement.

4) *Barren nodes*. What appear to be nodes without leaves are found on 30% of the plants in the greenhouse. As many a seven in a row have been observed (Fig. 4). Interestingly, the presence of barren nodes does not appear to change the total node number. If the plant counts vege-

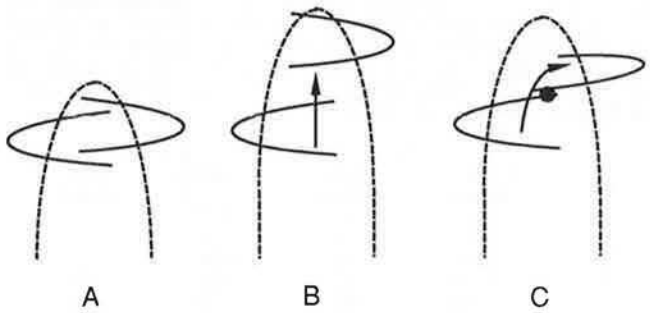


Figure 6. Proposed mechanism for twisted internodes. Dashed arch represents growing shoot. Solid horseshoes represent the bands of leaf initials. Leaf initials partially encircle the meristem and slightly overlap one another when viewed from the top (A). As growth proceeds, an internode develops and separates the leaf initials (B). In twisted plants, part of one node catches on the node below, binding the two bands of leaf initials (C, small filled circle). In the case illustrated, the leaf initials are bound on the rear side of the meristem. Internode growth is subsequently limited by the combined length of the leaf initials, and the internode twists to accommodate.

tative nodes (see Irish and Nelson, 1988, Planta 175:9-12), it probably does not count leaves .

5) *Twisted internodes*. About 30% of greenhouse plants have at least one twist. We think twisting results from part of one node (or major veins) getting "stuck" on the node before (Fig 5, 6). In the most extreme case twisting continues indefinitely until plant senescence (Fig. 1). In these "infinite twists", leaf width decreases and leaf number increases.

6) *Split plants*. About 20% of greenhouse plants have between two to four tassels (Fig. 4). There can be as many as four nodes between where the plant splits and where tassels are formed.

7) *Tube leaves*. About 20% of greenhouse plants have at least one leaf with fused margins (Fig. 4).

Ac11 acts early in plant development

--R. Kelly Dawe and Michael Freeling

The *Ac* (*Ac11*) element in the stock that gave rise to *Adh1-2F11* was found to induce large leaf and tassel sectors in plants expressing *bz2-m*, *a1-m3* and *a1-m4* (Dawe and Freeling, 1990, Dev. Biol. 142:233-245). About 15% of plants with *Ac11* (and an appropriate *Ds* reporter allele) have sectors as large as one half tassel branch, and sectors are often larger than half of the tassel circumference. *Ac11* has a similar effect on *Ds* elements that cause chromosome breakage (Scott Poethig, personal communication).

Ac11 was mapped 18 units distal to *wx* on chromosome 9 (and 2 units from *bz1*). The *Ac* element in the stock that gave rise to *Adh1-Fm335* (*Ac335*) was found to induce late plant sectors and to map 29 units from *wx* (three point crosses have not been made). Both of these stocks originally came from Drew Schwartz, so these *Ac* elements are presumably related by a transposition event(s). The difference in the timing of *Ac* activity is tissue specific. Though more research is needed, *Ac11* appears to act *later* in seed development than *Ac335*, the reverse of the situation in plant tissues.

Unfortunately *Ac11* is not at a known locus, and probably transposes to new sites at a high frequency where it may or may not retain its property of earliness. Nevertheless, as long as the element is maintained in stock where its activity can be monitored, it should prove useful in genetic mosaic studies where *Ds* is used to remove a wild type allele by chromosome breakage (Neuffer, MNL 63:61). Whether or not the earliness of *Ac11* affects the overall transposition frequency has not been investigated.

Lxm1-O suppresses the *Kn1-O* lateral vein phenotype

--Michael Freeling

Among the mutants that alter leaf development, a class of at least six genes, each defined by dominant mutant alleles, moves the position of the blade-sheath boundary in the leaf. This boundary is marked by the presence of the auricle and ligule. Mutants *Kn1*, *Rs1*, *Lg3*, *Lg4*, *Hsf1* and *Lxm1* all move the boundary out toward the tip of the blade, leading to the transformation of blade tissue to sheath tissue. No mutants have been observed that show the opposite polarity. Since *Kn1*'s coding sequence contains a homeodomain (Hake in Wessler and Hake, 1990, The Plant Cell 2:495), fundamental regulatory levels are probably involved.

A *Kn1-O* homozygote in a background conditioning severe knotting and ectopic ligule formation around lateral veins was crossed to a *Lxm1-O* heterozygote. *Lxm1-O* is much like knotted, but it affects the midvein rather than the laterals, either because it only acts in very early plastochrons, or because it is midvein specific; its phenotype is retarded development around the midvein resulting in the sheath extending all the way to the tip of the leaf over the midvein. The progeny of this cross were all *Kn1-O* heterozygotes, but half were *Lxm1-O* double mutants and half were knotted only. Of the 15 plants examined, 7 were normally knotted and 8 appeared to be lax midvein only, with the knotted phenotype suppressed (Family 10960 plant 1 and plant 14 exemplify these two phenotypes in Fig. 1).

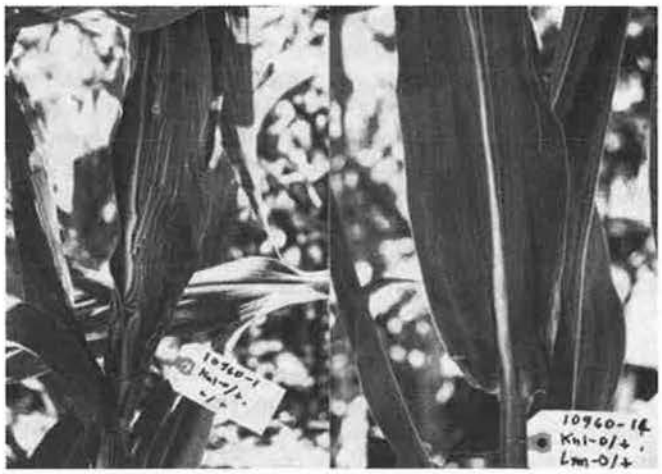


Figure 1.

There are two general ways this suppression could occur. Possibly, *Lxm1* is an upstream regulator of *Kn1* expression such that *Lxm1* "turns off" *Kn1* ectopic expression around lateral veins. A more biological and much more fun explanation is possible. Both *Kn1* and *Lxm1* mutants may be described as retarded (heterochronic) mutants in that the proper stage transitions from more juvenile to more adult move too slowly. *Kn1* retards the developmental "schedule" around lateral veins (Freeling and Hake, 1985, Genetics 111:617) at about plastochron 4 while *Lxm1* retards the developmental schedule for development controlled by the midvein. The suppression of *Kn1* phenotype by *Lxm1* is explained if the maturation of the midvein induces the laterals to begin their developmentally important inductive events. In this way, *Kn1* mutants normally encode cells in lateral veins that are "too young for their ages," and--when they receive signals from the midvein that say "stop dividing" or "switch to blade identity" they continue on as if deaf (incompetence). However, if the midvein is also developmentally retarded, leaf development returns to synchrony. Just an idea.

Developmental morphology of leaves using UV light-induced autofluorescence of cell wall components

--Anne W. Sylvester, Zac Cande and Mike Freeling

The pattern of cell wall deposition is both developmentally and spatially regulated in the maize leaf. We are using the fluorescence emission differences among wall components as developmental markers in several leaf mutants. The purpose of this analysis is to characterize the time when cells in the maize leaf are programmed to follow one developmental pathway or another. It is clear that differentiation of leaf parts in maize begins early during growth of the primordium. Cells of the blade, sheath, and ligular region differentiate from one another around plastochron 2 - 3, before the ligule itself is visible on the adaxial epidermal surface (Sylvester et al., Development, 1990, Nov. issue). The availability of developmentally specific markers in addition to epidermal cell markers (i.e. hairs, waxes, wall shapes, etc.) will allow for more precise analysis of stages when specific genes are acting during development.

Components of the phenylpropanoid pathway, particularly the monomers ferulic acid and p-coumaric acid, and the polymers, lignin and suberin, are known to emit excess energy in the form of autofluorescence when excited by UV light. Ferulic acid and p-coumaric acid absorb at around 310 nm and emit at 415 - 445 nm (Goulas et al., Photo. Res. 25:299-307, 1990), whereas lignin shows a maximum emission at 358 nm (Lundquist et al., Holzforschung 32:27-32, 1978). This shift in spectrum is useful for recognizing the time when lignin deposition begins. In the Gramineae, ferulic acid is bound to all cell walls (Harris and Hartley,. Nature 259:508-510, 1976) and can be recognized early in development by its characteristic fluorescence spectrum. Lignin on the other hand is deposited specifically in vascular tissue and in adjacent sclerenchyma cells later in development when cell growth has slowed. Using a standard Zeiss filter combination (exciter filter at 365 nm and a longwave pass filter at 418 nm), ferulic acid and associated monomeric phenylpropanoids appear white-blue whereas walls containing lignin appear dark purple-black. Histochemical tests using the standard metachromatic stain toluidine-blue O (TBO) at low pH confirmed that the observed fluorescence shift to purple-black corresponds to the presence of lignin. TBO staining, however, does not exhibit the subtle color gradations associated with the esterification of ferulic acid that is permitted by the use of UV excitation.

The lignin deposition pattern, based on shifts in UV emission, was characterized in seedlings and mature leaves of normal and heterozygous mutant siblings of *Lg3* plants. Seedling plants bearing a total of 8 -10 leaves were dissected, hand-sectioned, excited by UV light and photographed. Representative sections were fixed in buffered formaldehyde for histochemical staining. The blue fluorescence of walls in each of several leaf parts including the sheath, the ligular region, and the blade was recorded and compared. Preliminary observations show that the white-blue fluorescence characteristic of ferulic acid is visible as early as plastochrons 2-3 in normal plants. The blue intensity increases in sclerenchyma cells on either side of the vascular bundles (possibly due to esterification of prelignin monomers). By plastochron 6 the characteristic dark-purple fluorescence of lignin is first visible in local regions of the leaf. Sclerenchyma near the tip of the blade lignify first, in accordance with the age gradient of the leaf. Cells that are subjacent to vascular bundles in the adaxial sclerenchyma of the midrib lignify initially. As the leaf grows, lignification spreads until the adaxial midrib has a continuous band of lignified sclerenchyma. The adaxial and abaxial surfaces lignify at different rates with the abaxial sclerenchyma lignifying before the adaxial. There is also a lateral gradient of age: the shift from white-blue to bright-blue to purple-black occurs first at the midrib and then proceeds gradually toward the blade margin.

This method should prove useful for the analysis of developmental mutants that show alterations in the identity of entire regions of cells. For example, plants heterozygous for *Lg3* show alterations in the position of the ligule over the midrib region. By UV light analysis, *Lg3* plants show distinct changes in the lignification pattern. Deposition is delayed in the adaxial sclerenchyma but only over the midrib portion of the blade. The abaxial surface of the midrib appears to lignify normally as do both surfaces of the blade proper. There is an extra accumulation of lignified sclerenchyma cells at the junction between midrib and blade on the adaxial surface. This delay in lignification of the adaxial midrib continues past the time of ligule outgrowth, such that the ligule never joins at the midrib. Instead, the ligule stops at the site of extra lignin accumulation at the midrib/blade border. The continuous band of lignin at the adaxial midrib is absent except near the tip of the blade. This method of using wall autofluorescence should prove useful for further characterization of *Lg3* plants and other mutants with similar phenotypes. More detailed analysis of the fluorescence spectra for developmental study is underway.

Tissue-specific distribution of cytosolic 6-phosphogluconate dehydrogenase (6-PGD) isozymes

--Jonathan D. Tom and Julia Bailey-Serres*
*Present address: UC Riverside

Two PGD isozyme subunits, PGD1 and PGD2, are regulated in tissue specific fashion. We used PGD nulls to examine the localized distribution of cytosolic 6-PGD isozymes in the mature scutellum. A PGD1+PGD2 double null was constructed by crossing plants which were homozygous for *Pgd2-null* but wild-type at *Pgd1* to plants homozygous for *Pgd1-null* but wild-type at *Pgd2*. In the F2 progeny, we observed the expected 9:3:3:1 segregation of the four different 6-PGD isozyme dimer phenotypes: wild-type, PGD2null, PGD1null, PGD1+PGD2 double null. Fresh cross-sections of mature scutella from the four phenotypes were stained in situ for 6-PGD activity. The 6-PGD stained scutella were compared to scutella of the same genotype which had been stained without 6-phosphogluconate substrate (Fig. 1 Control column) and for alcohol dehydrogenase (ADH) (Fig 1, ADH column). In scutella from the inbred B73 (*Pgd1-3.8, Pgd2-5*) and *Pgd2-*

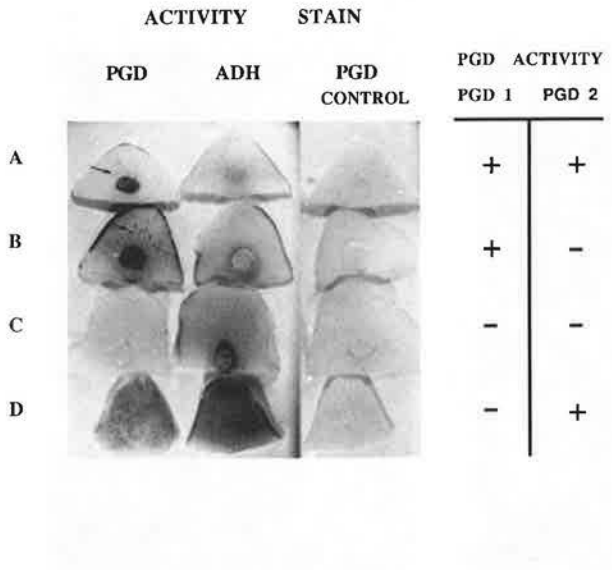


Figure 1. Fresh cross-sections of mature scutella were stained *in situ* for PGD, ADH, or PGD without 6-phos phogluconate substrate (control) Since no activity associ ated with the pro-vascular bundles in mature scutella was observed for ADH or PGD control activity stains, PGD activity exhibits tissue specificity. The arrows indicate the mature vascular bundles in scutella. (A) Scutella from the inbred, B73 (*Pgd1-3.8:Pgd2-5*) were understained to show mature vasculature. (B) Scutella from a recessive *Pgd2-null* mutant (*Pgd1-3.8: Pgd2-nu*ll). Note the PGD1 activity localized in the vascular bundles (see arrows) in (A) and (B). (C) Scutella from a recessive *Pgd1-null/Pgd2-null* double null mutant (*Pgd1-null: Pgd2-null*). No activity was detected. (D) Scutella from a recessive *Pgd1-null* mutant (*Pgd1-null: Pgd2-5*). Note the absence of PGD1 activity associated with vascular bundles. Scutella in (B), (C), and (D) are progeny derived from the same parents.

null, we observed specific PGD1 activity associated with pro-vascular bundles (See arrows Fig. 1A and 1B). In contrast, the PGD2 activity observed in scutella from *Pgd1-null* was uniform throughout the interior of the scutellum (Fig. 1D). In scutella from PGD1+PGD2 double nulls, no detectable PGD activity was observed (Fig. 1C). Since there are PGD1·PGD2 heterodimers in scutellar extracts (data not shown), it is likely that PGD1 is expressed in non-vascular as well as vascular tissue. Therefore, in mature scutella, PGD1 expression appears to be most intensely localized in the pro-vascular bundles whereas PGD2 occurs only in the non-vascular tissue.

The 6-PGD isozyme dimer patterns from primary roots, secondary roots, mature scutella, embryo axis, pollen and leaves were analyzed by native-polyacrylamide gel electrophoresis. Based on isozyme dimer ratios, we observed a variation in the tissue and organ-specific distribution of cytosolic 6-PGD isozymes among the tissues analyzed in the inbred B73 (data not shown). Using PGD nulls, we also demonstrated that the relative ratios of PGD1 and PGD2 isozymes were proportional to gene copy number (data not shown). In a moderate environment, the PGD1+PGD2 double null exhibited no obvious reduction in vigor, seed set, time to maturity, pollen viability, or germination when compared to its siblings. Even under anaerobic stress, we observed no reduction in germination for the PGD1+PGD2 double null as compared to PGD-positive seedlings. However, we are uncertain whether the pentose pathway was stressed under anaerobiosis. Since the PGD1+PGD2 double null's growth and development resembles that of wild-type, cytosolic 6-PGD activity appears to be dispensable. 6-PGD activity is localized the plastid as well as in the cytosol (Gottlieb, Science, 216: 373-380, 1982). Plastid isozymes may provide sufficient 6-PGD activity for the survival of plants that have no cytosolic PGD activity.

Work performed in the Freeling lab.

BOZEMAN, MONTANA
USDA-ARS and Montana State University

Isolation of repetitive DNA sequences specific to the genus *Tripsacum*

--Luther E. Talbert and Susan L. Moylan

Repetitive DNA sequences in plants tend to evolve rapidly, and as such, sequences specific to taxa have been isolated and used to document and monitor both artificial and natural interspecific hybrids. Perhaps the greatest use of repetitive DNA sequences as molecular markers has been with wheat and its relatives. Such markers have been used to monitor introgression of alien germplasm into wheat (e.g., Rayburn and Gill, 1987, Amer. J. Bot. 74:574-580) and to study the evolution of specific genomes in allopolyploid species (e.g., Dvorak et al., 1988, Genome 30:680-689; Talbert et al., 1991, Amer. J. Bot., in press). In the case of maize, previous work has shown that the transposable elements *Mu* and *Spm* are specific to the genus *Zea* (Talbert et al., 1989, J. Molec. Evol. 29:28-39), and this fact has been exploited to document an intergeneric hybrid involving *Zea* and *Tripsacum* (Talbert et al., 1990, Amer. J. Bot. 77:722-726). Given the long-standing interest in the use of *Tripsacum* germplasm as a source of beneficial genes for maize improvement, and the demonstrated utility of repetitive DNA sequences as molecular markers in several plant systems, it may be useful to isolate molecular probes for DNA sequences specific to *Tripsacum*. This report details the isolation of such molecular probes.

We cloned random *Taq*I fragments of *Tripsacum dactyloides* into the *Acc*I site of pUC18 and transformed recombinant plasmids into the *E. coli* host NM522. Recombinant plasmids were isolated from bacterial cultures, digested with *Eco*RI and *Hin*dIII to liberate the inserts, and Southern blotted onto nylon membranes. Membranes were hybridized consecutively to nick-translated total DNA from *Zea* and *Tripsacum*, respectively. A total of three inserts were identified that showed a stronger signal with *Tripsacum* DNA than with *Zea* DNA. Subsequent hybridization of these inserts to *Eco*RI/*Hin*dIII-digested DNA from several *Zea* and *Tripsacum* species verified the *Tripsacum*-specificity of the inserts. Figure 1 shows that the inserts from plasmid pTrip9 (Panel A), pTrip25 (Panel B), and pTrip36 (Panel C) hybridize strongly to DNA from

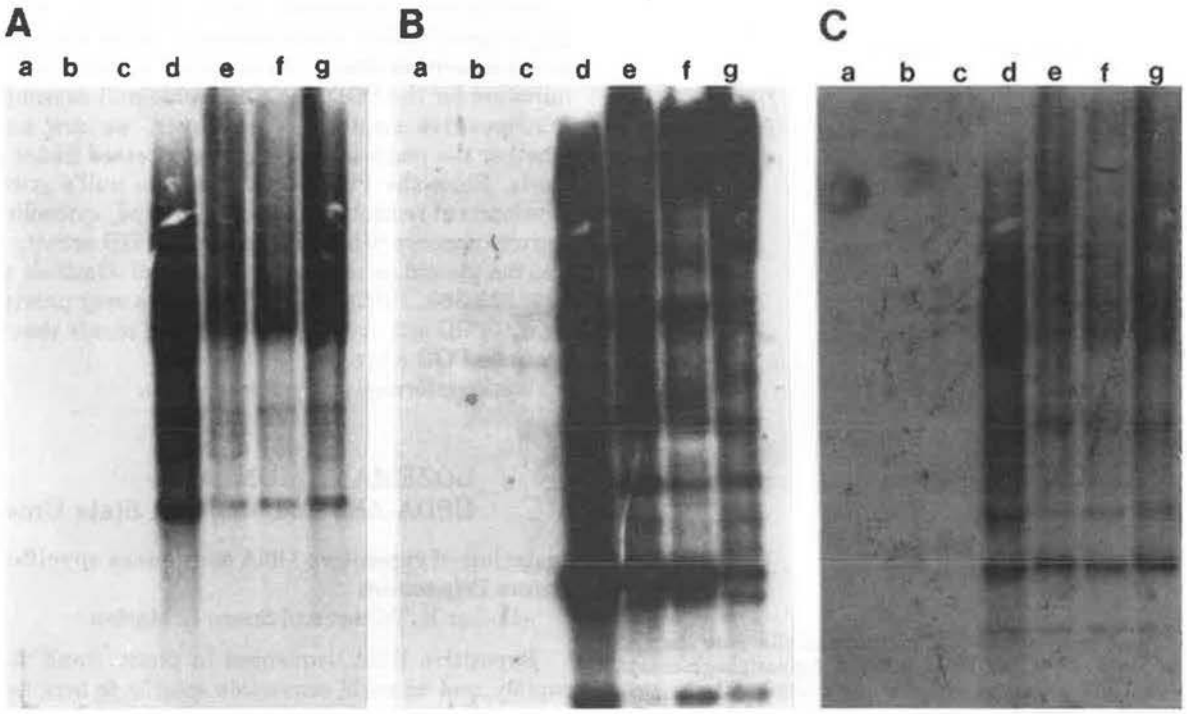


Figure 1. Southern blot of several *Zea* and *Tripsacum* species hybridized to *Tripsacum*-specific DNA probes. Lane a: *Zea mays* subsp. *mays*; b: *Zea perennis*; c: *Zea diploperennis*; d: *Tripsacum latifolium*; e: *Tripsacum peruvianum*; f: *Tripsacum andersonii*; g: *Tripsacum dactyloides*. *Tripsacum* DNAs were from J. F. Doebley, University of Minnesota. Panel A. Blot hybridized to pTrip9. Panel B. Blot hybridized to pTrip25. Panel C. Blot hybridized to pTrip36.

Tripsacum species (lanes d-g), but hybridize very little or not at all to *Zea* DNA (lanes a, b, and c).

Thus, three repetitive DNA sequences specific to the genus *Tripsacum* have been isolated. These sequences may complement the *Zea*-specific transposable elements *Mu* and *Spm* for documenting hybrids between *Tripsacum* and *Zea*, and monitoring introgression between the genera.

Assaying maize pollen phytic acid

--Victor Raboy, Glenn M. Magyar, Paolo Gerbasi

As part of a survey of pollen phytic acid (*myo*-inositol hexaphosphate) levels in 3 gymnosperm and 25 angiosperm species (the only survey of its kind in the literature), Jackson et al. (Phytochem. 21:1255, 1982) reported that the maize pollen sampled contained 2.6 mg phytic acid/gm dry weight. Since phytic acid is typically thought of (by plant biologists at least) primarily as a phosphorus (P) storage compound, these authors also assayed pollen total P, and determined the percent of total P represented by phytic acid P. Their maize pollen sample contained 5.3 mg total P/gm dry weight and phytic acid P (0.73 mg/gm) represented 14% of total P. They did not identify the maize stock tested in their survey.

To assay pollen phytic acid, these authors first ground pollen in liquid N_2, extracted in 0.02 M Na_4EDTA, heated the extracts in a boiling water bath, and following centrifugation, subjected aliquots of supernatant to high-voltage paper electrophoresis (0.1 M oxalic acid pH 1.5; 13 V/cm; 2 hr; Whatman 3mm paper). P-containing compounds were visualized using an acid molybdate stain. Spots co-migrating with standard phytic acid were visually quantified by comparison with a set of standards. They report that their lower limit of sensitivity was a spot containing approximately 0.66 µg phytic acid (1.0 n mol).

Our laboratory has been using an essentially identical method of paper electrophoresis with a similar lower limit of sensitivity to screen for EMS-induced mutations which perturb kernel phytic acid synthesis (Raboy et al., Maydica 35:383, 1990). We would like to use similar methods to screen for mutations at the level of pollen as well as seed. So, as a preliminary experiment, we used the methods of Jackson et al. (faithfully) to test pollen sampled from F_2 and F_3 progeny of an A632 x Mol7 cross, and from an "Early ACR composite," both kindly provided by M. G. Neuffer and used in our work. Despite repeated efforts, we could not detect phytic acid in these pollen samples using the methods of Jackson et al.

We therefore decided to use a more reliable approach to quantifying phytic acid, the "ferric-precipitation" method (described in Raboy et al., Maydica 35:385, 1990). Using this method, we found that the A632xMo17 progenies' pollen contained from 0.49 to 0.56 mg phytic acid/gm dry weight, and the "Early ACR composite" lines contained from 0.49 to 0.82 mg phytic acid/gm dry weight. These samples therefore contained from one-fifth to one-third the level of pollen phytic acid reported for maize by Jackson et al. However, we found similar levels of pollen total P, ranging from 5.2 to 6.2 mg total P/gm dry weight. Thus the lower levels of pollen phytic acid we observed relative to the report of Jackson et al. were not due to dramatically reduced supply of P to the developing pollen grain. In our

hands, maize pollen phytic acid P represents from 3% to 4% of pollen total P.

Since we still may not have been efficiently extracting and assaying maize pollen phytic acid, we continued to test a variety of combinations of methods (grinding in liquid N_2, different extraction media, freeze-thawing, heating, ferric precipitation, barium precipitation, paper electrophoresis, etc.). All tests gave values in the 0.5 to 1.0 mg phytic acid/gm pollen range, confirming our initial "ferric-precipitation" results.

Another possibility is that the discrepancy between the value reported by Jackson et al. and our results reflects a genotypic difference. We plan to conduct an informal survey this summer to test this hypothesis, and to continue efforts to develop rapid and reliable methods to assay pollen phytic acid.

BROOKINGS, SOUTH DAKOTA
South Dakota State University

Linkage data for opaque endosperm-1 (*o1*)

--R. H. Whalen

The 1990 working map in MNL 64 indicates that opaque endosperm-1 (*o1*) lies near *gl3* on chromosome 4L, but the order is unknown.

Three point data were obtained from fourteen families of the testcross *o1 + gl3* x *+ Tu +/o1 + gl3*, with the following results:

Region	Genotype	No.	Totals
0	*+ Tu +*	1799	
	o1 + gl3	1720	3519
1	*+ + gl3*	44	
	o1 Tu +	39	83
2	*+ Tu gl3*	174	
	o1 + +	157	331
1,2	*+ + +*	15	
	o1 Tu gl3	6	21
			3954

o1 - Tu = 2.6±0.3%
Tu - gl3 = 8.9±0.5%

There was no significant heterogeneity among the 14 families for the monohybrid ratios or for linkage. Each of the genes showed good fits to a 1:1 ratio. The data indicate the order to be *o1 - Tu - gl3*. The coefficient of coincidence was 2.295, due to an excess of the + + + double crossover class. Contamination by foreign pollen is the most likely explanation, since the heterozygote was used as the male parent.

BUENOS AIRES, ARGENTINA
CONICET and CIC

Perennial teosinte introgressed population of maize: behavior of half sib families and S1 derived lines

--J. L. Magoja

One hundred eight S1 lines and 108 half-sib families derived from a perennial teosinte introgressed population of maize (*Z. perennis*) (see MNL 60:82, MNL 61:66 and MNL 62:83) were tested during 87/88 in Llavallol, as part of an augmented randomized complete block design. Two commercial precocious hybrids (controls), with a similar evolutive cycle to one of the most studied progenies, also were part of the test.

A whole of 30 traits were evaluated, and 19 of them are the object of this article: (1) days to tassel (T), (2) days to pollen (P), (3) days to silking (S) - measured in days after emergence (dae)-, (4) protoandrous (PR=S-P) (days), (5) tassel branch number (TBN), (6) plant height (PH) (cm), (7) uppermost ear insertion height (EIH) (cm), (8) number of leaves (NL), (9) number of leaves above uppermost ear (LAE), (10) number of tillers (NT), (11) number of tillers in plants with low competence (TLC), (12) lodging resistance (LR) (0 = susceptible to, 2 = resistant), (13) ears per plant (EP), (14) ears weight per plant (EWP) (g), (15) kernel weight per plant (KWP) (g), (16) cob % (C), (17) uppermost ear weight (UEW) (g), (18) average ear weight (AEW) (g), and grain yield (Y) (kg/ha). The plants were grown at a density of 57,143 pl/ha and they were not sprinkled nor fertilized. Table 1 summarizes the results obtained and gives the inbreeding depression values (ID) resulting from the comparison among the S1 lines and half-sib family averages for each trait.

Table 1: Relevant traits of S_1 lines and half sib families derived from a maize population introgressed with perennial teosinte.

	S_1		Half sib		
Character	Mean ± SD	Range	Mean ± SD	Range	ID(%)
Tasseling(days aft.emerg.)	52.7 ± 5.3	36-64	53.6 ± 4.7	36-69	1.7
Pollination(days aft.emerg.)	57.7 ± 6.4	40-72	58.3 ± 5.5	40-75	1.0
Silking(days aft.emerg.)	60.1 ± 6.6	46-74	60.8 ± 5.6	51-78	1.2
Protoandrous(days)	2.7 ± 1.8	-3-9	2.7 ± 1.8	-5-11	-1.1
Tassel branch number	27.9 ± 9.5	10-60	32.8 ± 8.4	17-55	14.9
Plant height(cm)	196.6 ±24.0	136-255	222.2 ±21.7	150-263	11.6
Ear insertion height(cm)	89.7 ±20.9	33-140	107.6 ±18.8	49-145	16.6
Number of leaves	13.6 ± 1.9	10-18	14.9 ± 1.8	11-19	8.7
Leaves above ear	5.4 ± 0.8	4-7	5.6 ± 0.7	4-8	3.6
Number of tillers	1.2 ± 0.4	1-3	1.3 ± 0.5	1-3	7.7
Tillers in low competence	1.6 ± 1.0	1-6	2.0 ± 0.9	1-4	20.1
Lodging resistance	1.1 ± 0.6	0-2	1.1 ± 0.6	0-2	6.1
Ears per plant	1.6 ± 0.6	0.6-3.6	2.1 ± 0.7	1.1-4.4	24.5
Ear weight per plant(g)	120.3 ±49.2	37-273	195.8 ±62.7	54-400	38.6
Kernel weight per plant(g)	93.0 ±39.8	21-233	156.3 ±49.7	33-320	40.5
% of cob	24.5 ± 6.3	12-50	20.9 ± 3.7	10-38	-17.2
Uppermost ear weight(g)	104.2 ±29.3	43-183	130.0 ±27.9	67-217	19.8
Average ear weight(g)	77.7 ±24.5	21-151	96.1 ±24.7	25-163	19.1
Grain yield(kg/ha)	5233 ±2221	1194-12924	8755 ±2779	1891-17812	40.2

The evolutive cycle traits (T, P and S) show that the progenies tested are precocious, most as much as the commercial hybrids used as controls and some even more precocious.

The fact that the original population has arisen from hybridizations between perennial teosinte and Gaspé and then it was backcrossed with precocious maizes, conferred this particular characteristic to the material. These traits are the less variable ones, while most of the other traits show a relatively wide variation. The difference between the male and female flowering (PR) has an appropriate value, though it must be remarked that there is wide variation, from protogynous progenies (teosinte) up to variable protoandrous as in most maize.

Tassel branch number is relatively high, especially when the short evolutive cycle of the plants is considered.

The high mean value of this trait and its wide variation are associated characteristics--as in the case of prolificity--to the heterotic expression conditioned by the wild germplasm (Palacios and Magoja, Rev. Fac. Agrn. UNLP 59:81, 1983). Plant height is one of the less variable traits, the plants are relatively low, which is possibly and strongly associated to the evolutive cycle length and also with the number of leaves. The high variation for the uppermost ear insertion height could be possibly related to the different levels of prolificity of these plants.

The number of leaves (NL) and the number of leaves above the uppermost ear (LAE) are not very variable and they are adequately related to evolutive cycle and plant height. The plants have a low number of tillers (NT) when they are exposed to competence, but when the competence degree is low (plants disposed at the head of the plots), all the tillering capacity derived from teosinte is expressed and then a great part of the progenies are able to produce several fertile tillers (see TLC trait). It could be said that lodging resistance is good enough although very variable, and because of this it can be expected to find plants which tumble easily up to those which remain perfectly erect at harvest time.

Prolificity is the most remarkable trait among all those studied. This trait distinguishes the introgressed population of maize from the rest, through the number of ears per plant (EP). EP has a high value and it is really very variable. Probably this trait constitutes the main cause by which can be explained the high potential yield of some progenies. As repeatedly pointed out, prolificity is the most remarkable expression of the enormous heterosis produced by the interaction between wild and maize germplasm. The traits EWP, KWP, C, VEW and AEW are together with prolificity components of the grain yield and they also show a wide variability range. The average grain yields both for the S1 lines and the half-sib families are not too high, but the great variability between the S1 lines and half-sib families suggests that an adequate selection could engender a synthetic population with an enormous potential yield.

Considering the results obtained and bearing in mind that the commercial hybrids used as testers produced 10,119 kg/ha and 7,563 kg/ha, it can be deduced that 58 half-sib families (53.7% of the whole) and 11 S1 lines (10.2% of the whole) yielded significantly more than the less yielding tester while 18 half-sib families (16.7% of the whole) and 2 S1 lines produced significantly more than the most yielding commercial hybrid.

The inbreeding depression value, as seen in Table 1, is low and nonsignificant for some traits. All the opposite, that value is high when we consider yield and some of its components. A critical test for a better evaluation of the ID value consisted in comparing the S1 lines and the half-sib families derived from the same plant. For that, the first ear was self-pollinated and the second one was pollinated with a mixture of pollen taken from the whole population. The yield of the progenies derived from this was individually compared (Table 2). The S1 line yield is not related to the half-sib families (r = 0.04 nonsignificant). In mean, the S1 lines show an inbreeding depression value of 44%, which

Table 2: Comparison of grain yield between S_1 and half sib families derived from the same female plant.

	Yield(kg/ha)	
Female plant	S_1	Half-sib
01-03	8,966	7,323
02-01	4,772	6,909
02-14	4,596	8,612
03-05	6,104	8,747
04-05	4,490	7,799
04-10	9,895	7,840
05-02	4,732	12,417
05-03	7,942	10,390
05-13	1,874	10,069
06-17	3,871	6,395
07-05	3,393	7,135
07-11	6,466	10,813
07-14	4,164	6,561
08-02	5,323	6,193
09-15	8,238	10,724
10-02	4,451	16,116
10-13	5,742	17,564
11-07	2,394	9,014
11-14	3,674	8,793
12-05	5,871	10,746
Average	5,348	9,508

is very close to the estimated value considering the S1 lines and half-sib families have arisen from different plants of the population (see Table 1).

The results obtained to date document the usefulness that the wild germplasm has in the search for higher variability and the attainment of high yields. Bearing in mind that, as several authors point out, modern maize arose from a spontaneous introgressive process of its wild relatives (the teosintes) and that from the enormous variation produced by this primitive man led the evolution of the cultivated species, though with an empiric sense, the repetition of those events but managed with another criterion, can actually let us produce great expectancy for higher yields. As the variation produced in interspecific hybrids between maize and teosinte is well known, and as the female inflorescence (ear) has possibly been the base of selection of primitive man, it is easy to foretell the operated changes that led up to a modern maize with only one stem and one big ear. Probably, primitive man handled the criterion of the size of the edible organ and not yield per unit of area.

Actually, the variability generated in wild germplasm introgressed populations of maize possibly resembles the one available to primitive man, a long time ago. Then it is only necessary to get advantage of the fact, using a scientific criterion. Considering that one desires a more productive maize plant, then one with a different architecture cannot be discarded.

The results obtained let us infer that possibly those

plants with multiple ears and tillers (though these characteristics are moderately expressed), homogeneous maturation and growing at low densities, probably express the maximum potential yield of the species. The high production shown by some of those half-sib families tested (almost 18,000 kg/ha), without water supply or fertilization techniques are not usual in the traditional maize crop area of Argentina. This fact, obtained on the basis of prolific plants, constitutes one more evidence of the significant advance that can be obtained in maize breeding by using wild germplasm.

Diploperennial teosinte introgressed population of maize: isozyme variation

--I. G. Palacios and J. L. Magoja

The study of morphological and physiological traits--in its greatest part of agronomic importance--we have done on a diploperennial teosinte (*Z. diploperennis*) introgressed population of maize has revealed that, as a product of the interaction between the wild and cultivated germplasm, there is great variability for most of the quantitative traits, especially those associated with grain yields. With the purpose of giving a better measure of the whole genetic variation partially detected through the study of agronomic traits, we have initiated some studies to be able to establish isozyme variation.

One hundred ears (plants) obtained by controlled pollination and representative of that population (see MNL 60:82, MNL 61:65 and MNL 62:84) were used to obtain, through starch gel electrophoresis, the patterns of four systems: esterases (EST), alcohol dehydrogenase (ADH), malate dehydrogenase (MDH) and leucine aminopeptidase (LAP). With the purpose of obtaining a measure of variation and on the basis of presence-absence of each band revealed in the gel, it was considered that when the band was present in all the individuals, it belonged to a monomorphic locus and if it was present or absent according to the individual, is considered polymorphic. All the observed phenotypes and the frequency in which one of them appeared in the population, are drawn in Figure 1.

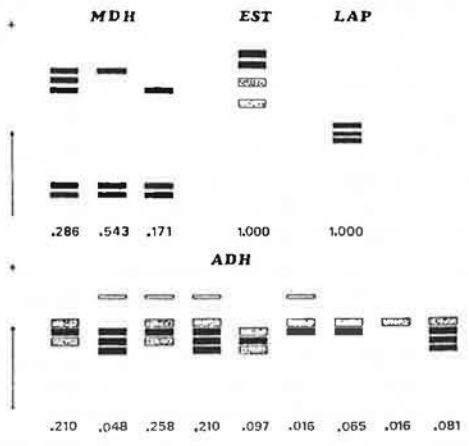


Figure 1: Phenotypic frequences of MDH, EST, LAP and ADH.

To make the comparisons of polymorphism easier, the polymorphic index (PI) is the frequency of each band and the number of loci for each isozymatic system. Bearing in

Isoenzymatic system	PI
EST	0.00
LAP	0.00
MDH	0.15
ADH	0.12

mind that PI varies from 0 and 0.25, the values found for ADH and MDH represent a high variation. The same pattern was observed in all the individuals analyzed in the EST and LAP systems. Considering that LAP does not show any variation in maize and that only a few systems were tested, the results can be considered congruent with the variability observed for agronomic traits.

The future availability of more information from the study of other systems that we are analyzing, in comparison with normal populations (without introgression), will let us deduce the variation generated by the introgression of wild germplasm.

Diploperennial teosinte-maize hybrids: expression of pollen grain size and pollen fertility traits

--V. R. Corcuera, M. B. Aulicino and J. L. Magoja

As part of our investigations on diploperennial teosinte-maize hybrids, two pollen grain traits were evaluated in teosinte, maize (Ever Green), and F1 and F2 progenies derived from them. Pollen grain size was evaluated for the main diameter and expressed in microns (μ). Pollen grain fertility was measured considering all those grains completely stained with an I-IK solution and expressed in percent. The results obtained are given in Table 1.

Table 1: Pollen traits in diploperennial teosinte (Zd), Ever green maize (Eg), and its F_1 and F_2 progenies

	Pollen grain size (μm)		Pollen fertility (%)	
	Mean	Range	Mean	Range
Zd	67.2 a	64–70	72.4 a	60–86
Eg	93.4 b	83–102	90.0 b	80–100
F_1	81.5 c	78–85	76.0 c	53–88
F_2	86.5 d	76–95	78.1 c	57–97
M.parent	80.3 c	—	81.2 d	—

Individual means within a column followed by different letters are significantly different at 5% level.

As can be seen, diploperennial teosinte has smaller pollen grains than maize, and the F1 and F2 mean values are intermediate. The F1 mean does not differ from the mid-parent, but the F2 mean is higher, probably because we couldn't dispose of a greater sample of this segregating population.

Compared with data obtained when pollen grain size

was studied in hybrids between perennial teosinte (*Z. perennis*) (see MNL 58:118) and maize, it can be noted a similar way of inheritance, where genes with additive effect prevail. In both cases, the F2 progeny mean is approximately similar to the mid-parent one. All the opposite, and as a direct consequence that perennial teosinte-maize hybrids are triploid (two doses of teosinte and one of maize) and that pollen grain size is controlled by the maternal genotype, this trait when studied in those hybrids showed that the F1 mean was very close to perennial teosinte.

As the difference in size between maize and the perennial teosintes is relatively important, and considering that the trait is quantitatively inherited, possibly this one could be used to detect wild germplasm introgression in the cultivated species (maize).

Contrary to what some authors have pointed out, the fertility of diploperennial teosinte-maize hybrids is not too high, unless for this particular case (see Table 1). Diploperennial teosinte "per se" hasn't got high pollen grain fertility, by which it can be considered that the F1 and F2 fertility cannot be explained only on the basis of normal phenomena in interspecific hybrids, as the smaller fertility of the wild parent must be considered too. In this particular case, no one of the parents has a fertility value near 100% and the values of the hybrid progenies are a little smaller than the mid-parent ones. In spite of this fact, and especially in the F2, as was expected, one can detect a wide variation of fertility values, from relatively low up to near 100% among individuals. An adequate selection of the most fertile individuals will consequently let us be successful in maize breeding programs using these wild genetic resources.

Diploperennial teosinte introgressed population of maize: kernel protein content

--L. H. Perini, G. Pischedda and J. L. Magoja

Zea diploperennis, like the other wild taxa of the genus *Zea*, has a high kernel protein content in relation to the cultivated maize (see MNL 59:61). The results we've obtained point out that diploperennial teosinte kernels have three times more protein than maize kernels. As was previously communicated, those progenies derived from crossings between perennial teosinte and maize keep a high protein level (MNL 59:69), but the trait dilutes while the plants look like maize, more and more, as for example, when wild germplasm introgressed populations of maize are analyzed (MNL 62:80).

Probably the high protein level of the wild relatives of maize is closely related to the little size of the kernel and the high proportion of hard endosperm. If this were absolutely true, the possibility of using these wild genetic resources to increase the protein content of maize kernels would be thrown away, as individuals combining a higher protein level with desirable yields could not be recovered.

Some previous results obtained when another perennial teosinte introgressed population of maize was studied (see MNL 62:80), pointed out that perennial teosinte germplasm produced a significant increase in the protein content and that this trait was not significantly associated with other plant, ear or kernel traits. This fact showed the certain possibility of increasing the protein content by using those wild genetic resources.

When analyzing a diploperennial teosinte introgressed population of maize (see MNL 61:65 and MNL 62:84), similar results to those previously communicated can be found (see Table). A sample taken at random constituted by ears (plants) belonging to S1 lines and full-sib families derived from this population, was employed as representative of that one. The samples were taken from the plants cultivated during the growing season 86/87 in two locations of the province of Buenos Aires: Llavallol (70 ears) and Pergamino (32 ears). The results show that the average protein content is higher than normal levels for maize. The variation is relatively wide, the trait was not affected by the location and not a single significant association among protein content and some other plant, ear or kernel traits was found.

Locality	Mean±SD	Range
Llavallol	12.0±1.9	8.6-16.0
Pergamino	12.5±1.3	9.4-14.7

It must be pointed out that the population has had an average yield of 5,500 kg/ha and that the full-sib families derived from it yielded between 2,000 and 11,000 kg/ha, when they were grown at a density of 57,143 pl/ha. Those individuals belonging to this introgressed population are prolific (2 to 4 ears/plant, average:3) and show a wide range of variation for different traits and kernels yield.

Presumably the absence of association among protein content and other traits, and the presence of families with a relatively high kernel protein content and a desirable yield, lets us suppose that diploperennial teosinte germplasm could be used to increase maize kernel protein content.

Gaspé germplasm for red flint corn improvement

--M. B. Aulicino and J. L. Magoja

The Argentine production of corn responds, nearly as a whole, to the red or orange flint kernel type, and the greatest concentration of the cultivation is placed between parallels 32 and 35 of south latitude. Expansion of the cultivation area to larger latitudes, which took place in the last years, made it necessary to count on corn adapted to more severe environmental conditions, especially due to the shortening of the period free from freezing and the lower summer temperatures.

The cultivars disseminated in these new areas came in from abroad, mainly from France and USA having the following characteristics: precocity, lesser thermal requirements and quick ripeness of grains. The fact that these imported corns are mainly of the dent type, with a lower price in our country, raised the concern to develop improvement plans on materials with the red flint kernel type, to produce cultivars adapted to the new cultivation areas.

As part of these projects we have begun to use Gaspé germplasm to introduce greater precocity in the red flint corn traditionally cultivated in Argentina. During the 1988/89 growth season, different materials were assayed with Gaspé germplasm, among which there were F1 ex-

perimental hybrids derived from the crossings between red flint inbreds of normal cycle and Gaspé.

In one of the assays there were, as part of a complete randomized block design with 3 replicates, 15 experimental hybrids (normal cycle line x Gaspé), their corresponding progenitors and three precocious commercial hybrids, normally employed in the cultivation zones. These assays were carried out in two localities: Llavallol (our working place) and Miramar, S. E. of Buenos Aires Province and representative of the new cultivation areas.

In these assays the thermal requirements were evaluated expressed in the sum of temperatures from sowing till flowering, yield and other agronomically important traits shown in Table 1. As can be seen, Gaspé germplasm is expressed in experimental hybrids, causing a sharp reduction of the evolutive cycle (sum of temperatures) as regards the mother lines. This fact is strongly associated with the expression of number of leaves, but not with the height of the plant because there is heterosis for the latter.

Table 1: Relevant traits of red flint inbreds, Gaspé, experimental hybrids (inbreds x Gaspé) and commercial hybrids.

Character	Inbreds	Gaspé	Experim. hybrids	Commerc. hybrids
Tasseling(sum temp.ºC)	961±60	441± 9	676±37	726±10
Silking(sum temp.ºC)	1164±78	696±23	853±30	871±16
Pollination(sum temp.ºC)	1121±79	657±16	824±31	846±13
Tassel branch number	16.9± 4.5	7.1± 2.0	18.0± 2.3	12.3± 9.3
Plant height(cm)	173.8±18.6	96.6± 3.6	202.0±10.9	226.3± 7.7
Ear insertion height(cm)	89.0±13.0	15.2± 4.8	75.4±10.6	86.1±16.2
Stalk diameter(mm)	16.5± 2.5	13.2± 1.1	18.0± 0.9	22.2± 0.9
Number of leaves	15.4± 1.3	8.7± 0.3	12.1± 0.9	13.4± 0.3
Leaves above ear	5.2± 0.8	4.9± 0.3	5.0± 0.4	5.0± 0.3
Ears per plant	1.2± 0.2	1.5± 0.2	1.1± 0.1	1.0± 0.0
Number of kernel rows	12.2± 1.7	9.8± 0.2	12.2± 0.6	13.7± 3.5
Kernel number/row	20.0± 5.6	20.6± 2.6	33.2± 3.6	35.5± 1.3
Ear lenght(cm)	12.2± 2.9	11.3± 0.7	16.3± 1.6	17.8± 1.3
Ear diameter(cm)	3.2± 0.3	2.8± 1.1	3.5± 0.2	4.0± 0.1
Cob diameter(cm)	1.9± 0.2	1.7± 0.1	2.1± 0.1	2.2± 0.2
Grain yield(kg/ha)	——	——	3370±566	4837±486

If the experimental hybrids are compared to the commercial ones, we can conclude that the former are significantly more precocious and represented by plants of smaller size; therefore and in consequence, they have a significantly smaller mean grain yield. In spite of this, there exist some combinations among the ones assayed, that preserve a red flint kernel type, and whose grain yield does not differ significantly from the hybrids used as controls. This is indicative of the possibility of using Gaspé as a precocity donor, making an adequate selection for yield and kernel type (red flint).

According to the previous results it can be inferred that, previous to the beginning of an improvement programme involving Gaspé, the starting point shall be those lines, varieties, composites, etc., that express good combining ability as regards Gaspé.

Due to the experience obtained to the present, it has been proved that certain inbreds, according to their origin, combine in a better way with Gaspé than others. Thus assays carried out during 1988/89 showed that the inbreds from Pergamino-INTA (Buenos Aires Province) produce 20% higher grain yields in combinations with Gaspé than the equivalent combinations with lines coming from Llavallol.

Wild germplasm for sweet corn improvement

--I. G. Palacios, R. Burak and J. L. Magoja

With the purpose of enlarging the genetic and germplasm base in sweet corn, the sugary endosperm trait (*su*) was incorporated into a corn population with diploperennial teosinte introgression (*Zea diploperennis*) (see MNL 60:82 and 61:65). In order to develop an adequate population in accordance with improvement projects, work is being carried out applying recurrent selection methods to such populations. During the growth season 1989/90 several sweet corn families were tested, among which there were sixty-four full sib families deriving from the introgressed population, being evaluated on the various characters by means of a comparative assay designed as an 8x8 lattice with two replicates.

The results obtained are provided in Table 1 and show that the families are quite precocious, prolific, and expressing low to very high variability according to the character. Those traits concerning the evolutive cycle, height of the plant and number of leaves are of low variation and very closely associated. The tassel branch number, ear insertion height, number of leaves above uppermost ear, ear size, number of kernel rows and kernel per row, are characters with great variation. Lastly, the rest of the characters studied is expressed with high variability, among which, the number of tillers, lodging resistance, prolificity and ear and kernel weight per plant are found.

Table 1: Relevant traits and heritability(h^2) in full sib families of sweet corn.

Character	Mean ± SD	Range	cv%	h^2
Tasseling(days aft.emerg.)	54.1 ± 2.6	49-66	4.8	
Silking(days aft.emerg.)	62.5 ± 3.1	54-72	5.0	
Pollination(days aft.emerg.)	56.4 ± 2.7	53-68	4.8	
Protoandrous(days)	6.1 ± 1.7	3-10	27.9	
Tassel branch number	32.5 ± 5.6	20-46	17.2	
Plant height(cm)	198.9 ±15.1	157-237	7.6	0.61
Ear insertion height(cm)	94.9 ±10.4	63-122	11.0	0.58
Number of leaves	15.4 ± 1.0	13-18	6.5	
Leaves above ear	5.5 ± 0.7	4-7	12.7	
Number of tillers	2.1 ± 0.6	1-5	28.6	
Lodging resistance	1.2 ± 0.5	0-2	44.1	
Ears per plant	2.7 ± 0.4	2-5	14.8	
Ear lenght(cm)	14.4 ± 1.9	9-19	13.2	0.83
Ear diameter(cm)	3.4 ± 0.5	2.1-4.4	14.7	0.87
Cob diameter(cm)	1.8 ± 0.5	1.1-2.6	27.8	
Number of kernel rows	12.8 ± 1.8	8-18	14.1	0.76
Kernel number/row	27.1 ± 4.4	15-35	16.2	
% of cob	15.1 ± 4.2	7-28.5	27.8	
Ear weight(g)	75.7 ±20.1	39-114	26.6	
Grain weight(g)	63.9 ±16.2	36-98	25.4	

The heritability values calculated for some of the characters, show the greatest part of the variation is genetic in nature. This leads to the assumption that the population under study may be adequate material to start a project of sweet corn improvement.

Another interesting fact to point out is that, in some of the families, high prolificity is expressed as a consequence of the production of several small ears per plant, which means that an acceptable yield in the production of baby corn would be easier.

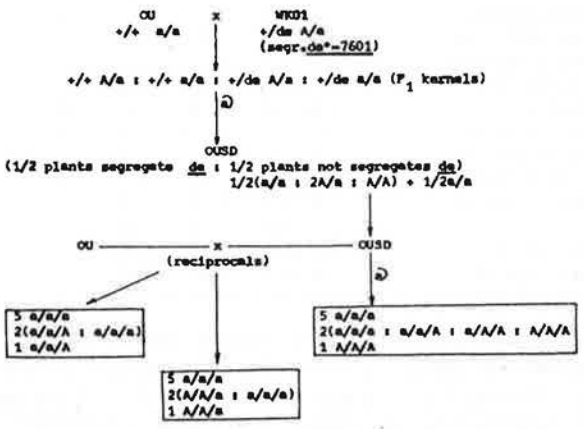
A probable codominant gene for tryptophan content of endosperm

--L. H. Perini and J. L. Magoja

As pointed out in other opportunities (MNL 58:120) high free amino acid level (a trait associated with high protein quality) is related to a recessive and spontaneous mutation, which conditions defective kernels (*de*-7601*) (MNL 56:108). In some cases the association of high protein quality and defective kernel wouldn't fulfill, and this is because it was tentatively deduced that the free amino acid levels seemed to be controlled by a codominant gene, presumably linked to *de*-7601*. It was also stated that this biochemical modification was produced in normal kernels (red flint phenotype), and because of this it was presumed that the responsible gene of this phenomenon was different from the defective and didn't condition other changes in kernel size or endosperm structure (see MNL 58:120). The probable fact that only one dominant gene could modify the kernel protein quality without affecting its normal phenotype has great practical importance. Because of this, we have recently proposed to give the necessary steps to try to isolate it from the defective gene, and make all the respective tests to characterize and verify its inheritance.

As can be seen in Figure 1, the WKO1 inbred, which segregates for *de*-7601*, was crossed by a normal (OU) inbred. According to preliminary evaluations (MNL 58:120) it was assumed that only one locus with two alleles (A/a) would be involved in the control of protein quality. The progenies derived from those crossings (OUSD) that do not carry the defective mutation were self-pollinated and reciprocally crossed by the OU line. The resulting materials and their hypothetic genotypes for protein quality are framed in Figure 1.

Figure 1: Genealogy of OUSD.



Three OU, 4 OU x OUSD, 6 OUSD x OU and 9 OUSD ears were available to analyze protein and tryptophan contents in the endosperm, as shown in Table 1. According to the stated hypothesis the reciprocal crossings (OU x OUSD and OUSD x OU) show significant differences for tryptophan content. There is a wide variation for protein quality among the nine ears analyzed in the OUSD progeny. Nevertheless, not a single homogeneous ear (plant) was detected for high tryptophan level according to the previous hope of meeting it in a 1/8 frequency.

Table 1: Protein and tryptophan content of OU, OUSD and its reciproccal crosses.

	Defatted endosperm	
	Protein (%)	Tryptophan (g/100g prot.)
OU	9.9±0.3	0.34±0.04
OU x OUSD	9.8±0.1	0.37±0.06
OUSD x OU	9.5±0.9	0.47±0.12
OUSD	9.4±0.9	0.46±0.12

Table 2: Endosperm protein pattern of selected OUSD progenies.

	Progenies			
Fraction(•)	1	3	13	33
Saline	6.7	4.6	8.2	8.7
Zein	38.0	51.3	42.1	39.5
Glutelin-1	16.4	15.7	15.5	16.4
Glutelin-2	6.2	5.8	5.8	6.3
Glutelin-3	28.5	19.5	20.9	24.9
Protein %	9.1	10.4	10.3	9.3
Lys(g/16gN)	3.4	1.7	3.1	3.2
Tryp(g/16gN)	0.9	0.5	0.7	1.0

(•): soluble nitrogen % of total. Results expressed on defatted endosperm basis.

In two OUSD ears obtained by self-pollination and with an intermediate average tryptophan content (0.6g/100g prot.) between the normal and high expected values, ker-

nels were individually analyzed for tryptophan content in the endosperm over a little sample (40 kernels) taken at random. This preliminary analysis let us establish that those ears were segregating for tryptophan content. The resulting average value was 0.6±0.2g tryp/100g prot. with a range between 0.3-0.9g tryp/100g prot. According to the fact assumed it could be deduced that the genotype a/a/a conditions normal tryp content (approx. 0.3g tryp/100g prot.), and the genotype A/A/A conditions approximately 0.9g tryp/100g prot.

Although the available data have been very few, they properly fit the expected segregation 1:1:1:1, as a quarter of the kernels have 0.3g tryp/100g prot., another quarter have 0.9g tryp/100g prot. and the rest are distributed in intermediate classes. Actually we obtained progenies from those ears segregating for tryptophan and we found as expected, homogeneous individuals for high tryptophan content.

Recently, several ears of OUSD progenies were analyzed for protein, lysine and tryptophan content, and in some of them the storage proteins of the endosperm were fractionated according to Landry-Moureaux (1970). Results are shown in Table 2. The results indicate that high quality protein OUSD is the consequence of zein repression.

High quality protein individuals will be crossed with normal lines, and then their progeny will be analyzed, to make a more critical test which will let us support or modify the stated hypothesis.

Variability and heterosis in maize-Balsas teosinte and maize-Guatemala teosinte hybrids

--M. B. Aulicino and J. L. Magoja

A maize inbred (OU) used as female parent was crossed by two of the wild taxa of the genus *Zea*: Balsas teosinte (*Z. mays* ssp. *parviglumis* var. *parviglumis*) and Guatemala teosinte (*Z. luxurians*). The parents and the F1 hybrids were cultivated during the growing season 87/88 as part of a complete randomized block design with three replicates.

The following traits were evaluated: (A) prolificity traits: (1) number of productive nodes per tiller (PN), (2) number of female spikes in the uppermost node (EUN), (3) number of female spikes per tiller (ET) and (4) number of female spikes per plant (EP); (B) plant traits: (5) number of tillers with female spikes (TE), (6) plant height (cm) (PH), (7) leaf width in the uppermost productive node (cm) (ULW), (8) leaf width in the 10th node (cm) (LLW), (9) leaf length in the uppermost productive node (cm) (ULL), (10) leaf length in the 10th node (cm) (LLL) (11) sheath length (cm) in the uppermost productive node (USL), (12) sheath length (cm) in the 10th node (LSL), (13) stalk diameter (cm) in the uppermost productive node (USD) and (14) stalk diameter (cm) in the 10th node (LSD).

The results obtained (see Table 1 and 2) show in the first place that the teosintes clearly distinguish themselves from the maize line for all the traits. The teosintes are much more prolific, with taller and thinner tillers than maize and also a greater number of them than in the cultivated species. In the second place our attention is called to the considerable (sometimes enormous) phenotypical variation that the teosintes show for most of the traits.

Table 1: Prolificity and plant traits in OU maize inbred, Balsas teosinte (Zmpp) and its hybrids.

		PN	EUN	ET	EP	TE	PH (cm)	ULW (cm)	LLW (cm)	ULL (cm)	LLL (cm)	USL (cm)	LSL (cm)	USD (cm)	LSD (cm)
OU	Mean	2.2a	1.1a	2.0a	2.0a	1.0a	188.7e	8.0a	6.3a	72.2a	74.3a	13.8a	16.5a	1.6a	2.0a
	SD	0.4	0.3	0.5	0.5	0.0	13.0	0.5	0.7	5.9	5.1	0.5	1.1	0.1	0.2
Zmpp	Mean	8.3b	2.4b	15.6b	710.7b	22.6b	280.0b	2.8b	1.8b	17.0b	93.0b	5.7b	20.5b	0.4b	1.0b
	SD	1.1	0.6	10.4	405.2	9.6	30.4	0.5	0.4	2.7	8.9	0.8	2.7	0.1	0.2
OUxZmpp	Mean	5.4c	3.5b	28.2b	272.3c	5.6c	249.1c	8.3a	7.2c	64.2c	77.1a	13.7a	16.2a	1.4a	2.0a
	SD	3.2	3.5	36.3	426.3	4.8	49.1	1.9	1.3	15.4	18.4	2.9	1.5	0.5	0.6
Mid parent		5.2c	1.8c	18.8b	356.4c	11.8d	234.4c	5.4c	5.0d	44.6d	83.6a	9.8c	18.5c	1.0c	1.5c

Individual mean within a column followed by different letters are significantly different at 5% level.

Table 2: Prolificity and plant traits in OU maize inbred, Guatemala teosinte (Z1) and its hybrids (OUxZ1)

		PN	EUN	ET	EP	TE	PH (cm)	ULW (cm)	LLW (cm)	ULL (cm)	LLL (cm)	USL (cm)	LSL (cm)	USD (cm)	LSD (cm)
OU	Mean	2.2a	1.1a	2.0a	2.0a	1.0a	188.7a	8.0a	6.3a	72.2a	74.3a	13.8a	16.5a	1.6a	2.0a
	SD	0.4	0.3	0.5	0.5	0.0	13.0	0.5	0.7	5.9	5.1	0.5	1.1	0.1	0.2
Z1	Mean	10.6b	3.7b	68.9b	941.2b	19.3b	300.5b	3.4b	4.7b	19.3b	117.0b	7.2b	20.9b	0.4b	1.3b
	SD	2.1	1.0	35.5	654.8	11.2	31.2	0.5	0.7	2.6	6.7	0.9	2.9	0.1	0.1
OUxZ1	Mean	9.0c	10.8c	94.4b	745.7b	8.3c	300.4b	7.4c	7.5c	61.7c	98.4c	12.6c	18.1c	1.1c	1.9a
	SD	2.2	5.3	47.7	332.5	3.6	32.9	0.8	1.0	11.2	13.7	1.4	2.0	0.2	0.2
Mid parent		6.4d	2.4d	35.4c	471.6c	10.2c	244.6c	5.7d	5.5d	45.8d	95.6c	10.5d	18.7c	1.0d	1.6c

Individual mean within a column followed by different letters are significantly different at 5% level.

Bearing in mind that one of the parents of these hybrids is an inbred (OU), the great variation shown by the F1 can be attributed to the diversity of the resulting genotypes as a consequence of the heterogeneity expressed by the teosintes.

This fact has a great practical meaning, as we could verify through teosinte introgressed populations of maize, that the most primitive taxa constitute the most valuable genetic resources to be used in maize breeding.

When comparing the values obtained for the traits studied in the hybrid progeny with respect to those calculated for the mid-parent (see Tables 1 and 2), it can be deduced that for most of the traits the F1 plants are higher than the estimated mean values. This fact is reflected in the percents of heterosis shown in Table 3, in which it can be noted that in most of the cases heterotic expression exists especially when prolificity traits are considered, though not always significant.

As was previously pointed out in preliminary studies using another kind of interspecific hybrids of maize, pro-

Table 3: Heterosis for prolificity and plant traits.

Character	% heterosis(•) OU x Zmpp	OU x Z1
NQ productive nodes/tiller	3.4	41.1
NQ ears in uppermost node	104.1	354.5
Ears/tiller	50.1	166.2
Ears/plant	23.6	58.1
Tillers with ears	52.5	21.2
Plant height(cm)	6.3	22.7
Uppermost leaf width(cm)	53.0	28.2
Lowermost leaf width(cm)	42.9	36.8
Uppermost leaf length(cm)	43.9	34.5
Lowermost leaf length(cm)	7.8	2.9
Uppermost sheath length(cm)	40.3	20.2
Lowermost sheath length(cm)	12.4	3.4
Uppermost stalk diameter(cm)	47.4	10.0
Lowermost stalk diameter(cm)	34.0	16.1

(•): % heterosis = $(F_1 - MP/MP) \cdot 100$

lificity traits exhibit the greater heterotic expression. From this fact it can be deduced that the OU-Guatemala teosinte hybrids are much more heterotic than the OU-Balsas. This is what we expected, considering that Guatemala teosinte is phylogenetically much farther away from maize than Balsas teosinte.

The results shown in this work arise from a project whose objective was hybridizing all the wild taxa of *Zea* by the same tester (maize). Unfortunately, it was difficult to cross the maize line (OU) with the perennial teosintes, and when it was crossed by *Z. mays* ssp. *mexicana* only a few seeds were obtained. This fact prevented us from doing a rigorous evaluation of the differential heterosis which could be generated in comparable crossings, as was planned.

While we have the results obtained from hybrids between maize and the perennial teosintes (*Z. perennis* and *Z. diploperennis*), the maizes used then were different, by which comparing the heterosis values estimated in those cases with the present ones would not be of great strength. Nevertheless, the experience we've accumulated through the last years lets us affirm that the heterosis degree expressed by teosinte-maize hybrids increases as the wild taxa used in the crossing phylogenetically separate more and more from the cultivated species.

The results we have obtained suggest that the hybrids using teosintes belonging to Section *Zea* are much less heterotic than those obtained when using the ones of Section *Luxuriantes*. Inside this last Section of the genus *Zea*, we have verified that *Z. luxurians*-maize hybrids are the less heterotic ones and that *Z. perennis*-maize hybrids are the most heterotic ones.

Protease activity during germination in high and low protein inbreds and its reciprocal hybrids

--J. L. Magoja and A. A. Nivio

All those phenomena related to nitrogen accumulation in kernels are of great interest, whenever one tries to explain the different causes by which there are plants able to produce very different protein levels in their kernels. Although several investigations were carried out to clear up the subject, there are still numerous dark aspects linked to the genetic and physiological basis of nitrogen accumulation in kernels.

Our works--initiated in 1974--let us establish that the kernel protein content depends on the female parent genotype. As the plant has only got a unique genotype, all those grains belonging to the same ear have a similar protein content which relies on the capacity shown by the female parent to translocate amino acids up to the ear, during grain filling period. As we worked with two different inbreds the first with a high relative protein content NH) and the second showing a low relative protein content (BP), considering that protein content is a stable characteristic through a series of years (see Table 1), we could demonstrate that the F1 kernels derived from reciprocal crosses between them do not differ in their protein content from the inbred used as female parent.

These conclusions, according to the ones given by other authors, have already been published some years ago

Table 1: Whole kernel protein content(%) of BP,NH and its reciprocal hybrids.

	Years						
	1974	1976	1977	1978	1980	1986(*)	Mean
BP	10.9a	10.9a	12.2a	11.9a	11.6a	10.5a	11.3a
BP x NH	10.7a	11.7a				11.2a	11.2a
NH x BP	16.0b	17.5b				14.0b	15.8b
NH	15.4b	15.6b	16.4b	15.1b	14.8b	13.6b	15.2b

(*): Data of endosperm
Individual means within a column followed by different letters are significantly different at 5% level.

(Magoja, Rev. Fac. Agron. 44:203, 1978). Afterwards we could also verify that during the grain filling period, those lines with a low relative protein content have a lower free amino acid level in their leaves than the ones expressing a high relative kernel protein content.

Inbreds studied have a similar total nitrogen content in their vegetative organs, which let us deduce that the capacity of accumulating more protein in the kernel depends on a greater availability of free amino acids during grain filling period, which is related to a greater proteolytic activity in leaves and a higher translocation rate (Magoja and Streitenberger, XVI Reun. Nac. Fisol. Veg. La Plata, Arg, 1985).

It is probable that the proteolytic activity is strongly influenced by the endosperm protein content (available substratum). This fact would initially mask (during germination stage) the truly proteolytic activity, which is able to condition the hybrid genotype in other development stages of the plant (e.g., during grain filling period).

In connection with finding out whether the differential proteolytic activity among high and low protein lines can be detected in early stages (e.g., during seed germination) and then be able to predict the protein content in the next generation, an experiment using the BP, NH lines and their reciprocal F1 progenies was planned. Seeds of those lines and their hybrids were germinated in Petri dishes inside the oven at 28 C in the dark. Seven days after germination (for the lines) and five days after (for the hybrids), when all the coleoptiles came to the same length, the seedlings were dissected and the endosperm was taken away, from which the proteases were obtained.

The crude extract was incubated using casein as substratum, and the proteolytic activity was evaluated through quantification of the amino acids set free relative to non-incubated testers, expressed in micromols of glutamic acid per 100 milligrams of endosperm per hour. The results obtained at two different incubation times are given in Table 2.

The proteolytic activity depends on the incubation time although the detected differences among lines and hybrids keep relatively constant. The low relative protein content line (BP) and its hybrid resulting from crossing with the NH line (BPxNH) have lower proteolytic activity than the high relative protein content line (NH) and its hybrid resulting from the crossing with the BP line (NHxBP).

An average of seven ears of each participant (lines and hybrids) were analyzed, and a sample of grains taken at random was considered from each one. In spite of this, the

Table 2: Protease activity during germination (μM Glu/100mg.h^{-1}) of BP,NH and its reciprocal hybrids.

	Incubation time	
	3hs	4hs
BP	3.5 ± 0.7	8.9 ± 0.5
BP x NH	3.2 ± 0.8	8.2 ± 1.0
NH x BP	4.0 ± 1.0	10.5 ± 0.6
NH	4.3 ± 1.0	13.7 ± 0.3

results obtained show a considerable error which is greater at a minimum incubation time.

Although considering the error owing to the method, the results let us deduce that the proteolytic activity during germination is strongly associated with kernel protein content in the lines and reciprocal hybrids studied. Nevertheless this kind of evaluation could not be used to foretell the protein content of the grains harvested from those plants (the next generation), since the reciprocal hybrids might show a similar proteolytic activity when compared to their parents and in an intermediate level between them.

CAMPINAS, SAO PAULO, BRAZIL
Instituto Agronomico

Linkages of interest around the latente Michoacán super-gene

--Luiz Torres de Miranda, Luiz Eugenio Coelho de Miranda, Osmar Villela and Sylmar Denucci

In MNL 64 the results were pooled on linkages of interest in mapping the Michoacán 21 latente-1 super-gene. In it *ltp* (reddish pericarp color on the presence of light after green corn stage) was tentatively mapped at position 20 in chromosome 2. In the present report we present new data in F2 of the cross, IAC Maya *B-W Ltp* with IAC Maya latente *B ltp*. In a population of size n=1013 the distribution was 362 *B Ltp*, 361 *B ltp*, 242 *B-W Ltp*, and 48 *B-W ltp*. By the product method this leads to a value of p between *B* and *ltp* of 28.4±1.3. Taking *B* as reference this puts *ltp* at position 17 in chromosome 2. For a Mendelian distribution there is an excess in the *B ltp* class. The "true" value of p must be greater.

CANBERRA, AUSTRALIA
CSIRO, Division of Plant Industry

Cold treatment does not appear to enhance the activity of a methylation sensitive *Ac* element

--Richard Brettell and Elizabeth Dennis

Previous work with *wx-m9 Ds-cy*, an inactive derivative of *Ac* in *wx-m9*, has shown that recovery of activity is associated with demethylation of cytosine residues in the 5' region of the transposase gene (Schwartz and Dennis, Mol. Gen. Genet. 205: 476-482, 1986). Recently we have shown that the frequency of reactivation is high among plants regenerated from tissue cultures initiated from embryos with *wx-m9 Ds-cy* (Dennis and Brettell, Phil. Trans. R. Soc. Lond. B 326: 217-229, 1990) and that the altered patterns of methylation are heritable (unpublished). As part of the study we examined the effects of other environmental influences on the activity of *Ac* in *wx-m9 Ds-cy*. Preliminary results of an experiment in which germinating seedlings were subjected to various cold treatments suggest that the methylation status and activity of the *Ac* element are not influenced by steady low temperature regimes.

Two lines of maize designated B and F, homozygous for *bz2-m*, were chosen from cobs which showed a low level of *Ac* activity (2-5% of kernels showing variegation, characteristic of an active *Ac* causing excision of *Ds* from *bz2*). The parent plants were later shown to be segregating for *wx-m9 Ds-cy*. Only kernels without spots were selected for planting, and were subjected to the following treatments: I, sown in a warm glasshouse (18 - 30 C) and after one week transferred to 3 C under low light for three weeks, then returned to the glasshouse; II, sown in a warm glasshouse and after one week transferred to 8 C under low light for three weeks, then returned to the glasshouse; III, sown at 8 C and maintained at 8 C in low light for four weeks before transfer to the glasshouse; IV, sown and maintained in the glasshouse.

The seedlings from treatment I succumbed to fungal rot and did not survive. For the other treatments DNA was extracted from young leaf tissue when the plants had reached a height of 300mm. The DNA was cut with the methylation-sensitive restriction endonuclease *Hpa*II and subjected to Southern analysis using the internal *Hin*dIII fragment of *Ac* as a probe. The size of the hybridising band gives a measure of the level of cytosine methylation within the transposable element (Schwartz and Dennis, 1986). No differences were seen in the hybridisation patterns between the samples taken for the different treatments, although a slightly smaller band was seen in the samples from line B compared to those from line F.

The plants were grown to maturity, self-pollinated and the resulting cobs scored for *Ac* activity. The data are given in Table 1 and show that there is no increase in the level of *Ac* activity for the cold temperature treatments. Our conclusion is that moderate cold treatment at an early stage of development does not enhance the activity of the inactive *Ac* element in *wx-m9 Ds-cy*.

Table 1. Percentage of kernels showing *Ac* activity in cobs harvested from *wx-m9 Ds-cy* plants exposed to different temperature regimes.

		Numbers of plants in each class defined by percentage of kernels showing *Ac* activity			
		0%	0.1-1%	1-5%	>5%
Line	Treatment				
B	II	3	0	2	0
	III	8	1	2	1
	IV (control)	7	2	1	2
F	II	1	1	0	0
	III	10	2	1	0
	IV (control)	8	2	0	2

CHESTNUT HILL, MASSACHUSETTS
Boston College

The prospect of using doubled haploids in breeding

--Y. C. Ting and K. Delorey

In the last few years many spontaneously doubled haploids via anther culture were obtained. However, more than 50 percent of these plants showed chlorophyll deficiency of varying degrees and died before they reached adult stage of growth. The other about 40 percent demonstrated abnormalities of different kinds: some died of lethal seedlings; others, of lodging, still others, of disease and insect attacks. Hence, only two to three percent of the doubled haploids survived and grew into reproductive stage. However, as soon as they had attained silking and anthesis period, some manifested protogynous characteristics. All of these hindered proper processes of fertilization and setting seeds, even though those processes were carried out under controlled operation. When we took all of the above into account only a very few percent of the regenerated plants were left for reproduction. By going through all of the effort, several genetically stable doubled haploids (inbreds) were selected (Ting and Gu, 1990). Those doubled haploids were crossed with several commercial inbreds such as B73. It was found that one of the combinations was outstanding in yield. It was estimated that this hybrid combination produced more than 200 bushels of grains per acre. During the growth period here, the hybrid plants were vigorous and free from diseases or insect pests. Therefore, it seems likely that through anther culture, genetically stable inbreds can be obtained and applied in a productive breeding program.

Continued studies on regeneration of haploid callus lines

--Y. C. Ting and L. J. Forastieri

It was consistently observed that haploid callus lines grew better on N_6 medium with 8-10 percent sucrose than those grown on N_6 medium with 1-2 percent of the same. In order to make a further study of this, three haploid callus lines, 88-S5, 87-S4 and SAN_1, were employed for testings in the last spring and summer. The media were prepared by following the standard N_6 medium formula with 100 g/l sucrose; 0.5 mg/l 2,4-D; 0.5 mg/l kinetin. After the callus lines had been on this medium for six weeks, somatic embryos were formed in most of the cultures. The embryos were creamy white, smooth surfaced and globular in shape. Particularly in the cultures of line 88-S5, about 60 percent of the calli differentiated into embryos while in those of lines 87-S4 and SAN_1 only less than 10 percent of them did the same.

When the above somatic embryos were transferred onto N_6 medium with 20 g/l sucrose but no hormones, over 50 percent of them from line 88-S5 germinated and grew into plantlets within two weeks. Of lines 87-S4 and SAN_1 only less than three percent of the embryos grew into plantlets. By the foregoing procedures, about 100 plantlets were obtained in the last year. However, to our disappointment none of these plants grew vigorously and reached reproductive stage. All of them died at the four to five-leaf stage. Even though some IAA (indole-acetic acid) and GA (gibberellic acid) of an amount of 0.5 mg per liter were added to the media, no improvement in the condition of plant growth and development was found. Therefore, it seems reasonable to conclude that if it is necessary to produce haploid plants which can continue to grow and set seeds from these callus lines, more research is needed.

COLD SPRING HARBOR, NEW YORK
Cold Spring Harbor Laboratory

Gene amplification at the *P-wr* allele

--Prasanna Athma and Thomas Peterson

P-wr is one of the few *P* alleles with differential expression in floral tissues: the *P*-specified pigments are present in cob glumes and absent in the pericarp. Preliminary results from Southern hybridizations indicate that the *P-wr* allele contains a 5-10 fold repetition, or amplification, of sequences present in *P-rr*. *P-rr*, which specifies red pericarp and cob, contains two 5.2 kb direct repeats separated by a 7 kb transcribed region (Lechelt et al., Mol. Gen. Genet. 219:225-234). The region amplified in *P-wr* represents most of the 7 kb transcribed region and one or both of the direct repeat sequences. Interestingly, a 700 bp fragment at the 3' end of the *P-rr* gene does not detect the *P-wr* amplified region; however, probes flanking this 700 bp fragment do hybridize to the amplified region. When restriction fragments outside the direct repeats were used as probes, the *P-wr* amplified region was not detected; instead similar banding patterns were observed in *P-wr* and *P-rr* alleles, indicating that some of the restriction sites are conserved in both alleles. Taken together, the Southern results suggest that the region amplified in *P-wr* represents approximately 12 to 21 kb of sequences present in *P-rr*. We do not know yet, either the size of the complete amplified unit or the arrangement of the repeats in the amplified region. Further experiments to determine the structure of the *P-wr* amplified region are in progress.

Since gene amplifications in other organisms are associated with developmentally regulated gene expression, we looked for differences in the *P-wr* amplification in different tissues of the maize plant. DNA was prepared from tissues in which *P* is expressed (silks, husks, cob glumes) or not expressed (leaves), digested with restriction enzymes, and analyzed by Southern hybridizations. No obvious differences in the intensities or sizes of the *P* hybridizing bands in the different tissues were found. Therefore, the *P-wr* amplified structure is not detectably altered during development. The *P-wr* transcriptional pattern is under investigation.

Cloning of a *P*-regulated chalcone-flavanone isomerase gene from maize

--Erich Grotewold and Thomas Peterson

Chalcone synthetase (CHS) is the first enzyme of flavonoid biosynthesis and converts malonyl-CoA and 4-coumaroyl-CoA into chalcone. The similarity of flavonoid biosynthesis in maize and other plants (petunia, bean, etc.)

suggests that maize should contain a second enzyme, chalcone-flavanone isomerase (CHI), responsible for the isomerization of the chalcone into the corresponding flavanone. Flavanone is the substrate for the synthesis of 3-deoxy and 3-hydroxy flavonoid derived pigments. The chalcone-flavanone isomerase enzyme should act between the *C2* and *A1* encoded products, which opened the possibility that its expression would be regulated by the *P* gene in the pericarp, as are *C2* and *A1* (Grotewold et al., submitted). However, CHI activity has not been reported in maize, nor has any mutant in this step been isolated.

To clone a maize CHI gene, we used highly degenerate primers made from sequences conserved between CHI proteins from several plants (we appreciate very much the suggestions provided by Dr. Arjen van Tunen regarding the sequences of the primers). We obtained a 210 bp cDNA by PCR, which was used as probe for screening a cDNA library from pericarps carrying a functional *P* gene (*P-rr*) (Grotewold and Peterson, 1990; MNL 64:37). We isolated nine independent clones about 650 bp long which showed identical sequences between them.

The translated sequence of the cDNA clones showed about 65% identity at the amino acid level with the sequence of two chalcone-flavanone isomerase proteins described in *Petunia hybrida*. The first amino acid encoded by the longest cDNA clone corresponds to amino acid 47 of the sequence of the petunia CHI-B protein (van Tunen et al., 1989; Plant Mol. Biol. 12:539-551), indicating that the obtained cDNA clones are not full-length.

Northern blot hybridizations of pericarp poly A RNA showed a single transcript of about 1 kb hybridizing with these cDNA clones. No transcript could be detected in pericarp RNA lacking a functional *P* gene, suggesting that the maize CHI gene could be regulated by *P* in the tissues in which *P* is being expressed.

We don't know yet how many CHI genes are present in maize, although our results suggest that a single gene would be transcribed in the pericarp under the control of *P*. In petunia, different CHI genes are transcribed in different tissues (van Tunen et al., 1988; EMBO J. 7:1257-1263). It remains to be shown if this is the case with the maize CHI gene. Since the step catalyzed by the product of the CHI gene is common to the synthesis of all flavonoid derived products, including the anthocyanins, it would be expected that other regulators of the pathway (for example *R, B, C1* and *Pl*) would also be able to regulate the expression of CHI in other tissues.

COLOGNE, GERMANY
Institut für Genetik

The synthesis or activation of a trans-acting factor is *not* the rate-limiting step in excision/transposition events

--Manfred Heinlein and Peter Starlinger

Barbara McClintock has observed a correlation between the excision of *Ds* from the *c-m1* allele and chromosome breakage at the site of *Ds* at standard position. If this would be a general effect, it might indicate that the activation or synthesis of a trans-acting factor is the limiting step in the transposition process. However, on the basis of our revised measurements of *Ac*-mRNA (Schein et al., MNL 64, 1990; Fusswinkel et al., Mol. Gen. Genet., in press), which yield 2 to 13 mRNA molecules per endosperm cell on the average, a limitation of transposition by the availability of *Ac* protein is not expected. Thus, the correlation of excision events was studied further (Heinlein and Starlinger, Maydica, submitted). We examined kernels on which the variegation patterns of two mutable alleles can be monitored. A lack of correlation is only diagnostic if all excision events lead to a visible phenotype. Due to excision "footprints" and other complications, we could not be sure to find such pairs of mutants. Therefore we looked for situations in which the correlation of events does not exist. This is the case when small sectors due to late events at the first mutable allele are located within larger sectors due to earlier events at the second locus. The small sectors are part of the larger sectors, meaning that the two events happened at different times but in the same cell clone. We observed such situations in several cases. Therefore we think that excision events are usually not correlated and are independent from each other. Thus, the low frequency of excision events cannot be explained by the lack of a trans-acting factor. B. McClintock's observation of correlated excision needs an additional explanation.

The *Ac*-dosage effect

--Manfred Heinlein and Peter Starlinger

Reciprocal crosses between *Ac* bearing and *Ds* bearing lines were done to learn more about the *Ac*-dosage effect (Heinlein and Starlinger, Maydica, submitted). The *Ds* carrying maize line was *C Sh bz-m2(DI) wx*, which was used as the tester line to measure the dosage effects of different *Ac*-elements in trans. The *Ac-containing* maize lines were homozygous *C sh bz wx-m9Ac*, *C sh bz wx-m7*, and *C sh bz wx Ac*. The latter *Ac*-element we call *Ac* u.p. for "unknown position" (on chr. 9). The dosage effects measured were compared with the dosage effect of *bz-m2Ac*, in which case the *Ac* is acting on itself. The reversion patterns seen on the kernels all were different. If *bz-m2(DI)* is transactivated by *wx-m9*, reversions occur later during development, with higher dose giving rise to smaller spots. However, if *wx-m7* is used, larger spots are seen with 2 *Ac* than with 1 *Ac*. We counted the number of spots of each revertant clone size separately and plotted the number of sectors against clone size. In every case the curves for 1 *Ac* and 2 *Ac* cross each other, which means that the sign of the dosage effects is altered during development. Whereas *wx-m9* shows a negative dosage effect during early developmental stages but a positive dosage effect during later stages, *wx-m7* shows a positive effect during early stages, but a negative effect at later stages. *wx-m9* is very similar to *bz-m2Ac* in respect to time and frequency of events as well as the dosage effect. Therefore we think that beside the known negative dosage effect positive dosage effects can also be measured in maize, and the positive dosage effect is therefore not restricted to heterologous plants transgenic for *Ac* (Jones et al., Science 244:204, 1989). The *Ac*-elements in *wx-m7* and *wx-m9* are identical in se-

quence but show opposite dosage effects in our test. This might either be due to position effects or to differences in the genetic background.

In vitro analysis of the *Ac* encoded ORFa protein and mutant forms after heterologous expression

--Siegfried Feldmar and Reinhard Kunze

The transposable element *Ac* is transcribed in the form of one 3.5kb long transcript. The first AUG codon of the *Ac* mRNA opens an 807 amino acid long open reading frame called ORFa. A corresponding protein with an apparent molecular weight of about 112kD is found in nuclear extracts by western blot analysis.

It was shown that the ORFa protein expressed in insect cells has DNA binding activity; the recombinant ORFa protein binds specifically to several subterminal fragments from both ends of *Ac*. The recombinant protein also binds to DNA fragments containing concatemers of an AAACGG motif that is repeated several times at both ends of *Ac* (Kunze et al., EMBO J. 8:3177, 1988). In order to identify the structural basis for the site specific DNA binding of the *Ac* ORFa protein we began to express the intact and mutant forms of the protein in *E. coli*.

The recombinant proteins were accumulated in an insoluble and inactive form in the bacteria. The protein aggregates were recovered by cell breakage and low speed centrifugation. After solubilization in guanidinium chloride and subsequent refolding by dilution the soluble fraction of the proteins was analyzed for DNA binding activity using the gel mobility shift assay.

We could demonstrate that renatured wildtype protein has DNA binding properties comparable to the ORFa protein expressed in insect cells. From these studies we concluded that the wildtype ORFa protein can be reactivated after denaturation in guanidinium chloride. Protein modifications that might occur in higher eukaryotic cells are not essential for site specific DNA binding.

To roughly localize the region of ORFa necessary for specific DNA binding, a series of N-terminal and C-terminal deleted forms of the protein were tested for their ability to bind specifically to the Ac5'-end and the AAACGG motif. We found the 537 amino acids can be removed from the C-terminus without disrupting the capacity to complex specifically the ORFa protein target sites. The N-terminus of the protein is not required for DNA-binding, too. A truncated version of the ORFa protein, having lost 135 amino acids from the N-terminus, still has full DNA-binding activity.

From these studies we conclude that a DNA binding domain of the protein resides somewhere between residues 136 and 370. The N-terminal half of this segment has a rather basic character, whereas the C-terminal half reveals a weak homology to the helix-loop-helix motif. Such motifs are found in the DNA-binding domains of some proteins (Murre et al., Cell 56:777, 1989).

Nuclear factors bind to hexamer motifs in subterminal sequences of *Ac*

--Heinz-Albert Becker and Reinhard Kunze

The transposable element *Ac* encoded ORFa protein can bind to AAACGG motifs in subterminal regions of the transposon but not to the 11bp terminal inverted repeats (Kunze et al., EMBO J. 8:3177, 1989). Host factors are known to be involved in the transposition mechanism of some other transposons. Therefore DNA-protein interaction studies were carried out looking for such factors.

Last year it was reported that three fragments of the *Ac* ends formed strong retarded complexes in electrophoretic mobility shift assays (EMSA) using crude nuclear extracts from kernels and pdIdC as an unspecific competitor. Fragment 1 (*Ac*-pos. 1-181), fragment 2 (*Ac*-pos. 4195-4419) and fragment 3 (*Ac*-pos. 4419-4565) showed homologous and heterologous competition in 10-fold excess of the non-labeled fragment.

To analyse the DNA-protein interactions in more detail all three *Ac* fragments were subjected to indirect DNaseI footprint analysis. Fragments 1 and 3 were used alternatively labeled at both strands. These experiments resulted in identification of a sequence motif which seems to be responsible for complex formation of the three *Ac* fragments. In regions where protected bases were clustered a hexamer motif could be identified. The sequence is GxTAAA, where x is a G in 4, and A in 2 cases and a T in one case. A GCTAAA doesn't occur in *Ac*. The GxTAAA motifs lie in between the AAACGG hexamers known to be recognized by the *Ac* ORFa protein. The hexamer is the only consensus sequence among the protected sites. Under the conditions applied the protected regions containing the GxTAAAs were the only ones in the three *Ac* fragments. No protection of the terminal 11bp inverted repeats was detectable. The hexamer is found 3 times in the first 181bp of *Ac* and 6 times in the last 369bp. At 7 positions protected bases can be seen. One position shows no clear protection and the last hasn't been analyzed so far.

For efficient DNA-protein complex formation in EMSAs more than one hexamer seems to be necessary per fragment. Each of the three strongly complexed *Ac* fragments contains more than one motif. When fragment 1 is split at *Ac* pos. 75 the fragment pos. 76-181 is strongly complexed. It still contains 2 GGTAAAs. Fragment 1-75, which has only one hexamer, is only very weakly complexed.

These observations are in coincidence with the use of isolated host factor motifs. The hexamer at *Ac* pos. 4533 was subcloned with two upstream and downstream bases as a 14bp oligonucleotide with Sau3A spacers in vector pUC19. A copy number of four seems to be necessary for efficient and specific complex formation in vitro. It can compete in 10-fold excess complex formation of the *Ac* fragments 1 and 3, but the reverse competition experiment wasn't successful. The subcloned GGTAAA motif seems to be stronger complexed than the *Ac* fragments with the different GxTAAAs. This observation and the fact that one of the 9 GxTAAAs in the *Ac* ends showed no clear protection leads to the assumption that neighbour bases might have an additional influence on the strength of complex formation.

So far there is no evidence for a role of the observed DNA-protein interactions in the transposition mechanism of *Ac*.

A transient assay for *Ac* excision in parsley (*Petroselinum crispum*)

--Ralf Lütticke and Reinhard Kunze

A transient excision assay has been developed using *Petunia hybrida* protoplasts cotransfected with two plasmid DNAs (Houba-Herin et al., MGG 224:17, 1990): one contains a *Ds*-element (*Ac*Δ) between the octopine TR-DNA 1'-promoter and the *E. coli* ß-glucuronidase (GUS) gene (pNT 100). The other plasmid carries the *Ac*-coding sequence (ORFa) or mutants of ORFa behind the 2'-promoter. After expression of a functional *Ac* protein the *Ds* element (*Ac*Δ) can be excised, thereby restoring the activity of the ß-glucuronidase gene. GUS activity is detected by histochemical staining (Jefferson et al., EMBO J. 6:3901, 1987) as blue stained protoplasts. The number of blue protoplasts after fixation on a filter is considered to be *Ac*Δ-excision frequency. As only leaves from young sterile plants of a certain age can be used as a protoplast-source, a continuous supply of young plants is required for routine usage.

We tested the suitability of a fast growing parsley suspension culture (obtained from Dierck Scheel and Klaus Hahlbrock, MPI für Züchtungsforschung, Cologne) as a source of protoplasts for the cotransfection experiments. The background frequency of blue spots after transfection with the target plasmid pNT100 (1'Pr. - *Ac*Δ - GUS) alone is slightly higher (2 to 3 blue spots per 10^5 protoplasts) compared to *Petunia* protoplasts. Cotransfection of the parsley protoplasts with pNT100 and a plasmid carrying the wildtype *Ac* element resulted only in a 2-3-fold increase above background. Very similar numbers were obtained when cotransfecting a genomic ORFa fragment or a cDNA ORFa fragment behind the 2'-promoter, respectively.

Li and Starlinger have shown that the 102 N-terminal amino acids of the *Ac* ORFa protein are dispensable. The truncated ORFa protein mobilized a *Ds* element with an even higher frequency than the full length ORFa protein (Li and Starlinger, PNAS 87:6044, 1990). This truncated genomic (= intron containing) ORFa placed behind the 2'-promoter (plasmid pNT804) enhanced the *Ac*Δ excision-frequency in *Petunia* protoplasts even about 15-fold compared to the full length ORFa protein, although the ATG codon at amino acid 103 was preceded by 7 out-of-frame ATGs (Houba-Herin et al., MGG 224:17, 1990). In parsley protoplasts we have results from only 3 independent transfections. Therefore we consider these results to be preliminary, pNT804 increased the *Ac*Δ excision frequency as it does in *Petunia*, but only about 2-3-fold compared to the wildtype *Ac* element. Cotransfection with an equally truncated cDNA fragment, however, resulted in a 6-fold increase.

We conclude that parsley protoplasts can be used as an alternative to *Petunia* protoplasts. The *Ac*Δ excision frequencies are slightly lower than in *Petunia*, but the qualitative results seem to be identical. The sensitivity of both assay systems is very limited, as only excision frequencies above the level triggered by the wildtype *Ac* ORFa protein can be detected reliably. The advantages of the parsley cells are their quick and nearly unlimited availability.

We have begun to investigate derivatives of the truncated ORFa protein in the parsley cell system. Initial experiments revealed that removal of the 7 upstream ATGs by placing the 10 in-frame ATG immediately behind the 2' promoter has virtually no influence on the excision frequency. Preliminary experiments indicate that an N-terminally truncated ORFa protein starting with amino acid 137--that is immediately behind a tenfold repeat of the dipeptide Pro-Gln/Glu--abolishes the transposition function. A construct lacking 220 amino acids from the C-terminus of the ORFa protein is transposition deficient, too, or induces such events only with a very much reduced frequency. If these results can be confirmed, we have to conclude that essential functions of the 807 amino acid *Ac* ORFa protein are located between amino acids 103 and 136, and within the 220 C-terminal amino acids, respectively.

DNA methylation of *Ac* sequences in maize and in transgenic tobacco

--Thomas Ott, Birgit Nelsen-Salz, Hans-Peter Döring

From previous studies with the transposable element *Ac* in transgenic tobacco cells (Coupland et al., EMBO J. 7:3653, 1988; Coupland et al., Proc. Natl. Acad. Sci. USA 89:9385, 1989) it is known that both subterminal regions of *Ac* carry *cis*-acting sequences which are required for the transposability of the element and which are likely to be recognized by the *Ac*-encoded protein. Within the 5' subterminal region the hexamer motif AAACGG is present in nine copies in either orientation. In several transgenic tobacco cultures carrying one or two copies of a transposable *Ac* or *Ds* element we examined the 5' subterminal region of *Ac* (position 70-220) for the presence of 5-methylcytosine. We used the recently developed ligation-mediated polymerase chain reaction-aided genomic sequencing (Mueller and Wold, Science 246:780, 1989; Pfeifer et al., Science 246:810, 1989) with minor modifications. This method gave good results with 10µg of genomic tobacco DNA for each cleavage reaction according to Maxam and Gilbert. We analysed 60 cytosine positions of both DNA strands and detected no 5-methylcytosine. Thus, DNA methylation is not needed for the transposability of *Ac* and *Ds* in transgenic tobacco plants. These findings confirm other results with similar transgenic plants on the lack of de novo methylation at most of a lot of different restriction sties of the *Ac* sequence (Nelsen-Salz and Döring, Mol. Gen. Genet. 223:87, 1990).

Maize DNA containing the inactive *Ac* element of the *wx-m9Ds-cy* allele (Schwartz and Dennis, Mol. Gen. Genet. 205:476, 1986) was cleaved with *Sal*I and size-fractionated on a sucrose gradient. The 5-6kb fraction which contains the *Ac* element was used for genomic sequencing. We sequenced one DNA strand and showed that the hexamer motifs between position 120-160 as well as the three cytosines at or next to the *Bam*HI site at position 181 are unmethylated. Southern analysis with maize DNA containing the same allele and restricted with *Bam*HI showed that the majority of the *Ac* molecules were cleaveable. This finding suggests that *Bam*HI was unable to cleave at completion, or alternatively, that a small fraction of the *Ac*

sequences are methylated and had gone undetected upon genomic sequencing.

If we compare the DNA methylation patterns of the maize lines *wx-m9Ac/wx* and *wx-m9Ds-cy/wx* by means of the methylation-sensitive restriction enzymes, we find pronounced differences in DNA methylation of the GC-rich, untranslated leader sequence of the *Ac* transcription unit. The active *Ac* element of the *wx-m9* allele is unmethylated at the *Ava*I site (position 86) and the *Mlu*I site (position 434). At least one of the *Eco*RII sites (position 332.340), one of the *Rsr*II sites (position 546.610.657), one of the *Sac*II sites (position 609.656.740), and one of the *Eco*RII sites (position 840.880) are unmethylated. The latter sites are so densely spaced that it could not be determined which of the sites was unmethylated. Therefore it is well possible that all 12 restriction sites are unmethylated. The inactive *Ac* element of the *wx-m9Ds-cy* allele is heavily methylated at all 12 sites. Only a minute fraction of the inactive *Ac* molecules are unmethylated at position 332 or 340. The *Ac* sequences of both alleles are completely methylated at position 938 (*Eco*RII). There is also no significant difference between both strains with respect to methylation in e transcribed region at positions 1320, 3556, and 3844 (*Pvu*II, *Ava*I, *Pvu*II). In all cases it is only a fraction of the *Ac* sequences which is unmethylated. We also examined these restriction sites in the *wx-m9Ds* allele which is a 194bp deletion derivative of the *Ac* element. In this allele the methylation pattern was almost identical to the pattern which we found in the *Ac* element of the *wx-m9* allele. It was shown previously (Kunze et al., Mol. Gen. Genet. 214:325, 1988) that a specific transcript is produced in the *wx-m9 Ac* and the *wx-m9Ds* alleles, whereas no transcript is found for the *wx-m9Ds-cy* allele. If DNA methylation is involved in the control of *Ac* activity the decisive cytosines are located in the GC-rich untranslated leader sequence of the *Ac* transcription unit.

COLOGNE, GERMANY
Max-Planck-Institut

Integration of *dSpm* in transgenic tobacco

--Guillermo Cardon, Julio Reinecke, Monika Frey, Heinz Saedler and Alfons Gierl

An excision assay system was recently established in transgenic tobacco (Frey et al., EMBO J. 9:4037-4044, 1990) for the *En/Spm* transposable element. In this model system two *En/Spm* encoded functions, TNPA and TNPD, are required for excision of a *dSpm* reporter element. TNPA is the product of the most abundant 2.6kb mRNA that has been previously identified as the suppressor component of *En/Spm* (Grant et al., EMBO J. 9:2029-9035). TNPD is the product of the less abundant 5.9kb mRNA (Masson et al., Cell 58:755-765, 1989). The sequence alterations (footprints) generated during excision are similar to the ones observed in maize. This suggests that the mechanism of excision promoted by TNPA and TNPD is similar, if not identical, to the one existing in maize. Based on these results a model for *En/Spm* excision was proposed (Frey et al., 1990): Binding of TNPA to the subterminal region of the element induces complex formation between both ends by a "zipper"-like mechanism, where the bound TNPA molecules represent the teeth of the zipper. This leads to "correct" alignment of the terminal inverted repeats (TIRs). Binding of TNPD to the TIRs leads to endonucleolytic cleavage and release of *En/Spm* from the site of integration accompanied by healing of the chromosome breaks.

In order to analyse transposition in this transgenic system, four independent *dSpm* integration events were molecularly cloned into the lambda EMBL4 vector. All clones represent completely transposed *dSpm* elements, and their insertion generated the characteristic three base pair target site duplication. Therefore, the mechanism of integration in the presence of TNPA and TNPD in the transgenic system seems to be identical to the one present in maize. Three of the transposed *dSpm* elements moved into single copy DNA that is relatively undermethylated, as judged by methylation sensitive restriction enzymes. One of the transposed *dSpm* elements moved into middle repetitive, methylated DNA. The regions surrounding the new insertion sites do not show any significant homology to *En/Spm* specific sequences. These results suggest that this system might be suitable for gene tagging in transgenic systems.

Does *A2* encode a dioxygenase?

--Adriane Menssen, Heinz Saedler and Alfons Gierl

The *A2* gene encodes an enzyme acting late in anthocyanin biosynthesis in the aleurone tissue of kernels or other parts of the maize plant. The biochemical role of the *A2* enzyme in anthocyanin biosynthesis is not well understood. Inter-tissue complementation assays (Reddy and Coe, Science 138:149-150, 1962) suggest that the *A2* enzyme acts after the *A1* gene product (dihydroflavonol 4-reductase), and converts cis-leucoanthocyanidin to anthocyanidin (cis-leucocyanidin to cyanidin or cis-leucopelargonidin to pelargonidin depending on *Pr*), the first colored component of the anthocyanin enzyme cascade. This step requires two biochemical reactions: trans-elimination of H_2O and oxidation of carbon No. 2.

The *A2* gene was cloned by transposon-tagging and its transcription unit was determined (Menssen et al. EMBO J. 9:3051-3057, 1990). The *A2* gene is intronless and the open reading frame encodes a putative protein of 43.5kD. An amino acid sequence homology search revealed a possible function. A 26% identity and 48% similarity was found with the pTOM13 mRNA encoded protein. pTOM13 probably encodes a polypeptide involved in the conversion of 1-aminocyclopropane-1-carboxylic acid to ethylene by the ethylene forming enzyme (ACC-oxidase, Hamilton et al., Nature 346:384-387). A similar amount of homology (29% identity and 53% similarity) was found to the flavanone 3-hydroxylase cDNAs of *Antirrhinum majus* (C. Martin and A. Prescott, personal communication) and *Petunia* (G. Forkmann, personal communication). The flavanone 3-hydroxylase is a dioxygenase that is 2-oxoglutarate and Fe^{2+} dependent (Forkmann et al., Z. Naturforsch. 35c:691-695, 1980). In this case, flavanone is converted to dihydroflavonol by introduction of a hy-

droxyl group in the 3-position. Therefore, it is assumed that the *A2* enzyme is also a 2-oxoglutarate dependent dixoygenase, as well as the ACC-oxidase. In the case of the *A2* reaction the oxidation of carbon No. 2 may occur via introduction of a hydroxyl group. The resulting triol might be an unstable intermediate, which is spontaneously dehydrated, probably because the activation energy for the elimination of water is reduced by the generation of the aromatic system. Another possibility is that in addition to the *A2* enzyme, a dehydratase is involved in the synthesis of anthocyanidin. However, no corresponding mutant allele has been found.

The *inc3* (*candica*) gene of *A. majus* probably represents the homologue of the *A2* gene, since both gene products share 60% amino acid identity and 70% similarity (C. Martin and A. Prescott, personal communication), as would be expected for monocots vs. dicots. Scattered patches of amino acid motifs are identical in all five proteins mentioned above. These motifs may represent conserved binding-sites of the common co-substrate 2-oxoglutarate or the common cofactor Fe^{2+}. Experiments are now on the way to unravel this last unknown step in anthocyanidin biosynthesis.

Molecular analysis of the two maize chalcone synthase genes *C2* and *Whp* (white pollen)

--Philipp Franken, Ursula Niesbach-Klösgen, Heinz Saedler and Udo Wienand

The *C2* locus has been cloned from a *Spm* induced mutant *c2-m1* using transposon tagging (Wienand et al., MGG 203:202, 1986). The analysis of a *C2* specific cDNA confirmed that the locus is coding for a chalcone synthase (CHS) (Niesbach-Klösgen et al., J. Mol. Evol. 26:213, 1987). Genetic analysis indicates that *C2* is not the only gene in maize encoding CHS. A second gene which can complement *C2* function has been previously identified as the white pollen gene (*Whp*) (Coe et al., J. Hered. 72:318, 1981). This gene is of particular interest since the loss of CHS activity (in a double recessive mutant *c2 c2 whp whp*) leads to the production of sterile, white pollen.

Because of the predicted homology in function (and sequence) of both genes we used *C2* specific cDNA as a molecular probe to clone the *Whp* gene. A homologous fragment segregating with the *Whp* locus could be identified from F2 progeny analysis of a *Whp* heterozygous population. This fragment was cloned, sequenced and compared to the genomic sequence of the *C2* locus.

The two genes share high homology in the codogenic parts (94%) while the introns are very different in sequence and size (1524 bp for *C2* and 2157 bp for *Whp*). The *Whp* clone isolated represents a functional gene since cDNA fragments homologous to the genomic clone have been isolated and sequenced. The putative amino acid sequence deduced from the genomic and cDNA sequences indicates that the two CHS proteins are very similar. Both proteins are 400 amino acids in length and differ in 21 amino acid positions. Sequences 5′ and 3′ of the codogenic sequences are significantly divergent indicating a different mode of regulation for the two genes.

Gene specific probes for both the *C2* and the *Whp* gene have been isolated to analyze expression of both genes in various genotypes and different tissues. These analyses showed that *Whp* and *C2* expression in aleurone is regulated differently.

Molecular analysis of the dominant inhibitory *C2* allele *C2-Idf*

--Udo Wienand, Irmgard Weisskirchen, Philipp Franken and Heinz Saedler

The dominant inhibitory mutant *C2-Idf* (colorless kernels) is allelic to *C2* (Greenblatt, MNL 49:23, 1975) and decreases kernel pigmentation if crossed to a wildtype allele. Since the *C2* locus has been cloned (Wienand et al., MGG 203:202, 1986) and thus *C2* specific probes were available we were interested in analyzing the molecular nature of the *C2-Idf* mutation.

The genomic structure of the *C2-Idf* allele was analyzed and compared to a wildtype *C2* allele by Southern experiments. With a *C2* specific sequence as a probe on *Eco*RI digested DNA several bands light up in the *C2-Idf* mutant compared to a single band in wildtype DNA (Line C), indicating the presence of several *C2* genes in the *C2-Idf* line. For further analysis of the *C2-Idf* allele a genomic library was prepared from homozygous *C2-Idf* DNA in lambda EMBL4 and screened with a *C2* specific probe. Five positive clones were isolated and compared to the wildtype *C2* gene isolated previously. Restriction fragment analysis and hybridization experiments showed that the *C2-Idf* clones contained deletions or rearrangements or the wildtype *C2* gene (instead of a full size wildtype *C2* gene).

Transcript analysis of the homozygous *C2-Idf* mutant using the *C2* cDNA as a probe showed that no *C2* specific transcript could be detected (in developing kernels, 30 DAP). Interestingly, besides *C2*, the *C2-Idf* mutation also affects expression of other anthocyanin genes like *A1* and *Bz1*. In contrast no effect of the mutation on the second chalcone synthase gene, *Whp* (white pollen), could be detected.

COLUMBIA, MISSOURI
University of Missouri

Linkage of *D9* and *Nl2* on chromosome 5

--M. G. Neuffer

The EMS induced dominant mutants *Nl2* and *D*-2319* have been placed on chromosome 5 by T *wx* tests. The following information will place *D8-2319* in the short arm of chromosome 5 and since there are no known dwarfs in that part of the genome the symbol *D9* is proposed. Backcross linkage crosses of the two mutants with the recessive tester *a2 bt1* have produced the following values:

Nl2-10-*a2*-3-*bt1*
D9-32-*a2*-5-*bt1*

Crossing the double heterozygote *D9* +/+ *Nl2* onto a + + stock produced a map distance of 19cM between them.

These results indicate that these mutants are on the short arm of chromosome 5. The order with approximate map distances is *D9*-19-*Nl2*-10-*a2*-5-*bt1*.

Indeterminate dwarf: an EMS-induced double mutant

M.G. Neuffer and S. Chao

The indeterminate dwarf mutant, *idd*-2286A*, reported in MNL63:62 and located (MNL64:52), has exhibited some interesting characteristics. The mutant seedlings are shorter than normal but respond to GA. At the time when normal sibs are mature (60 days) mutant plants are about 1/4 normal height with no evidence of flower formation. At 100 days, mutant plants which are then as tall as normal sibs are still green and growing with approximately 32 leaves and no tassel. As the days become shorter and as a consequence of other still unknown conditions, a heavy tassel with small, culturable plantlets growing from between the glumes appear. Some viable pollen is also produced.

The mutant has been located on the long arm of Chromosome 1 by B-A chromosome tests. A cross with *id1* has indicated allelism for the indeterminate aspect but not for dwarfing. Crosses of the mutant with the radiation-induced double mutant, *an-6923*, which is thought to be deficient for the *an1* and *bz2* loci, gave a positive allelism test for *an1* but negative for *bz2*. These results suggest that *idd*-2286A* may be a deficiency for the *id1* and the *an1* loci and that the order of the genes in question is: centromere, *id1*, *an1*, *bz2*.

As part of an interval mapping project, which is to incorporate all the available visible markers onto the RFLP based map, we have located the mutant segment with reference to RFLP markers using F2 materials generated from *idd*-2286A*. The preliminary data have indicated the gene order as: centromere - *UMC83* - *id1* - *an1* - *bz2*. The map distance between *UMC83* and *id1* is under detailed investigation.

The integrated mapping project: Chromosome nine

--Casey Howell, Ed Coe, Jack Gardiner, Susan Melia-Hancock and Shiaoman Chao

The 1990 News Letter reported, by chromosome, projected naked-eye polymorphisms (NEPs) to be mapped in this project. Mapping of chromosome nine is nearly complete, and work is in progress on the other chromosomes.

The F1 that was selfed to produce an F2 mapping population was heterozygous for the following ten NEPs: *yg2, c1, sh1, wx1, d3, gl15, bk2, Wc1, Bf1, bm4*. All alleles were in a cis arrangement in the F1. We selected 116 individuals among a family of 185 F2 plants (excluding 69 phenotypically normal plants). Typing was carried out on 110 plants showing various recessive phenotypes, and on 6 normal plants. Hetero-fertilization appears likely in a few individual plants, for traits that were characterized by endosperm classification *(c1, sh1, wx1, Wc1)*, but progeny tests that would check the classifications were not available for these specific individuals. Probing with clones of known function (e.g., *C1, Sh1, Bz1, Wx1*) has not yet been completed. Of the RFLP markers tested 16 were polymorphic. The data were analyzed using Mapmaker Macintosh V1.0. The accompanying map was constructed using a maximum recombination fraction of 0.40, a mini-

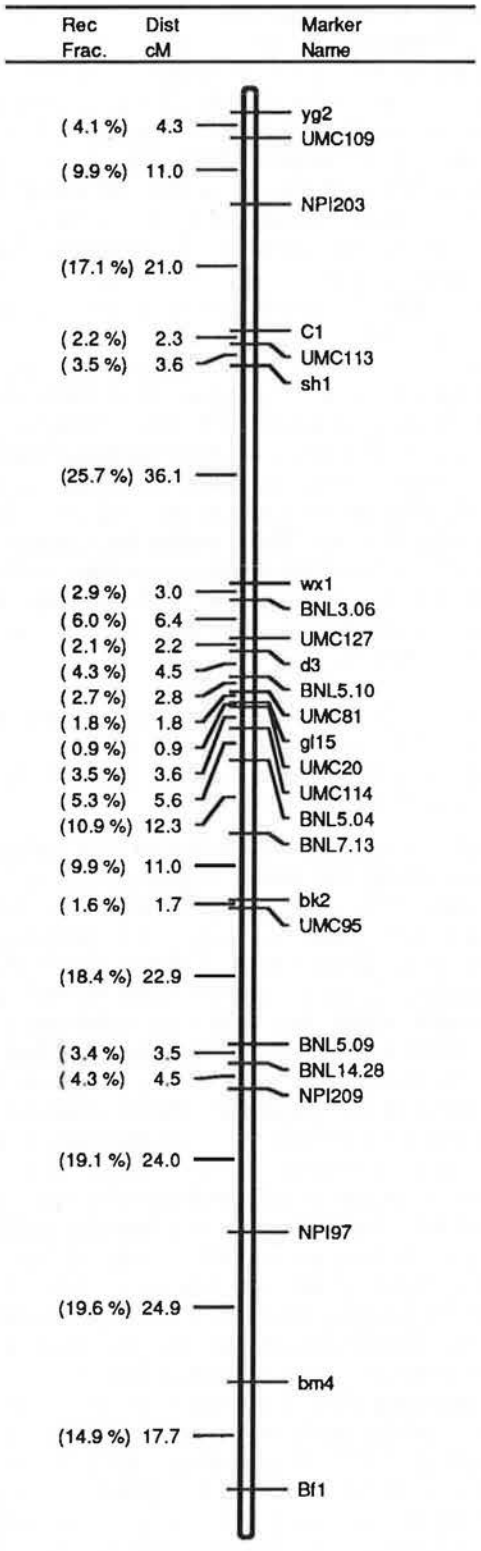


Figure. Map of 9 NEPs and 16 RFLP loci in chromosome 9 (recombination percentages and distances in cM), derived with F2 data from 110 plants showing one or more recessives and 6 all-dominant plants.

mum LOD of 3.0, and applying the Haldane function.

The distal three NEPs on 9L, *Wc1, Bf1, bm4,* mapped with unexpectedly large intervals and with a "best fit" order for *Bf1 and bm4* that conflicts with previous data. The individual-plant data for *Wc1*, while placing it in the established region, indicate far too many double crossovers, and the classifications for this marker, which is famously difficult, must be considered suspect. This marker has been excluded pending new tests. The classical order of *Bf1* and *bm4* is rather firmly defined by earlier data (MNL 38:10), and the present data are confounded by the *Wc1* situation. In addition we suspect that the conflict may be related to the skewed ratios that occurred in this region. These ratios were: *Wc1* 76:40; *Bf1* 105:11; and *bm4* 113:19. Unfortunately *NPI97* segregates with presence-absence (also in cis) rather than codominant alleles, meaning that the data for all 4 distal markers are not robust statistically in any case. A current analysis of new F2 and testcross individuals with the distal 9L markers, verified (and completely classified) by progeny tests, should clarify the orders and interval distances in this region.

Integrated Mapping Project: interval mapping strategy

--Ed Coe and Shiaoman Chao

About 125 NEP variants and isozymes (see MNL 64:47) that have been mapped over the years into the "classical map" are currently being placed on the RFLP Core Map as part of an ongoing NSF-supported mapping project, by interval mapping. The target number of NEP loci to be interval mapped on that project in its next phase is approximately 250 selected loci. The principle of interval mapping (Lander and Botstein, 1986) is that segregating markers nearest to a target gene remain linked with the target and tend to become homozygous when it becomes homozygous. The stock materials required for interval mapping of NEPs are segregating families in which multiple-point polymorphism is present; in fact the extent of polymorphism in maize is such that virtually any segregating family inherently has sufficient interval marking. Interval mapping is typically done individual-by-individual, by determining association of markers and target gene in 25-30 homozygous individuals, directly giving a measure of frequency of recombination.

Expansion to high-density interval mapping, potentially locating every available visible variant, is the goal of the following plan we have developed with the help of Tim Helentjaris and Dave Weber.

Materials for interval mapping include any and all segregating (e.g., F2) progenies that may be made available by maize investigators. These may be the segregating family in which a mutant is found or from which it is being recovered following outcrossing. Progenies segregating for over 2800 characterized mutants, 800 or so of which have been located to some degree in the genome, are ready in Gerry Neuffer's materials to be used in this part of the mapping. In addition there are in this resource many, unnumbered mutants of "common" classes such as albinos and virescents, and new cases of all kinds are currently being screened from M2s of mutagenized families. A collection of unanalyzed mutants is available in the Maize Genetics Stock Center, and in the stocks of scores to hundreds of investigators. The problem is one of prioritization rather than supply. The focus of our selection will be, first, on partially located mutants of all types; second, on certain more-characterized types (e.g., biochemically or physiologically characterized traits); third, on seed and seedling expressions; and fourth, on unique growth types and others.

The strategy we now intend to use, in order to achieve large numbers of interval-mapping evaluations, is a pooled-sample approach. In place of plant-by-plant interval mapping, an approach is needed that can be more bold in the determination of location and map order than distance. The principle of our approach is that, for alleles of RFLP loci more and more closely linked with the target locus (M/m), two changes occur in their average distribution: In dominant individuals (MM and Mm) they change from a 1:1 ratio (independence) to 2:1 (complete linkage), and in recessive individuals from 1:1 to 0:1. In pooled samples, reduction or disappearance of one of two bands in the recessive class, accompanied by its enhancement in the dominant class, evidences linkage and has its own internal control.

Resolution of autoradiograms in our experience generally permits easy discrimination of 2:1 from 1:1, and dilution series indicate that decrements of a band down to 9:1 or less can be distinguished clearly from more-equal proportions or from 10:0. Among 10 pooled recessives containing 20 strands, 5% recombination will average one recombinant strand per pool (in a series of pools: 0, 1, 2...), so this resolution becomes dependent upon the "disappearance point" for the particular band.

Our specific plan at this time, subject to refinements, is to pool 2 samples of 10 dominant individuals each and 3 samples of 10 recessive individuals each, prepare DNA from each of the 5 pooled samples, and digest with 4 restriction enzymes. The 20 samples, plus markers, can then be loaded to produce as many as 8 blots in one casting and one run, sufficient for up to 80 probings. For loci that already have been placed to chromosome arm (e.g., within 50 cM), 50-80 probings potentially could define the position of the target gene with a precision finer than 1cM (limited only by the size of population and the "disappearance point"). For loci that have not yet been placed, 60 probings followed by 2-3 increasingly focussed probings potentially can define the position within 10cM or less (genome size estimated to be 2300cM per D. Grant and W. Beavis, personal communication). We consider these estimates of precision to be conservative. Pooled-sample interval mapping, if consistently successful, could establish a new and generally applicable technique for rapid and efficient mapping of mutants.

Among the considerations to be weighed for the pooled-sample approach are (1) limitations for particular classes of mutants and (2) equivalence of tissue representation in the pool. Mutants expressed in kernels can be mapped if they will germinate, simply by growing the two classes and pooling seedlings (heterofertilization, averaging 1%, will reduce precision only to that extent). Mutants that are

inviable and will not germinate (e.g., defective or germless kernels, both of which are very common) can be mapped by growing the normal class and evaluating pools for 2:1 ratio vs. 1:1 ratio; densitometry readings will be tested as a method for refining the precision of this approach. The inviable class of mutants is particularly important, because these represent losses of essential, cell-limited functions required in the development and survival of the zygote and developing endosperm, in contrast to ubiquitous housekeeping functions, which will not generally survive the gametophyte screen (the latter, however, will be represented among cDNAs). Because band ratios have internal controls through comparison of dominants with recessives, modest precautions will be sufficient to ensure that tissue from each seedling represents approximate equivalence (i.e., not so little as one-half relative to the others in the pool). It should be noted again in this context that the intent of most of our present interval-mapping tests is not so much to determine distance of a target gene from the markers as to determine order of the loci, even if only approximately.

We would be pleased to have comments or suggestions from Cooperators on this approach to mapping of NEPs.

Database and network for genome data

--Ed Coe

A prototype for a database on maize is being developed as part of the recently authorized and funded Plant Genome Initiative. Dr. Jerome P. Miksche, Director of the Office of Plant Genome Mapping with USDA-ARS, has asked me to develop a proposal to plan and to initiate this prototype. One of the targets of the PGI is development of database and network systems for genetic data, analysis of data, and linked access to sequences, clones, biosynthetic pathways, and the like, for crop plant species. Prototypes are being defined that will include selected monocot species (i.e., maize), a dicot (soybean), and a gymnosperm (loblolly pine), to be developed in coordination among the species insofar as possible and with mutually effective components. Of course we expect to benefit from substantial parts of the structure and rationale of the effort in humans and in microbial species, especially *E. coli*. Following is a brief sketch of the maize prototype project I have proposed, following discussions on short turnaround with a number of Cooperators. Your comments, suggestions, and advice will be appreciated.

Objectives: Define data and resources needed in the database, assemble first-priority components, and implement trials of accessing and usage, by September, 1992.

Approach: (1) Examine database strategies, analyses, and software that have been developed for other genome initiatives, including those for human and for bacterial genetics, and for other comparable programs (e.g., biotech and seed industry); (2) Evaluate adaptability of existing products for this project; and (3) Initiate programming modifications and new software needed for mapping, searching, pedigreeing, and information accessing; (4) Develop, initiate, and enhance an on-line database and interfaced network that provides access to current resources and data on the genetics of maize and is readily available to research scientists; (5) Ensure that genetic stock materials (variant strains; intensively characterized populations) and molecular materials (clones; probes) are documented in the database and that the materials are maintained with backup reserves. Solicit from specialists (1) identification and prioritization of user needs; (2) standardizations and defining of data resource structures (example: QTL data); (3) releases of data and scheduled updating; (4) surveys and inventories and compilations of key data; (5) accomplishment of essential cyological, morphological, molecular, and physiological characterizations; (6) essential laboratory analyses; and (7) derivation of key stock materials.

A preliminary outline of the primary components that should be available for access via the prototype:

Variants and reference points
(Mutations & variations; loci & alleles; translocations, inversions, deficiencies; centromeres & telomeres; knobs, etc.; QTLs)
Descriptions
Expression, modifiers, regulation, biochemistry
Mapping Data
Strains and Stocks
Probes and Clones
References
Utilities

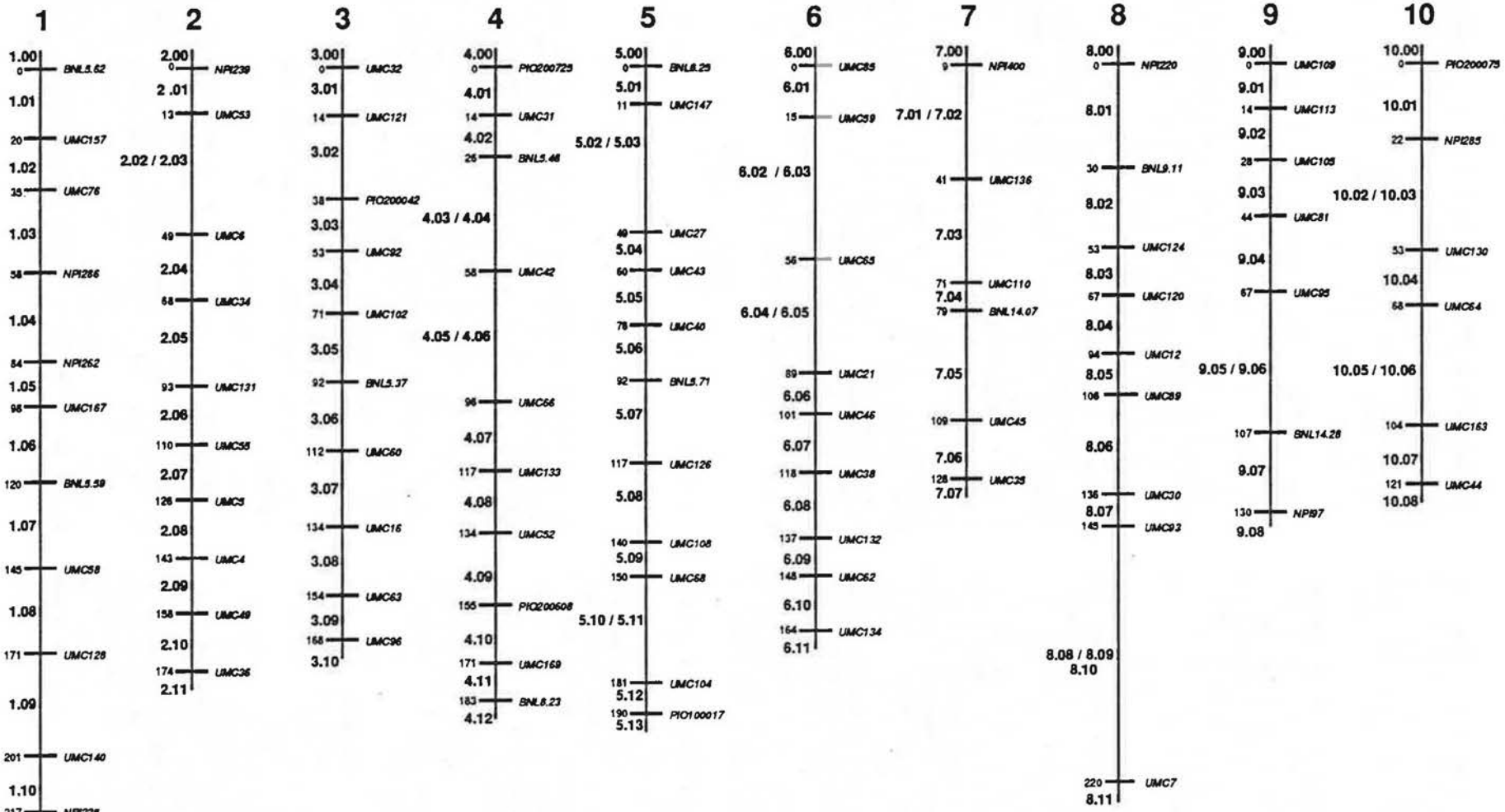
A set of core RFLP markers for maize

--Jack Gardiner, E.H. Coe, Susan Melia-Hancock, D.A. Hoisington, and Shiaoman Chao

Maize researchers are fortunate in having not one but several well developed RFLP linkage maps. Many of these maps have numerous markers in common, which allows an approximate correlation of the various public and private sector maps. Still there is not a complete correlation between maps and choosing the proper RFLP probes to flank a trait of interest is often a trial and error process dependent upon finding a suitable probe-enzyme combination. Clearly, a standardized set of chosen RFLP markers would be a useful starting point for any maize researcher wanting to map a particular trait. In this article, we would like to propose a set of core markers that define a series of bins and a core map.

To establish materials for a core map, an immortalized Tx303/CO159 F2 population consisting of 56 individuals was developed. This was accomplished by planting 40 F3 seeds from each of 56 F2 ears in two 20-plant rows, each planted with half delayed 7 days. Pollen from 5 F3 plants in one row was bulked in one pollination cycle and used to pollinate 5 F3 plants in the other row. Using this approach for two or more pollination cycles, as many as possible of the 40 F2 ears were pollinated. This procedure allows the heterozygosity of the 56 individual F2 ears to be maintained, minimizing drift and selection, while at the same time allowing the production of large amounts of seed. A minimum of 20 ears were bulked in order to recapture the heterozygous constitution of the F2. Using this procedure only 40 of the 56 F2 families are complete, due to localized poor stands in 1990. The remaining 16 families will be completed in the summer of 1991. Limited distribution of

UMC MAIZE RFLP CORE MAP JANUARY 1991



seed will be available to anyone interested in doing their own RFLP mapping. All that one need do to recapture the heterozygosity of the F2 mapping population is to plant out 10 or more seeds from each family - by bulking samples at the leaf collection stage it is only necessary to isolate 56 DNA samples.

A complete UMC RFLP map consisting of 187 markers was generated on a SUN 4.0 workstation using Mapmaker V. 2.0, the genetic mapping program developed by Eric Lander that uses maximum likelihood equations to generate a multipoint map. A maximum allowable recombination fraction of 0.4 and a LOD score of 3.0 were used.

In the present immortalized Tx/Co F2 population, of the 187 RFLP and isozyme markers that have been mapped, 94 were selected as core markers (please see the accompanying map). The selection criteria were as follows. Ideally, a core marker should be a single copy probe which gives an easily interpretable pattern and is polymorphic across a variety of inbred lines. For the most part, core markers fulfill this requirement but a few represent the only probe available in a sparsely marked region of the genome. Secondly, a strong preference has been given to those probes that are widely used and are available to researchers in both the public and private sectors. Finally, core markers were chosen to be spaced no more than 30cM (22% recombination) apart, with an optimal spacing of 15-20cM. In 13 cases, lack of a suitable core marker that fit the above criteria necessitated regions of greater than 30cM to be defined between a pair of core markers. It is anticipated that over the next year these regions will be subdivided by core markers, which are in the process of being determined.

Even with our best refinement of the data, a few orders must be considered uncertain because of low LOD scores (i.e., data are not definitive). Specifically the end of chromosome 1L is uncertain and we have chosen the order on the basis of previous work and consensus with other maps.

The designation of 94 core markers in addition to the 13 to be designated allows the maize genome to be divided into a series of 116 "bins". We appreciate the suggestions and encouragement of Tim Helentjaris toward this approach. Each bin is defined by a pair of RFLP markers. For example, bin 1.01 is defined by *BNL5.62* and *UMC157*. Bins are numbered according to the chromosome on which they reside, with further subdivisions following the decimal point being created by numbering sequentially from the most distal short arm core marker to the most distal long arm core marker. As stated above, 13 regions spanned a distance greater than 30cM. These bins will be subdivided upon finding a suitable core marker that bisects this region. This bin system has the advantage that any genetic trait can be localized to a small region of the genome by using a limited number of agreed-upon markers. Once localized to a particular bin, the bin number serves as a computer sortable tag which will allow all markers in the same bin to be identified, should more precise map order within a bin be desired. The other advantage of a bin system for collection, organization and retrieval of genetic information is that it is expandable and allows bins to be infinitely subdividable. For example, bin 6.10, defined by *UMC62* and *UMC134*, can be subdivided into 9 computer sortable sub-bins by appending 0.1 - 0.9 to 6.10 to define bins 6.10.1 - 6.10.9. In the future this will become a necessity as more RFLP probes are mapped.

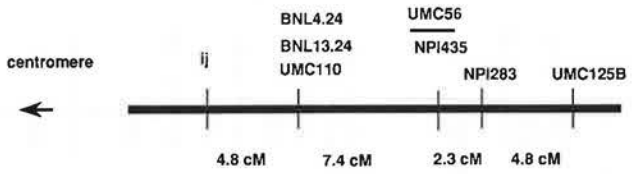
We look forward to receiving comments or suggestions on the Core Map and the utility of "bins".

Mapping of the *ij* (iojap) locus with RFLP markers

--Chang-deok Han[1] and Ed Coe

[1]present address: Cold Spring Harbor Laboratory

As part of experiments for the transposon tagging of *ij*, the RFLP (Restriction Fragment Length Polymorphism) markers on the long arm of chromosome 7 were examined to obtain molecular markers linked with the *ij* locus. The markers located on the long arm of chromosome 7 were obtained from D. Hoisington (University of Missouri), B. Burr (Brookhaven National Lab.), and T. Helentjaris (Native Plants Incorporated). The following RFLP markers were used for this project; *UMC56, UMC110, UMC125B, BNL13.24, BNL4.24, NPI435,* and *NPI283*. The *ij* seedlings were examined among F2 progeny of the cross between Oh51a inbred and *ij* in Mo17 inbred background. Around 50 *ij* F2 seedlings were examined. There were limited amounts of DNA because many of the *ij* seedlings die at the 2-3 leaf stage. Because of the shortage of DNA, not all the seedlings were tested by the same probes. With the help of D. Hoisington, the RFLP markers were mapped with the computer-aided maximum likelihood program, Mapmaker. The figure



shows the calculated linkage map for *ij*. All the iojap seedlings we examined with markers *BNL13.24* and *BNL4.24* showed the same patterns of polymorphism as with *UMC110*. This indicates that the population we worked on was not large enough to detect recombination among the three loci. We did not examine the possibility whether either of the markers, *BNL13.24* or *BNL4.24*, is homologous to *UMC110*. The DNA marker closest to *ij* is *UMC110*, 4.8cM proximal to the gene.

Compatible maize parents for maize x *Tripsacum* hybridization employing a simplified crossing technique

--B. Kindiger and J. B. Beckett

Identification of maize stocks that produce viable seed when pollinated by *Tripsacum dactyloides* L. (L.) should facilitate the transfer of useful *Tripsacum* germplasm into maize. To this end, a large number of dent stocks, inbred and open pollinated popcorn stocks, popcorn hybrids and popcorn x dent derivatives were pollinated by several diploid and tetraploid *T. dactyloides* accessions. After preliminary tests, all crosses were made by applying

Tripsacum pollen to maize silks in the manner normally used in maize x maize pollinations. However, cutting back was delayed until the silks near the tip of the cob were receptive. In addition, ears were cut back far enough to expose the tip of the cob. The modified technique is not only more efficient but also greatly reduces damage from fungi and insects. Typically, four or five pollinations, utilizing pollen from several 2n and 4n *Tripsacum* sources, were made in order to rate the quality of hybrid seed produced by each maize stock.

Ears that had 50 or more hybrid kernels with plump endosperm were rated excellent. Ears with plump endosperm but fewer than 50 kernels were rated good. Ears showing less well-developed hybrid seed were rated fair, and ears that failed to produce obvious hybrid seed (because of early abortion) were rated poor.

The table lists the crosses that gave good to excellent hybrid seed development.

Maize Parent	Description	Kernel quality
Popcorn and popcorn-related stocks		
SG1533	Population	Excellent x 4n *Tripsacum*
SA194	Population	Excellent x 4n
Germplasm source #1	Population	Excellent x 4n
PI222648	Population	Excellent x 4n, good x 2n
Germplasm source #2	Population	Excellent x 4n
311254	Experimental	Excellent x 4n
311255	Experimental	Excellent x 4n
HP303W	Hybrid	Excellent x 4n
Sg32	Inbred	Good x 4n
Sg16	Inbred	Good x 2n & 4n
P608	Hybrid	Good x 4n
IaDS53	Hybrid	Good x 4n
PI186206	Population	Good x 2n & 4n
White Cloud	Hybrid	Good x 2n & 4n
IADS28	Hybrid	Good x 4n
Ladyfinger Pop	Population	Good x 4n
Cornbelt material		
W23*	Inbred	Good x 2n and 4n
W23 x Kys	Hybrid	Good x 4n
K55 x W23	Hybrid	Good x 2n and 4n
L289 x W23	Hybrid	Excellent x 4n; good x 2n
L289 x N6	Hybrid	Good x 4n

*Other tests indicate poorer results in some years

Selected kernels from five popcorn stocks, one popcorn x dent derivative (Germplasm source #2) and two Midwest dent stocks were tested for viability. Complete results are not presented here, but germination ranged from 20% to 96%; six crosses gave at least 50% germination.

In general, it appears that the successful production of viable hybrid kernels depends primarily on the maize stock used. Within ploidy levels, differences between *Tripsacum* sources were not detected. As established many years ago, 2n maize is much more compatible with 4n *Tripsacum* than with 2n *Tripsacum*.

Two new B-A translocations involving the long arm of chromosome 4

-J. B. Beckett

Several years ago, an effort was made to generate new B-A translocations on the short arm of chromosome 6. In order to use Bor-Yaw Lin's procedure (MNL 46:193), a recessive endosperm factor was required on the short arm of chromosome 6. Because such factors were not available at that time, the reciprocal translocation T4-6(003-16) was employed to add a portion of the long arm of chromosome 4 onto 6S so that *c2* could be used as the endosperm factor.

Immature tassels of a stock carrying T4-6(003-16), *R-scm* and several B chromosomes were X-rayed when regions with microspores at or just beyond the first division could be located. About a week later, when pollen from the appropriate regions began to shed, it was applied to silks of a *c2* stock. The occasional colorless kernels, often with colorless scutella, were planted out and 87 individuals were testcrossed onto ears of a *c2* stock. Two likely and four possible B-A translocations were identified. The two likely translocations were confirmed this summer.

One stock appears to carry a simple B-A translocation, hereby designated TB-4Lh. The only individual checked for pollen fertility had 5% aborted pollen. Because only colorless kernels with colored scutella had been planted, all plants should have been hyperploid heterozygotes, which often have little pollen abortion. Several testcrosses onto a hybrid tester stock carrying *c2*, *dp1* and *R-scm* yielded ears segregating nearly normal-sized colorless kernels with colored scutella. In the sandbench, the colored kernels yielded progenies that segregated distal pale seedlings. Thus, the presence of a B-A translocation appears to be virtually confirmed, as the male parents did not carry *dp1*.

The second translocation, here temporarily designated TB-4L13474, appears to be compound rather than simple. Colorless kernels with colored scutella were planted. The only plant examined had approximately 60% aborted pollen, which is my usual estimate of pollen abortion in hyperploid compound translocation heterozygotes. Several plants were testcrossed onto the same hybrid tester stock mentioned in the preceding paragraph. The resulting ears segregated large and small kernels. Large colorless kernels had colorless scutella; small colorless kernels had colored scutella. In the sandbench, kernels from testcrosses of four plants were tested. The large colored kernels gave only normal seedlings at first, but after another day tiny narrow-leaved seedlings emerged, most of which displayed the distal pale phenotype. As with TB-4Lh above, the appearance of distal pale seedlings in testcross progenies virtually confirms the presence of a B-A translocation.

Although X-rays generate a variety of chromosomal aberrations, it is likely that TB-4L13474 is a compound involving the short arm of chromosome 6 and the the long arm of chromosome 4. If so, plants grown from the tiny colorless kernels with colored scutella should be hyperploid heterozygotes of the following constitution: 4, 6, 6-B, B-6S-4L, B-6S-4L. The appropriate designation for the translocation would then be TB-6Se-4L003-16. If the translocated segment of 4L can be removed by crossing over, the resulting simple B-A translocation should be designated TB-6Se. Testcrosses involving *rgd* and other likely 6S genes will be made in the greenhouse this winter in order to determine whether the translocation does indeed involve 6S.

Chromosome arm dosage analysis - Identification of potential QTLs on the short arm of chromosome 5

--- E.A. Lee, D.R. Baxter, L.L. Darrah and E.H. Coe, Jr.

Considerable effort is now underway to identify and mark chromosome segments (QTLs) associated with quantitative traits. The approach being used involves mapping QTLs using isozymes and restriction fragment length polymorphisms (RFLPs). An alternative to this approach is to examine the phenotypic changes in a standard hybrid background when specific chromosome arms are either added or lost (dosage effects). The approach is based on the expectation that dosage effects and differential effects between parental inbred lines can be used to identify chromosome segments influencing quantitatively inherited characters. The results presented below are from a pilot experiment examining chromosome arm dosage effects in order to identify chromosome segments (QTLs) associated with quantitatively inherited characters.

Mo17Ht and B73Ht (hereafter shown as Mo17 and B73) versions of TB-5Sc (Katsuta and Coe, MNL 64:48, 1990) were used to generate reciprocal F1 hyperploid and hypoploid hybrids. The aneuploid plants were evaluated in the field during the summer of 1990 at one location with four replications. The seed was planted in three-row plots with hyperploid and hypoploid plants segregating within the same plot. Identification of the aneuploid plants was done initially on the basis of phenotypic characters (plant height, leaf width, overall vigor, etc.). Putative hypoploid and hyperploid plants were further verified by examining percent pollen abortion. Hypoploid plants should have approximately 50% aborted pollen grains while hyperploid plants should have approximately 10 to 15% aborted pollen grains. Competition within the plots was adjusted by 'topping' the non-aneuploid plants in the plot. A normal F1 check plot was included in each replication to serve as a control. Observations were made on 10 competitive plants per plot except for the following characters, which were evaluated on a plot basis among classified types of plants: days to silk, days to anthesis, and pollen-silk interval.

Loss or gain of doses of 5S affected days to silk in all cases (Table 1). Plants having only the Mo17 5S arm also were significantly affected for number of days to anthesis. The interval between anthesis and silk emergence (pollen-silk intv.) was greatly affected by loss of 5S or by the gain of 5S coming from the Mo17 source. Differential effects between B73 and Mo17 were observed in the hypoploids for pollen-silk intv., further suggesting that genes affecting nicking of silk emergence with anthesis are located on the short arm of chromosome 5. Dose of 5S was directly proportional to rind puncture resistance (a measure of stalk strength). Rind puncture resistance may be influenced by internode length or stalk circumference. Yet, stalk circumference exhibits the same dosage effect pattern that rind puncture resistance exhibits, while internode length does not exhibit the same dosage effect pattern. Loss or gain of 5S affected leaf length, and plant and ear height in all cases. Loss of 5S also affected tassel branch number, leaf width, and the number of leaves above the ear. Based on these observations, genes (QTLs) influencing silk emergence, stalk strength, stalk circumference, leaf length, tassel branch number, nicking of silk emergence with anthesis, and plant and ear height may be located on the short arm of chromosome 5.

Genetic materials have been prepared for a full-scale field study involving 18 of the 20 chromosome arms, which will be conducted during the summers of 1991 and 92.

Table 1. Pedigree means for 12 traits measured in a single environment for genotypes with varying doses of chromosome 5S.

	Pedigrees				
	Hypoploid		Hyperploid		F1 Check
Trait	B73xMo17†	Mo17xB73‡	B73xMo17§	Mo17xB73¶	B73xMo17
Days to silk	79.0 ab*	80.8 b	77.8 a	79.3 ab	72.5 c
Days to anthesis	72.0 b	76.5 a	74.8 b	77.5 a	72.3 b
Pollen-silk intv. (days)	-7.0 b	-4.3 a	-3.0 a	-1.8 c	-0.3 c
Rind puncture (load-Kg)	7.4a	8.6 a	15.1b	13.5 c	10.4 d
Stalk circum. (cm)	5.8 a	6.2 a	10.6 b	10.1 c	9.0 d
Internode length (cm)	14.5 a	15.9 b	15.0 a	15.8 b	18.3 c
Plant height (cm)	133.3 a	160.3 b	183.6 c	186.8 c	232.6 d
Ear height (cm)	43.1 a	73.6 b	89.0 c	94.4 c	113.0 d
Leaf width (cm)	7.9 a	6.1 a	12.2 b	11.9 b	10.6 b
Leaf length (cm)	65.7 a	73.3 b	75.2 bc	76.5 c	89.9 d
Tassel branch no.	4.5 a	5.9 a	9.2 b	10.2 b	9.3 b
Leaf no. above ear	4.5 a	4.9 b	5.5 c	5.5 c	5.6 c

* Means with the same letter(s) within a row are not significantly different from one another at the 0.05 probability level.

† Cross having a single-dose of chromosome 5S from B73.

‡ Cross having a single-dose of chromosome 5S from Mo17.

§ Cross having three doses of chromosome 5S, two from Mo17.

¶ Cross having three doses of chromosome 5S, two from B73.

Parker's flint contains *orp1*

--Allen D. Wright

The orange pericarp phenotype requires the duplicate homozygous recessive loci *orp1* and *orp2*. A cross was made of Parker's flint by a pollen source heterozygous *orp1* and *su* (in repulsion) and homozygous *orp2*. Approximately 50% of the selfs of this progeny segregated 3 normal to 1 orange, instead of the expected 15:1 segregation, indicating that Parker's flint contained one of the *orp* genes. The other selfs did not segregate for orange pericarp, but segregated for sugary kernels, indicating that the *orp* gene in Parker's flint is *orp1*.

We had previously determined that *orp1* was present in the original stock that was mutagenized to produce orange pericarp. Longfellow Flint was one of the lines used to produce this stock. The testing of several lines for *orp1* is currently underway. Appropriate tester material will be provided to anyone interested.

Whether the presence of *orp1* has adaptive significance for Parker's Flint and related Northern Flints, or is simply something that it can live with is not at present clear.

COLUMBIA, MISSOURI
University of Missouri
BELTSVILLE, MARYLAND
USDA Plant Hormone Laboratory

Tryptophan not required for IAA biosynthesis?

--Allen D. Wright, M. G. Neuffer, Michael B. Sampson, Jerry D. Cohen, Lech Michalzcuk and Janet P. Slovin

Tryptophan is generally assumed to be the precursor to the plant auxin indole-3-acetic acid (IAA), although other pathways have been postulated. The indole-accumulating mutant, orange pericarp, lacks tryptophan synthase activity and thus should be a good system for testing the hypothesis that IAA is derived from tryptophan. Embryos were excised from developing kernels, grown on MS media for 10 days under aseptic conditions and analyzed for IAA using a procedure which involved column cleanup, HPLC, derivitization, and GC-MS using 13C6-IAA as an internal standard (Cohen, Baldi and Slovin, Plant Physiol. 80:14-19). Surprisingly, IAA levels in the mutant were about 50 times greater than those of the non-mutant, implying that tryptophan is not a necessary precursor to IAA in maize. Addition of tryptophan to the media did not alter these results. In another study, mutants grown in 30% D-2O were found (by mass spectrometry) to have incorporated deuterium into stable ring positions of IAA, indicating de novo synthesis had occurred. The evidence indicates that IAA can be made without passing through tryptophan.

DAVIS, CALIFORNIA
University of California
STANFORD, CALIFORNIA
Stanford University

Products of *Mu* insertion and excision at the bronze-1 gene

--Anne Bagg Britt and Virginia Walbot

Last year we reported the position of the *bz-mu1* and *bz-mu2* insertion sites, as well as the sequence of several somatic excision products of *Mu1* from *bz-mu1*. Here we report the sequence of two germinal revertants of *Mu1* from *bz-mu1*, and revise our report of the *bz-mu2* insertion site.

We recovered two independent germinal revertants of *bz-mu1*. Both were isolated from *bz-mu1* homozygous plants which produced ear sectors segregating for revertant (purple) kernels. *Bz1-R1* was isolated from a self-pollinated ear, while the ear which produced *Bz1-R2* was crossed by *bz* tester. DNA isolated from each of these germinal revertants was employed as a template for PCR amplification of the *bz-mu1* target site. The resulting products were cloned and sequenced. Because the sequence of the cloned product for *Bz1-R1* was identical to that of a previously cloned somatic excision product, the amplification, cloning, and sequencing procedures were performed a second time, and yielded identical results.

The sequences of these two germinal revertants are as follows:

bz-mu1...GCCG**CAAACAGGG(MU1)CAAACAGGGTG**GACG...
Bz1-R1 ...GCCG**CAAACCCAAACAGGGTG**GACG...
Bz2-R2 ...GCCGACG...

Like the two germinal revertants of *bz-rcy* sequenced by Schnable, Peterson, and Saedler (MGG 217:459, 1989), these revertant alleles are the result of imprecise excision of *Mu1*. *Bz1-R1* carries a deletion of the four bases 5' to the *Mu* target site, as well as a single base insertion 3' of the target site. *Bz1-R2* includes a deletion of the entire 9 bp repeat, as well as an additional 3 bp deletion 3' of the original *Mu* insertion. Unlike the two revertant alleles sequenced by Schnable et al., both of these excision products restore the original *Bz-W22* reading frame.

In order to determine the points of insertion of the *Mu2* element in *bz-mu2*, PCR was used to amplify the *Mu* ends and their contiguous *Bz* sequences at both the 5' and 3' ends of each insertion. We found that the amplification protocol recommended by Perkin-Elmer Cetus produced a very nonspecific product in this region of the *Bz1* gene. We therefore modified our regular three step cycling procedure (1 min. at 94°, 1 min. at 55°, 1 min. at 74° 30 reps) to a two step procedure recommended to us by Tom Sullivan (1 min. at 94°, 1 min. at 74° 30 reps.). This modified reaction produced a very specific product. The amplified fragments were then cloned and sequenced. The sequence of the 9 bp repeat of *bz-mu2* is GCCCAACTG.

This sequence is located just 3' of the *Xho*II site in *Bz1*. Previously published Southern blot analysis had indicated that the *Mu2* element had inserted 5' of the *Xho* II site. Last year we published an estimation of the position of the insertion site based on Southern blot data which indicated that the element had inserted, paradoxically, 5' of the *Xho* II (AGATCC) site, but 3' of the *Sau*3a (GATC) site located within that *Xho*II site. The sequence reported above, however, indicates that the insertion is located 3' of both the *Xho*II and the *Sau*3a sites.

Why then did the digests reported by Taylor and Walbot (Genetics 117:297, 1987) indicate that the element was positioned 3' to the *Xho*II site? *Xho*II is sensitive to methylation, and will not digest sites at which C is methylated in the 5' position. The *Xho*II site in *Bz1* (**GAGATCCG**) includes two C's which are susceptible to methylation. If this site was methylated in the DNA preparation analyzed by Taylor and Walbot, their results (but not their conclusions) would be consistent with the sequence presented here.

DEFIANCE, OHIO
Defiance College

Paramutation: a tassel mosaic controlled by temperature

--Bernard C. Mikula

In MNL 64 we reported differences in the level of paramutation of an *R-g* allele received from Native Seeds/SEARCH resulted from temperature conditions administered to *R R-lst* seedlings during the first three

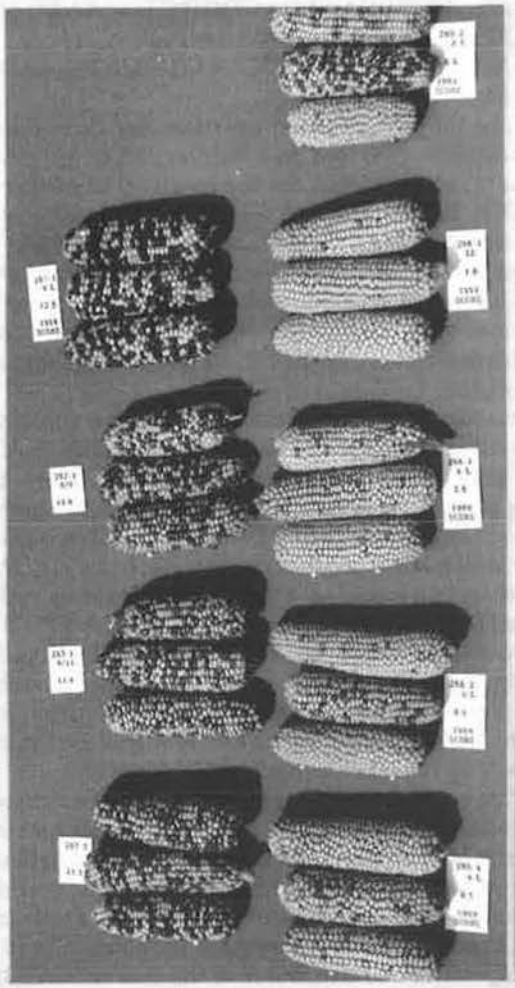


Figure 1

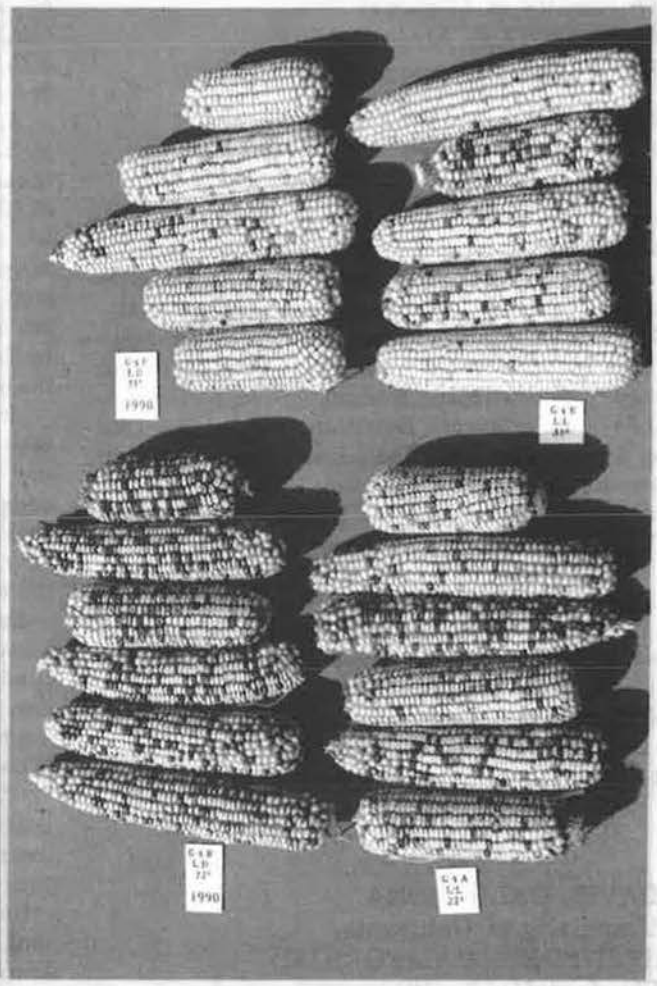


Figure 3

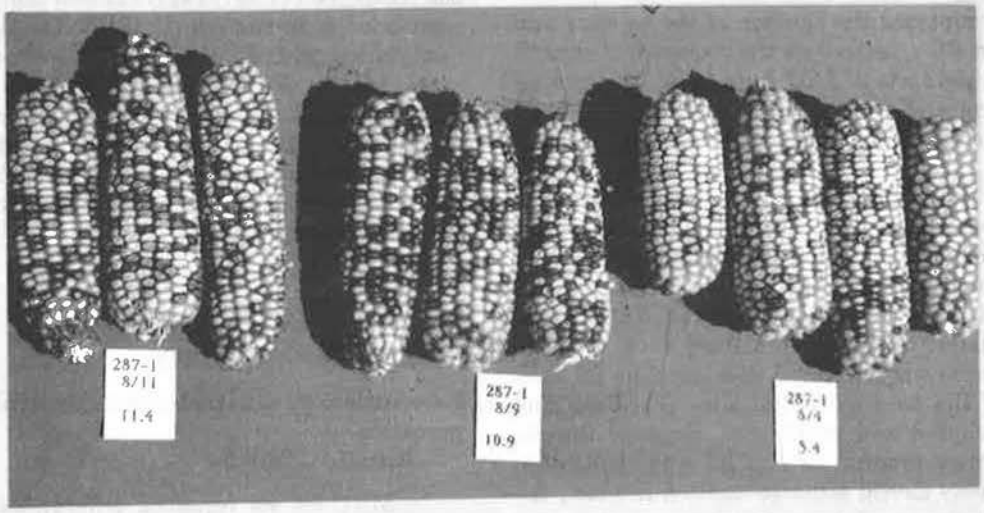


Figure 2

weeks of development. Temperatures of 28 C produced plants whose *R*-expressions in testcrosses were significantly lighter than those from plants grown at 22 C for the same period. Seeds of 1989 testcrosses were grown out and testcrossed in 1990 to test for heritability. Fig. 1 shows that the relative differences of the previous year are still apparent in the testcrosses of 1990. Four sets of testcrosses on the left of Fig. 1 represent four different testcross ears from 1989 which scored 10 or above for level of pigmentation (on a scale of 0 to 20). Five sets of testcrosses on the right represent five testcrosses from 1989 which scored 1.0 or less for paramutated *R*-gene expression.

When *R R-lst* plants are testcrossed over the seven days that pollen is shed, a tassel mosaic for paramutated *R*-gene expression can be noted. Seeds from each of the seven testcrosses of the previous year were grown out and testcrossed for heritability of the tassel mosaic; differences can still be observed in paramutated *R*-gene expression (Figure 2). The three ears on the right are from testcrosses from the earliest pollen collection (8/4); the two sets of three ears to the left represent collections made five and seven days later (8/9 and 8/11). Tassel mosaics were more readily observed with the new *R*-genes from the Southwest than from the more highly inbred *R*-genes in W22 background which often tend to be "subliminal" and require tedious scoring procedures.

Does inbreeding reduce the range of tassel mosaicism? In the early days of getting the paramutation phenomenon accepted, it was essential to set aside the arguments for segregating modifiers. The climate for accepting "unstable" genes within the canons of orderly Mendelian behavior demanded an inbred background if paramutation was to be taken seriously. Our results over the past few years seem to be telling us that "unorthodox" genetic behaviors will require careful attention to how conventional procedures may inhibit the investigations of some of the more challenging questions facing genetics. "Inbred" habits are hard to overcome!

Four new *R-g* alleles from Native Seeds/SEARCH were made heterozygous with an *R-lst* allele received from the Maize Coop. When plants from *R R-lst* seedlings, grown at 31 C or 22 C for the first three weeks, are testcrossed a clearly visible difference in the level of paramutation is noted. Fig. 3 shows an example of how one of the alleles responded to these temperature differences to which seedlings were exposed. The bottom half of Figure 3 shows testcrosses of plants whose seedlings received 22 C; the upper half of the photo shows testcrosses of those plants whose seedlings received 31 C. All of the other three alleles tested show similar differences. It must be pointed out that all male-transmitted differences similar to those shown here have been found to be heritable. Heritable responses of a gene to temperature at a critical stage in development may be considered a case of transgenerational, programmable, genetic modification. Jacob summarized the 20th Century dogma when he said the genetic "program does not learn from experience". Is it possible that with this paramutation evidence the dogma can be challenged, using "unstable alleles" such as *R* and *R-st*? The transacting paramutagenic alleles are probably but one example of the many two-element transacting systems whose utility for discussions of the most interesting and fundamental biological questions must still be determined.

Before passing judgment on Jacobian dogma, we would advise caution. It must be pointed out as did Rhoades when he first described a two element system in 1941, there are some who would consider unstable genes "sick". The "sick genes", occupying the attention of genetics the last half of this century, may, however, provide the therapy essential for initiating 21st Century genetics!

EAST LANSING, MICHIGAN
Michigan State University

Metabolism of the host-selective phytotoxin HC-toxin

--Robert B. Meeley and Jonathan D. Walton

The maize pathogen *Cochliobolus* (*Helminthosporium*) *carbonum* race 1 is highly virulent on maize that is homozygous recessive at the *Hm1* locus, which is on the long arm of chromosome 1. This virulence is due to production by race 1 (but not other races) of a cyclic tetrapeptide known as HC-toxin (MNL 57:53-54). HC-toxin contains an unusual amino acid, L-2-amino-8-oxo-9,10-epoxidecanoic acid; both the epoxide and the vicinal ketone are required for activity. To test the hypothesis that the dominant resistance to HC-toxin controlled by *Hm1* is due to metabolism of the toxin, especially at the labile epoxide group, we prepared radiolabelled HC-toxin by feeding the fungus tritiated D-alanine. In leaf uptake studies, HC-toxin is metabolized to a single product, which was purified and analyzed by HPLC, TLC, and mass spectrometry. Surprisingly, the metabolite retains an intact epoxide but the vicinal ketone is reduced to the corresponding alcohol. In a time course study, no detectable difference between the ability of resistant (*Hm1/hm1*) and susceptible (*hm1/hm1*) maize leaves to metabolize the toxin was found. The conversion of HC-toxin to the 8-alcohol occurs also in vitro: the activity is sensitive to boiling and protease treatment and uses NADPH as co-substrate. The enzyme has an apparent molecular weight of 42,000 by gel filtration, similar to carbonyl reductases found in other plants. Studies in progress on the kinetic characteristics of this enzyme are addressing its role in host-selective reaction to HC-toxin and its relation to the *Hm1* locus.

FREIBURG, GERMANY
University of Freiburg

Tissue-specific differences of maize HMG proteins

--Klaus D. Grasser and Günter Feix

High mobility group (HMG) proteins represent a class of small (Mr 10,000-30,000) nonhistone chromosomal proteins which have been isolated from a variety of eukaryotic organisms including several plants. They have been shown to be preferentially associated with transcriptionally active chromatin and, in the case of maize and soybean, to bind to A/T-rich stretches of duplex DNA (Maier et al., Mol. Gen. Genet. 221:164-170, 1990; Jacobsen et al.,

Plant Cell 2:85-94, 1990). Maize HMG proteins, furthermore, exhibit a specific binding to CCAAT- and TATA-boxes of the P2 promoter of the zein gene pMS1 (Grasser et al., J. Biol. Chem. 265:4185-4188, 1990).

In continuation of this finding we were interested to see whether differences in the HMG protein pattern could be detected between different maize tissues and developmental stages. As shown in the figure, clear differences can be

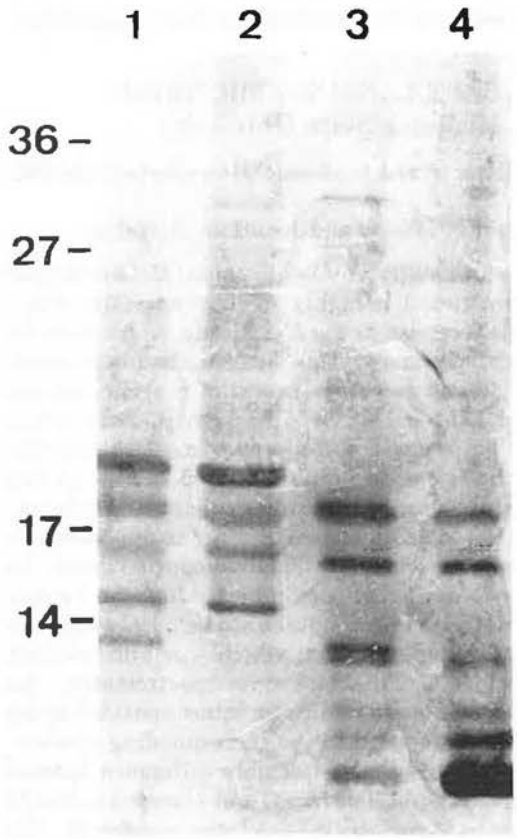


Figure. Silver stained protein pattern of SDS-PAGE separated HMG proteins prepared from 7 and 14 dap endosperm tissue (lanes 1 and 2 respectively), from seedlings (lane 3) or 60 day old leaves (lane 4). Numbers on the left indicate molecular weight markers in kDa.

observed between HMG proteins isolated from nuclei of endosperm and leaf tissue (especially with respect to the smaller proteins visible only in leaf tissue), while only slight differences exist between the developmental stages tested. Additional evidence for differences comes from phosphorylation experiments with a casein type II protein kinase activity from endosperm nuclei (Grasser et al., Biochem. Biophys. Res. Commun. 162:456-463, 1989) which phosphorylates all the major HMG proteins from endosperm, as well as the HMG proteins of the corresponding sizes from seedling tissue, but not the smaller proteins which are unique to leaf extracts. The HMG proteins of 60 day old leaves, however, can be phosphorylated only very weakly compared to HMG proteins of other tissues. Differences were also evident in western blots with an antiserum against the small proteins from seedling tissue which does not cross-react with any HMG protein from the endosperm preparation. However, antisera against the two larger HMG proteins, both from endosperm or leaf tissue, exhibit strong cross-reactions indicating a close immunological relation. Work is currently in progress to clone the genes coding for the HMG proteins of maize. Hopefully, this will allow a more detailed analysis of the function of the HMG proteins in gene regulation.

GAINESVILLE, FLORIDA
University of Florida

The sweet corn "Silver Queen" contains two genes conditioning white seed

--L. C. Hannah and D. R.. McCarty

For a number of years, we have used the sugary sweet corn 'Silver Queen' as a *su* tester. 'Silver Queen' is a white sweet corn which is quite popular in Florida. In our initial series of testcrosses, all resulting kernels were yellow, indicative of a recessive white being carried by 'Silver Queen'. However, during the past 6 to 7 years we have noted that testcross ears segregate approximately 1/2 yellow and 1/2 white. This occurred when several yellow lines were crossed onto 'Silver Queen'. These results are consistent with two hypotheses: (1) Silver Queen now contains two alleles of one locus. One allele is a dominant white while the other is a recessive white. (2) There are now two genes in this hybrid which condition a white seed. 'Silver Queen' is homozygous for a recessive white but heterozygous for a non-allelic dominant white.

To distinguish between these two possibilities, white plump seed were selected from a cross of yellow, *sh2* corn 'Florida Stay Sweet' by the *su* 'Silver Queen'. Plants were grown, self-pollinated and yellow plump seed were selected. Nine plants derived from such seed produced, upon self-pollination, four progenies that segregated for white seed. Were there one locus conditioning white seed in Silver Queen, selection in the original cross for white seed would also have selected against the presence of the recessive white allele being present in the F1 seed. Thus yellow seed, derived from the F2, should have been homozygous for the yellow allele at this locus and should not have produced white seed in subsequent generations. The finding of segregation in 4 of the 9 F3 progenies clearly rules out the "one gene-two white allele" hypothesis.

We conclude that there exist two genes conditioning loss of carotenoid pigmentation in 'Silver Queen'. The cultivar is homozygous for a recessive white and heterozygous for a non-allelic dominant white. If there is not linkage between the two loci, we would have expected 6 of the 9 progeny above to have segregated for white seed. The finding of 4 segregating progeny is consistent with very loose or no linkage between these two genes.

GAINESVILLE, FLORIDA
University of Florida and USDA/ARS

Mutation of the miniature 1 (*mn1*) locus is associated with loss of invertase activity

--Michael E. Miller and Prem S. Chourey

We have used histochemical methods of localizing in-

vertase activity on kernel sections as described by Doehlert and Felker (Physiol. Plant. 70:51-57, 1987), to screen large populations of immature kernels for possible mutations showing loss of invertase activity. Among many genotypes tested, including various endosperm starch mutants, only the miniature 1 (*mn1*) mutant (Lowe and Nelson, Genetics 31:525-533, 1946) was lacking invertase activity (Fig. 1). Further analyses included *Mn* and *mn* kernels segregating on the same F2 ear and again the mutants showed no detectable activity.

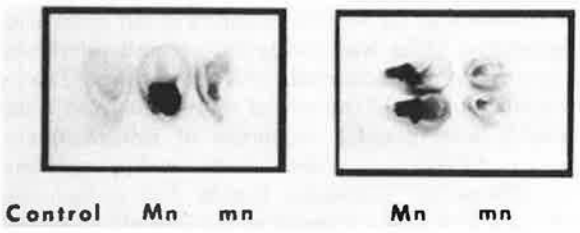


Figure 1. Kernels showing histochemical staining for invertase activity. Control = -sucrose, *Mn* and *mn* = + sucrose

Spectrophotometric assays for enzyme activity (Tsai et al., Plant Physiol. 46:299-306, 1970) on 12 DAP kernels, harvested from two different crops, confirmed earlier observations based on histochemical assays (Table 1). However, a low level of residual activity was consistently observed in the mutant kernels. Table 1 shows mean values

Table 1. Mean specific activity (μM glucose/mg/min) in 12 DAP kernels (pedical & lower 1/3 endosperm.

	Mn Mn		mn mn	
	soluble	bound	soluble	bound
Spring 1990	1.73	1.24	.016	.02
Fall 1990	2.10	--	.02	--

of specific activity derived from 3-5 independent extractions for each genotype. No detectable levels of invertase were found in the upper 2/3 portion of either *Mn* or *mn*. In miniature, invertase levels were highly reduced for both cell wall bound and soluble forms indicating that both forms may be due to a single gene. No invertase-inhibitor was exhibited by miniature over normal when predialyzed samples were combined, dialyzed overnight, and assayed. Specific activities of invertase remained as high in the mixed sample as those observed in the mixture of *Mn* with buffer. Kernels at the 12 DAP stage of development were used as a baseline for this study since invertase levels are known to peak at this stage (Tsai et al., 1970). Spectrophotometric assays on root extracts of *Mn* and *mn* showed no detectable differences in invertase activity. Further studies are in progress to determine biochemical, cellular, and molecular basis of loss of invertase activity in the *mn* genotype.

Variations in the structure and expression of shrunken alleles

--William K. Johnson and Prem S. Chourey

The shrunken (*sh1*) alleles from a total of 20 mutant lines (obtained mainly from O. Nelson and P. Peterson) of independent, spontaneous origin (*sh-Woodman* was provided by J. Woodman and derived from tissue culture) were analyzed at the DNA, RNA and sucrose synthase (SS) protein levels for variations in gene structure and expression. Approximately one-half of the mutant alleles appeared to be associated with transposable elements (*Ac/Ds* or *En/I*) and there was great variation among the restriction fragment patterns from all of the lines. Two of the alleles (*sh-bz-m4* and *sh-82-6466*) are gene deletions, since no hybridization was detected on Southern and Northern blots and no *Sh*-encoded (SS1) protein was found on Western blots for either line. Eight of the *sh* mutants expressed multiple transcripts (from 1.9 to 7.2 kb in size), but these were not necessarily correlated with probable (*-En/I* control, while the two alleles [*sh-76-3014* and the *sh* derivative of *Sh* revertant 1 from *sh-m5933* (provided by B. Burr)] that yielded evidence for *Ac/Ds* control each expressed single, but aberrant-sized transcripts (2.5 or 4.4 kb respectively).

In terms of tissue-specific RNA accumulation, nearly all of the *sh* alleles exhibited very low levels of steady-state transcripts in 22 DAP kernels, although *sh-79-4507* with wildtype sized (2.8 kb) transcripts and *sh-80-5142* with truncated (2.2 kb) transcripts accumulated *sh* RNA to near wildtype levels. A few other alleles (*sh-81-6302*, *sh-7107* and *sh-7731*) showed elevated kernel transcript levels compared to other *sh* mutants, but still low when compared to wildtype kernels. In seven-day-old seedling roots, the *sh* alleles expressed low levels of transcripts (usually about one-quarter of wildtype root levels) and anaerobic induction resulted in at least a five-fold increase in transcript levels in all cases. In general, anaerobically stressed roots provided the most abundant source of *sh* mRNA for Northern analysis.

In terms of protein accumulation, Western blots revealed a pattern that was very different from what was expected based on steady-state RNA levels. Only six *sh* mutants yielded detectable SS1 proteins (wildtype = 96 kD). The *sh-7342* mutant had nearly normal levels of SS1 protein (96 kD) in both induced and control roots, while it had almost undetectable protein levels in 22 DAP kernels. The other five mutants (*sh-80-5142*, 70 kD; *sh-76-3018*, 96 kD; *sh-7731*, 96 kD; *sh* deriv. of *Sh-rev1*, 90 and 92 kD; and *sh-79-4507*, 94 kD) showed low levels of SS1 protein in 22 DAP kernels (about 5% of wildtype kernel levels), but no readily detectable SS1 protein in induced or control roots (except for *sh-79-4507*, which had slightly more than the other four). In no case (*sh* or *Sh* genotype) was there a detectable increase in the amount of SS1 protein in anaerobically induced roots when compared to control roots of the same genotype.

These results seem to indicate that there is some mechanism that leads to the accumulation of SS1 protein in 22 DAP kernels. Also, the control of *sh* gene expression is generally post-transcriptional in nature, since there were many mutants that had abundant transcripts (wildtype sized and/or aberrant sized), but undetectable levels of SS1 protein. In the case of *sh-7107*, *sh* transcripts are found in polysomes, but no SS1 protein was detectable. Finally, it is also interesting to note that the only difference in *Sus* (codes for SS2 protein) RNA and protein size and accumu-

lation in the *sh* lines was found in the deletion mutant *sh-82-6466*. In this mutant, SDS-PAGE gels showed a normal-sized SS2 polypeptide, but native gels revealed a banding pattern suggestive of hetero-tetramerization among different SS2 subunits, indicating a duplication of the *Sus* locus as a possible explanation for these results.

HARLAN, IOWA
Orsan/Wilson Hybrids, Inc.

Development of a tertiary trisomic (A A B-A) stock carrying indeterminate gametophyte (*ig*)

--Bryan Kindiger

The *ig* gene, first identified by Kermicle in 1969, is known to be a simply inherited single recessive gene. This gene allows the development of androgenetic monoploids at a frequency of 1-3%.

Utilizing the TB-3Ld B-A translocation in a W23 *R-nj* background, a tertiary trisomic (A A B-A) and of the genetic constitution *ig ig* B-A(+) has been generated. The breakpoint position of the TB-3Ld translocation is apparently close enough to *ig* to disallow frequent crossing over in the region.

Classical maintenance of the gene in the heterozygous condition yields only 21% *ig/ig* individuals (Kermicle, Amer. J. Bot. 58:1-7, 1971). Since homozygous *ig* individuals are male sterile, it is impossible to maintain them in a homozygous condition. By having *ig* in a tertiary trisomic stock, this may allow 40-50% regeneration of the *ig/ig* genotype and successful maintenance of homozygosity by selfing.

Transmission of the B-3Ld chromosome is 50% through the female and about 2% through the pollen. Chromosome counts of 31 root tips showed 14 with 20 chromosomes, 14 with 20 + 1 B-A, 1 with 10 chromosomes and 2 with 20 chromosomes + 2 B-A's. By utilizing the tertiary trisomic stock as male, about 98% of the gametes should carry the *ig* gene.

Seed of this stock is being supplied to the Maize Genetics Cooperation Stock Center.

IRKUTSK, USSR
Siberian Inst. Plant Physiol. Biochem.

Oligonucleotide DNA-probe for detecting linear mitochondrial plasmids

--E. L. Tauson, S. I. Belicov, J. M. Konstantinov, V. P. Kumarev

The molecules of the linear plasmid-like DNAs (S1, S2, 2.3kb plasmid or S3) found as individual elements of mitochondrial genome of maize are structurally unique in that they contain terminal inverted repeats significant for their biological functions. There are 16bp of perfect homology between terminal inverted repeats (TIRs) of these linear plasmids, which probably are protein binding sites (Fig. 1) (Bedinger et al., MGG 205:206-212, 1986). The S episomes of maize constitute a unique group of replicons with a common origin. At the present time it has become obvious

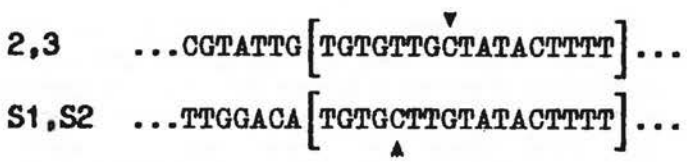


Figure 1. Nucleotide sequence comparison of S plasmids and 2.3kb plasmid. Differences are indicated by the arrow.

that the genetic functions of these plasmids are not limited only by their role in the cytoplasmic male sterility trait.

Radiolabeled DNA-probes with specific sequences are a useful instrument for identification of small quantities of corresponding DNA without isolation and purification. The application of specialized DNA probes should be helpful in investigations of the role of some structural zones of these additional genetic elements of mitochondria of maize. In addition such probes can be used in experiments on recombination processes inside the mitochondrial genome and probable exchange of the genetic material between organelles. In the present work we have obtained such a DNA probe for S plasmids of maize mitochondria.

A 16bp DNA probe that has a perfect homology with three linear plasmids (2.3; S1 and S2) and free of mismatched nucleotides was synthesized according to a method of Frochler and Mattenci (Tetrahedron Lett. 27:469-472, 1986). The first and the second strand of the fragment was arranged with *PstI* sites (Fig. 2), phosphorylated with T4-polynucleotide kinase, annealed and inserted into pBR322 by a standard method (Maniatis et al., Molecular Cloning, NY, 1982).

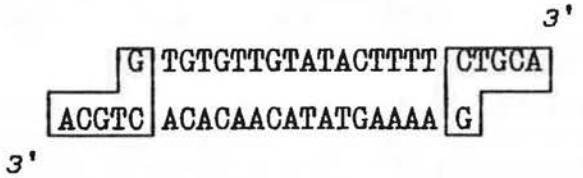


Figure 2. 16bp DNA sequence of synthetic oligonucleotide, homologous to the linear plasmids of maize mitochondria. *PstI* sites marked with boxes.

The recombinant DNA (pmtZET16) was used to transform *E. coli* ΔM15. Cells were plated on agar containing 15μg tetracycline/ml. Colonies were transferred on Whatman 542 and hybridized with [^{32}P] radiolabeled oligonucleotide probe. Colonies giving a strong signal were picked and transferred on other plates of LB-agar with tetracycline (15μg/ul) and LB-agar with tetracycline (15μg/ml) and LB-agar with tetracycline (15μg/ml) and ampicillin (50μg/ml). Colonies that were resistant to Tc, but sensitive to Ap, were selected. DNA was isolated from fresh cells of each clone, nick-translated and dot-hybridized with S2 plasmid-like DNA isolated and purified from mitochondria of etiolated seedlings of maize hybrid "Bekke LLO" (Fig. 3).

It can be concluded from the results of hybridization that recombinant plasmid pmtZET16 will be of use as a probe for recognizing corresponding DNAs. Moreover, it is very likely that localization of linear mitochondrial plasmids could facilitate the investigations of protein-binding sites in these DNAs.

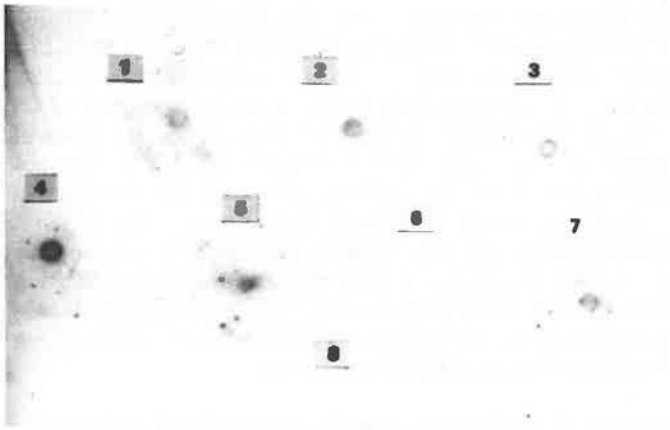


Figure 3. Hybridization of radiolabeled DNA of several pmtZET16 clones to S2 maize mitochondrial plasmid and pBR322; 1-5.7-clones with oligonucleotide insert; 8-pBR322.

The possibility that the same protein recognizes and binds the termini of all of the linear maize mitochondrial plasmids was proposed by Bedinger and coauthors (MGG 205:206-212, 1986). At present, however, it is unknown whether or not the 16bp fragment with a perfect homology within the TIRs of linear mitochondrial plasmids has functional significance in DNA-protein complex formation. The use of the cloned sequence in terms of the slight changes introduced into its synthesis, might be one of the ways to elucidate this problem.

JOHNSTON, IOWA
Pioneer Hi-Bred International, Inc.

Computerized two-dimensional electrophoretic protein profiles of 37 inbred lines and one hybrid line

--J. W. Higginbotham, J. S. C. Smith, and O. S. Smith

A dataset of protein spot densities obtained from 69 computerized two-dimensional (2-D) electrophoretic protein profiles of 37 inbred lines and one hybrid line has been constructed. Fluorographs showing the separation of 35-S methionine labeled seedling proteins were generated at Cold Spring Harbor Laboratory, Cold Spring Harbor, NY. They were, subsequently, scanned, spot detected, and spot quantified at Protein Databases, Inc., Huntington Station, NY. The fluorographic images were matched with the aid of PDQUEST^{tm} software. Densities for over 1500 protein spots are included in the resulting dataset. Eleven inbred lines are represented by one protein profile image. Twenty-three inbred lines and the hybrid line are represented by two protein profile images each. For the 23 inbred lines, each fluorographic image was obtained from a different gel. For the hybrid line, two fluorographs of differing exposures from the same gel are included. Three images each are included for two inbred lines. The three images were obtained from two different gels with two exposures of one of the gels being included. Four images are included for one inbred line and represent two exposures each of two different gels. To summarize, the 69 protein profile images represent 64 different gels. Two exposures per gel were included only when the appropriate exposure could not be determined a priori. The methodology used in constructing this dataset is given in Higginbotham, Smith, and Smith (Electrophoresis, in press).

Because many inbred lines are represented by more than one gel, it is possible to select by analysis of variance only those protein spots which vary significantly among the inbred lines. Details of this method of spot selection are given in Higginbotham et al. (in press). This is not the only method by which subsets of spots have been selected. Another subset consists solely of spots varying in their presence or absence among the inbred lines.

Information from this 2-D dataset is being used to determine genetic relationships among inbred lines, to resolve heterotic groups of lines, and to identify spots or spot clusters which diagnose lines.

The 2-D data are being used in comparisons with field derived, other lab derived, and pedigree data, as well. The 2-D data and RFLP data (for 35 of the 37 inbred lines in the 2-D dataset) are being modeled, separately and in combination, against heterosis yield and F1 yield. Correlations between 2-D data and morphological traits are being generated as well. For these correlations, the 2-D data are being divided into a subset of spots which varies among the lines qualitatively and another subset which varies among the lines quantitatively. The objective is to determine if the two kinds of spot variability yield different information and, if so, which is more informative (Damerval, Hebert, and deVienne, Theor. Appl. Genet. 74:194, 1987).

Correlations between restriction fragments and protein spots: integrating two-dimensional electrophoretic data with RFLP data

--J. W. Higginbotham

Restriction fragment length polymorphism (RFLP) data exist for 35 of 37 inbred lines used to construct a two-dimensional (2-D) electrophoresis dataset of seedling proteins. An introduction to the 2-D dataset appears above (Higginbotham et al., this Newsletter) and in Higginbotham et al. (Electrophoresis, in press).

Genes conditioning the expression of protein spots are being assigned to putative chromosome locations by correlating protein spots with restriction fragments. The probe library in use at Pioneer Hi-Bred was constructed from genomic DNA. A few probes are known to contain transcription regions, but the vast majority of them are anonymous pieces of DNA. By linking the 2-D protein profile data to the RFLP data, information on sites of active gene expression will be acquired. Furthermore, protein spots which appear to be associated with fragments known to be linked to quantitative trait loci (QTLs) may merit further scrutiny.

Table 1 lists seven putatively allelic pairs of proteins and three other proteins. The putative locations of the genes conditioning the expression of these proteins are shown along with the probes on that chromosome arm which were correlated with the proteins. Many of these probes are genetically linked to a number of QTLs which

Table 1. Protein spots, RFLP probes, and quantitative traits which appear to be associated. The chromosome arm locations of the RFLP probes are given.

Protein Spot #	Chromosome Arm	Probe ID	Quantitative Trait
4204	1S	PIO200689	Heat units to pollen shed
4205		PIO200603	Delay between shed and silk
		UMC094	% moisture at harvest
		PIO200640	
8835	1L	UMC128	
		UMC033	
		PIO200668	
	8L	UMC089	Heat units to pollen shed
		UMC030	Heat units to silk
		UMC007	
112	1L	PIO200557	
144		UMC084	
318	2	UMC139	% moisture at harvest
401		PIO200005	Stay green
		UMC034	
		BNL06.20	
3806	3L	PIO200509	
4808		PIO200576	
		PIO200558	
311	4L	BNL07.65	Ear height
312		PIO200071	
		BNL10.05	
		UMC019	
		UMC066	
5405	5L	UMC040	Test weight
5406		PIO150018	Cob diameter
		PIO150024	Ear diameter
		PIO200589	Row length
		PIO200715	Row number
224	7L	PIO200593	Ear height
		BNL08.39	Stay green
		BNL08.32	% moisture at harvest
		PIO200684	
		PIO200708	
		PIO200746	
301	9S	UMC109	Row number
		PIO100005	Yield in bushels per acre
		PIO200052	100 kernel weight
		UMC081	Plant height
			Heat units to pollen shed
			Heat units to silk
3804	10L	BNL10.13	
3808		UMC044A	

are given in Table 1.

Because this is a correlative study rather than a mapping project, the power of the analysis is limited. It is, however, a method of ascertaining probable sites of active gene expression in the absence of a cDNA linkage map. Proteins visualized on 2-D gels may, themselves, be used as markers especially when the segment is poorly marked. The short arm of chromosome 9 is such a segment.

Numbers of RFLP probes necessary to show associations between lines

--O.S. Smith, J.S.C. Smith, S.L. Bowen, R.A. Tenborg

Fifty-five pairwise distances ranging from 0.109 to 0.660 were computed from RFLP profiles scored for 125 probes that were well dispersed through the genetic map of maize. In cases where a probe was hybridized against more than one restriction enzyme digest of genomic DNA, only the probe/restriction enzyme digest combination that showed the highest level of polymorphism was utilized for these computations. Genetic distances between lines were calculated (Nei and Li, Proc. Natl. Acad. Sci. USA 76:5256-5273, 1979) for multiple sets of probes in increments of 5 probes. Confidence intervals (CIs) (4x standard error at the 95% level of probability) were computed for pairwise distances between lines. CIs (in parentheses) for the various numbers of probes (not in parentheses) used to generate distances were as follows: 10(0.51), 20(0.37), 30(0.30), 40(0.25), 50(0.23), 60(0.21), 70(0.19), 80(0.18), 90(0.17), 100(0.16), 120(0.15), 140(0.14), 160(0.13), 180(0.12), 200(0.115). Observations of the relative narrowing of CI around the distance value, in terms of the numbers of probes used to generate that distance, allows a more objective decision to be made concerning the numbers of probes to be used for the derivation of such data. When it is important to provide distance data in order to show associations among lines that reflect genetic constitution then the use of 100 probes can provide a reasonable degree of accuracy and is a technically feasible proposition given the current state of RFLP technology.

Choice of probes and restriction enzymes to generate RFLP data for the calculation of inter-line genetic distances and for the presentation of associations among inbred lines

--O.S. Smith, J.S.C. Smith, S.L. Bowen, R.A. Tenborg

Thirty-seven inbred lines that represent a broad array of diversity for that utilized in the U.S. Corn Belt and which included pairs of lines related by pedigree from 0% to 95% (Malecot's Coefficient of Parentage) were profiled by RFLPs for 157 probes and 257 combinations of probes by restriction enzyme digests of genomic DNA. Pedigree data and listings of probes have been given by Smith et al., (Theor. Appl. Genet. 80:in press, 1990). Distances were calculated between lines (Nei and Li, Proc. Natl. Acad. Sci. USA 76:5256-5273, 1979) from RFLP profiles for all probe/restriction enzyme combinations that were run and also for the following subsets of probes or restriction enzyme digests: 1) probes versus *Bam*HI digests; 2) probes against *Eco*RI digests; 3) probes against *Hin*dIII digests; 4) only probes that showed >10 variants across the 37 lines; 5) Pioneer Hi-Bred International, Inc., probes only; 6) University of Missouri (UMC) probes only; and 7) Brookhaven National Laboratory (BNL) probes only. Correlation coefficients of pairwise distances between lines for different sets or subsets of probes and restriction enzymes ranged from a low of r = 0.85 (UMC versus BNL probes) to a high of r = 0.994 (complete set of probe enzymes versus probe/enzymes showing >10 variants). Correlation coefficients for different restriction enzyme combinations versus the complete dataset were r = 0.96 (*Bam*HI), r = 0.96 (*Eco*RI), and r = 0.97 (*Hin*dIII). Correlation coefficients for different sources of probes versus the complete dataset were r = 0.94 (BNL), r = 0.95 (UMC), and r = 0.98 (Pioneer). If a subset of probes was to be chosen in order to limit the number of gels and hybridizations that would need to be made for a survey of diversity among lines then use of probes and restriction enzymes that reveal most polymorphism would be recommended on the basis of the results obtained from these data. It would be important that any set or subset of probes used to generate profiles for the showing of associations between lines or hybrids be selected so that probed sites are well dispersed throughout the genome and thus can provide a thorough sampling of the genome.

Associations among 45 hybrids widely grown in the U.S.; comparisons of groupings shown by isozymic versus RFLP data

--J.S.C. Smith, B.A. Orman, S. Wall

Forty-five widely grown proprietary named hybrids released from a total of 8 organizations were profiled for 19 isozymic loci and also by 80 RFLP probes well dispersed through the maize genome. Modified Rogers' Distances were calculated between all pairs of hybrids from the isozymic data; distances from RFLP data were computed according to Nei and Li (1979). Associations among hybrids were revealed by cluster analysis performed upon each of the respective distance matrices. Correlation of distances between hybrids for isozymic versus RFLP data was r = 0.70. Isozymic data placed 17 hybrids into 5 groups within which hybrids had indistinguishable profiles. Twenty-eight (62%) of hybrids had unique isozymic profiles. All hybrids had unique RFLP profiles. However, some distances, on the basis of RFLP data, were small and RFLPs confirmed the associations for 11 of 13 hybrids into 3 groups that were previously shown by isozymic data. Within each of these groups, hybrids shared more than 95% of RFLP variants. There were no examples of hybrids with different isozymic profiles yet with similar RFLP profiles. Isozymic data could provide a relatively cost effective and timely screen for hybrids with either similar, or different germplasm constitutions. Additional and more detailed analyses to test for similarities among hybrids or to show associations among hybrids should utilize data from additional genetic sites that will more completely sample the genome. Knowledge of the frequencies of occurrence of bands across lines or hybrids can allow calculations of match probabilities to be calculated. Data from relatively few probes might be sufficient to show very long odds that a matched profile could have been independently derived. As the frequencies of the bands across a relevant set of germplasm decreases, the numbers of probes needed to indicate long odds will also decrease. Such data can be provided by RFLPs.

Associations among 150 publicly available inbred lines as revealed by cluster analysis of RFLP data

--J.S.C. Smith, S. Wright, M. Walton, O.S. Smith, and S. Wall

One hundred fifty publicly available inbred lines, including those most widely used in U.S. maize breeding and agriculture from 1930-1990, were profiled for 46 well dispersed RFLP probes. Distances between lines were calculated according to Nei and Li (1979) and associations among lines were revealed by cluster analysis. There was a major split between lines at a distance of and below 70% dissimilarity. Iowa Stiff Stalk Synthetic lines such as B14, B37, B73 and lines with a Krug background partitioned into one side of the total cluster with Oh43, C103 (both non-Stiff Stalk) and Wf9 derived lines falling into the other side of the cluster. Lines were associated broadly in agreement with family assignments made on the basis of known pedigree. Of the 8 families into which public lines are usually classified (Seedsman's Handbook, Mike Brayton Seeds, Ames, IA, 1978), 4 (Wf9, C103, Oh43, and SSS) were represented by many lines and were further split into 2 subclusters for C103, Oh43, and Wf9; SSS split into 3 subclusters. Four families were either virtually extinct as clusters of lines (CMV3, CO109 x Early Butler, and W153R) or were represented by a few dissimilar lines (Hy). RFLP data revealed 3 additional small and diverse families. These were Krug, Clarage, and Illinois Synthetic. B14 and B73 appeared more similar to each other than was either to B37. Fourteen of the 16 original members of the Iowa Stiff Stalk Synthetic constituent lines were included in the study. They were spread throughout the cluster showing that collectively they represent a broad diversity of germplasm. Conspicuously, none of the Iowa Stiff Stalk Synthetic progenitors that were studied clustered within or between the Oh43 and C103 related lines. Approximately 33% of all lines clustered with one or more lines at or below a dissimilarity level of 25% (lines ≥75% similar). Only 15 lines clustered at or below a dissimilarity level of 10% (lines ≥90% similar). Most pairs of lines were more than 75% different with regard to their RFLP profiles. Most lines with more than 75% RFLP similarity were related through backcrossing in their pedigrees.

Ability of RFLPs to uniquely identify and to show associations among 160 elite lines used within a proprietary breeding program during 1930-1990

--J. S. C. Smith, O.S. Smith, S.L. Bowen, R.A. Tenborg, S. Wall, and D.N. Duvick

Inbred lines of key importance in the proprietary U.S. maize breeding program of Pioneer Hi-Bred International, Inc. from 1930 through to 1990 are being profiled for 100 RFLP probes; preliminary data for 160 lines and 37 probes are presented herein. RFLP probes were selected on the basis that they were well dispersed throughout the genome, that they revealed a high degree of polymorphism among previous screens of elite U.S. maize germplasm (Smith et al., Theor. Appl. Genet., 80:in press, 1990), and that banding profiles were readily scorable in terms of discrete variants. Distances were calculated between all pairs of lines according to Nei and Li (1979) and associations among lines were revealed by cluster analysis. All lines gave unique profiles. Two lines that were 94% related on the basis of pedigree (Malecot's Coefficient of Parentage) had 98.5% of their variants in common and were the most similar according to RFLP data, as would have been expected from their pedigrees. The most dissimilar lines, on the basis of RFLPs, had 22% common variants. Seventy percent of the lines clustered at a level of >30% profile dissimilarity between lines. Two hundred and eighty-six variants (mean of 7.73 variants per probe) were scored. Collectively the data show an abundant breadth of genetic diversity among these elite inbred lines. Cluster analysis of the RFLP data resulted in seven large groups of lines in broad agreement as would be expected from pedigrees. Correlation of RFLP distances between lines versus distances calculated from pedigree records (1 - Malecot's Coefficient of Similarity) was r = 0.58. This value was lower than that (r^2 = 0.81) reported previously (Smith et al., Theor. Appl. Genet., 80:in press, 1990) for 37

elite Corn Belt lines. However, among these 160 lines there were no connections by pedigree breeding for 66% of the pairs of lines. For the majority of pairs, therefore, there was no information upon which to compute a reliable estimate of distance other than to use RFLPs (or other data such as F1 yield or heterosis that are extremely resource-consuming to obtain). RFLPs can be very useful to show associations among lines on the basis of their genetic constitutions. Studies that track genotypes through generations of breeding are one means by which an index of agronomic worth could be assigned to RFLP profiles. Such information could assist in further objective utilization of available genetic resources by breeders.

Choice of restriction enzyme digests of genomic DNA in profiling inbred lines

--J.S.C. Smith, O.S. Smith, D. Grant, S.L. Bowen, and R.A. Tenborg

The abilities of single and multiple individual digestions of genomic DNA to reveal polymorphisms using 6 restriction endonucleases on 13 inbred lines of maize that encompass a broad array of diversity in the U.S. Corn Belt were investigated. Genomic DNA of each line was extracted by CTAB (Saghai-Maroof et al., Proc. Natl. Acad. Sci. USA, 81:8014-8018, 1984). DNAs were individually digested by *Bam*HI, *Bgl*II, *Eco*RI, *Eco*RV, *Hin*dIII, and *Kpn*I. Electrophoresis, transfer to membrane, random-prime labelling, hybridization, and washes were performed as described in Smith et al. (Theor. Appl. Genet., 80:in press, 1990). Each genomic digest was probed separately with 100 clones that have been mapped and were, therefore, known to be well dispersed throughout the maize genome. Approximately equal numbers of probes from Brookhaven National Laboratory, the University of Missouri at Columbia, and Pioneer Hi-Bred International, Inc. were used.

*Kpn*I digests were often unscorable because of blurred profiles or bands that migrated closely together; these digests were, therefore, excluded from further analysis. The mean values per probe for the percentage of all possible pairs of inbreds that revealed polymorphism (in parentheses) for single digestions of genomic DNA were as follows: *Bam*HI(77%); *Bgl*II(80%); *Eco*RI(77%); *Eco*RV(77%); and *Hin*dIII(80%). Percentages of inbred pairs that were polymorphic for 2 individually digested and separately probed DNAs ranged from 77% to 86% (mean 81%). Percent inbred pairs polymorphic for 3 individually digested genomic DNAs ranged from 83% to 89% (mean 86%). The mean percentage of pairs that were polymorphic when 5 individual digestions were probed was 91%.

Use of a single restriction enzyme digest revealed, on average, 86% of the maximum variation that was revealed when 5 individual digestions of DNA were probed separately. The absolute percentages of polymorphism that are revealed will depend upon the degree of difference among the genotypes under study. Nevertheless, these data indicate that the use of additional probes rather than the use of additional restriction enzymes is a far more effective means of revealing variation. The employment of additional probes provides the further advantage of allowing the genome to be surveyed in greater detail. Therefore, it is proposed that for routine studies, profiles be acquired using a single restriction digest per probe. The particular restriction digestion of genomic DNA that is used for each probe can be determined so as to maximize the opportunity to reveal polymorphism and to reproducibly generate autoradiograms of high quality with variants expressing obviously discernible migration and molecular weight differences.

Standards for declaring and computing matching probabilities between genotypes of maize in cases where identities, pedigrees, and ownership of germplasm are in dispute

--J.S.C. Smith, B. Bowen, D. Grant. B.A. Orman, R. Fincher, and O.S. Smith

Disputes over the identity, pedigree, and ownership of lines can be very costly to resolve in respect of both monetary resources and time spent by breeders and other researchers away from the development of further improved genotypes. Methods to derive laboratory data that are repeatable and reliable would help resolve issues of genetic identity and veracity of pedigree. Acceptable testing methods could reduce both the length and the number of disputes. A decline in both the frequency and expensiveness of disputes would benefit agriculture since it would allow the concentration of more resources into independent, productive, and innovative breeding rather than their siphoning out into defensive strategic enterprises. In the field of human forensics, much valid criticism has been made of the use of DNA as evidence (Lander, Nature 339:501-505, 1989). In plants, all of the problems encountered in human forensics, i.e. lack of abundant, high quality, and uncontaminated DNA, are obviated by the usual availability of numerous seeds of each genotype. There remain, however, important lessons to be learned from the experiences that human forensic scientists have encountered. These include: 1) quality checks on DNA; 2) checks on sample identity and protection against mislabelling errors; 3) use of adequate molecular weight marker standards; 4) running of samples unmixed and mixed as final tests of band matches; and 5) the availability of a database of band frequencies that can be used to compute match probabilities.

We have prepared a document citing these various factors and including some proposals as to what should be done to evolve usable standards of procedure in the derivation of RFLP data to resolve identity, pedigree, and ownership disputes. We would like to send this manuscript to any individual or organization in order for them to be able to add any comments to its content. Therefore, we ask you to write to one of us (J.S.C. Smith) if you would like to have an opportunity to review the current proposals. A revised set of standards then could be considered for use as a means to expedite or to reduce the need for lengthy disputes over germplasm ownership. In this respect, it is very important that one or more independent laboratories be able to perform these procedures and that they have at their disposal a database of RFLP band frequencies that encompasses the most important public in-

bred lines and proprietary commercial hybrids. An investment by the corn industry to develop a means of rapidly solving or even preventing disputes would be very small compared to the costs that would be entailed by numerous lengthy disputes.

Methods to precisely estimate molecular weights of fragments constituting RFLP "fingerprints" and a means to standardize measurements within and across laboratories

--J.S.C. Smith, R.A. Tenborg, B. Roth, D. Grant, O.S. Smith, T. Brumback, and T. Beghtol

In order to build a database of RFLP profiles for inbred lines and hybrids using probes that are repeatedly used against membranes made from different gel runs, it is imperative to be able to report data in terms of an inherent property of the DNA fragments themselves. Relative migration in a gel (Rf) is a result not only of the size of the fragment but also of other factors including agarose concentration, buffer strength, temperature, and volt hours applied. Molecular weight is an inherent property of the DNA fragment that, once determined, properly describes that variant. Therefore, we are running gels to profile lines and hybrids with 3 lanes of molecular weight ladder. The ladder is constructed by isolating individual digested fragments of lambda with the final concentrations of each band adjusted so that the individual rungs of the marker ladder are exposed using random prime labelled lambda in approximately equal amounts. Although several commercially prepared marker ladders are available, they provide problems either in 1) having molecular weight components (below 2.0kb) that migrate into and obscure the separations coming from other combs placed lower in the gel; 2) having unequal amounts of DNA at the various rung positions, therefore, producing both over and under exposed components; and 3) having sparse representation of standards in the region of the gel from approximately 23kb to 10kb where small changes in migration equate with relatively large molecular weight differences. The rungs in the molecular weight ladder (restriction digest used to obtain the fragment in parentheses) in current use are as follows: 23.13kb (*Hin*dIII); 17.06kb (*Kpn*I); 13.29kb (*Bgl*II); 9.42kb (*Hin*dIII); 6.56kb (*Hin*dIII); 4.36kb (*Hin*dIII); 2.32kb (*Hin*dIII); and 2.03kb (*Hin*dIII). In addition, each lane of genomic DNA is spiked with the slow moving (23.13kb) and fast moving (2.03kb) bands. In order to facilitate the preparation of the ladder, we have cloned lambda fragments below 10kb so that high yields of the smaller components of the ladder can be obtained. Thus, the time consuming testing of suitable dilutions of the ladder components need be done only infrequently. We are also seeking to replace some of the larger rungs of the ladder by using mixes of other digests of lambda. These mixes individually give multiple rungs but with no large disparities between ladder components in their ability to expose film so that adjustments in amount of fewer individual components of the ladder will be necessary. In that respect, a combination of lambda digested by *Eag*I and *Xho*I provides 5 rungs between 33.5kb and approximately 12kb.

We have tested the potential ability of a *Bam*HI/*Eco*RI digest of AdenovirusII (AdII)to provide a ladder that can 1) be purchased and would, therefore, obviate the need to isolate individual components of the ladder; 2) give approximately equal exposures of bands throughout the ladder; and 3) give an acceptable coverage of molecular weight standards in profiling. This digestion of AdII provides 13 rungs from 35.94kb to 1.74kb. There are four components above 10kb (35.94, 21.34, 14.33, and 10.68kb). Random prime labelled lambda did not hybridize with the AdII fragments. Individual components of the commercial product (purchased from IBI) gave approximately equal exposures after hybridization with random prime labelled AdII. This product could be used as a standard within and across laboratories to facilitate accurate and repeatable estimates of molecular weight of genomic DNAs. It could be used in one of two ways. First, as a molecular weight ladder loaded into lanes specifically designated as marker lanes. Second, as a spike in every lane of genomic sample. In this latter case, it and digest(s) of lambda would be placed in specific marker lanes also. The first hybridization of the membrane would then be made by lambda and AdII. Subsequent hybridizations would be made with lambda and genomic DNA. The lambda bands would allow an interface to be made of the genomic DNA profile and the 13 AdII *Bam*HI/*Eco*RI bands that were co-electrophoresed in the genomic lanes. This latter procedure would provide the most precise means of estimating molecular weights for genomic DNA profiles. In either case, AdII is a commercially available product that could provide a good means of standardizing profile scores within and across laboratories.

List of probe and restriction enzyme digest combinations of genomic DNA that are useful for "fingerprinting" inbred lines and hybrids

--J. S. C. Smith.

Not all combinations of probes and restriction enzyme digests result in clearly readable and interpretable RFLP banding profiles that show polymorphism. We have surveyed some four hundred DNA clones from the Brookhaven National Laboratory, University of Missouri, and Pioneer Hi-Bred International, Inc. libraries against DNAs of 13 inbred lines of maize that were individually digested by *Bam*HI, *Eco*RI, and *Hin*dIII. Probe/enzyme combinations that gave clearly readable polymorphisms against these lines were subsequently used to profile 37 inbred lines of maize. Approximately 100 of these probes were then used to profile 350 inbred lines. This set of 100 probes, using one restriction enzyme digest with each probe, constitutes our set of probes that are routinely used to profile maize. The map locations show that these probes collectively provide a good coverage of the genome. In order to circumvent the need for other organizations and researchers to conduct much of their own screening of probes for utility in RFLP profiling, I will send the list of probes and restriction enzymes to anyone who requests it.

A linkage map based on information from four F2 populations

--William D. Beavis and David Grant

Several genetic maps using RFLP and other molecular markers and based on data from one or more F2 populations have recently been reported in this Newsletter (Helentjaris et al. 1986; Murray et al. 1988; Working Map 1988 and 1989). Unfortunately, because the mapping was done using different populations and often with different markers, it has been difficult to define a single genetic map which includes all of the information available. As a first step in the construction of such a composite map, we have

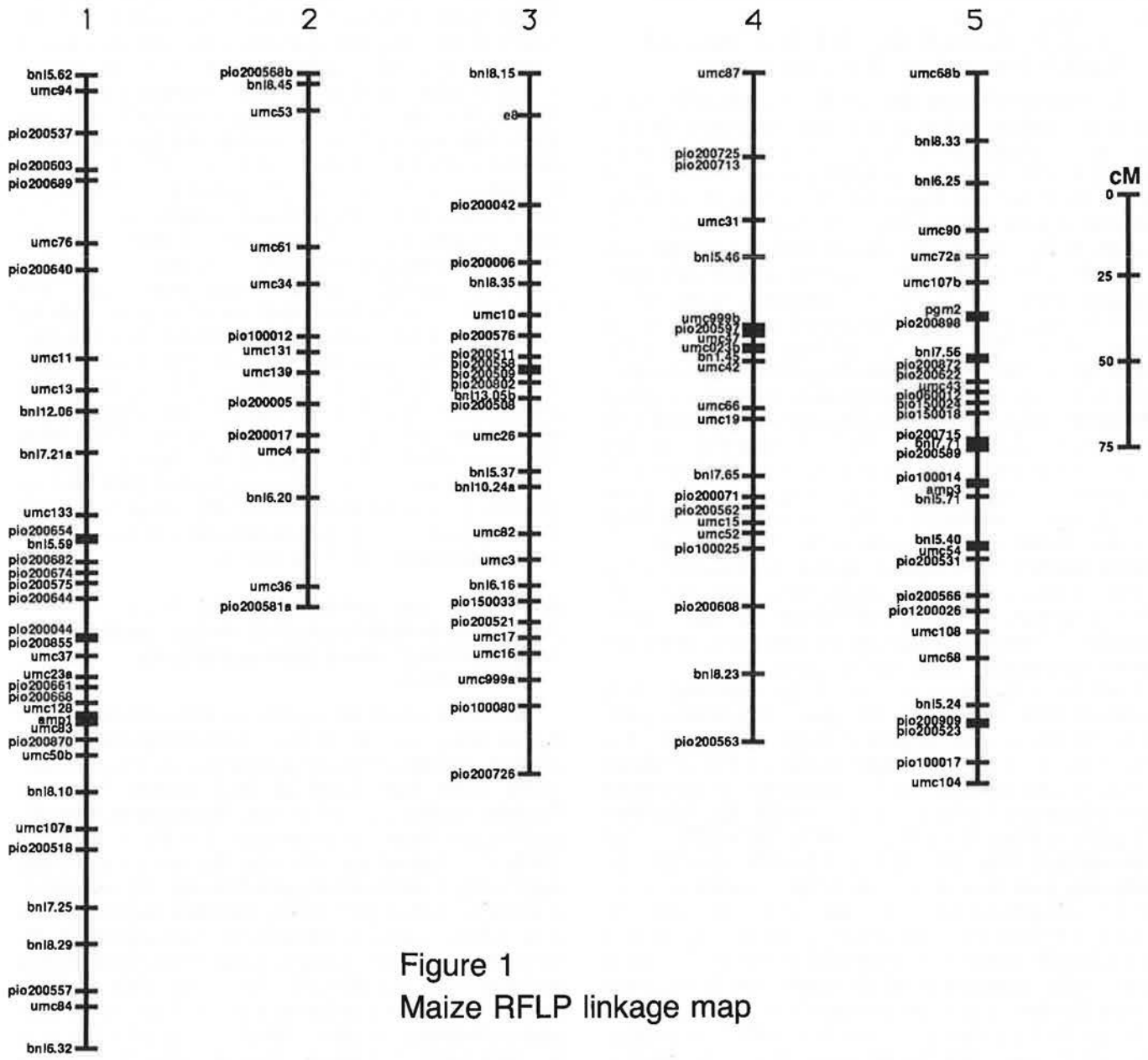
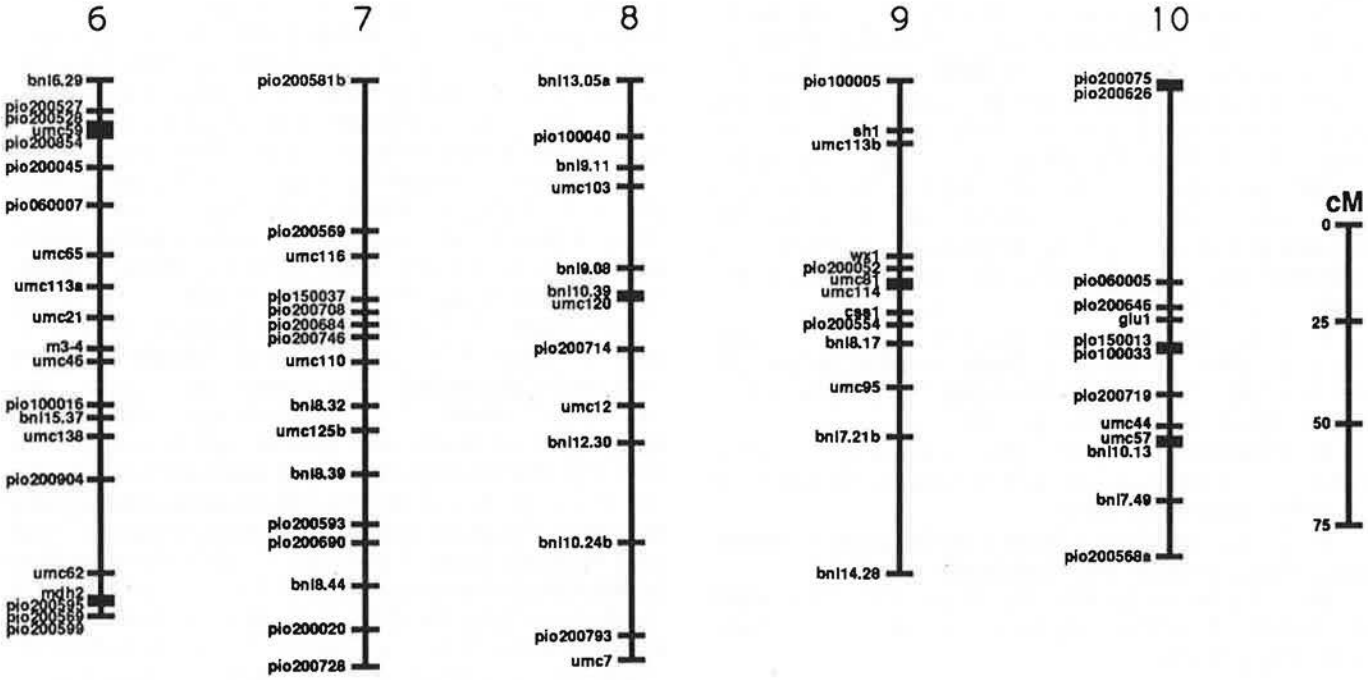


Figure 1
Maize RFLP linkage map

developed a method for combining data from different populations prior to generating an RFLP map. Specifically, we utilize the log likelihood statistic to test for homogeneity of recombination among populations and to genetically map partially overlapping sets of markers. A more detailed description of the methods can be found in Beavis and Grant (Theor. Appl. Genet. 1991, in press).

In this report we present a composite map composed of 209 markers and based on data from four F2 populations (B73/Mo17, B73/G35, K05/W65 and J40/v94). Population sizes were 112, 112, 144 and 144 individuals, respectively. The probes used came from Ben Burr (bnl), Univ. of Missouri (umc) and Pioneer Hi-Bred (pio). All molecular biology steps were done using standard conditions. Genetic maps were constructed using MAPMAKER (Lander et al. 1987).



KRASNODAR, USSR
Krasnodar Research Inst. Agric.

Genetic control of maize variability

--A. S. Mashnenkov

Actual progress in evolution and breeding is attributed to different types of genetic variability: mutation, recombination, combination, introgression. The interaction of these sources of variability controlled by inheritance is not known to the author.

At first we managed to reveal a curvilinear association between maize inbred sensitivity to N-nitroso-N-methylurea in M1 and their mutability (MNL 59:104). Some insensitive and sensitive low-mutable maize inbreds proved similar to Uvr$^+$RecA$^+$ and Uvr$^-$RecA$^-$ of *E. coli* strains respectively, and those of moderate sensitivity high-mutable inbreds to strain Uvr$^-$RecA$^+$. We supposed the existence of an ancient system of genome maintenance providing distinction and removal of any distortions in genetic substance (Genome 30:159, 1988). Proceeding from this hypothesis the following considerations may be drawn:

1. In case of distant hybridization with low-mutable lines insensitive to mutagen effects hybrid kernels would not develop. In high-mutable lines the maximum frequency of hybrid seed set is expected.

2. In low-mutable sensitive inbreds elimination of alien genetic stock produced through distant hybridization or transformation rarely occurs.

3. In supersensitive inbreds recombination processes are partially or completely suppressed.

Our experience in crossing maize with *Tripsacum dactyloides* (2n=72) mainly proves consequence 1 of the general hypothesis.

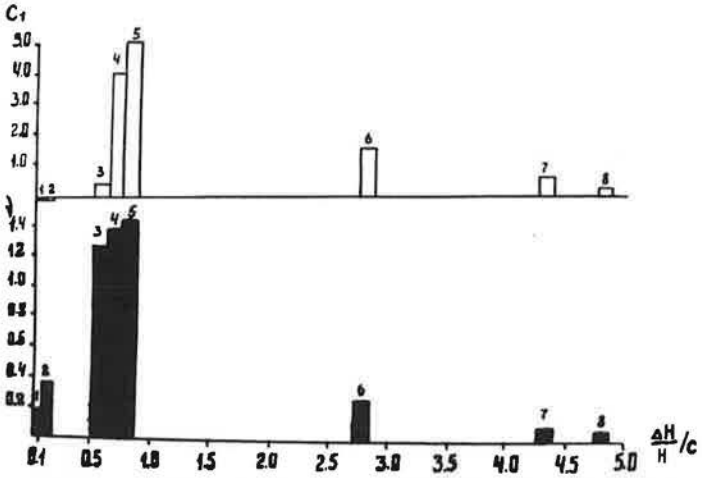


Figure. Sensitivity, mutability and compatibility of maize inbreds. Symbols. Inbreds: 1 - T22, 2 - Gk26, 3 - W23, 4 - A344, 5 - PLS61, 6 - Gb834, 7 - Hy2, 8 - F2. ($^{\Delta}$H/H)/c - unit depression of plant height in M1, where: $^{\Delta}$H - plant height decrease, H - control plant height, c - NMU concentration, mM, ν - mutation frequency in optimal test variants, C1 - coefficient of *Zea* and *Tripsacum dactyloides* genome compatibility (see E. P. Erygina and A. S Mashnenkov's contribution to this issue).

Some considerable deviation from the theoretically expected C1 value was only found in W23. According to mutation theory any factor in an abnormal state for this organism can induce mutation. Therefore, *T. dactyloides* genome in the first approximation may be taken as a mutagenic factor. Hence, the possibility to compare values ν and C1 is feasible.

The individuals of *Zea mays* L. are specialized by the main alternative functions: inheritability and variability. The function of *Zea* genome preservation is realized in inbreds that are low-mutable sensitive and insensitive to mutagen effect. The effect of species preservation is achieved by principally different methods. In sensitive inbreds self-elimination of cells and organisms with distortions in the genome takes place. In insensitive inbreds this effect is realized by active distinction and correction of all distortions or by degradation of an alien genome. Due to the latter, high frequency of haploids in F1 progeny may be expected. The function of genetic variability is concentrated in the lines of moderate insensitivity. These lines are generators of species variability where probably all types of genetic variability are mostly intensified. As for the latter group of inbreds, the majority of maize mutations earlier unknown was induced (MNL 60:70-71, 1986). Genetic analysis showed digenic control of mutability induced by NMU (Genome, 30:159 1988). Probably the systems of unspecific control of the ratio "variability:inheritability" are conditioned by superloci. The digenic type of control provides preadaptation of the species or evolutionally valuable population to any environment. Let us examine the case of gradual tension increase of mutagen factor in time and space. First of all supersensitive aabb inbreds give a response. Suffering from phenotype depression induced even by small doses of the mutagen they decrease their contribution to the genetic pool of the next generation. It results in an increase of the share of high-mutable aaBB, aaBb genotypes in the population. In case of further increase in tension of the factor the stage of ultimate tension of all types of variability is observed. Search for new approaches to adaptation to changed environment is under way. At last only insensitive low-mutable genotypes AABB, AaBB, AABb, AaBb, improved by new specific factors for stability obtained by crossing with highly variable inbreds, remain. In nuclei of insensitive inbreds adaptively valuable alleles are accumulated and concentrated. The species transits into a genetically closed state where new genotypes mainly occur under the control of the locally specified system. With lessening of mutagen factor tension the species (population) recovers its genetically open evolutionarily valuable preadaptive state (i.e. containing all three types of lines) because of segregation.

Lack of money does not allow the author to thoroughly check all the consequences of the hypothesis. It is necessary for evolutionists, geneticists and molecular biologists to cooperate to realize the project "Genetic Control of Corn Variability".

Effect of inbred genotype on kernel development in maize-*Tripsacum* hybrids

--Elena P. Erygina and A. S. Mashnenkov

It was found that inbreds differed much in frequency of seed set in maize-*Tripsacum* crosses (MNL 63:87, 1989). In 1989 11 maize inbreds were crossed with *Tripsacum dactyloides* (2n=72). Ten ears were pollinated in each in-

bred. Depending on rate of ovary development four types of maize-*Tripsacum* hybrid kernels were distinguished:

1. kernels with well-developed germs and endosperms (test weight of a kernel--m=30-80mg),
2. kernels with poorly developed endosperms (m=15-30mg),
3. kernels with well-developed pericarps and initiated germs and endosperms (m=6-15mg),
4. kernels with initiation of pericarps, germs and endosperms.

All four types of kernels were included in the index-percentage of developed ovaries (Table).

Maize inbreds	Ovaries per ear	Developed ovaries, %	F1 kernels with germs and endosperm, %	Coefficient of kernel development, P	Coefficient of compatible F1 genomes, C1
Gk26	4020	0.72	0.00	0.000	0.000
Tm10	3030	4.90	0.00	0.000	0.000
T22	2256	9.66	0.00	0.000	0.000
Gk30	1922	29.08	18.73	0.144	2.697
W23	4200	19.50	3.55	0.092	0.327
A344	3596	48.28	28.20	0.139	3.920
PLS61	3726	48.77	39.99	0.125	4.999
Gb834	2040	18.14	14.17	0.109	1.545
Hy2	3322	17.40	7.92	0.076	0.602
F2	1876	43.07	3.62	0.065	0.235
A663	5070	20.63	12.96	0.152	1.970

Coefficient of kernel development (P) was determined as a quotient of division of the value of test weight of hybrid kernels with germs and endosperms into the value of inbred per se test weight. Coefficient of genome compatibility (C1) is equal to the product of F1 kernel development rate (P) by percentage of kernels with endosperms.

Three inbreds, Gk26, Mangelsdorf's Tester 10 (TM10) and T22 showed complete incompatibility with *Tripsacum dactyloides* genomes. According to rate of kernel development other inbreds may relatively be divided into three groups: P = 0.65-0.92; P = 0.109-0.139; P = 0.144-0.152.

In the case of *T. dactyloides* ample pollen on cut maize silks, all egg cells are probably fertilized but kernel development stops at different stages of organogenesis.

In this case the value P mainly expresses rate of accumulation of stored substances in kernels. Coefficient C1 considers time that kernel development stops, i.e. stability of organogenesis. Evidently these traits are independently inherited. For instance, for inbreds PLS61 and A344 the most stable organogenesis is observed at moderate rates of stored substance accumulation in endosperms. Inbreds A663 and Gk30 are characterized by a contrasting combination of these traits.

To improve efficiency of hybridization the genetic factors governing maximum expression of both traits should be combined.

LENINGRAD, USSR
Institute of Plant Industry

Macrosporogenesis in both normal plants and *mei*-mutants

--N. A. Avalkina and I. N. Golubovskaya

Recently an enzyme squash technique on isolation of archeosporic cells undergoing meiosis (Jongedijk, Stain Technol. 62:135-142, 1987) has been adapted to maize. It permits the study in detail of both female meiosis and embryo-sac development. In normal plants the main distinction between the male and female meiosis is revealed at

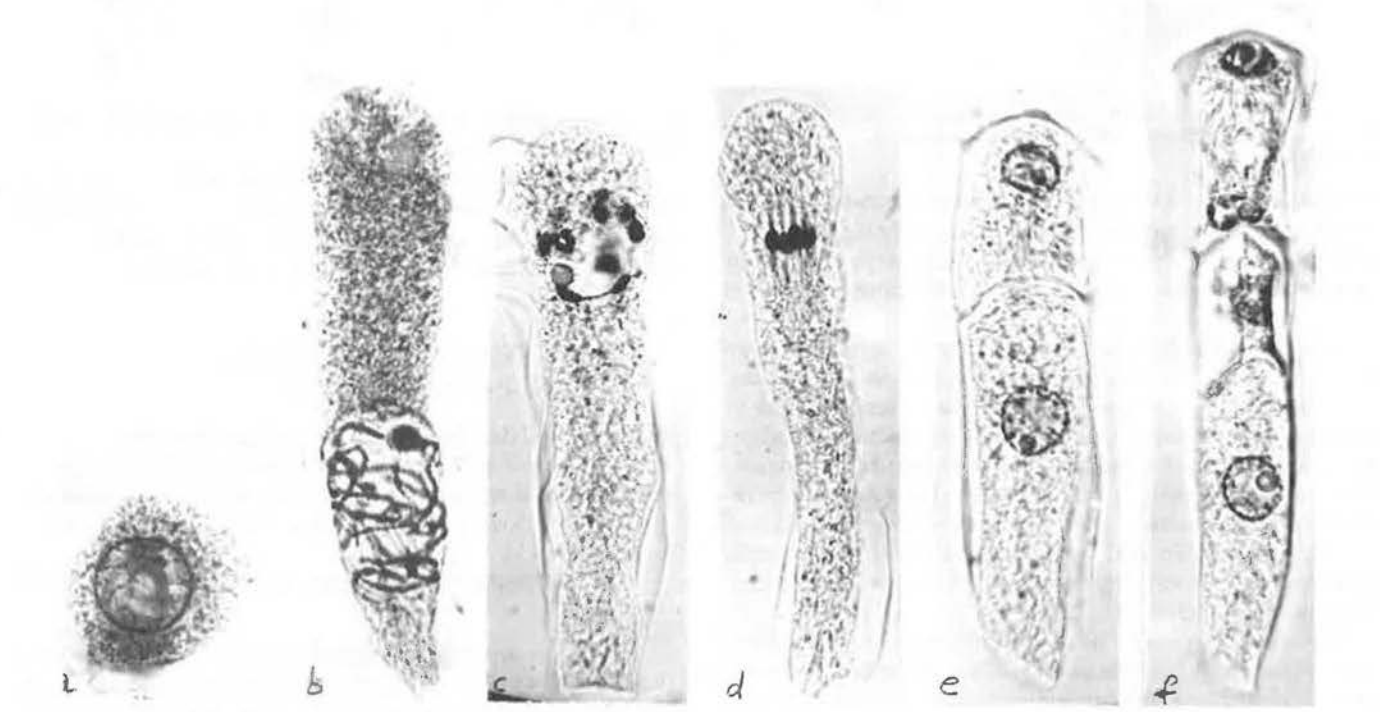


Figure 1. Female meiosis in normal fertile plants. a) interphase, b) pachytene, c) diakinesis, d) metaphase I, e) dyad cell, f) tetrad.

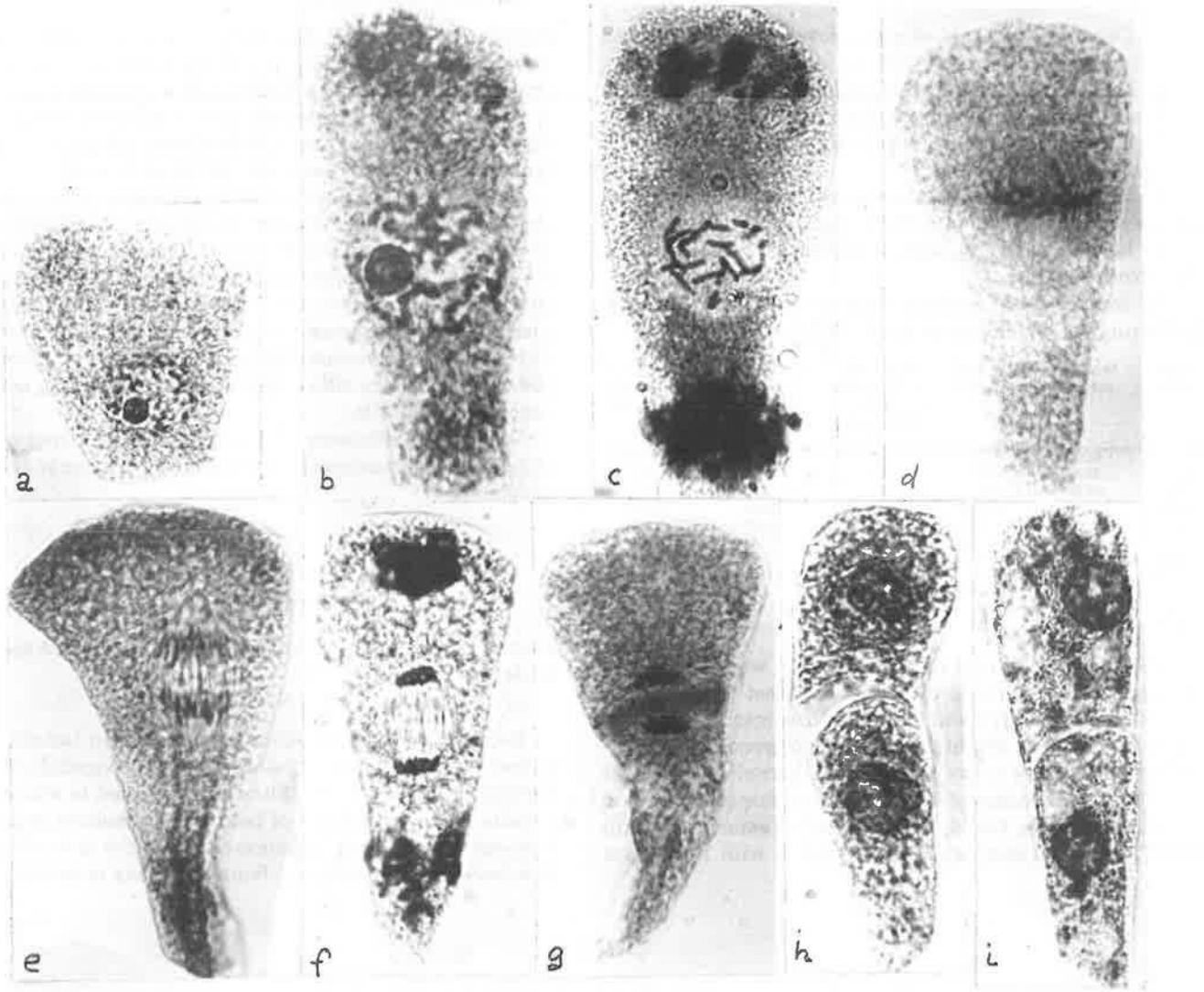


Figure 2. Female meiosis in ameiotic *mei*-mutants. a) interphase, b) prophase, c-d) metaphase, e) anaphase, f-g) telophase, h) dyad cell, i) degenerated dyad cell.

second division. As a rule in macrosporogenesis the 2nd meiotic division proceeds nonsimultaneously in dyad cells, and in micropylar cells the second division is delayed or completely stopped at metaphase 2 - telophase 2 (Figure 1).

The squash technique is convenient for solving cytogenetic problems, for example, an effect of *mei*-genes, impairing different key cytogenetic events of meiosis, both in micro- and macrosporogenesis. The character of meiosis in an ameiotic maize *mei*-mutant is shown in this paper. The ameiotic recessive allele in the homozygous state is responsible for meiosis substitution by mitosis. In microsporogenesis in meiocytes of ameiotic homozygotes, synchronous mitosis proceeds instead of meiosis (Palmer, Chromosoma 35:233-246, 1971).

The female meiosis in ameiotic homozygous mutants has been studied, and the pattern of irregularities in female meiosis is similar to ones in male meiosis. Different stages of ameiotic mitosis (interphase, prophase . . . telophase) are observed (Figure 2,a-h). The ameiotic mitosis in macrosporogenesis is completed by formation of dyad cells, but a great number of them are degraded (Figure 2i). Rare triads or tetrads occur.

For the first time we have the opportunity to visualize the real effect of *mei*-genes in female meiosis. The enzyme squash technique used here is convenient for solving some questions of the great problem of genic control of meiosis.

LLAVALLOL, ARGENTINA
UNLP-CONICET-CIC

Variation of the frequency of multivalents in *Zea diploperennis* x *Z. perennis* (2n=30 and 2n=40) hybrids treated with dilute concentrations of colchicine

--C. A. Naranjo, L. Poggio*, M. C. Molina and E. A. Bernatené

*Also affiliated with Depto. de Cs. Biológicas, FCE (UBA).

Having managed immature panicles with colchicine (0.5×10^{-4}M) for 12 hours, we have shown the existence of cryptic homoeology in maize (*Z. mays* ssp. *mays*) and *Z. perennis*, forming quadrivalents in the first one and increasing the frequency of quadrivalents in the second one

(Poggio et al., Theor. Appl. Genet. 79:461-464, 1990). These results confirm the allotetraploid nature of maize proposed by Molina and Naranjo (Theor. Appl. Genet. 73:542, 1987) and Naranjo and Molina (MNL 61:62) and suggest that the species have homoeologous genomes (*Z. mays* ssp. *mays*, $A_2A_2B_2B_2$; *Z. perennis*, $A'_1A'_1A''_1A''_1$ $C_1C_1C_2C_2$ which fail to pair, probably due to the present of *Ph*-like genes (Poggio et al., ibid).

To complete the analysis of genome relationships we have analyzed two artificial F1 hybrids, *Z. diploperennis* x *Z. perennis*; 2n=30 ($A'_1A''_1A_1$ $B_1C_1C_2$) and 2n=40 ($A'_1A''_1A_1A_1B_1B_1C_1C_2$). The results indicate that the frequency of six or more trivalents was increased from 25.6% in the untreated material to 90% in the treated material of *Z. diploperennis* x *Z. perennis* (2n=30). Meanwhile the frequency of five or more quadrivalents was increased from 46.3% in the untreated material to 69.4% in the treated material of *Z. diploperennis* x *Z. perennis* (2n=40). Differences between treated and untreated materials were subjected to a test of homogeneity and were found highly significant.

Moreover, *Z. diploperennis* (2n=20, $A_1A_1B_1B_1$) was treated but we have not found multivalent formation.

The results pointed out greater homoeology among B and C genomes with respect to the untreated material and not between these genomes and the A as shown by the absence of hexavalents and octavalents in the 2n=30 and 2n=40 taxa (MNL 62:74-75).

Meiotic behavior and DNA content in two stable lines of maize with cytoplasm of *Z. mays* ssp. *mexicana*

--L. Poggio, C. Tito, L. Mazoti and C. A. Naranjo

Cytoplasm of *Z. mays* ssp. *mexicana* is responsible for several inherited morphological and physiological characters when it is combined with genotypes of *Z. mays* ssp. *mays*. Mazoti (1950, 1958, 1963, 1978) pointed out that some of these characters are: a) vegetative period (response to photoperiod), b) yield in grains, c) height of seedling and plants, d) number of spikelets per plant.

In the "Multiple Dominant" line (Z line), with cytoplasm of *Z. mays* ssp. *mexicana* (E line), Mazoti and Velazquez (1962) found a greater percentage of pollen sterility, greater variation in the diameter of nucleolus, stickiness among meiotic chromosomes and, in sections of anthers, very frequent intercellular contacts. Mazoti (1975, 1978) showed that Z line has the gene *C2-IE7002* in its own cytoplasm, which induces instability, and the cytoplasm of E line could act as an activator of this gene. Mazoti (1987) reported that the knobs in pachytene of the E line have larger size and higher DNA content.

Poggio et al. (MNL 64:71-72, 1990) found that Z and E lines differ significantly in the total DNA content and in their meiotic behavior. These authors found that the E line possesses higher DNA content and heterochromatin than Z line and, in some places of the panicle, a high percentage of meiotic irregularities (desynapsis, cytomixis, cellular fusion, presence of diffuse nucleolar bodies, pseudomultivalents, etc.).

In the present work the DNA content and meiotic behavior is studied in two stable lines of maize (*gl ij* and *c*-tester) and these lines with E cytoplasm. The aim of this work is to analyze the question raised by Mazoti (1987) if "the plasmon of *Z. mays* ssp. *mexicana* induce knob variation only in presence of the genes of the Multiple Dominant Line".

The pure lines utilized were maintained in the IFSC since their introduction. Maize *c*-tester and *gl ij* lines were introduced in Argentina in 1933 by Ing. Agr. S. Horowitz. The lines *gl ij* (E) and *c*-tester (E) were obtained using the pure lines of maize as male recurrent parent during five backcrosses, onto plasmon from *Z. mays* ssp. *mexicana* (Florida variety, Huixtla, Mexico).

The meiotic behavior of both lines is indicated in Table 1. Total DNA content is shown in Table 2. The *c*-tester vs. *c*-tester (E) and *gl ij* vs. *gl ij* (E) did not show any differences in the average of total and closed bivalents. However lines with E cytoplasm showed two groups of five bivalents each with high frequency in prophase I. This separation is more notorious than that reported by Naranjo et al. (Acad. Nac. Cs. Ex. Fix. Nat., Buenos Aires, Monogr. 5:43-53, 1990). Moreover, each group of five bivalents is slightly asynchronic in respect to the other group in the development of meiotic prophase I. The total DNA content is slightly higher in lines with E cytoplasm (Table 2). The quantitative change in DNA content is accompanied by larger C+ bands in mitotic and meiotic metaphases when C banding technique is applied in Z and E lines (Poggio et al., MNL 64:71-72, 1990).

TABLE 1. Meiotic behaviour in Metaphase I (MI) and Diakinesis (Diak)

Line	Nº of cells studied	$\bar{X}$ II ± S.E. (range)	$\bar{X}$ I ± S.E (range)
c-tester	109 (MI)	9.91 ± 0.03 (9-10)	0.18 ± 0.05 (0-2)
c-tester (E)	61 (MI)	9.90 ± 0.07 (6-10)	0.2 ± 0.13 (0-8)
gl ij	32 (MI)	9.59 ± 0.14 (8-10)	0.81 ± 0.29 (0-4)
gl ij	82 (Diak)	9.77 ± 0.06 (8-10)	0.46 ± 0.12 (0-4)
gl ij (E)	69 (Diak)	9.92 ± 0.37 (8-10)	0.14 ± 0.07 (0-4)
gl ij (E)	68 (MI)	9.04 ± 0.07 (8-10)	1.91 ± 0.13 (0-4)

TABLE 2. Nuclear DNA content

Line	Nº of measured nuclei	DNA content (2C) pg ($\bar{X}$ ± S.E.)
c-tester	217	5.783 ± 0.036
c-tester (E)	144	6.269 ± 0.049
gl ij	50	5.998 ± 0.080
gl ij (E)	69	6.117 ± 0.055

Summarizing, the results obtained suggest that the E cytoplasm promotes, through some still unknown mechanism, an increase of highly repetitive DNA in the zone of the knobs. This effect would be independent of the presence of the "Multiple Dominant Line" genotype since the stable lines *gl ij* (E) and *c*-tester (E) have greater DNA con-

tent than *gl ij* and *c*-tester in their own cytoplasm. Another effect of the nucleus-cytoplasmic interaction seems to be the alteration of the spatial distribution of genomes and the asynchronism detected during the development of prophase I.

Recalibration of "*c*-tester" line as a standard to estimate nuclear DNA content/pg by Feulgen microdensitometry

--C. Tito, L. Poggio and C. A. Naranjo

When nuclear DNA content is measured by microdensitometry using Feulgen stain, the results are obtained in arbitrary units (A.U.). It is assumed that there is a proportionality between stain density and DNA amount, and through a standard species the arbitrary units can be converted into absolute units in picograms (pg).

Bennett and Smith (Proc. Roy. Soc. London B 274:227-274, 1976) advised using as a standard a species, cultivar or seed of defined population with DNA content similar to one of the species being studied. Moreover, they should be easy to culture under laboratory conditions, and root-tips should be readily available for use as standards throughout the year. This procedure tends to minimize some technical errors inherent in Feulgen microdensitometry. These authors pointed out that the accuracy of absolute DNA amounts calculated by this method depends "first, on the accuracy with which each species is measured relative to the standard species used for calibration, and secondly, on the accuracy of the assumed DNA amount of the standard species". The majority of plant DNA amounts measured by Feulgen microdensitometry have been calibrated using *Allium cepa* as a standard (2C=33.55 pg). Bennett and Smith did not find evidence of detectable intravarietal variation in DNA amount in *A. cepa* var. Ailsa Craig, which was considered suitable for use as a standard for calibrating other species. Moreover, this species was used to recalibrate several species as other standards.

The data available for species of *Zea* in the literature vary between 2C=4.96 pg for Gaspe Flint and 2C=11.36 pg for *Z. perennis* (Laurie and Bennett, Heredity 55:307-313, 1985; Rayburn et al., J. Exp. Bot. 40:11-1183, 1989). The standard used by these authors is not easily available to us and the available ones (e.g., *A. cepa* var. Ailsa Craig) have remarkably high 2C value to be used when the genus *Zea* is being studied.

The *c*-tester inbred line has all the characteristics suggested by Bennett and Smith to justify its use as standard. Therefore, three experiments were done on different days to recalibrate carefully the 2C value of *c*-tester line, against *A. cepa* var. Ailsa Craig.

DNA content was measured in telophase nuclei (2C) of the root apex of germinating seeds. Seeds were placed in Petri dishes on wet filter paper. Roots of 0.5-1 cm length were fixed in 3:1 (absolute ethanol:acetic acid) during 1-4 days. After fixation the roots were rinsed for 30 minutes in distilled water. Hydrolysis was carried out with 5N HCl at 20 C. Different times of hydrolysis were investigated and the optimum period was found to be 30 minutes. They were then given three washes in distilled water for 10 or 15 minutes and stained for 120 min in Schiff's reagent at pH 2.2. The material was then rinsed three times in SO_2 water for 10 minutes each rinse, kept in distilled water (5 to 15 minutes) and squashed in 45% acetic acid. The coverslip was removed after freezing with CO_2 and the slide dehydrated in absolute alcohol and mounted in Euparal. The amount of Feulgen staining per nucleus, expressed in arbitrary units, was measured at a wavelength of 570 nm using the scanning method in a Zeiss Universal Microspectrophotometer (UMSP 30). The DNA content per basic genome expressed in pg was calculated using *A. cepa* var. Ailsa Craig as a standard. The differences in DNA content were tested through an analysis of variance and comparisons between means using Scheffe's method.

The results obtained are shown in the table.

Experiment	Individual	No. nuclei measured	DNA content (2C) pg X	S.E.
1	1	24	5.9289	0.1103
	2	36	5.5411	0.1055
2	3	26	5.8013	0.1085
	4	36	5.7059	0.1033
3	5	34	5.7817	0.0673
	6	33	5.8196	0.1044
	7	28	6.0128	0.0762

An Anova test was made, and it was determined that the results do not show significant differences among themselves ($F=2.43$; $p<0.01$). The results obtained point out a DNA average amount for "*c*-tester" of 2C=5.78±0.0965 pg.

In vitro culture of 0.15, 0.25 mm immature embryos. I. Picloram effects

--M. D. Garcia, M. del C. Molina and O. Caso

Plants have been obtained from isolated immature embryos cultured on nutrient media in the absence of any plant growth regulator (Haagen-Smit et al., Science 101:243, 1945; Sheridan et al., MNL 52:88-90, 1978; Van Lammeren, Acta Bot. Neerl. 37:49-61, 1988). However, no plants were regenerated from embryos smaller than 0.3 mm in length (about 9 days after pollination). Picloram and kinetin combinations increased the growth opportunity of 0.3 to 0.6 mm length maize embryos excised and cultured in vitro (Garcia et al., unpublished).

The aim of this study was to obtain maize plants through 0.15 to 0.2 mm length. Three different Picloram concentrations (in combination or not with kinetin) were evaluated.

Plants of cv. Ever Green were grown under field conditions and auto- or sib-pollinated in the summer of 1989-1990. Embryos were excised from caryopses between 7 and 9 days after pollination (0.15 to 0.25 mm length) and cultured on 6 differed solidified nutrient media. All of them contained the inorganic components of N6 medium (Chu, Proc. Symp. Plant Tissue Culture, 43-45, 1978) with added 0.025 mg/l Na_2MoO_4, 0.025 mg/l $CuSO_4$, 0.025 mg/l CoCl, vitamins of Haagen-Smit (1945), 1500 mg/l asparagine, 5% sucrose and 6 different combinations of Picloram and kinetin (Table 1).

The embryos and subsequent seedlings were incubated at 28-30 C with a 16 hour photoperiod from cool white fluorescent lights with an intensity of 2500 Lx. Plant frequency (P.F.) obtained in every culture medium was

Table 1. Plant growth regulator concentrations of culture media

Plant growth regulator (mg/l)	Kinetin (0.05)	Without kinetin
Picloram 0.05	G	J
Picloram 0.1	H	K
Picloram 0.2	I	L

calculated as No. of embryos which formed plants/total no. of embryos x 100. After a month in culture, plants were transferred to pots with a mixture of soil-sand (2:1), placed in the greenhouse and grown to maturity.

Embryos in culture showed different responses (Table 2), but most of them germinated prematurely and formed plants (Graphic 1).

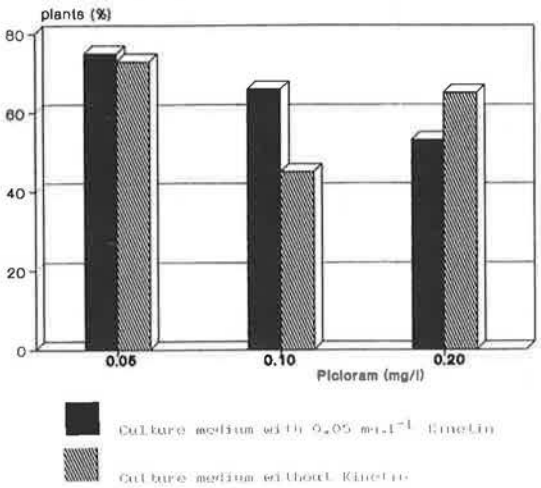
Table 2. Abnormalities observed on embryo germination.

Culture media	% of non-growing embryos	% of callus	% of plants with radicular callus	% embryos producing root development	% of growing scutellums without germination
G	0	0	0	0	23.5
H	5.5	0	16.6	0	5.5
I	5.8	17.6	23.53	0	0
J	0	0	13.3	0	0
K	15	0	10	5	0
L	0	21.7	21.7	0	0

The embryos on medium J germinated at about 5 days in culture, before the other ones, and showed the least number of abnormalities. However, plants obtained from these were small, not very vigorous, and old leaves got necrotic before a month in culture. 15.79% of these embryos died after germination.

Embryos on medium G formed the largest frequency and the most vigorous plants. Plants obtained on media H, I and L showed very curled leaves.

Graphic 1: Plant frequency obtained from embryos on different culture media.



The largest and more heterogeneous scutellum growth before germination was on media with kinetin (Table 3).

In conclusion, the results demonstrate that through the use of a low concentration of Picloram and kinetin (0.05 mg/l of each one) in the culture medium it is possible to obtain a high maize plant frequency from embryos less than 0.3 mm length through premature germination.

Table 3. Average of scutellum growth before germination.

Culture media	Average of scutellum growth (mm)
G	3.34
H	3.53
I	3.34
J	2.04
K	1.96
L	1.79

In vitro culture of 0.15-0.25 mm immature embryos. II. 2,4-dichlorophenoxy acetic acid (2,4-D) effects

--M. D. Garcia, M. del C. Molina and O. Caso

2,4-D has been the most commonly used plant growth regulator to induce callus formation from immature embryos of maize and other *Zea* species. 2,4-D has frequently induced regeneration by organogenesis (Green and Phillips, Crop Sci. 15:417-420, 1975; Springer et al., Protoplasma 101:269-281, 1979; Lowe et al., Plant Sci. 41:125-132, 1985) or somatic embryogenesis (Lu et al., Theor. Appl. Genet. 62:109-112, 1982; Vasil et al., Protoplasma 127:1-8, 1985; Franz and Schel, Acta Bot. Neerl. 36:247, 1987, among others). Regeneration ability depends mainly upon culture conditions, genotype (Tomes and Smith, Theor. Appl. Genet. 70:505-509, 1985) and embryo age (Rapela, MNL 59:59, 1985).

The aim of this work was to obtain a method of regenerating a high frequency of maize plants through organogenesis or somatic embryogenesis. This method could be used to get plants from difficult genotypes.

Plants of cv. Ever Green were grown under field conditions and auto- or sib-pollinated in the summer of 1989-1990. Embryos were excised and cultured on media as in the preceding article, with 6 different combinations of 2,4-D and kinetin (Table 1).

Table 1. Plant growth regulator concentration of culture media.

Plant growth regulators (mg/l)	Kinetin (0.05 mg/l)	Without kinetin
2,4-D 0.05	A	D
2,4-D 0.1	B	E
2,4-D 0.2	C	F

The embryos and subsequent calli and plantlets were incubated at 28-30 C with a 16 hour photoperiod from cool white fluorescent lights calculated as No. of embryos which formed embryoids/total no. of embryos x 100. After two months in culture, regenerated plants were transferred to pots with a mixture of soil-sand (2:1), placed in the greenhouse and grown to maturity.

No plant regeneration was obtained through zygotic embryo germination. Some embryos developed roots or soft non-embryogenic calli near the coleorhiza. These calli turned yellow to brown and died or produced roots. A low number of embryos showed no growth (Table 2).

The other ones showed a compact, white scutellum

Table 2. Percentage of embryos producing non-embryogenic callus

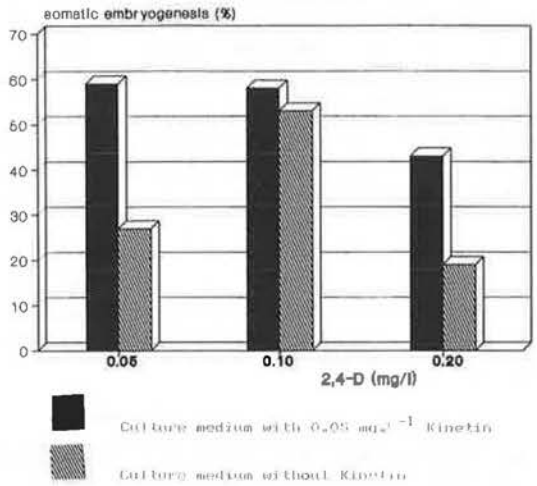
Culture media	% of non-growing embryos	% of embryos forming roots or radicular callus
A	0	41.20
B	21.05	21.06
C	6.25	43.75
D	6.66	40.00
E	13.33	33.33
F	14.28	66.66

growth. At the beginning of culture the surface was smooth. About 4 to 7 days in culture, swellings were seen along the scutellum and soft calli derived from the radicle embedded in the medium. White scutellum-like structures appeared at the surface after a week in culture. Green coleoptiles emerged from these structures. Scutellar notches were either median (like zygotic embryos) or terminal. Root growth was also observed and often covered somatic embryos.

Somatic embryos kept on 2,4-D containing medium either showed slow germination or fused together forming a white, compact callus. Further production of plants from this callus was by proliferation of adventitious shoots on media with 0.5 to 1 mg/l 2,4-D.

Regeneration was obtained only from the white, compact tissue that arose from scutellum, also called type I by Armstrong and Green (Planta 164:207-214, 1985).

Graphic 1:Somatic embryogenesis frequency induced by different 2,4-D concentrations.



Embryos on medium with 0.05 mg/l kinetin produced the largest frequency of somatic embryogenesis. Similar frequencies were obtained on media with 0.1 mg/l 2,4-D with or without kinetin (Graphic 1).

Somatic embryos germinated when they were transferred to a medium with 0.1 mg/l Picloram.

LLAVALLOL, ARGENTINA
Universidad Nacional de La Plata

Maize-Balsas teosinte and maize-Guatemala teosinte hybrids: inheritance of plant traits

--Víctor Raúl Corcuera (2)

In connection with the study of the possibilities offered by Balsas teosinte (*Zea mays* ssp. *parviglumis* var. *parviglumis*) and Guatemala teosinte (*Z. luxurians*) as wild genetic resources of maize (*Z. mays* ssp. *mays*), six plant traits were measured in F1 and F2 progenies derived from crosses between maize x Balsas teosinte and maize x Guatemala teosinte. The same traits were also measured in their respective parents, and in all the cases on the basis of individual plants.

The traits measured in the different populations were the following: 1) plant height (PH), 2) stalk diameter (ST), 3) number of leaves per tiller (NLT), 4) leaf width (LW), 5) leaf length (LL) and 6) number of tillers per plant (NTP).

Taxonomically, maize and Balsas teosinte are grouped in Section *Zea* whilst Guatemala teosinte belongs to Section *Luxuriantes* (Doebley and Iltis, 1980). Maize has short and narrow leaves, few tillers per plant, and is not very tall, with a low number of leaves and great stalk diameter compared to teosinte species.

The descriptors evaluated are quantitatively inherited, and according to the results shown in Tables 1 to 4, the following can be deduced:

1) Stalk diameter and plant height average values found both in F1 and F2 progenies are very similar to the mid-parent value. This fact denotes that these traits are controlled by genes with additive effect.

2) The same phenomenon is observed when one analyzes what happens with the trait, number of leaves per tiller.

3) Maize-Balsas teosinte and maize-Guatemala teosinte F1 and F2 hybrids show leaves as short and wide as maize. This fact lets us deduce that maize is dominant over teosinte species for these traits.

4) Finally the F1 and F2 progenies derived from both crossings show number of tillers lower than the mid-parent, and displaced to the left side (maize). Thus it could be said that maize is not completely dominant for this trait

Table 2. Differences between means for plant traits of OU maize inbred line (P1), Balsas teosinte (P2), F1 and F2 populations and mid-parent value (MP)

	PH (cm)	ST (cm)	NLT	LW (cm)	LL (cm)	NTP
P1	150,0 a*	1,6 a	14,6 a	7,3 a	74,5 a	1,0 a
P2	280,0 b	0,4 b	18,0 b	3,8 b	93,0 b	22,6 b
F1	221,0 c	0,8 c	19,0 b	8,3 a	64,2 c	8,8 c
F2	238,0 d	0,9 c	16,3 c	6,3 c	65,2 c	3,6 d
MP	215,0 c	1,0 c	16,3 c	5,5 c	83,7 d	11,8 e

*Individual means within a column followed by different letters are significantly different at 1% level.

Table 4. Differences between means for plant traits of OU maize inbred line (P1), Guatemala teosinte (P3), F1 and F2 populations and mid-parent value (MP).

	PH (cm)	ST (cm)	NLT	LW (cm)	LL (cm)	NTP
P1	150,0 a*	1,6 a	14,6 a	7,3 a	74,5 a	1,0 a
P3	300,5 b	0,4 b	21,2 b	3,4 b	117,0 b	19,5 b
F1	241,0 c	0,9 c	21,3 b	7,3 a	61,7 c	6,5 c
F2	229,0 d	1,0 d	17,5 c	7,4 a	62,0 c	2,9 d
MP	225,2 d	1,0 d	17,9 c	5,3 c	95,7 d	10,2 e

*Individual means within a column followed by different letters are significantly different at 1% level.

Table 1. Means, standard deviation and ranges for plant traits in the OU maize inbred line (P1), Balsas teosinte (P2) and F1 and F2 populations.

	PH* (cm)			ST (cm)			NLT			LW (cm)			LL (cm)			NTP		
	Mean ±	SD	Range	Mean ±	SD	Range	Mean ±	SD	Range	Mean ±	SD	Range	Mean ±	SD	Range	Mean ±	SD	Range
P1	150,0	20,0	100-180	1,6	0,18	1,2-2,0	14,6	1,4	10-17	7,3	0,7	5,5-8,5	74,5	6,2	55-82	1,0	0,0	---
P2	280,0	30,4	243-325	0,4	0,05	0,3-0,5	18,0	3,8	16-24	3,8	0,4	3,2-4,7	93,0	8,9	80-106	22,6	9,6	11-43
F1	221,0	25,0	152-267	0,8	0,10	0,5-1,0	19,0	1,5	16-22	8,3	1,9	4,0-11,5	64,2	15,4	29-87	8,8	3,0	3-14
F2	238,0	30,0	165-295	0,9	0,26	0,6-1,4	16,3	2,5	13-22	6,3	0,9	5,0 7,5	65,2	10,4	51-78	3,6	2,0	2-11

*see abreviations in paragraph 2.

Table 3. Means, standard deviation and ranges for plant traits in the OU maize inbred line (P1), Guatemala teosinte (P3) and F1 and F2 populations.

	PH* (cm)			ST (cm)			NLT			LW (cm)			LL (cm)			NTP		
	Mean ±	SD	Range	Mean ±	SD	Range	Mean ±	SD	Range	Mean ±	SD	Range	Mean ±	SD	Range	Mean ±	SD	Range
P1	150,0	20,0	100-180	1,6	0,18	1,2-2,0	14,6	1,4	10-17	7,3	0,7	5,5-8,5	74,5	6,2	55-82	1,0	0,0	---
P3	300,5	31,2	260-330	0,4	0,06	0,3-0,5	21,2	2,5	18-25	3,4	0,5	2,0-4,0	117,0	6,7	102-132	19,5	11,2	8-57
F1	241,0	23,0	180-290	0,9	0,10	0,7-1,0	21,3	1,7	16-25	7,3	0,8	5,5-9,1	61,7	11,2	27-77	6,5	1,9	3-10
F2	229,0	45,0	130-330	1,0	0,30	0,6-1,4	17,5	2,9	14-24	7,4	0,8	6,0-8,4	62,0	5,3	56-72	2,9	1,7	3-7

*see abreviations in paragraph 2.

(dominance in low degree).

The explanations given in the previous paragraphs are completely coincident with the intermediate phenotype between maize and teosinte shown by the F1 and F2 plants of both crossings. This fact is clearly different from what has previously been seen in diploperennial teosinte-maize hybrids (Corcuera and Magoja, 1988).

Last, it can be added that those differences between maize and the teosinte species somehow let interspecific hybrids between them express a wide range of variation. This variation is generally high and useful for maize breeding projects. In addition the high variability shown by plant traits in F1 as well as in F2 progenies is another sign of all possibilities offered by wild relatives to increase the narrow genetic base of maize crops.

Maize-Balsas teosinte and maize-Guatemala teosinte hybrids: inheritance of prolificity

--Víctor Rául Corcuera

Generally, hybrids between maize and its nearest wild relatives--the teosintes--are highly heterotic, especially when considering prolificity traits. This fact is easy to appreciate in 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 and MNL 62:77, 1988). Evidently, prolificity is a 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, teosinte species are really very prolific, though in different intensity depending on the species considered. Nevertheless, prolificity reaches its maximum expression in interspecific hybrids between maize and its wild relatives.

Prolificity traits were measured in F1 and F2 progenies derived from crossings between maize and Balsas teosinte, maize and Guatemala teosinte, and their respective parents. The traits considered in this study were: 1) number of productive nodes per tiller (NP), 2) number of ears in the uppermost node (EUN), and 3) number of ears per tiller (ET). All of them were measured on the basis of individual plants, when these were completely mature.

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) and diploperennial teosinte-maize hybrids (see MNL 62:77, 1988). These results are shown in Tables 1 to 4.

As can be seen in Tables 2 and 4, the average values found for the F1 and F2 progenies highly exceed the mid-parent. In addition the simple observation of the average values for the number of ears in the uppermost node (EUN) demonstrates that it is not only higher than the mid-parent average value, but significantly exceeds the most prolific parent values too. Even a similar fact can be

Table 1. Means, standard deviation and ranges for prolificity traits in the OU maize inbred line (P1), Balsas teosinte (P2) and F1 and F2 populations

	NP			EUN			ET		
	Mean ±	SD	Range	Mean ±	SD	Range	Mean ±	SD	Range
P1	1,2	0,4	1-2	1,0	0,0	---	1,2	0,4	1-2
P2	8,3	1,1	6-9	2,3	0,6	1-3	35,6	10,3	21-58
F1	7,1	1,6	4-12	4,6	1,5	3-9	33,9	11,2	17-53
F2	5,8	2,5	3-11	5,4	2,5	3-12	48,3	27,1	14-84

Table 2. Differences between means for prolificity traits of OU maize inbred line (P1), Balsas teosinte (P2), F1 and F2 populations and mid-parent value (MP)

	NP	EUN	ET
P1	1,2 a(*)	1,0 a	1,2 a
P2	8,3 b	2,3 b	35,6 b
F1	7,1 b	4,6 c	33,9 b
F2	5,8 c	5,4 c	48,3 c
MP	4,7 d	1,6 d	18,4 d

(*) Individual means within a column followed by different letters are significantly different at 1% level.

Table 3. Means, standard deviation and ranges for prolificity traits in the OU maize inbred line (P1), Guatemala teosinte (P3) and F1 and F2 populations.

	NP			EUN			ET		
	Mean	± SD	Range	Mean	± SD	Range	Mean	± SD	Range
P1	1,2	0,4	1-2	1,0	0,0	---	1,2	0,4	1-2
P3	10,6	2,7	8-12	3,7	1,0	2-5	68,9	35,5	23-157
F1	7,4	1,3	5-10	4,9	1,9	3-12	32,5	13,2	13-87
F2	9,2	2,9	4-11	6,2	1,7	3-14	57,4	26,6	16-95

Table 4. Differences between means for prolificity traits of OU maize inbred line (P1), Guatemala teosinte (P3), F1 and F2 populations and mid-parent value(MP)

	NP	EUN	ET
P1	1,2 a(*)	1,0 a	1,2 a
P3	10,6 b	3,7 b	68,9 b
F1	7,4 c	4,9 c	32,5 c
F2	9,2 b	6,2 d	57,4 d
MP	5,9 d	2,3 e	35,0 c

(*) Individual means within a column followed by different letters are significantly different at 1% level.

pointed out if one considers the number of ears per tiller (ET) in the hybrids between maize and Balsas teosinte.

Prolificity traits frequency distribution is completely displaced to the right side, relative to their parental frequency distributions. From the results obtained, it can be settled down that prolificity traits express a high degree of heterosis, especially if one refers to number of ears in the uppermost node (EUN) and ears per tiller (ET) in hybrids between maize and Balsas teosinte.

According to the results obtained, it can be stated that prolificity traits are transgressively inherited in a positive direction (heterosis), as happens in diploperennial teosinte-maize hybrids (see MNL 62:77, 1988). It could also be added that maize-Balsas teosinte hybrids seem to be more prolific than those obtained between maize and Guatemala teosinte.

Finally it is important to point out that this particular mechanism of inheritance for prolificity traits has a high practical value, as through the utilization of wild germplasm the prolificity degree of cultivated maize could be significantly increased.

Maize-Balsas teosinte and maize-Guatemala teosinte hybrids: inheritance of evolutive cycle

--Víctor Rául Corcuera

At the latitude in which most cultivated maize is concentrated in Argentina (35° southern latitude), normally all the teosinte species express a strong photoperiod response, flowering during autumn (April-May). Thus, it can be said that they show a long evolutive cycle. Under these environmental conditions, annual teosintes like diploperennial teosintes have an evolutive cycle measured in number of days from plant emergence up to tasselling that is 3 times longer than cultivated maize (190 days against 60 days for maize). It can also be added that annual teosintes must accumulate 560 C to tassel, whilst maize only needs approximately 250 C.

Evolutive cycle traits were measured in 7 different populations to obtain the basic information for future projects in which annual teosinte germplasm could be used for maize genetic improvement. The 7 populations studied are: maize inbred line OU (P1), Balsas teosinte (P2), Guatemala teosinte (P3), and the F1 and F2 progenies derived from crossing maize with each one of these teosinte species.

On the basis of individual plants, cycle traits measured were: 1) days to tassel (T), 2) days to silking (S) and 3) days to pollen (P). The results obtained are summarized in Tables 1 to 4. It can easily be noted that in both crossings the average values both for the F1 and F2 progenies are smaller than the mid-parent value (MP). At the same time,

Table 1. Means, standard deviation and ranges for evolutive cycle traits in the OU maize inbred line (P1), Balsas teosinte (P2) and F1 and F2 populations

	Days to tassel			Days to silking			Days to pollen		
	Mean	± SD	Range	Mean	± SD	Range	Mean	± SD	Range
P1	60,8	2,5	59-70	71,0	2,1	66-74	73,0	1,4	69 76
P2	181,5	4,3	176-198	186,7	3,0	179-197	193,8	3,5	187 204
F1	110,2	7,0	98-136	130,0	2,3	120-133	124,7	3,6	119-133
F2	90,4	14,5	62 121	108,7	12,6	76-136	100,4	14,5	70-128

Table 2. Differences between means for evolutive cycle traits of OU maize inbred line (P1), Balsas teosinte (P2), F1 and F2 populations and mid-parent value (MP).

	T	S	P
P1	60,8 a(*)	71,0 a	73,0 a
P2	181,5 b	186,7 b	193,8 b
F1	110,2 c	130,0 c	124,7 c
F2	90,4 d	108,7 d	100,4 d
MP	121,1 e	128,8 c	133,4 e

(*) Individual means within a column followed by different letters are significantly different at 1% level.

Table 3. Means, standard deviation and ranges for evolutive cycle traits in the OU maize inbred line (P1), Guatemala teosinte (P3) and F1 and F2 populations

	Days to tassel			Days to silking			Days to pollen		
	Mean	± SD	Range	Mean	± SD	Range	Mean	± SD	Range
P1	60,8	2,5	59-70	71,0	2,1	66-74	73,0	1,4	69-76
P3	185,5	2,9	176-198	188,1	2,7	179-198	200,1	4,1	194-206
F1	108,0	6,5	96-125	130,5	1,9	129 136	124,3	4,3	119-136
F2	94,8	16,5	62-123	109,7	14,9	71-134	104,6	14,9	68 129

Table 4. Differences between means for evolutive cycle traits of OU maize inbred line (P1), Guatemala teosinte (P3), F1 and F2 populations and mid-parent value (MP).

	T	S	P
P1	60,8 a(*)	71,0 a	73,0 a
P3	185,5 b	188,1 b	200,1 b
F1	108,0 c	130,5 c	124,3 c
F2	94,8 d	109,7 d	104,6 d
MP	123,1 e	129,5 c	136,5 e

(*) Individual means within a column followed by different letters are significantly different at 1% level.

the amplitude of variation for each trait considered is wide enough to reach their parent extreme values (see Tables 1 and 3).

The results obtained point out that cycle traits are quantitatively inherited: a short cycle (maize) is dominant, though in a medium degree, over a long cycle (teosinte). This fact indicates that maize-Balsas and maize-Guatemala teosinte hybrids behave in a similar way to diploperennial teosinte-maize hybrids (see MNL 62:77, 1988).

In addition, the evolutive cycle traits measured are closely entailed among themselves and their hereditary behaviour is very similar. Otherwise, as in previous publications (see MNL 62:77, 1988), these hybrids are protandrous.

The results obtained demonstrate that it would be relatively easy to select short evolutive cycle individuals derived from these crossings, though this is not a surprising fact, as previous investigations we have done already demonstrate it. Nevertheless, it is really very important and advantageous that individuals derived from these non-traditional crosses and expressing a short cycle duration can be chosen amongst the whole, as they may be grown in the field and mature under normal environmental conditions.

LLAVALLOL, ARGENTINA
Universidad Nacional de La Plata
LOMAS DE ZAMORA, ARGENTINA
Lomas de Zamora University

Diploperennial teosinte-maize hybrids: inheritance of male spikelet outer glume traits

--V. R. Corcuera and J.. L. Magoja

According to Doebley and Iltis (1980), two characteristics of the male spikelet outer glume, number of veins between primary lateral veins (VBL) and total vein number (TV), are of such taxonomical importance that the two different sections of the genus *Zea* can be clearly distinguished through them. We have verified this singular observation (see MNL 60:81) when those traits were quantified in the teosintes, and we have also pointed out that both VBL and TV are characteristics which can be used to detect teosinte (belonging to Section *Luxuriantes*) germplasm introgression in maize (see MNL 61:64).

Bearing in mind what has previously been exposed, it can be deduced that VBL and TV are two traits which would perfectly let us discriminate the different taxa between the two sections of the genus *Zea*, but not within sections (see MNL 60:81). When hybrids among different taxa belonging to different sections of the genus *Zea* are done, the clear differences existing for those traits of the male spikelet outer glume let one study the way of inheritance of them.

Continuing the studies referred to on the inheritance or expression of specific traits or ones of agronomic importance in hybrids between diploperennial teosinte (*Z diploperennis*) and maize (see MNL 62:77), we have recently evaluated VBL and TV in these hybrids and their parents.

A sample of male spikelets taken at random from the primary branches of the medium third of the tassel was used to evaluate these traits (VBL and TV) in a diploperennial teosinte population, another one of a sweet maize variety (Ever Green), and the F1 and F2 progenies resulting from the cross between the two species.

The outer glumes were diaphanized and stained with safranine and then observed with a stereomicroscope to count the number of veins. The results shown in this work were obtained after averaging all the data belonging to four male spikelet outer glumes taken from one tassel of each plant.

In Tables 1 and 2 the clear differences for the traits studied between diploperennial teosinte and maize can be seen, and the F1 and F2 mean values are nearer to maize than to teosinte. When the averages obtained for the four populations studied are statistically compared it can be deduced that the F1 and F2 mean values are significantly lower than those of the mid-parent.

Table 1. Number of plants (N), means and range for VBL and TV traits in diploperennial teosinte (Zd), Ever green maize (Eg) and F_1 and F_2 populations.

		VBL		TV	
	N	Mean ± SD	Range	Mean ± SD	Range
P_1 (Zd)	25	8.8 ± 0.6	6-11	14.2 ± 0.8	11-17
P_2 (Eg)	25	4.1 ± 0.4	3-5	8.1 ± 0.4	7-10
F_1	21	5.6 ± 0.3	5-7	10.5 ± 0.5	9-12
F_2	75	5.3 ± 1.0	2-9	9.7 ± 1.4	6-13

Table 2. Differences between means for VBL and TV traits of diploperennial teosinte (Zd), Ever green maize (Eg), F_1 and F_2 population and mid-parent values (MP).

	VBL	TV
P_1 (Zd)	8.8^{a} (*)	14.2^{a}
P_2 (Eg)	4.1^{b}	8.1^{b}
F_1	5.6^{c}	10.5^{c}
F_2	5.3^{c}	9.7^{d}
MP	6.5^{d}	11.2^{e}

(*) Individual means within a column followed by different letters are significantly different at 5% level.

The frequency distributions shown in Figure 1 show a relatively wide range of variation for the F2, but without

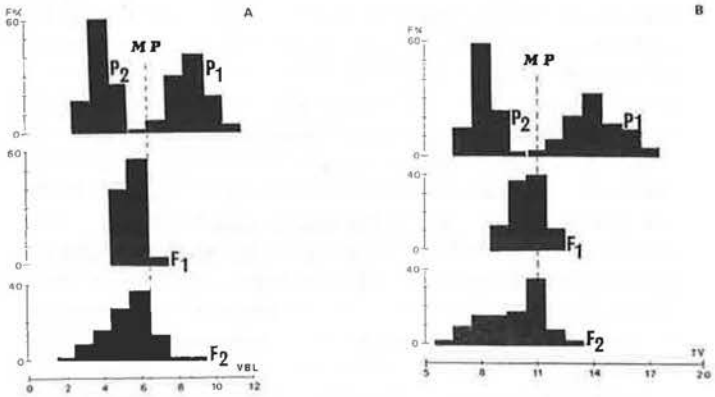


Figure 1. Frequency distributions for VBL (a) and TV (b) traits in diploperennial teosinte (P1), Ever green maize (P2) and its F1 and F2 populations - MP: mid-parent value.

reaching the greater extreme values (teosinte). The displacement to left of those distributions (toward maize) and the significant differences between the F1 and F2 mean values with respect to the mid-parent, suggest that both VBL and TV are quantitatively inherited, considering partial dominance of a low total vein number (maize) over high (teosinte). Both traits are strongly associated; the correlation coefficient among the mean values of the four populations considered is 0.99 (significant at 1% level) and among the F2 progeny plants is 0.77 (also significant at 1% level).

The study of the expression of these specific traits has given enough information to explain the causes by which our teosinte (belonging to Section *Luxuriantes*) introgressed populations of maize show higher values for VBL and TV than the normal ones (see MNL 61:64).

Bearing in mind that these populations were obtained after backcrossing them with maize (recurrent parent) and that almost all the traits of the cultivated species (maize) have been recovered, explaining the causes by which high VBL and TV values remain as introgression signs is only satisfactory on the basis of the partial dominance of high vein number over low.

LONDON, ONTARIO, CANADA
The University of Western Ontario

Transient expression of *B-peru* gene in bombarded (PDS-2000) immature shoot apical meristems

--V. R. Bommineni, D. B. Walden and J.C. Sanford*

*New York State Agricultural Research Experiment Station, Cornell University, Geneva, New York

A helium gas driven device (PDS-2000, 'wand' design) was used to transform immature shoot apical meristems (MNL 63: 87-88; Plant Cell Tissue Organ Culture 19:225-234, 1989) with a dominant anthocyanin *B-peru* gene color marker (gene construct was provided by Dr. Vicki L. Chandler, University of Oregon).

Shoot apical meristems were dissected from 12-14 days old pollinated ears of *a C R b pl*, and *yg2 c sh wx b pl* genotypes and arranged in a circle on the agar plate in such a way that the apical dome of the meristem was facing towards the microprojectile source. The procedure for preparation of DNA was described by Klein et al. (Bio/Technology 6:339-563, 1988) with the following modifications. Mix and vortex all the components (gold particles, DNA, $CaCl_2$, and spermidine) and leave the solution in the microtubes for 5 min. Decant the supernatant and rinse the particle mixture with 140 µl of 70% EtOH and pulse vortex, decant the supernatant and rinse with 140 µl of 100% EtOH. Pulse vortex again, decant the supernatant and resuspend the particles with 48 µl of 100% EtOH. Sonicate the mixture three times at one second intervals. Place 5 µl of suspended microprojectile solution on flying disc (kapton membrane) in a ring, and set them in a small dessicator in the culture hood until use. The design and operation of the gun was described by Johnston (Nature 346: 776-777, 1990).

Table 1. Transient expression (96h) of *B-peru* gene in bombarded* immature apical meristems.

Genotype and treatment**	No DNA** (N)	No DNA** # of spots	Number of explants bombarded	Number of Explants expressed *B-peru* gene	Mean number of brown spots (±S.E)** S	P	R	Mean total # of spots (± S.E.)
a C R b pl (2 µg/µl)								
1x								
1st shelf	10	0	32	30	16.3(2.5)	7.6(1.1)	5.4(0.7)	29.3(3.5)
2nd shelf	10	0	41	31	3.0(0.8)	1.8(0.4)	1.6(0.4)	6.4(1.0)
3rd shelf	11	0	19	11	4.5(1.9)	2.2(0.6)	2.5(1.5)	9.2(3.0)
Total	31	0	92	72	8.8(1.4)	4.3(0.6)	3.3(0.5)	16.4(2.1)
3x								
1st shelf	9	0	18	17	6.9(1.6)	3.5(0.3)	4.4(0.8)	14.8(2.2)
2nd shelf	12	0	13	12	8.8(2.3)	6.6(1.2)	6.8(1.7)	22.2(4.3)
3rd shelf	9	0	22	19	11.6(2.3)	7.1(2.3)	8.1(1.4)	26.8(4.2)
Total	30	0	53	48	9.3(1.3)	5.7(1.0)	6.4(0.8)	21.4(2.3)
yg c sh wx b pl (10 µg/µl)								
1x								
1st shelf	15	0	18	17	22.2(4.5)	38.4(8.1)	43.4(6.7)	104.0(14.3)
2nd shelf	14	0	21	19	12.9(2.4)	6.3(2.0)	23.4(5.7)	44.6(9.1)
3rd shelf	15	0	18	15	4.8(2.9)	3.4(1.5)	13.5(9.0)	21.7(13.1)
Total	44	0	57	51	13.6(2.2)	16.1(3.6)	27.3(4.5)	57.0(8.5)

*Helium gas at 900 psi; 1 µm gold particles were used. 1st shelf = most distant (11 cm) from delivery source.
**(N)=number of meristems; S=scutellum portion; P=plumule (leaves); R=radical portions of a growing plant at the time of transient assay; S.E.=standard error of the mean.

A summary of the transient expression (96 h) of *B-peru* gene in bombarded shoot apical meristems is provided in Table 1. Many of the bombarded explants expressed the *B-peru* gene. A larger number of spots was observed in the meristems bombarded once (1x) on the lowest (1st) shelf than on the 3rd shelf; however, bombardment three times produced a fewer number of spots in the meristems located on the 1st shelf than on the 3rd shelf. The pattern of *B-peru* gene expression was consistent among the meristematic portions (scutellum, plumule or leaf, and radicle). A similar pattern was observed between the two genotypes, but approximately four times more expression was observed with 10 µg/µl DNA (*yg2 c sh wx b pl*) than 2 µg/µl DNA (*a C R b pl*) in the microprojectile solution.

Mature plants from cultured shoot apical and tassel meristems

--V. R. Bommineni and E. Banasikowaska

Mature plants from cultured shoot apical meristems through "jiffy pot" technique: We reported the successful recovery of plantlets through shoot apical meristem culture (MNL 63: 87-88 and 64: 79-79; Plant Cell Tissue Organ Culture, 19: 225-234, 1989). The in vitro culture technology imposes stress upon the developing plantlets. We have developed a "jiffy pot" technique to minimize some of the stress-related conditions in the transfer sequence of excised meristem to plantlet, and at the same time to enable developing plantlets to be shipped in large numbers from a laboratory to a remote nursery.

The jiffy pots can be obtained from a garden/plant nursery store. First, the jiffy pots were soaked in water (in a tray) until the pots expand completely. The pots were then placed in Magenta(TM) plant cell culture vessels (3" x 3" x 4") (Sigma Chemical Company, USA) and the vessels autoclaved.

Murashige and Skoog's basic nutrient agar medium

Table 1. Plants from cultured immature apical meristems (*a C R b pl*).

Treatment	Number			
	meristems cultured	plantlets recovered	plants matured	percent of plants recovered
Agar block on jiffy pot (M&S medium)	40	27	4	10
Cultured in petri dishes 72 h; explant removed with agar block; placed on jiffy pot	20	20	20	100
Cultured in petri dishes 3 weeks; transferred to peat pots (control)	21	17	17	81

(MNL 63: 87-88; Plant Cell Tissue Organ Culture 19: 225-234, 1989) was used to support the explant apical meristems. One and one-half cm diameter agar blocks were removed and pressed into the autoclaved jiffy pot (top centre) under sterile conditions. The meristems were excised and placed on the agar blocks as described in Table 1. Two different methods were followed to compare the recovery of plantlets with the existing method (control, Table 1).

Recovery of fertile plants through in vitro culture of tassel meristems in Magenta plant cell culture vessels: We reported a summary of the recovery of plantlets from cultured tassel meristems in 125 ml conical flasks (Maydica 34: 263-275, 1989) derived from Oh43 and K21 genotypes. However, the number of plantlets recovered and the number of plantlets matured through that culture method were very low. To improve the success rate and increase the scale of the effort, Magenta plant cell culture vessels were used in the place of 125 ml conical flasks. As reported 40 ml of Murashige and Skoog's nutrient medium was placed into the vessels and the vessels were autoclaved.

The initial length of tassel meristems ranges from 1.0 to 1.5 cm; the tassel meristems were dissected by removing the sheath leaves, the dissecting meristems were placed into the Magenta vessels and incubated at 26+2 C for 25+ days. The plantlets which emerged after 25+ days of culture were allowed to grow further in the vessel until they were ready to be transferred to soil pots in the glasshouse.

Table 2. Recovery of plants from cultured tassel meristems (Oh43).

Treatment	Number			Percent of plants recovered***
	tassels cultured	plantlets obtained	plants matured	
(a)	(b)	(c)	(d)	(e)
Flask	40 5	5*	2	5
Magenta(TM) Vessels	36	22**	7	19

* 1 - one plantlet, and 2 - two plantlets/flask.
** 7 - one plantlet, 2 - two plantlets, 1 - three plantlets, and 2 - four plantlets/Magenta(TM) vessel.
*** e = d x 100/b.

A summary of the recovery of plantlets from cultured tassel meristems in both flasks and vessels is included in Table 2. A higher percentage of plantlets (19%) were recovered through tassel meristems cultured in Magenta vessels comparing to meristems cultured in 125 conical flasks (5%).

Tolerance of maize and oat shoot apical meristems to brief cold temperatures and recovery of mature, fertile plants

--V. R. Bommineni, and E. Banasikowaska

The shoot apical meristems from 72 h imbibed mature seeds of maize, and immature grains of oat (summer grown crop plants), were used to examine the effect of a brief cold temperature (1 h) on the explant material. The plumules of maize and the immature whole grains of oat were surface sterilized in 10% "javex" for 30 min and exposed gradually to the low temperatures.

An 'Endocal Refrigerated Circulating Bath' (NESLAB, Model LT 50DD) was used to lower the temperatures at designated intervals. The samples were kept in a sterile test tube and the methanol temperature was lowered 1 C per every 5-10 min until the required temperatures were

Table 1. Recovery of mature plants from cold shocked apical meristems (72h imbibed seeds).

Genotype	Shock Temperature	Number		Percent mature Plants recovered
		Meristems Cultured	Plantlets recovered	
Oh43:				
	Control	20	10	50
	0° C	20	15	75
	-5° C	20	11	55
	-10° C	20	0	0
	-15° C	20	0	0
wxwx:				
	Control	10	8	80
	0° C	10	8	80
	-5° C	10	8	80
	-10° C	10	1	10

Table 2. Recovery of mature plants from cold shocked young apical meristems of oat (var. Donald).

Shock Temperature	Number		Percent mature Plants recovered
	Meristems cultured	Plantlets recovered	
Control	46	40	87
0° C	20	17	85
-5° C	18	16	89
-10° C	30	19	63
-15° C	21	1	5
-20° C	21	0	0
30° C	22	0	0

established (Tables 1 and 2). After attaining the appropriate temperature, the temperature was held at that level for 1 h and the samples removed. Similarly, the remaining samples were treated at different temperature levels for 1 h. Then the samples were kept (approximately 12 - 24 h) in a refrigerator (5 C) until the explants were cultured.

The shoot apical meristems were exposed under the microscope and transferred to Murashige and Skoog's nutrient medium (MNL 63: 87-88; Plant Cell Tissue Organ Culture 19: 225-234, 1989). After 2-3 weeks, the plantlets from the explanted meristems were transferred to pots in the glasshouse and grown in the glasshouse to maturity.

MADISON, WISCONSIN
University of Wisconsin

Preliminary results from the molecular analysis of intragenic recombination at the waxy locus

--Ron J. Okagaki

The waxy locus has been a focus for research on intragenic recombination in higher plants for almost 40 years. Most of the information on intragenic recombination is limited to genetic phenomenology, however this situation should change with the molecular characterization of these events. Ultimately we hope to come to an understanding of the rules governing the behavior of mutant heteroallelic combinations. In this report, data from Nelson's genetic studies on intragenic recombination at the waxy locus (MNL 50:109, 1976) is combined with molecular studies on the locus from the Wessler lab (PNAS 82:4177, 1985; PNAS 87:8731, 1990; and unpublished data) and other sources (Klosgen et al., MGG 203:237, 1986). This extensive body of data is available at the waxy locus first because of the large numbers of mutant alleles that have been recovered and second because the waxy phenotype can be determined by staining pollen with iodine. It was this ability to screen thousands of *wx* pollen grains for rare revertant *Wx* pollen grains that has made these studies possible.

Two aspects of intragenic recombinations are discussed here, the relationship between intragenic recombination frequency and the physical distance separating mutant lesions and second the effect of the size of mutant lesions on the ability of a mutant allele to recombine. Lastly, the outline of an ongoing project designed to study gene conversion and estimated the size of conversion tracts is presented.

A simplistic model of intragenic recombination would assume that the probability of intragenic recombination occurring between two sites is largely determined by the distance between the two sites. According to this model as the physical distance between two mutant lesions increases the frequency of recovering nonmutant alleles also increases. Figure 1 presents a graph of this relationship in waxy alleles which shows a weak association between increasing recombination frequency and increased physical distance. However, distance alone is not a strong predictor of recombination frequency between two alleles suggesting that additional factors influence intragenic recombination.

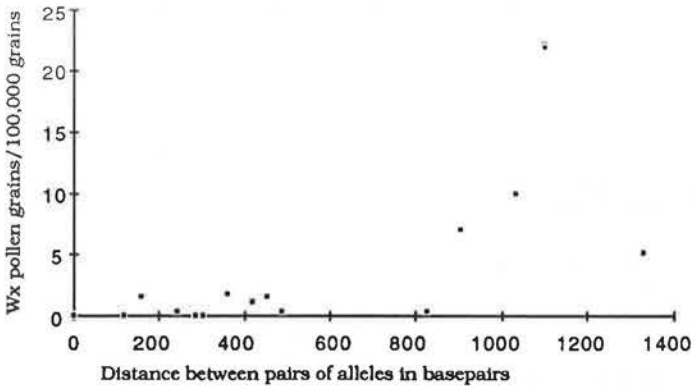


Figure 1.- The relationship between intragenic recombination frequency and physical distance. Recombination frequencies are presented as the number of nonmutant Wx pollen grains per 100,000 pollen grains counted. Frequencies below 1 Wx pollen grain per 100,000 are unreliable as nonmutant pollen grains can be detected at this frequency in crosses between two overlaping deletions (Ralston et al. TAG 74: 471, 1987). Each point represents one pair of wx alleles.

One possible explanation for the scatter in the data is the size of the lesion. Work at the bronze-1 locus has shown that *Ds* insertions compress the genetic fine structure map (Dooner, Genetics 113:1021, 1986), and to date only insertions and deletions have been placed on the waxy physical map. Insertions in the waxy locus range in size from approximately 0.15kb for *wx-B2* to over 5kb for *wx-Stonor* and *wx-M*, and deletions range from 30bp for *wx-C* and *wx-BL2* (note, these alleles have identical breakpoints) to *wx-C34* which deletes the entire locus. This approach takes into account both the distance between lesions and the size of lesions, and predicts that increasing the size of the lesion will decrease the rate of recombination. Data at the waxy locus do not allow the testing of this possibility. The difficulty lies with the confounding of the variables distance and size. It is likely that the size of lesions will affect intragenic recombination as has been found at the bronze-1 locus, but demonstration of this will require additional data. One observation can be made for alleles with large insertions, such as *wx-M* and *wx-Stonor* which are larger than the waxy locus itself; these insertion alleles are able to recombine with other alleles within the waxy locus. Insertions and deletions can inhibit intragenic recombination, but this suppression may be limited to short distances.

In an attempt to learn more about intragenic recombination a project to study gene conversion and estimate the size of conversion tracts was initiated. The molecular descriptions of the three mutant waxy alleles used in this study, *wx-C34*, *wx-B1*, and *wx-I*, have previously been published (PNAS 82:4177, 1985). *wx-C34* is a deletion of the entire waxy locus, and crossing the *Wx** alleles produced by intragenic recombination with *wx-C34* permits the analysis of *Wx** alleles without the complication of other waxy sequences in the genome. *wx-B1* and *wx-I* were chosen because Southern blot analysis can distinguish the lesions causing the mutations as well as the 5′ and 3′ ends of the alleles (Figure 2).

Homozygous *wx-B1* and *wx-I* plants were crossed to

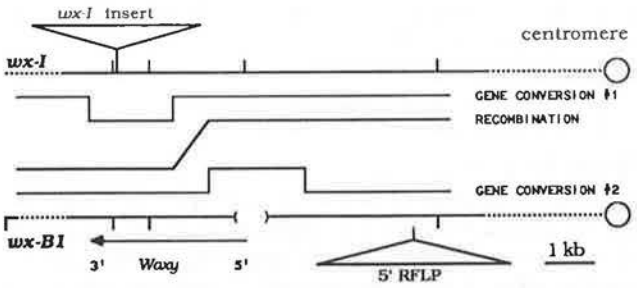


Figure 2.- Structures of alleles used in this study. The arrow dipicts the transcription unit oriented with the centromere. wx-I contains a large insertion in the 3' end of the gene and wx-B1 contains a deletion at the 5' end of the gene. Vertical ticks on the maps represent the SalI restriction enzyme sites used for restriction mapping. The 5' ends of the parental alleles were distinguished by an insertion polymorphism in wx-B1; the 3' SalI fragment was 16 kb in wx-B1 and too large to measure in wx-I. Two possible gene conversion events, types 1 and 2, and one cross-over event can generate a nonmutant waxy allele.

produce the heteroallelic mutant; these plants were crossed by *wx-C34* and nonmutant *Wx** plants. *Wx* kernels arising from pollination by stray *Wx* pollen were identified at this stage. Contaminants will contain a nonmutant *Wx* allele and either a *wx-I* or *wx-B1* allele instead of a *Wx** allele over the deletion of the waxy locus.

*Wx** alleles arising from gene conversion versus recombination were also distinguished by Southern blot analysis. Recombination generates *Wx** alleles with 5' flanking RFLPs from *wx-I* and 3' RFLPs from *wx-B1* (Figure 2). *Wx** alleles created through gene conversion should resemble one of the parental alleles unless the conversion tract is very large. To date four *Wx** alleles have been partially characterized. One allele arose through recombination, and three alleles have the flanking RFLPs of *wx-I* and apparently arose by gene conversion of the type 1 class (Figure 2). one putative *Wx** allele was determined to be a contaminant, and 35 additional *Wx** alleles have been isolated and await analysis.

Mosaic pericarp does not result from an *Spm* insertion

--Oliver Nelson

In the last issue of the Newsletter (64:81), I suggested that the unstable *P* allele, *P-mo*, could have resulted from the insertion of an autonomous *Spm* in a *P* allele. The basis for this suggestion was the observation that three different mosaic pericarp stocks from R. A. Brink's collection of *P* mutants all contained more than one active *Spm*. The test was made by crossing mosaic pericarp plants by *bz-m13* (a *dSpm* insertion in a *Bz* allele), and then crossing the F1 plants (*P-mo/P-wr; Bz/bz-m13*) by a *sh bz wx*; no *Spm* tester. If the F1 plant contains one or more *Spm*'s, then one observes a segregation for *bz*-variegated kernels. In a more extensive test of one of these F1's, almost every mosaic pericarp plant in the resulting progeny segregated *bz*-variegated (*bz -> Bz*) kernels showing that the plants contained an active *Spm*. The few exceptions--mosaic pericarp plants that were *Bz/Bz* or *Bz/bz*--were explicable on the basis of *dSpm* excision sufficiently early in development that the entire ear was derived from the cell in which that event occurred or by contamination. In addition, many nonmosaic pericarp plants segregated *bz*-variegated kernels. In all, 72% of the plants in the progeny had some *bz*-variegated kernels.

The three mosaic pericarp stocks from the Brink collection had each been backcrossed five times to the inbred, 4Co63. Tests made as outlined above of the 4Co63 line that I have been carrying and that also came from the Brink collection indicated that the inbred did not have an active *Spm* thus suggesting that the *Spm*'s were derived from the mosaic pericarp stocks and were carried along through five BC's to an inbred that lacked the transposable element. Evidence from the past summer shows, however, that *Spm* is not involved in the *P-mo* allele. A second approach to testing whether *P-mo* has resulted from an *Spm* insertion is to derive plants that are *bz/bz; P-mo/P-wr* and test whether all such plants activate *bz-m13*. The answer is that although derived from a *P-mo/P-ww* line with more than one *Spm* present via successive outcrosses to stocks (*bz-m13* and the *sh bz wx* tester) not carrying an *Spm*, not all such plants do. Therefore, *P-mo* does not result from an *Spm* insertion in a *P* allele.

In the same Newsletter in which I reported that 4Co63 does not contain an active *Spm*, Peter Peterson reported (64:8) that 4Co63 does contain an active *En (Spm)* in the homozygous condition. Dr. Peterson kindly provided his line of 4Co63, which also came from the Brink collection via J. Kermicle, and his tester stock, *c-m(r)*. Tests here in the summer of 1990 using the Peterson tester substantiated the previous observations. No plant of my 4Co63 line had an active *Spm(En)*, while all plants of the Peterson line were homozygous. It's not clear when these lines of 4Co63 diverged. Nevertheless, it seems that an obvious source of the *Spm*'s present in the mosaic pericarp stocks that I have been testing is the recurrent 4Co63 parent used by Dr. Brink.

It was noted above that in the F1 progeny (*P-mo/P-wr*; *Bz/bz-m13*) tested extensively in the summer of 1989 by crossing by a *sh bz wx* stock 72% of the plants segregated for *bz*-variegated kernels suggesting that the *P-mo/P-ww* plant crossed by *bz-m13/bz-m13*; *P-wr/P-wr* had two unlinked *Spm*'s. It was also noted that there were a few mosaic pericarp plants on which the kernels were either *Sh Bz/Sh Bz* or *Sh Bz/Sh bz*. Since these constituted exceptions to expectations if *P-mo* resulted from an *Spm* insertion, the kernels from these plants were tested. In 1989, there were five mosaic pericarp plants that had only *Sh Bz/Sh Bz* kernels. In 1990, the progeny of each of the five were tested as female parents by crossing times *bz-m13/bz-m13* and times a *sh bz wx*; *+Spm* tester. The results were the same for the five progenies; no plant had an active *Spm* as shown by failure to activate *bz-m13*, and some plants segregated *bz*-variegated kernels when crossed by the *sh bz wx*; *+Spm* tester so these had a responsive *bz-m13*. Therefore, the failure to produce *bz*-variegated kernels in 1989 was the absence of *Spm* from the genome. This constitutes further evidence that *P-mo* does not result from an *Spm* insertion.

The plants from the *Sh bz/sh bz* kernels on the two plants (41128-3 and -4) that had *Sh Bz/sh bz* and *Sh bz/sh*

bz kernels in 1989 when crossed by a *C sh bz wx*; no *Spm* tester were crossed by *bz-m13 / bz-m13* to ascertain whether the parental plants had an active *Spm*. In the progeny of 41128-3, 90% of the 112 plants tested had at least one *Spm*, and many clearly had more than one. In the progeny of 41128-4, 85% of the 88 plants tested had at least one *Spm*, and again many had more than one. The results suggest that both 1989 parental plants were heterozygous for three unlinked *Spm*'s. It is not possible to exclude definitively the possibility that the *Sh bz* chromosome came from a contaminating gamete from a distant *C Sh bz wx* tester in 1985, but it is intriguing that both these plants with a chromosome carrying a presumptive change very early in development from *Sh bz-m13* to *Sh bz* apparently had three *Spm*'s as shown by their progeny tests.

Locations of new mutations on 9S
--Oliver Nelson

This report covers investigations of the last few years on a group of mutants isolated by Gerry Neuffer and identified by him as being on 9S. I have reported on several of these mutants in mapping sessions at the Maize Meetings but not in the Newsletter. Data concerning some of these mutants have also been reported by the Missouri group.

There are two recessive virescent mutations in the distal part of 9S. These are *v28* (formerly *wlv-pg-585*) and *v31* (formerly *gry-wlv-828*). Both mutants are uncovered by the white deficiency (*wd*), and neither is allelic to *yg2*. Limited data from backcrosses, *V sh bz wx/v Sh Bz Wx* x *V sh bz wx* followed by selfing plants from different classes of kernels recombinant for the seed markers and then ascertaining the percentage of plants in each class segregating *v/v* plants gave estimates of 20% recombination between *v28* and *sh* and 23% recombination between *v31* and *sh*.

The dwarf mutation *d*-660B* proved to be an allele of *d3* and should now be designated as *d3-660B*.

The mutant adherent-glossy-512B is not located on 9S since it showed no linkage to any marker on that arm.

The dominant Zebra stripe-8 (*Zb8*) mutation is located between *bz* and *wx*. Data from an F2 population (*sh bz + Wx/Sh Bz Zb8 Wx*) selfed showed 12% recombination between *bz* and *Zb8*.

The dominant mutation Cross-banded*-1456 (*Cb*) is also located between *bz* and *wx*. Data from a backcross progeny (*Sh Bz Cb Wx/sh bz + wx*) x *sh bz + wx* place *Cb* 3 map units distal to *wx*. A test of allelism of *Zb8* and *Cb* has not yet been made. The phenotypes of plants heterozygous for these mutations are somewhat different. The *Zb8*/+ plants have a sharper transverse banding pattern on the leaves and are more vigorous than *Cb*/+ plants.

A third presumed dominant mutant, *G6*, was also received. I have not been able to identify *G6*/+ plants by their phenotype here in Madison in any of the four years in which I have grown progenies that should contain plants of this genotype. However, some plants in these progenies when selfed segregate for a bright yellow, lethal seedling that is apparently the homozygote. Since the heterozygote can be identified in Columbia, Missouri but not in Madison, Wisconsin, one should be cautious about attempting to use this mutation as a dominant marker. I have no data that locate this mutation on 9S.

Ac dosage and transposon mutagenesis
--Jerry Kermicle

What does of *Ac* should be chosen when planning transposon mutagenesis experiments? In particular, in what circumstances will the negative effect of increased *Ac* dosage on transposition rate more than offset the increased number of *Ac* donor copies? To address this question use was made of a revertant to *R-nj* of the *R-nj::Ac* mutable allele isolated by Irwin Greenblatt. This particular revertant, number 136 in the series established by R. A. Brink and E. Williams, yields new mutables at high frequency, presumably because *Ac* is closely linked to *R*. Indeed an initial test for linkage has indicated that most separations of *Ac* from *R* in *R-nj:rv-136* occur by transposition rather than by meiotic recombination.

Stocks of *R-nj:rv-136* with and without active *Ac* were isolated from an inbred W23 subline that was homozygous for *R-nj:rv-136* but segregating for *Ac*. Plants in the homozygous *Ac* lineage were selfed for two-dose *Ac* test progenies and crossed to the *Ac*-minus lineage for one-dose *Ac* progenies. Plants in these progenies as well as control *Ac*-minus ones were pollinated with *r-g r-g* and kernels expressing unstable -navajo phenotypes were then tested for heritability.

Mutable *R-nj* alleles established from homozygous *R-nj:rv-136* plants with different doses of linked *Ac*.

Ac dosage	Kernel population	Mutable *R-nj* derivatives Number	Frequency per 10^4
0	16,500	0	nil
1	30,900	31	10.02
2	24,500	6	2.45

Not surprisingly, the testcross population involving *R-nj:rv-136* lacking *Ac* produced no mutable-navajo derivative. Moderately larger populations of one and two-dose *Ac* populations yielded 31 and six cases at respective frequencies of 10.02 and 2.45 per 10^4. Thus the heterozygous *Ac* parents produced four times as many mutables as homozygous ones despite the fact that only half of their gametes are eligible to have received *Ac* from a linked source.

The four-fold difference in mutable allele frequency was unexpectedly large in light of an estimate of the germinal reversion frequency of another *Ac* mutable system. Using inbred W23 variegated pericarp (*P-vv*) stocks, R. I. Brawn obtained 257 full-red revertant ears among 6,786 progeny of homozygous parents (3.79%) and 390 full-reds from a total of 8,070 plants from heterozygous parents (4.83%), a difference of only 28%. The experiments involving *R* and *P* differ of course in the portion of the life cycle during which mutation can occur to yield a recoverable variant. In particular, mutations occurring post-meiotically through the stage where seedlings or young plants are capable of yielding whole ear variants would be included in the *P-vv* screening system but excluded from the *R-nj* tests. Moreover, because only half of the gametes from heterozygous parents carry *P-vv*, the higher incidence of post-meiotic mutations expected from

P-vv P-vv homozygotes will tend to counter the negative effect of *Ac* dosage in the parent sporophyte.

MILAN, ITALY
C.N.R. - Istituto Biosintesi Vegetali

Nitrogen requirements of wildtype and opaque-2 endosperm cultures

--L. A. Manzocchi and S. Faranda

Cells from immature endosperms differentiate in culture into storage cells, corresponding in most physiological features to the intact tissue (reviewed by Felker and Goodwin, Plant Physiol. 88:1235, 1988). After the first reports on the presence of zein and protein bodies in cultures (Shimamoto et al., Plant Physiol. 73:915, 1983; Felker, Am. J. Bot. 74:1912, 1987), recent results on endosperm suspension cultures have evidenced that active zein accumulation is associated with the negative logarithmic phase of cell growth cycle, and therefore coincides with the termination of cell divisions (Lyznik and Tsai, Plant Sci. 63:105, 1989; Manzocchi, Plant Cell Rep. 1991, in press). This further analogy to the in vivo condition makes suspension endosperm cell cultures a promising in vitro system for the study of the regulation of reserve protein synthesis and nitrogen metabolism in maize endosperms at the cellular level. With respect to kernel cultures (Cully et al., Plant Physiol. 74:389, 1984) and endosperm tissue in vitro maturation (Balconi et al., Plant Sci. 1990, in press), endosperm cell suspension cultures offer, together with an easily controlled environment, the possibility to obtain protoplasts able to express foreign DNA in transient expression experiments (Manzocchi, Plant Cell Rep. 1991, in press).

Although nutritional requirements for endosperm cell growth have been described (Shannon, in Sheridan (ed.), Maize for Biological Research, p. 397, 1982), we began experiments on nitrogen requirements for cell division and zein accumulation in A69Y wt and *o2* cultures. A medium containing 30 g/l sucrose, 0.4 mg/l thiamine, 200 mg/l inositol and Murashige and Skoog salts (MS-N) was used to grow cells with inorganic nitrogen supply (40 mM NO^{3-}, 20 mM NH^{4+}). Amino acid supplemented media were prepared from MS-N medium by addition of 15 mM asparagine (MS-A) or glutamine (MS-G), proline (MS-P) or asparagine and glutamine (7.5+7.5 mM: MS-AG). Control growth curves were obtained with medium MS-O, with the components of MS-N with the exception of nitrogen salts.

MS-N medium supported for both wt and *o2* cells a growth rate approximately one half of growth obtained with MS-A (the medium normally used for endosperm cell culture); nitrogen salts, and not amino acids eventually derived from dead cells in the cultures, represent the nitrogen source, as can be stated from the complete absence of growth in MS-O medium (Fig. 1). Cells have been cultured in MS-N medium for over 5 months without modifications of growth rate; their ability to accumulate zein (expressed as protein/mg cells) was maintained through subcultures, although slightly reduced with respect to cells grown in MS-A.

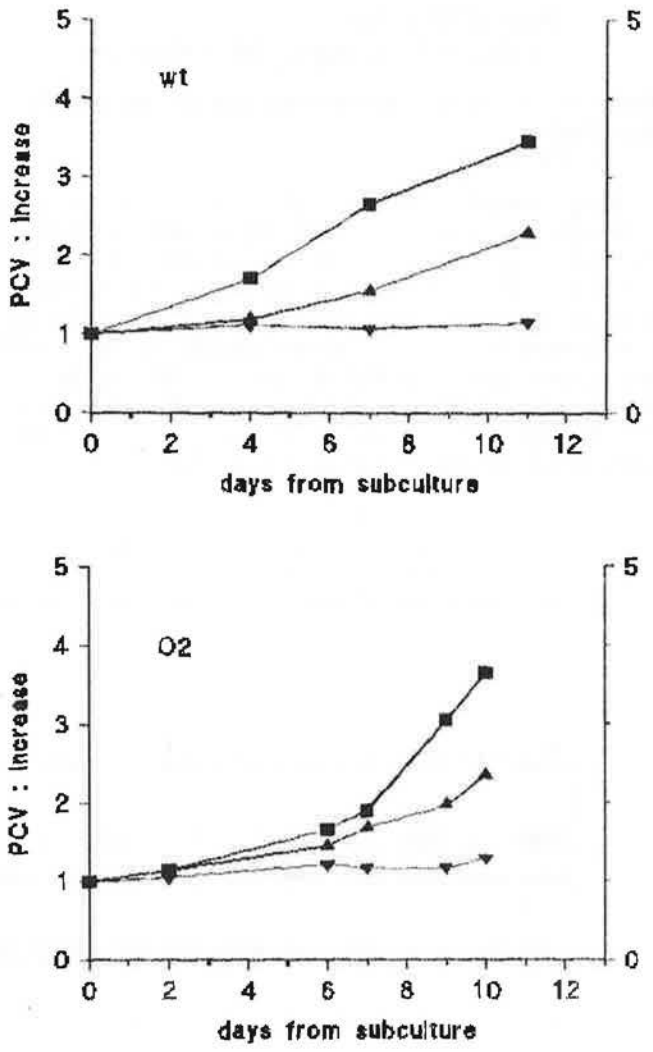


Figure 1. Growth curves of wildtype (wt) and opaque-2 (*o2*) A69Y endosperm cell cultures in MS-A (■), MS-N (▲) and MS-O (▼) medium.

MS-A, MS-G, MS-AG stimulated cell growth rate at the same extent; MS-P was less effective. Zein accumulation was similar in MS-A, MS-G and MS-P; a considerable increase was observed in MS-AG. Asparagine and glutamine, the amino acids supplied to the developing kernels by maize plants (Lyznik et al., Phytochemistry 24:425, 1985) represent a good nitrogen source for wt and *o2* cultured cells, as reported for A636 cultures (Lyznik and Tsai, Plant Sci. 63:105, 1989). The ability of cells to grow and accumulate zein in the absence of an organic nitrogen source suggests the capacity of endosperm cells for a consistent amino acid synthesis from inorganic nitrogen; experiments are in progress to elucidate the levels in cultured cells of nitrogen metabolism enzymes, whose levels during endosperm development have been reported with conflicting results (reviewed by Muhitch, Physiol. Plant. 74:176, 1988).

MOSCOW, USSR
All-Union Inst. Agric. Biotechnology

Esterase and peroxidase patterns in leaves of A188 somaclones

--Emil E. Khavkin

Fully developed 4th and 5th leaves of light-grown seedlings were employed for zymographical analysis of SC2 generation in eight somaclonal variants described in the note by Dolgykh et al. (MNL, this issue). Variable bands of anodic esterases (marked by arrows in Fig. 1A and B) could be tentatively assigned to *E5*, *E7*, *E9* and *E4* loci and non-identified bands 42/43 and 59 as described by MacDonald and Brewbaker (Hawaii Agr. Exp. Sta. Tech. Bull. No. 89, 1975). Among anodic peroxidases (Fig. 2A) variable bands appeared immediately following the most mobile zone described as PRX6 by Brewbaker and Johnson (MNL 46:29, 1972) and between two slowly moving zones assigned to PRX3 and PRX7, this interval could be tentatively identified as PRX9 and PRX10 zones. Only quantitative changes in staining were found in the cathodic peroxidase spectrum (Fig. 2B).

Two features of these isozyme patterns fall in line with our previous observations made with inbred 346 somaclones (MNL 64:91, 1990). First, the extent of variation found within several samples of sibs produced from the same SC1 ear (e.g., in the case of somaclones 11, 15, 27, 53 or 54b) sometimes exceeded the deviation from the standard (control, A188). Second, some of the bands active in somaclones but absent from the leaves of the standard were previously described by Brewbaker et al. not as leaf-specific but as characteristic of other tissues, e.g., root-spe-

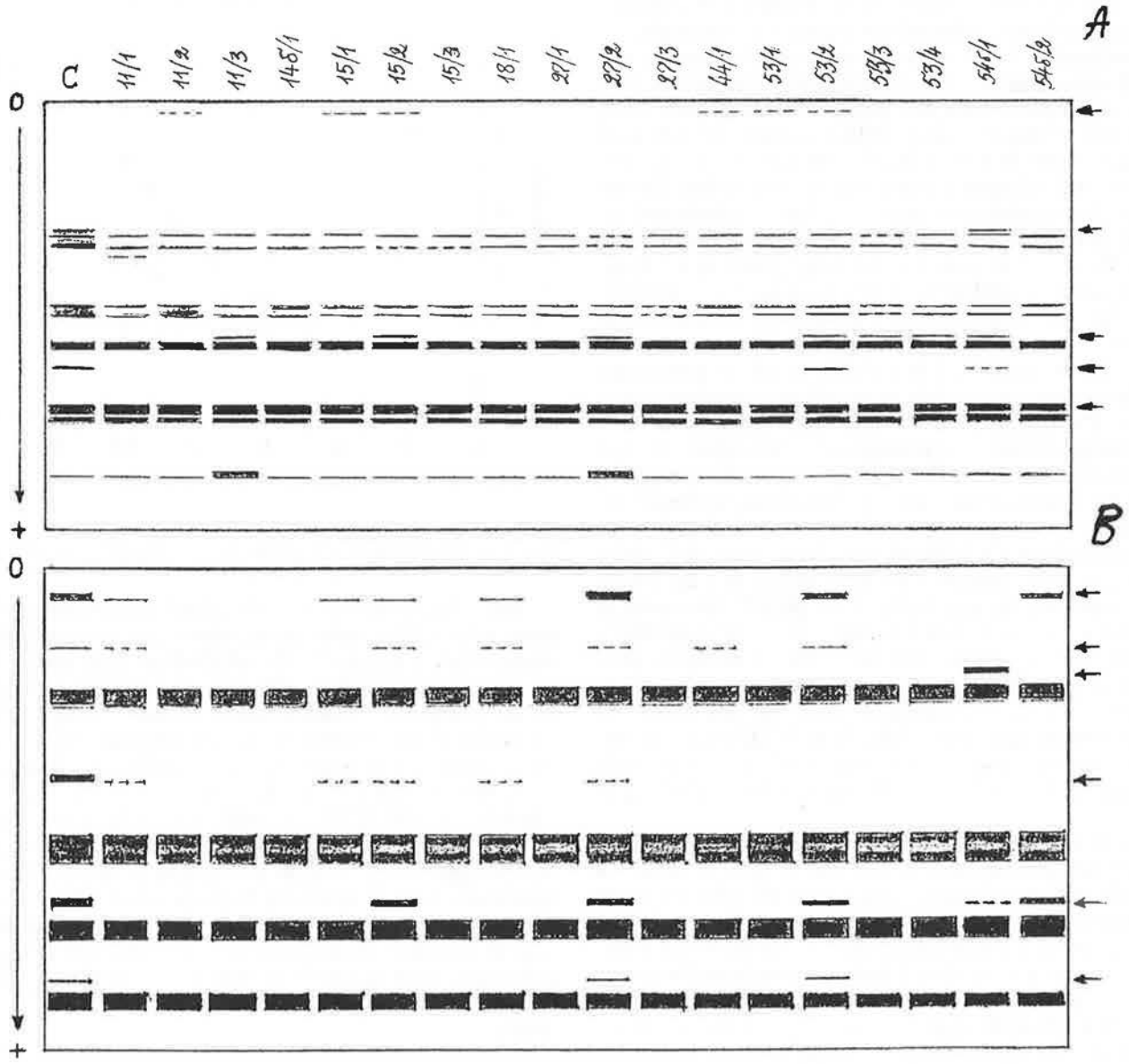


Figure 1. Esterase patterns of control (C) and somaclone leaves as resolved by PAGE after Davis (A) and Taber and Sherman (B).

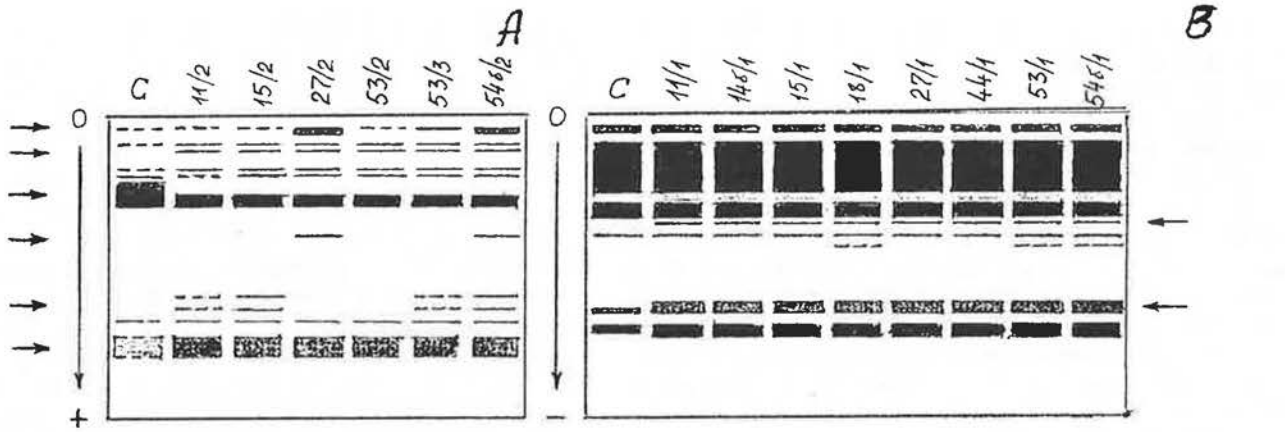


Figure 2. Peroxidase patterns of leaves as resolved by PAGE after Davis (A) and Reisfield et al. (B).

cific *E4* or root-, coleoptile- and mesocotyl-specific PRX9 and PRX10. It is also noteworthy that in both inbreds some loci seemed to be more sensitive to somaclonal variation and affected more frequently than others: virtually the same organ-specific anodic esterases and peroxidases exhibited significant changes in contrast to invariant non-specific isozymes, e.g., *E8* or PRX3.

These observations, along with our earlier data on isozyme patterns of intact and isolated tissues (Biochem. Physiol. Pflanzen 174:431, 1979; MNL 55:47, 1981), suggest that there is a specific control system ordering tissue- and organ-specific isozyme spectra that might be affected by somaclonal variation at the early steps of a regulatory cascade ruling cell and tissue differentiation. By misgoverning some of temporal and spatial signals for isozyme loci expression, somaclonal variation could produce numerous, apparently non-linked (judging by sibs) and probably non-heritable, changes in isozyme patterns.

Quantitative changes of telomeric heterochromatin in A188 somaclones

--T. B. Dubrovina and D. M. Atayeva

C-banding patterns were compared in the root tip mitotic cells of inbred A188 and SC2 seedlings obtained as described by Dolgykh et al. (MNL, this issue). While the structural patterns (distribution of C-heterochromatic blocks) did not change within investigated samples, there

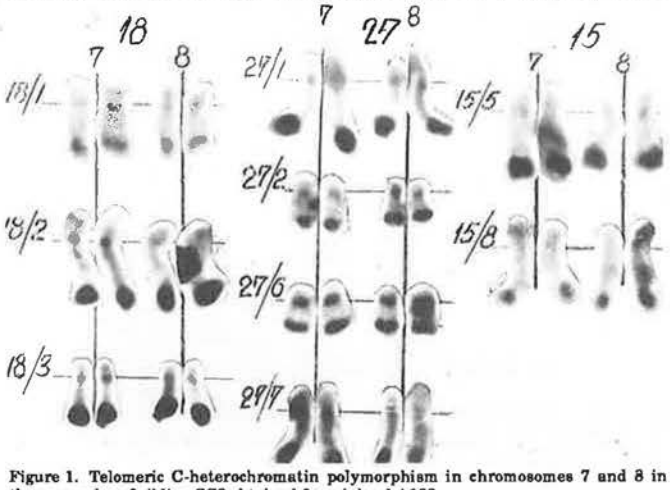


Figure 1. Telomeric C-heterochromatin polymorphism in chromosomes 7 and 8 in three samples of sibling SC2 obtained from inbred A188.

were significant quantitative variations within groups of sibs as to the amount of telomeric heterochromatin (Fig. 1).

MOSCOW, USSR
K. A. Timiriazev Inst. Plant Physiology
KRASNODAR, USSR
Krasnodar Research Institute of Agric.

Somaclonal variability in inbred line A188

--Y. I. Dolgykh, Y. V. Bolonkina, S. H. Larina and Z. B. Shamina

Regenerated plants (SC1) were produced in 2 to 14-month callus culture from immature embryos of inbred line A188. Plants from 2-month culture differed in several quantitative traits and manifested numerous morphological abnormalities: reduced plant height, changed number of kernel rows per ear, bisexual tassels, branching, chlorophyll deficiency, male sterility, etc. Only reduced plant height and increased number of kernel rows per ear were inherited in SC2 after self-pollination. The progeny of several morphologically normal SC1 plants segregated in branching, the number of kernel rows and anther color; the latter probably due to recessive mutation. Two regenerated plants have produced mosaicism in the number of midribs (1 to 3 in the same plant) and chlorophyll deficiency in SC2.

Plants from 10 to 14-month callus culture had low viability: about 80% died after placing into soil. All regenerated plants were sterile, mostly with shortened internodes and chlorophyll deficiency; frequency and expression of these abnormalities increased with the time of callus cultivation.

These results suggest that in vitro cultivation leads to genome destabilization which is apparent in several seed generations as epigenetic variations and mutations.

MOSCOW, USSR
All-Union Inst. Agric. Biotechnology
KRASNODAR, USSR
Krasnodar Research Institute of Agric.

Development of heterochromatin polymorphism in inbred lines

--A. B. Romanova, D. M. Atayeva, V. S. Shcherbak and M. V. Chumak

When C-heterochromatic patterns were compared in

root-tip mitotic chromosomes of B55, W132, H95, Mo17, W64, B84 and Cg25 inbreds, the size and distribution of heterochromatin blocks significantly differed in homologous chromosomes. Similar observations were previously reported in rye inbreds (Weimarck, Hereditas 79:293, 1975; Lelleg et al., Can. J. Genet. Cytol. 20:307, 1978).

In an attempt to determine more precisely when this intra-inbred polymorphism might appear, we investigated seven dihaploid lines produced in Krasnodar and once again found that homologous chromosomes differed by their heterochromatic patterns. We suggest that the physiological implications of the de novo development of heterochromatin polymorphism described above could possibly include mechanisms of both adaptation and somaclonal variation in maize.

MUNICH, GERMANY
University of Munich
STUTTGART, GERMANY
University of Hohenheim

Comparisons among strains of inbreds for RFLPs

--J. Boppenmaier, A. E. Melchinger, E. Brunklaus-Jung and R. G. Herrmann

We have analyzed RFLP patterns of different strains of five inbred lines, developed by W. G. Pollmer from the University of Hohenheim (Table 1). The lines have been released between 1968 and 1981. They had been maintained in separate breeding programs in Germany for one to eight generations by selfing or sibbing and rogueing off-type plants.

The various strains from each inbred were analyzed for their respective RFLP patterns using equal quantities of leaf tissue harvested from five seedlings per strain. Genomic DNA was digested separately with restriction enzymes *Eco*RI and *Hin*dIII. RFLP analyses were performed with a total of 101 genomic DNA clones kindly provided by D. Hoisington (University of Missouri, Columbia, MO) and B. Burr (Brookhaven National Laboratory, Upton, NY).

For inbred DK105, two strains (B and C) showed identical RFLP patterns for 101 DNA probes with both restriction enzymes. These two strains had been highly inbred (S12) before being maintained separately for two to eight generations. Strain A had identical RFLP patterns with strains B and C for 99 of the 101 DNA probes. However, different RFLP variants in A than B and C were found with both enzymes for DNA probes *BNL5.09* and *UMC106*, mapping to chromosomes 9 and 1, respectively.

For inbred D140, the two strains showed different RFLP banding patterns with both enzymes only for a single DNA probe (*UMC116*), mapping to chromosome 7. The other three inbreds (D503, D406, and D44) showed identical RFLP patterns for both strains with all 101 DNA probes examined.

The small numbers of polymorphic RFLP loci found between different strains of the same inbred lines indicate that the lines had been highly homozygous as expected from the respective number of selfing generations before separate strain maintenance. In addition, our findings are in agreement with an earlier report (Evola et al., Theor. Appl. Genet. 71:765-771, 1986) that RFLPs are stably inherited over several selfing generations. In contrast, Godshalk and Lee (MNL 64:58, 1990) reported a significant number of polymorphic RFLP variants in different strains of maize inbred W22 maintained in separate breeding programs. In combination with high level of polymorphisms at RFLP loci found in maize (Melchinger et al., Crop Sci. 30:1033-1040, 1990), our results corroborate that RFLPs should be a valuable tool for identification of maize inbred lines for plant variety protection, registration, and patenting.

Table 1. Line maintenance of five maize inbred lines in separate breeding programs and number of DNA probes revealing RFLPs among different strains.

	Inbreds									
	DK105			D140		D503		D406		D44	
	A*	B	C	A	C	A	B	A	B	A	B
Year of release	—— 1968 ——			- 1978 -		- 1979 -		- 1981 -		- 1977 -	
Selfing generation before separate maintenance of strains	- S_9 ———	- S_{12} -		-- S_9 --		-- S_9 --		-- S_8 --		-- S_{11} -	
No. of generations of separate line maintenance§	1	8	2	3	2	3	2	3	2	2	2
No. of probes out of 101 revealing RFLPs among strains	-- 2 ———	-- 0 --		-- 1 --		-- 0 --		-- 0 --		-- 0 --	

* A, B, and C refer to strains of the line maintained in separate breeding programs.
§ Lines were maintained by selfing or sibbing and rogueing off-type plants.

PEORIA, ILLINOIS
Plant Polymer Res., USDA, ARS, NCAUR

New zein genes mapped to chromosome 7 by IEF and HPLC

--C. M. Wilson

A number of zein genes have been mapped to chromosomes 4 and 7 by determining linkages using IEF in agarose (C. M. Wilson, Theor. Appl. Genet. 77:217, 1989). The genes on chromosome 7 were identified as Zp B9/22, Zp B9/10, and Zp B8/38, where the first nomenclature element refers to SDS-PAGE and the second to the IEF position. Zp B9/21, found only in R801, W22, and H55, was thought to be on chromosome 7. R801 was pollinated by WF9 or Oh45, which have Zp B9/10 and Zp B9/22 with genes on chromosome 7. The F1 was pollinated by B57 or by W64A, inbreds which have only Zp B9/38 mapped to chromosome 7. The resultant seeds were assayed by IEF for the presence or absence of IEF bands 10, 21, and 22. For the cross (WF9xR801)xB57, 34 seeds had bands 10 and 22, while 30 had band 21. For (Oh45xR801)xW64A, 59 seeds had bands 10 and 22, while 60 had band 21. The conclusion is that genes for these three zeins are closely linked at the same position on chromosome 7, with IEF band 21 being in repulsion to bands 10 and 22.

Serial analysis by reversed-phase high pressure liquid chromatography (HPLC) in addition to IEF and SDS-PAGE identifies more zeins than can be found by IEF alone (C. M. Wilson, Plant Physiol. in press). IEF band 38 produces two bands on HPLC, but the linkage experiments (TAG 77:217, 1989) suggest that the genes for the two band 38 zeins are tightly linked on chromosome 7. IEF band 21 from R801 elutes from the HPLC column at the same time as the second IEF band 38 from inbreds B57, W64A, and N28. These results show that no single assay for separation of zein polypeptides can give unique identifications to the many zeins in a single inbred.

Identification by IEF of zeins from genes located in recombinant inbreds

--C. M. Wilson

Zeins of individual seeds from two sets of maize recombinant inbreds derived from F2 populations T232xCM37 (48 lines) and CO159xTx303 (41 lines) (Burr et al., Genetics 118:519, 1988) were assayed by IEF in agarose (Wilson et al., Theor. Appl. Genet. 77:217, 1989). Each band of the parental inbreds was given an IEF number by relating it to the positions of zeins in standard inbreds. The occurrence of zeins in three seeds from each of the recombinant inbreds was determined. Only 3 or 4 inbred lines appeared to be segregating. Loci positions were kindly assigned by B. Burr (see MNL64, 1990 maps).. Eleven zein genes were placed on chromosome 4S, four on chromosome 4L (near the centromere), and 3 on chromosome 7S (Table 1). The recombinant inbred linkages agreed with the positions suggested by the earlier work.

IEF bands previously reported (Wilson, TAG, plus MNL report above) are associated with the linkage groups as follows:ZpL1a-f (4S): 17.5, 14/19, 32, 35, 36, 37, 60; ZpL2a/3a (4L): 28, 30.5, 32y, 33, 33.5, 49, 54.5; ZpL2b (7S): 10, 21, 22, 38.

Table 1. Linkage groups for zein genes in recombinant inbreds.

Linkage Group	Chrom.	Inbred CM37	Inbred T232
ZpL1a	4S-29	18	20
ZpL1b	4S-31	32	31.5
ZpL1c	4S-31	36	29.5
ZpL1d*	4S-34	33.5	33.5
ZpL2a	4L-74	47.5	45

*33.5 bands differentiated by intensity.

Linkage Group	Chrom.	Inbred CO159	Inbred Tx303
ZpL1e	4S-30	17.5	18,60
ZpL1f	4S-33	35	32,36
ZpL2b	7S-21	21.5,53	38
ZpL3a	4L-78	44	44.5

Genes for zein bands 32 and 33.5 occurred on both arms of chromosome 4. Totals for identifiably different (by IEF position alone) zeins which have been mapped are 13 for ZpL1a/f, 11 for ZpL2a/3a, and 6 for ZpL2b, for a total of 30 genes for AB zeins in the four recombinant inbreds plus the 13 crosses studied earlier. Recent work (C. M. Wilson, Plant Physiol. 1991, in press) suggests that these are minimum numbers, for serial analysis by HPLC in addition to IEF shows that apparently identical zein IEF bands in two inbreds may differ by HPLC.

PHILADELPHIA, PENNSYLVANIA
University of Pennsylvania

gl15 is a heterochronic mutation

--Matthew M. S. Evans and Scott Poethig

We have been searching for mutations that accelerate the transition from juvenile to adult growth by screening for mutations that have an abbreviated expression of juvenile traits, such as epicuticular wax, and precociously express adult traits, such as epidermal hairs. A mutation with this phenotype has recently been identified in a group of glossy mutations obtained from Dr. Sprague. We have confirmed his observation that this mutation is an allele of *gl15* and have agreed to call this new allele *gl15-2*.

In families segregating for *gl15-2*, wild type plants have at least some wax on the first 5 or 6 leaves whereas their mutant siblings only possess epicuticular wax on the first 3 or 4 leaves. In both wild type and mutant plants, the disappearance of epicuticular wax is correlated with the appearance of epidermal hairs. Thus, mutant plants express epidermal hairs as early as leaf 3, in contrast to their wild type siblings, which do not produce epidermal hairs until leaf 5 or 6. The distribution of epicuticular wax and epidermal hairs on *gl15-2* juvenile/adult transition leaves is similar to that on wild type transition leaves. Histological analysis demonstrates that *gl15-2* also accelerates the change in epidermal cell shape that normally accompanies the transition from juvenile to adult growth. However, at least two other phase-specific traits -- root production and cuticle thickness -- do not seem to be affected by this mutation. In segregating families, wild type and *gl15-2* plants are about the same size and have the same tassel morphology, ear position, and number of leaves. It is difficult to

compare the expression of *gl15-2* with the first allele of this locus (*gl15-1*) because these mutations are in different genetic backgrounds. Available stocks of *gl15-1* have a phenotype similar to that of *gl15-2* , but weaker.

The phenotype of these alleles of *gl15* suggests that this gene regulates some aspect of phase change in maize. Because the character of transition leaves in mutant plants is similar to that of wild type transition leaves, we conclude that phase change occurs in the same manner in the mutant as the wild type, only earlier. *gl15* maps to the long arm of chromosome 9 and is therefore not a recessive allele of Corngrass or the Teopod mutations. Because *gl15* only affects a subset of the traits affected by these other heterochronic mutations, we believe that *gl15* may act downstream of the Corngrass and Teopod genes.

New alleles of Teopod2

--Mark Dudley and Scott Poethig

Teopod2 (*Tp2*) is a semi-dominant, neomorphic mutation that causes the inappropriate expression of juvenile characteristics (such as the presence of leaf wax and prop roots, and a characteristic leaf shape) in normally adult phytomers of the plant. One explanation for this heterochronic phenotype is that a "juvenile program" of development , which is turned off at the transition to an adult phase of growth in normal plants, is expressed constitutively in *Tp2* plants. To test this hypothesis we have been isolating and characterizing new alleles of *Tp2*.

To date we have isolated five new alleles of *Tp2* (Table 1) by screening for loss of the semi-dominant Teopod phenotype in progeny of crosses in which *Tp2* was the pollen parent: +/+ X *Tp2*/*Tp2* or +/+ X *Tp2*/--.

Table 1. New *Tp2* alleles.

Allele	mutagen	plants screened
Tp2-E1	EMS	8,300
Tp2-E2	EMS	"
Tp2-Mum1	?	16,200
Tp2-x2	x-ray	9,400
Tp2-x16	x-ray	"

These five alleles were recovered in three separate experiments. *Tp2-E1* and *Tp2-E2* were isolated in 1987 from EMS-mutagenized *Tp2* pollen; *Tp2-Mum1* was recovered in 1986 in a Robertson's mutator (*Mu*) background. *Tp2-Mum1* has a stable and consistent phenotype, and no *Mu*-homologous band cosegregates with it, suggesting that the insertion of a *Mu*-like element is not directly responsible for this allele. *Tp2-x2* and *Tp2-x16* were recovered from x-ray mutagenized *Tp2* pollen. In each of these experiments the progenitor *Tp2* allele was linked to the recessive golden (*g*) marker, 2 map units distal to *Tp2* on 10L, to control against contamination.

Partial Revertants. Three revertants, *Tp2-E1*, *Tp2-E2*, and *Tp2-Mum1*, display a partial or attenuated teopod phenotype. The order of severity of teopod expression of these alleles is:

Tp2 > *Tp2-E2* = *Tp2-Mum1* > *Tp2-E1* > +

Tassel morphology of these alleles is a characteristic aspect of the revertant phenotype. Similarly to *Tp2*, tassel branching in the revertants is reduced; however, in contrast to *Tp2*, spikelet morphology is normal. The revertant alleles are fertile even in homozygous condition. The *Tp2* progenitor allele and all partial revertants are highly pleiotropic and dependent on genetic background. In a background that enhances their expression, such as A632, the severely reduced number of tassel branches and partial vegetative transformation of the tassel characterize these revertants as teopod-like. In mild genetic background such as W23, the range of expression in + and *Tp2-E1* plants overlaps, so that it is not always possible to differentiate them.

Knock-out Revertants. Preliminary investigations of *Tp2-x2* and *Tp2-x16* suggest that they are large deletions. Plants heterozygous for either *Tp2-x2* or *Tp2-x16* are indistinguishable from wild type siblings, suggesting complete loss of *Tp2* function. Transmission of either allele through the male gametophyte has not been observed. In heterozygous plants, transmission of both alleles through the female gametophyte is reduced; *Tp2-x2* is transmitted maternally at a frequency of approximately 33%; *Tp2-x16* is transmitted at a frequency of approximately 16%. We attempted to use TB-10L translocations to uncover the *Tp2-x2* and *Tp2-x16* alleles in hemizygous plants using the following cross:

Tp2-x g r-/+ + *r-* x TB-10L19 (*R-scm2*)/+ *r-*

For *Tp2-x2*, approximately 1/3 of the seeds with colored endosperm (potentially hypoploid embryos) had no detectable embryo. For *Tp2-x16* approximately 1/6 of the colored endosperm kernels had no embryo. This is consistent with the frequency with which these alleles are transmitted and suggests that these alleles result in embryonic lethality in hemizygous condition. Together with their relatively poor transmission through both the microgametophyte and megagametophyte, this result suggests that these alleles are large deletions. Further physical and genetic characterization of these alleles is underway.

These new alleles of *Tp2* are potentially useful in several ways. For instance, the partially revertant *Tp2* alleles may allow us to isolate genetic enhancers and suppressors of the teopod phenotype. Knock-out revertants will be useful in examining the effects of gene dosage on *Tp2* phenotype, as well as elucidating the function of the wild type allele of *Tp2*. Thus, these alleles greatly enhance our prospects for understanding the genetic regulation of phase change in maize.

ST. LOUIS, MISSOURI
Monsanto Agricultural Company
WOODLAND, CALIFORNIA
PetoSeed, Inc.
ST. PAUL, MINNESOTA
University of Minnesota

Development and availability of germplasm with high Type II culture formation response

--C. L. Armstrong, C. E. Green and R. L. Phillips

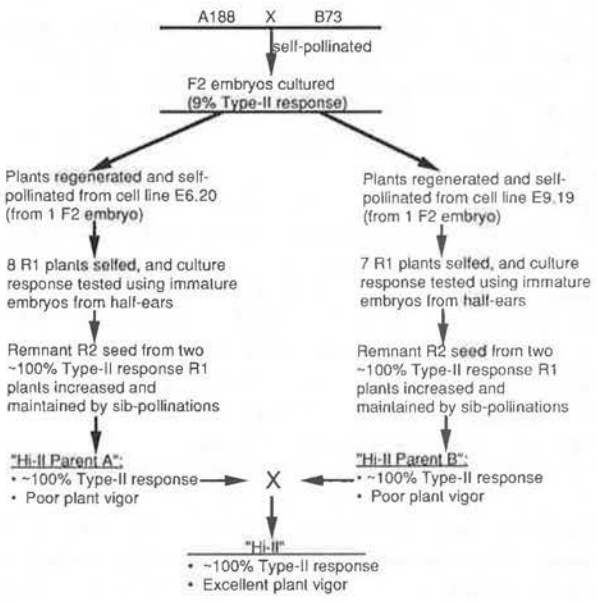
Type II (friable, embryogenic) cultures of maize were first developed and described in the early 1980's (Green, Plant Tissue Culture, ed. A. Fujiwara, pp. 107-108, 1982).

These cultures have been used successfully in a variety of applications, including the recent production of fertile, transgenic maize plants following microprojectile bombardment (Fromm et al., Bio/Technology 8:833-839, 1990; Gordon-Kamm et al., Plant Cell 2:603-608, 1990). In anticipation of increased interest from the maize genetics community in utilizing this tissue culture technology, we describe here the development and availability of "user-friendly" germplasm for the establishment of Type-II cultures.

Most published reports on Type-II cultures have utilized the inbred lines A188, B73, or the F1 hybrids between these two lines. Using published procedures, initiation frequencies for B73 are extremely low (typically less than 1 in 100 immature embryos). Initiation frequencies are much better for A188 and the F1 hybrid, but are variable and dependent on environmental conditions. For the inexperienced researcher, high quality Type-II cultures from A188 and the F1 can be difficult to maintain. Establishment of embryogenic suspension cultures from these genotypes also requires a reasonable amount of tissue culture experience.

We selected two partially inbred lines (Hi-II Parent A and B) out of the cross between A188 and B73 which have a greatly improved Type-II culture response (Fig. 1). Each parent was derived from a different F2 embryo. The Type-II culture initiation frequency from immature embryos from each line is nearly 100% when incubated on a modified N6 medium contain 1 mg/L 2,4-D and 25mM L-proline (Armstrong and Green, Plant 164:207-214, 1985). The cross between the two selected lines has been designated "Hi-II", and has much better plant vigor than the two parental lines while maintaining the excellent culture response. While selection was based on immature embryo response, immature "Hi-II" tassels also form Type-II cultures at a high frequency (William Petersen, Monsanto, unpublished results). Embryogenic suspension cultures can be established relatively easily from "Hi-II" callus, and such suspensions have been used to generate fertile transgenic plants (Fromm et al., Bio/Technology 8:833-839, 1990).

Figure 1. Development of "Hi-II" germplasm.



The "Hi-II" germplasm should be useful in any experiments requiring a high-frequency Type-II initiation response from immature embryos or tassels. Many F1 combinations with the "Hi-II" germplasm have formed excellent Type-II cultures, and therefore it should be useful for researchers desiring to produce Type-II cultures of specific dominant or cytoplasmic genetic stocks which are in a recalcitrant background. We feel this material should be particularly useful for researchers with little or no experience with corn tissue culture. It is more "forgiving" than most genotypes, and will respond reasonably well under a wide variety of in vitro culture conditions.

The "Hi-II" germplasm has some significant limitations which must also be considered. The parents are only partially inbred. One consequence of this is that regenerated plant vigor is somewhat variable from line-to-line. While generally quite acceptable, regenerated plants are not as vigorous as from A188xB73 F1 cultures. A second limitation is that the parental lines are somewhat difficult to propagate.

Samples of seed of "Hi-II" and the "Parent A" and "Parent B" lines can be obtained by contacting Chuck Armstrong (Monsanto, Mail Zone GG4H, St. Louis, MO, 63198). Recommended culture media and growth conditions are as described by Armstrong and Green (Planta 164:207-214, 1985), but modified to contain 0.2% Phytagel™ (Sigma) in place of 0.7% Difco-Bacto agar, and including 10μM $AgNO_3$ (Dr. Dave Songstad, Monsanto, unpublished results).

ST. PAUL, MINNESOTA
University of Minnesota

Maize plastid genome *Bam*HI 14 fragment has conserved intron-containing open reading frame

--Jaakko Kangasjarvi, Dennis E. Mathews and Burle G. Gengenbach

In previous studies (MNL 63:105, 1989) we have found that the plastid transcript accumulation pattern differs between chloroplasts and amyloplasts. A 2.4 kb transcript from the plastid *Bam*HI 14 fragment accumulated progressively during endosperm development and was more abundant in older endosperm than in leaf total RNA. No known genes have been identified on maize *Bam*HI 14, but the corresponding region of three other species has conserved intron-containing open reading frames (ORF170 in rice). No function is known or has been proposed for the hypothetical protein. Our subsequent PCR analysis (MNL 64:102, 1990) suggested the presence and expression of a similar intron-containing open reading frame in the maize plastid genome, located between *psaA* and *rps4*. We have now sequenced the maize plastid genome *Bam*HI 14 fragment and the cDNA of the fully spliced putative ORF to verify the existence of the similar ORF in maize and to establish the splicing sites. We also determined its pattern of expression by PCR amplification of total and polysomal

RNA isolated from various tissues and endosperm developmental stages.

*Bam*HI 14 fragment was subcloned from a cosmid clone. Mature ORF170 was cloned with PCR from reverse transcribed leaf total RNA using ORF170 specific primers. Both were sequenced using the dideoxy procedure. Total RNA was isolated from 7 d old shoots and roots, 8-12 DAP endosperm and developing leaves. Polysomes were isolated from developing endosperm, roots and seedling leaves. All the RNAs were treated with RNase-free DNase. cDNA was synthesized using random primers and amplified with PCR. Cloned *Bam*HI 14 fragment and mature ORF170 were used as specific controls for the full length transcript and fully spliced transcript, respectively.

The nucleotide sequence of maize plastid *Bam*HI 14 fragment (3.1 kb) revealed the expected open reading frame containing three exons and two introns, as found in other species where the homologous region has been sequenced. 5' and 3' ends of the introns contained splicing sequences conserved in plastids and the cDNA verified the expected splicing sites. As in other species, maize exons I and II start with ATG and have an in-frame stop codon inside the introns.

ORF170 is expressed in maize chloroplasts and amyloplasts, but the developmental processing of the full length transcript is different in these two plastid types. The fully processed transcript was present at the early stages of endosperm development during the time of differentiation of proplastids to amyloplasts. When RNA from 8-12 DAP endosperm was used in the cDNA synthesis, there was a proportional increase in the amount of unspliced transcript (1.9 kb) compared to mature message at older stages; the abundance of the intermediate processing product (1.2 kb, one intron removed) decreased. The decrease in the abundance of the partially processed form and the accumulation of the unspliced form coincided with differentiation and the onset of starch accumulation. Transcription of ORF170 continued through development of endosperm at least up to 16 DAP and the regulation of expression appeared to be at the level of transcript processing as has been suggested for plastid intron-containing genes. In contrast to endosperm, the completely processed transcript (0.45 kb) was the most abundant amplified product for root, old and young leaf and seedling RNA of maize. The splicing pattern of ORF170 transcripts did not change in subsequently older leaves and two intermediate processing products were observed which was different from the pattern found in endosperm.

Total polysomal RNA from various tissues contained transcripts from the *Bam*HI 14 region. Only the mature transcript was detected in roots, shoots and 8 DAP endosperm. Polysomal RNA from 10 DAP endosperm contained the unspliced transcript and at 13 DAP both were present. The intermediate processing product was not detectable in any of the polysome samples. The presence of the transcripts from the *Bam*HI 14 fragment in polysomal RNA suggests that the gene is being translated in all the tissues and endosperm developmental stages studied. In leaves, roots and 8 DAP endosperm this was as expected, because the mature transcript was most abundant in the total RNA. The association of the unspliced transcript with polysomes at 10 and 13 DAP is surprising; however exons 1 and 2 are open reading frames and thus could be translated separately. This possibility is under study. We are currently making antibodies against the ORF170 protein expressed from the cloned cDNA in *E. coli* to determine whether a corresponding protein is actually synthesized in plastids.

Altered acetyl-CoA carboxylase confers herbicide tolerance and may affect fatty acid biosynthesis

--Lorelei C. Marshall, Robin A. Keith, Donald L. Wyse, Burle G. Gengenbach, and John W. Gronwald

Acetyl-CoA carboxylase (ACCase) activity from five mutants exhibiting partially dominant tolerance to ACCase-inhibiting herbicides (Parker et al., PNAS 87:7175, 1990) was 16- to 350-fold less inhibited by these herbicides than ACCase activity in wildtype (susceptible) maize. Seedlings of the homozygous tolerant mutants survived 2- to > 130-fold higher rates of these herbicides than wildtype seedlings in greenhouse tests. The tolerance to representatives of two classes of ACCase-inhibiting herbicides (cyclohexanediones, e.g. sethoxydim, and aryloxyphenoxypropionates, e.g. haloxyfop) cosegregated and increased in parallel for plant and ACCase responses in the mutants. Based on allelism tests, the mutations appeared to be located in a single gene or in closely linked genes. These results suggested that we have identified at least four alleles of the ACCase structural gene--one wildtype susceptible allele (*acc*) and at least three alleles representing distinguishable mutant phenotypes. All mutants conferred moderate tolerance to haloxyfop, but each conferred different levels of tolerance to sethoxydim. The *Acc-H1* allele had no sethoxydim tolerance, *Acc-H2* had intermediate tolerance and *Acc-S1*, *Acc-S2*, *Acc-S3* exhibited tolerance to high rates of sethoxydim. Differences in cross-tolerance to the two herbicides suggested that sites on ACCase that interact with the different herbicide chemistries do not completely overlap.

The single gene inheritance, high levels of herbicide tolerance, and retention of ACCase catalytic activity in the mutants suggested that the tolerance alleles may be of agronomic interest. Supporting this, the herbicide-tolerance alleles were expressed in diverse genetic backgrounds including F1 and backcross combinations with the inbreds A188, A619, A641, A665, A682, B73 and W153R. The tolerance trait has been genetically stable over at least four generations. Herbicide treatments at rates recommended for control of annual grass weeds did not adversely affect grain yield or quality of tolerant lines. Incorporation of the tolerance trait was not associated with deleterious effects on grain yield or quality in the absence of herbicide. These results warrant efforts to transfer the tolerance trait into elite maize lines.

The mutants will be useful in studying maize ACCase. ACCase is the first committed step in fatty acid biosynthesis and may regulate lipid biosynthesis and thus kernel oil deposition. Homozygous *Acc-H1* and *Acc-H2* mutants had ACCase activity in the absence of herbicide that was

about 25% lower than wildtype, suggesting alterations in ACCase that affected some catalytic function. Homozygous *Acc-S1*, *S2*, and *S3* mutants had wildtype levels of ACCase activity and a slight tendency for increased kernel oil concentration in the absence of herbicide treatments (overall mean of 9% more than wildtype). These results suggested that alteration(s) conferring reduced herbicide inhibition of ACCase activity may alter the regulatory properties of ACCase in controlling fatty acid biosynthesis and thereby affect lipid biosynthesis.

To further investigate the role of ACCase in kernel oil deposition, we are currently comparing the herbicide inhibition of mutant and wildtype ACCase extracted from seedling leaves and developing embryo and endosperm. Our preliminary results indicate that the herbicide-tolerant form of ACCase is expressed in all three tissues. Furthermore, the herbicide-tolerant ACCase is expressed throughout kernel development in both embryo and endosperm. These results lead us to speculate that a single ACCase gene is expressed throughout the plant and that it produces precursor for synthesis of fatty acids for both the housekeeping function of membrane maintenance and for oil deposition in the developing kernel. Based on this one gene model it is possible to predict that an *acc-null* allele will be lethal provided that other ACCase isozymes do not exist. A recessive mutant exhibiting a substantial reduction in ACCase activity should have low kernel oil and be an albino lethal; bleached leaves are observed when wildtype seedlings are treated with sublethal rates of the ACCase inhibiting herbicides presumably because of a reduction in ACCase activity. This predicted phenotype is similar to the description of *loc1* (Plewa, MNL 53:93, 1973). Such a mutant would be of great interest to us and we would appreciate any guidance to a source of *loc1* or similar phenotype.

Purification of maize leaf acetyl CoA carboxylase

--Margaret A. Egli, Burle G. Gengenbach, John W. Gronwald[1], David A. Somers, and Donald L. Wyse
[1]USDA-ARS

Acetyl CoA carboxylase (E.C. 6.4.1.2; ACCase) catalyzes the initial step of fatty acid synthesis, carboxylation of acetyl CoA to form malonyl CoA, the substrate for fatty acid elongation and also for synthesis of flavonoids and other secondary metabolites. ACCase of most monocots, including maize, is inhibited by cyclohexanedione and aryloxyphenoxypropionate herbicides. Herbicide-tolerant maize lines having altered ACCase activity conferred by partially dominant nuclear mutations have been obtained from tissue culture (Parker et al., PNAS 87:7175-7179,1990). We plan to isolate the ACCase gene from wildtype maize to more fully examine its expression and regulation in normal and herbicide-tolerant genotypes. Our immediate objectives are to purify ACCase from wildtype maize, generate ACCase antibodies, and screen expression libraries to obtain ACCase mRNA sequences.

Approximately 60 ug ACCase was obtained from 50 g maize leaves (A619) extracted in buffer containing 0.2 mM PMSF (PNAS 87:7175) and purified as described in Table 1. Similar to other plant and animal ACCases, purified

Table 1. Purification of ACCase from A619 leaves.

step	Units[2] /mg	-fold purification	activity yield (%)	protein yield (%)
crude extract	0.002	1	100	100.00
30-40% $(NH_4)_2SO_4$	0.068	32	475	15.00
S-300	0.377	175	780	4.50
Blue Sepharose	2.23	1030	113	0.11
FPLC Mono-Q	5.84	2704	35	0.013

[2]incorporation of $[^{14}C]HCO_3$ into acid-stable products.

maize leaf ACCase has a native MW of approximately 490 kD (Superose 6 gel filtration), a pI of 6.9, and appears to be a dimer of biotinylated subunits of 220 kD (SDS-PAGE).

High-titer antiserum (rabbit) was obtained by subcutaneous injections of 20 to 100 ug of 220-kD ACCase peptide in SDS gels. Whole immune serum bound to Protein A-agarose immunoprecipitated ACCase activity. On western blots of crude leaf extracts, antibodies bound primarily to one 220-kD polypeptide. We are currently using the ACCase antiserum to screen a maize seedling lambda gt11 cDNA library (S. Gantt, Univ. Minnesota).

ACCase is known to be localized in plastids, the site of fatty acid synthesis, however, mitochondrial and cytosolic forms of similar MW occur in animals (Allred and Roman-Lopez, Biochem. J. 251:881-885). Plants may also contain different ACCase enzymes. We observed two biotinylated polypeptides of approximately 220 kD on western blots of crude leaf extracts probed with avidin, but only one was strongly recognized by ACCase antiserum. The possibility of ACCase isozymes will be further examined by using avidin and antiserum to test for 220-kD polypeptides in different maize organs and tissues, and by using an ACCase mRNA to estimate the number of ACCase genes.

STANFORD, CALIFORNIA
Stanford University

Germinal revertants of *bz2-mu1*, an allele with a *Mu1* element in a background with a typical, late pattern of somatic reversion

--Virginia Walbot

The *bz2-mu1* allele contains a *Mu1* element in the second exon (J. Nash et al., Plant Cell 2:1039, 1990). Like other *Mu*-induced mutable alleles, *bz2-mu1* yields a fine spotting pattern in the aleurone and other tissues. Only one germinal revertant allele has been verified: purple kernels in a sector encompassing about one-eighth of the ear (A. A. Levy et al., 1989 Dev. Genetics 10: 520) were shown to contain an extra proline residue in the *Bz2* sequence. Given the late sectoring pattern typical of the allele, occasional (est. $1/10^4$) individual purple kernels were ascribed to rare contamination and not analyzed further.

To determine the reversion rate more precisely, a large test was made in 1989. *bz2-mu1* homozygous and *bz2-mu1/bz2* plants were pollinated by *bz2*. Progeny ears were obtained from 339 plants; 83 of the ears had no spotted kernels indicating that 24% (83/339) of the individuals had lost Mutator activity. One ear had a small sector of 5 pur-

ple kernels. Of the total population of 38,000 spotted kernels, there were 9 additional purple kernels. By Southern blotting, three of these were demonstrated to be contaminants, leaving 7 putative, independent revertants. All of these transmitted normally, although one revertant (I124-8) gives a dark pink rather than full purple color. Using PCR we plan to determine the nature of the putative revertant alleles, and will accept alterations at the site of *Mu1* insertion as proof that these are revertants rather than contaminants. If all 7 events are verified to contain novel bases, the reversion frequency to a functional *Bz2* allele is 1.8×10^{-4} for this population.

Early events in Mutator lines: I. Germinal revertants

--Virginia Walbot

Last year (MNL 64:107) I reported on the gradual selection of a *bz1-mu1* line that yields early somatic excision events in the aleurone including half-kernel somatic and full function, germinal revertants. The *Mu1* allele is inserted just beyond the 3' intron boundary of the gene (A. B. Britt, unpublished data) in precisely the same location as the *bz-rcy* allele reported by P. S. Schnable et al. (Mol. Gen. Genetics 217:459, 1989). Two germinal revertants from ear sectors in this stock have been verified at the DNA sequence level (A. B. Britt, unpublished data). In 1990, the *bz1-mu1* early excision stock was grown in an isolation plot; about 900 ears were recovered, and the majority of these contain one or more germinal revertants.

A detailed analysis is complete for a smaller population of a second early excision line. This line was recovered in 1989, and involves an exceptional *bz2-mu2* plant (I216-3). The I216 family was derived from a faintly spotted stock, H210-1 (X), that appeared to be losing Mutator activity; other I216 family members showed near complete loss of Mutator activity on selfing and outcrossing, but I216-3 gave large spots on selfing and outcrossing to *bz2* tester. These progeny were verified by Southern mapping to contain a *Mu1* insertion near the 5' terminus of the coding region and are not *bz2-Ds* contaminants (C. Warren, unpublished data). From the 1990 crop, 20 progeny ears of 6 plants (one *bz2-mu2* and five *bz2-mu2/bz2*) crossed onto *bz2* tester have been analyzed for sector distribution. The Mutator plants are "turning off" and give about 25% sectored kernels (rather than 100% or 50%).

Phenotype	Number	Percent
purple	149	4.0
large spots	360	9.7
fine spots	354	9.5
bronze	2,853	76.8

Progeny of 14 selfed ears were also examined and germinal revertants were found. Although the data set is small, germinal reversion events are twice as likely in the pollen outcross test than in selfed ear progeny (1.9% in the ear vs. 4% in the pollen, on a per allele basis). This bias is more striking considering that loss of Mutator activity was more severe in pollen compared to ear progeny. *Mu* element excision frequency rate may differ significantly in the lineages leading to sperm and eggs. The data are consistent with the possibility that all of the purple kernels in the selfed test represent events transmitted through the pollen.

It is possible that early excision activity depends on a segregating factor. Kernels with the large spotting pattern give rise to progeny with both patterns; similarly kernels with the typical Mutator late spotting pattern yield progeny with both large and small sectors. Thus kernels with both sector types have the factors required to yield sectors of all sizes; the early pattern could depend on the copy number of a factor present in all stocks.

Early events in a Mutator line: II. Somatic reversion

--Virginia Walbot

This is chapter 2 of the story of I216-3, an exceptional plant carrying *bz2-mu2* with an early excision pattern. The sectoring is radically different from the typical *Mu*-induced allele in which few sectors larger than 250 cells are found (A. A. Levy and V. Walbot, Science 248:1534, 1990). In *bz2-mu1*, the highest excision frequency occurs near the end of aleurone development, resulting in sectors with 32 (2^5) or 64 (2^6) cells. In contrast, among the 714 large + fine spotted *bz2-mu2/bz2* kernels in the derivatives of I216-3 examined from an outcross population, early somatic events were common.

Sector	Cell Number	Number of Events	Frequency (x 10^{-3})
1/2 kernel	2^{17}	7	4.90
1/4	2^{16}	30	10.5
1/8	2^{15}	57	9.98
1/16	2^{14}	88	7.70
1/32	2^{13}	148	6.48

Frequency was calculated by dividing the number of events by the number of possible events (product of 714 and the reciprocal of sector size). These frequencies are similar to those recorded previously for *bz2-Ds* and *c2-Spm*.

The ratio of large:fine spotted kernels varied widely among the ears examined, and is hypothesized to reflect the activity state of Mutator rather than segregation of a single factor regulating excision timing. Virtually the entire population of *bz2-mu2* individuals (those described above and two additional groups with 31 more plants) show signs of losing Mutator activity: fewer than expected spotted kernels, some kernels with just a few spots per kernel. Further support for the hypothesis that Mutator activity state is affecting the large spotting phenotype comes from examination of the progeny of fine spotted I216-3 kernels with the typical Mutator phenotype. When fully active, these gave rise to ears with the full spectrum of phenotypes: germinal revertants, large spots and fine spotted kernels. Kernels with rare spotting indicative of initial loss of Mutator activity, on the other hand, gave kernels with no or small sectors only.

By Southern blotting the reporter alleles in large spot stocks appear unchanged. In progress are trans-activation tests to determine whether the early excision *bz1-mu1* and *bz2-mu2* stocks can change the sector size in *c2-mu1* and

a2 mutable alleles. I want to determine whether the early excision pattern is dominant and whether such lines can reactivate cryptic Mutator reporter alleles efficiently. Using a *bz2-mu2 R-r* stock I plan to determine whether early excision also applies to tassel development. Also in progress is a test of the transmission of exceptional "large spots" found in typical Mutator material. Twelve exceptional *bz2-mu1* kernels were found among 38,000 in the reversion test described in an accompanying note. Eight of these kernels had one spot each of ~256-512 cells, and the remaining 4 kernels each had one very large sector (estimated sizes: ~2,000, 5,000, 10,000 and 16,000 cells). If these exceptional kernels represent heritable changes in Mutator activity, then this change is relatively common (3 x 10^{-4}).

Sectors of null activity on somatically unstable kernels

--Virginia Walbot

In examining somatic instability in the early excision *bz2-mu2* line described in an accompanying note, I found null sectors in addition to purple sectors. In material with more than one dose of the reporter allele, null sectors are very likely to represent loss of Mutator activity. In outcross ears with just one dose of the reporter allele, however, null sectors could represent either loss of activity or excision events that fail to restore sufficient *Bz2* activity to condition a purple phenotype. Because *Mu* somatic excision is not very precise, usually resulting in small deletions (A. B. Britt, unpublished data), only one-third of *Mu* excisions from an exon are expected to restore the reading frame. Seven ears -- outcross progeny of 3 different plants -- were analyzed, and null sectors were found to be about 3 times more common than purple sectors of the same size.

Sector Size	Purple Sectors	Null Sectors	
		Obs.	Exp.
1/2 kernel	6	23	12
1/4	16	52	32
1/8	34	95	68

The excess of null sectors compared to predictions based solely on restoration of the reading frame (Exp. column) could result from [1] loss of Mutator activity (fewer than expected spotted kernels were recovered in this population), and [2] deletions or alterations that mutate an essential gene element, i.e. loss of the initiator or termination codon, loss of a site required for splicing, or amino acid changes that destroy protein function. The precise location of the *Mu1* insertion in *bz2-mu2* is not known but by Southern mapping it is in the reading frame near the ATG. Alterations of the reading frame would, therefore, be expected to yield null function.

The excess of single kernel revertants compared to ear sectors

--Virginia Walbot

A cluster of purple kernels on an ear containing a mutable gene of the anthocyanin pathway is likely to represent a single reversion event that occurred during ear ontogeny. The larger the sector, the earlier the event. If the frequency of excision is constant during development then a simple "powers of two" model would predict the number of clusters of each size, i.e. twice as many single kernel events as two kernel events, twice as many two kernel events as four kernel events, etc. Yet it is obvious that there is an excess of single kernel events. Does this mean that reversion frequency is much higher late in development?

Single kernel events are a complex class: an unknown number of cell divisions intervene between separation of lineages for individual kernels. Of events that occur just prior to meiosis, half will be lost during meiosis as a result of cell death. Among events that occur somewhat earlier, half will be lost as a result of lower floret abortion. Thus a single kernel may be the only visible event in a small sector; when a reversion event occurred early enough to affect 8 or more kernels, then it is highly likely that 2 purple kernels will be found and the sector will be correctly scored as occurring long before megaspore formation.

To determine the bias for single kernel events, the distribution of single kernels vs. clusters was scored for *a1-m4 Ac* crossed by *a1* tester in two populations of 33 and 16 ears, with an estimated 12,250 kernels scored. This test ignores the possibility of coincidental reversion of the two alleles contributed by the female parent and of contiguous lineages each undergoing a reversion event, because overall recovery of revertant kernels was low (191/12,250).

Sector Size	Number of Events
One kernel	156
Two kernels	19
3-4 kernels	9
5-8 kernels	3
9-16 kernels	1
17-32 kernels	3 (17, 19, 29 kernels)

There is clearly a bias for single purple kernels. With the larger sectors there is a trend that fits a "powers of two" notion of ear development: the events with two purple kernels are about twice as frequent as the 3-4 kernel class, etc. There is, however, considerable uncertainty in classifying the smaller sector sizes. Does a single kernel really represent a two or even four kernel event masked by segregation at meiosis? Do 3 kernels represent an event involving 4 or 8 kernels, etc.? Chance events should affect all categories equivalently, but a change in the frequency of reversion or in the number of mitotic events separating when it is possible for two kernels vs. one to be revertant from a single excision event will preferentially affect specific categories. More information on the ontogeny of the ear is required to resolve this dilemma. In my opinion it seems likely that there are more cell divisions in the lineages restricted to producing a single kernel than in the developmental events separating a two kernel from a four kernel lineage. At least in aleurone tissue, however, the frequency of excision changes over developmental time (A. A. Levy and V. Walbot, 1990 Science 248: 1534) so per-

haps there is an increase in *Ac* activity in the terminal stages of kernel development and/or gametogenesis as well.

Mu9, a new 5 kb member of the Mutator family of transposable elements

--Jane Hershberger, Christine Anne Warren and Virginia Walbot

The Mutator family of transposable elements causes high rates of forward mutation. Eight elements, named *Mu1* through *Mu8*, have been previously characterized. They range from 1.0 - 2.2 kb in size, and they have unique central regions flanked by homologous 215 bp terminal inverted repeats (TIRs). In a recent screening for new Mutator-induced insertions in genes of the anthocyanin pathway, this laboratory isolated *bz2-mu4*, an unstable allele of the Bronze-2 gene. Like many other Mutator insertions, *bz2-mu4* gives rise to small, frequent sectors of somatic excision and infrequent germinal revertants. Southern analysis of this mutant using probes from *Bz2* revealed the presence of a large (approximately 5 kb) insertion in the second exon of *Bz2*. To determine whether this large insertion was related to other large transposable elements, we digested genomic *bz2-mu4* DNA with a panel of diagnostic restriction endonucleases and blotted it. From the derived restriction map it was clear that the insertion was neither *Ac*, *Spm/En*, nor any simple deletion derivative of these elements. Hybridizing the same blots with a probe from the 215 bp TIR of the *Mu1* element did not, however, conclusively prove that this insertion belonged to the Mutator family of transposable elements. Therefore, we cloned the insertion to analyze it in more detail.

The insertion, flanked by about 5 kb of *Bz2* sequences, was cloned on two *Eco*RI fragments from a lambda zap II (Stratagene) genomic library. The restriction map of the isolated clones matches the one derived from our analysis of the genomic Southern blots. We have used several fragments derived from these clones to examine the copy number of this insertion and its derivatives in the maize genome. On Southern blots of maize genomic DNA, both active Mutator lines and non-Mutator lines contain multiple hybridizing fragments. A 500 bp probe from the central region of the cloned insertion hybridizes to 5-10 bands per haploid genome; a 450 bp fragment adjacent to the 3' TIR hybridizes to 10-15 bands per haploid genome. Thus, it appears that this sequence or deletion derivatives thereof exist in most or all lines.

Sequence analysis of the cloned DNA indicates that this 5 kb insertion from *bz2-mu4* is a new member of the Mutator family of transposable elements; we have named it *Mu9*. The insertion is flanked by 9 bp direct repeats of the sequence TCCTGGAGG. *Mu9* has the characteristic 215 bp TIR of the Mutator family, and these are 80-90% similar to the TIRs of other known Mutator elements. The *Mu1* TIR that was used as a probe on the genomic Southern blots mentioned above is only about 82% similar to the *Mu9* TIR, thus, hybridization between the two TIRs would be weak under the high stringency wash conditions we used.

As the sequence analysis of *Mu9* is still in progress we cannot yet draw firm conclusions about the protein(s) encoded by this element. However, multiple open reading frames of 100 amino acids or more are present throughout the sequence. We will use both RNAse protection and cDNA cloning to determine the exon/intron structure of *Mu9*. The previously identified Mutator elements do not have long open reading frames; because *Mu9* is more than twice as large as any of these, it has the potential to encode proteins that may be involved in the transposition process. Computer analysis should enable us to identify DNA-binding regions and other functional domains if any are present in the *Mu9*-encoded protein. Because *Mu9* has transposed into *Bz2*, we will be able to analyze this element and any proteins it encodes at both the molecular and the genetic level.

HPLC analyses of bronze pigments

--John Bodeau and Virginia Walbot

Although the exact enzymatic role of the *Bz2* gene product is still unknown, cross-feeding studies and phenotypic similarity of *bz2* mutants to *bz1* plants suggest that it acts very late in the pigment biosynthetic pathway. Recent findings of acylated anthocyanins in maize following mild extraction procedures (Harborne and Self, Phytochemistry 26:2417, 1987) suggest the possibility that *Bz2* encodes an acyl-transferase enzyme which acts after the *Bz1* encoded enzymatic step. To test this idea we tried to identify a pigment component (a cyanidin malonylglucoside?) of wildtype plants which is reduced in *bz2* plants.

We extracted soluble pigments from fresh young leaves of greenhouse-grown *bz1 B Pl*, *bz2 B Pl*, and *Bz1 Bz2 B Pl* plants of W23 background. Leaf slices were macerated in MAW (Methanol/Acetic Acid/Water, 8:1:1, pH~5) and allowed to equilibrate overnight at 4 C. Raw pigments were applied to a high-capacity LH-20 column run with MAW, to remove most simple sugars. Fractions with peak pigment were pooled and lyophilized. HPLC was performed with the help of Dr. Vern Singleton, and Eugene Trousdale, in the Department of Enology, University of California at Davis. Pigments were eluted on a C18 column using an acetonitrile acidic phosphate gradient. Elution peaks of cyanidin and cyanidin 3-glucoside were identified by spiking samples with these compounds.

In wildtype extracts, cyanidin 3-glucoside and trace amounts of cyanidin were present, while *bz1* pigments accumulate virtually no glucoside, as expected from the previously determined identity of the *Bz1* product as UDP glucose-flavonoid glucosyl-transferase. In *bz2 B Pl* leaf tissue no wild-type peaks appreciably decreased, including cyanidin 3-glucoside. Additionally, both *bz1* and *bz2* extracts accumulated high levels of an unidentified pigment which eluted at nearly the same point as purified cyanidin aglycone, and which was an extremely minor component of the wildtype extract.

While our hope of finding a "missing peak" which would provide a clue as to the function of *Bz2* was frustrated, the presence of cyanidin 3-glucoside in *bz2* pigment extracts provides further evidence that the *Bz2* gene product acts after *Bz1*.

Regulated transcription of the maize *Bz2*-promoter in electroporated BMS protoplasts

--John Bodeau and Virginia Walbot

We found that electroporating BMS protoplasts with gene constructs containing components of the maize anthocyanin genes is a simple and efficient technique for studying regulation of this set of genes. We tested promoter activity of a 700 bp upstream region of Bronze-2 using luciferase as a reporter gene, with the first intron from maize *Adh1* inserted into the 5' untranslated region (Bz2-I-Luc). Similar constructs with the *A1*- and *Bz1*-promoters (A1-I-Luc and Bz1-I-Luc) were obtained from Michael Fromm, while constructs expressing the *R* and *C1* regulatory genes from cDNAs expressed under the control of the CaMV 35S promoter were kindly provided by Susan Wessler and Steven Goff, respectively. As shown in Table 1, following electroporation of protoplasts the *Bz2*-promoter was activated by *R* and *C1* over 640-fold, while a 33-fold and 150-fold activation of the *A1*- and *Bz1*- promoters occurred. No luciferase expression was observed from a construct containing the *Bz2*-promoter in reversed orientation, either with or without *R* and *C1*. Absolute induced levels of luciferase expressed from the *Bz2* promoter were approximately twice those of a similar 35S-promoter construct, and approximately 100-fold above induced levels from *Bz1*- and *A1*- promoters. Similar results were also obtained using the particle gun to introduce these constructs into intact aleurones of mature seeds (data not shown).

Table 1. Expression of anthocyanin gene promoter constructs in electroporated BMS cells.

Plasmids	Relative expression[1]	Fold enhancement by C1 and/or R	Color
Bz2-I-Luc	0.36	1.0	-
Bz2-I-Luc, 35S-R	0.29	0.8	-
Bz2-I-Luc, 35S-C1	0.56	1.6	-
Bz2-I-Luc, 35S-C1,35S-R	229.03	639.6	Red
Bz1-I-Luc	0.02	1.0	-
Bz1-I-Luc,35S-R, 35S-C1	2.36	115.8	Red
A1-I-Luc	0.10	1.0	-
A1-I-Luc,35S-R, 35S-C1	3.26	33.0	Red
35S-I-Luc (pJD312)	52.88	1.0	-
35S-I-Luc,35S-C1, 35S-R	85.25	1.6	Red

[1] Each DNA mixture included an equal amount of a GUS-encoding plasmid (35S-I-GUS). Relative expression is expressed as light units produced/moles GUS converted.

Additionally, introduction of *R* and *C1* into BMS protoplasts activated the endogenous anthocyanin structural genes, resulting in red protoplasts within 24 hours. By 48 hours, approximately 30% of surviving protoplasts appear pigmented.

RNA was isolated from electroporated protoplasts, and RNase protection demonstrated that induction levels of luciferase and visible pigment reflect differences in RNA message abundance of both introduced and endogenous genes. Both *Bz1* and *Bz2* messenger RNAs are undetectable in protoplasts not receiving *R* and *C1*, while cells receiving *R* and *C1* accumulate high levels of endogenous *Bz1* message, and a much lower level of *Bz2* message. Luciferase message expressed from the *Bz2*-promoter is induced from undetectable to high levels comparable to the induced level of endogenous *Bz1* message.

We are intrigued by the discrepancy between the high activity of the isolated *Bz2*-promoter, and the low accumulation of endogenous *Bz2* message. Possible explanations for this difference include silencer elements in the endogenous gene which are not present in the 700 bp promoter fragment, and low stability of the *Bz2* message.

STANTON, MINNESOTA
Northrup King Co.

Near-isogenic line localization of MDMV resistance to chromosome 6S

--Ed Weck, Diana Beckman, Doug Mead, Christi Bredenkamp and Mary Trainor

A single major gene for MDMV resistance (*Mdm1*) was mapped to chromosome 6S between probes *UMC85* and *BNL6.29* (McMullen and Louie, Mol. Plant-Microbe Interactions 2:309, 1989) in two backcross populations using the inbred Pa405 as a source of resistance because of its symptomless response to MDMV strains A and B. Near-isogenic lines are a genetic resource for the integration of conventional and molecular marker linkage maps (G. J. Muehlbauer, et al., Crop Sci. 28:729, 1988). We have confirmed the chromosome 6S location for MDMV resistance using two independently created near-isogenic lines. Selections for MDMV resistance with 6S markers confirm the location of MDMV resistance.

Table 1. Lines analyzed and their level of MDMV resistance.

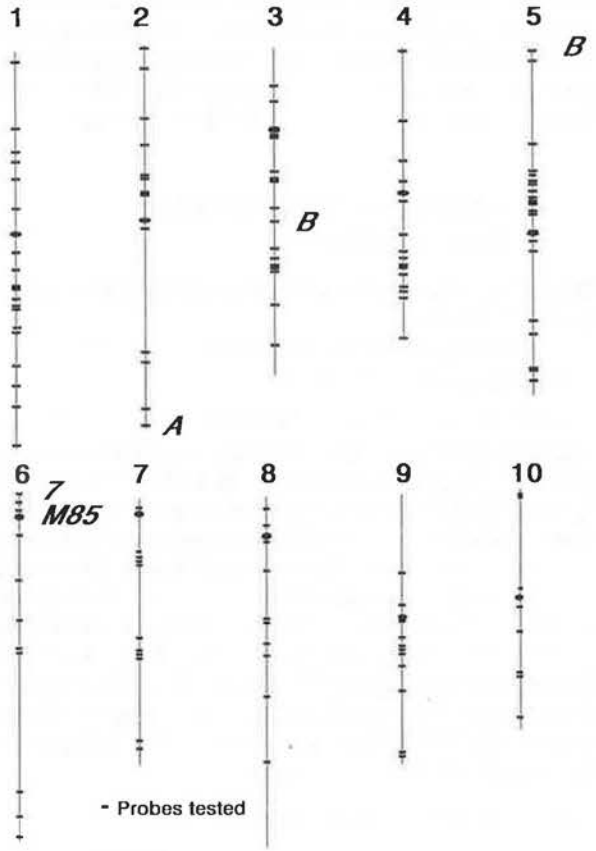
Line	% MDMV Resistant
Pa405	100%
NKA	0%
NKA-MDM (*Mdm1* from Pa405)	100%
NKB	0%
NKB-MDM (*Mdm1* from Pa405)	66%
NKC = (NKC*8/Pa405) 7x	86%
NKD = (NKD*9/Pa405) 7x	58%
NKE = (NKE*8/Pa405) 6x	21%

Greenhouse inoculations were with Maize Dwarf Mosaic Virus Strains A and B. Symptoms were measured 7, 14, 21, and 28 days after virus inoculation. Plants resistant on all 4 days were termed resistant. (NKC*8/Pa405) 7x indicates a cross of Pa405 to NKC that was backcrossed 8 times to NKC and selfed 7 times.

Two independently created MDMV resistant lines (NKA-MDM,NKB-MDM), the near-isogenic non-resistant lines (NKA,NKB) and the source of MDMV resistance (Pa405) were analyzed with 139 RFLP probes distributed over all corn chromosomes, see Figure 1. Two probes, *NPI7* and *UMC85* on chromosome 6S, showed a common pattern for the MDMV resistant lines: NKA-MDM; NKB-MDM; and Pa405. The presence of this chromosome 6S region in two independent MDMV resistance conversions and in the backcross populations of McMullen and Louie (Mol. Plant-Microbe Interactions 2:309, 1989) indicates that this is an important region in MDMV resistance.

Other regions from Pa405 appeared in the MDMV resistance conversions, NKA-MDM and NKB-MDM. Two probes represented by the letter B, showed an identical banding pattern for NKB-MDM and Pa405 and one probe, represented by the letter A, showed an identical banding pattern for NKA-MDM and Pa405. Letters A and B indi-

Figure 1., Probe distribution



cate regions of the Pa405 genome that were transferred to MDMV conversions NKB-MDM or NKA-MDM respectively. The additional region in NKA-MDM or the two additional regions in NKB-MDM could result in NKA-MDM being 100% resistant while NKB-MDM is only 66% resistant.

The location of *Mdm1* on chromosome 6S was used to guide MDMV resistance selection in lines segregating for MDMV resistance which had not yielded to breeders attempts to fix the resistance. In the summer of 1988 three lines were planted: (NKC*8/Pa405)7x, (NKD*9/Pa405) 7x and (NKE*8/Pa405) 7x. Table 2 shows the selections planted in the field and greenhouse and the percentage of plants resistant to MDMV. Pollinations from these populations were analyzed for MDMV resistance in the winter of 1988 in the greenhouse. Selections were planted in the summer of 1989, analyzed with chromosome 6S probes, and tested for MDMV resistance in the greenhouse in the winter of 1989. The winter 1989 MDMV viral greenhouse screen showed 99% (204/206) of the individuals to be MDMV resistant following selection with 6S probes *NPI7* and *UMC85* in the summer of 1989. This indicates that these two 6S probes are strongly correlated with MDMV resistance when using Pa405 as a source.

Probe patterns for 1988 and 1989 field grown plants are summarized in Figure 2. MDMV resistance data for these plants is shown in Table 2. Genomic DNA was digested with enzymes *Sst*I and *Hin*dIII in an attempt to explain the different patterns observed with probe *UMC85*

Figure 2., Chromosome 6S probe patterns

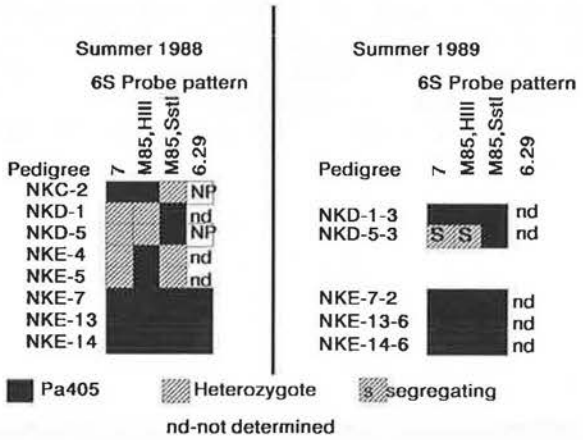


Table 2.
MDMV Resistance of Breeding Populations

Pedigree MDMV resistance	Field 88	Greenhouse 88 % MDMV res	Field 89	Greenhouse 89 % MDMV res	
(NKC*8/Pa405) 7x	NKC-2	67			
				NKD-1-3-2	100 (16/16)
(NKD*9/Pa405) 7x	NKD-1	30	NKD-1-3	NKD-1-3-4	100 (22/22)
				NKD-1-3-10	100 (5/5)
	NKD-5	71	NKD-5-3		
			NKD-5-4	NKE-13-6-1	100 (10/10)
(NKE*8/Pa405) 6x	NKE-4	14		NKE-13-6-6	100 (10/10)
				NKE-13-6-10	100 (8/8)
	NKE-5	67		NKE-13-6-12	100 (9/9)
				NKE-13-6-15	100 (9/9)
	NKE-7	100	NKE-7-2	NKE-13-5-1	60 (3/5)
			NKE-7-6	NKE-13-5-2	100 (10/10)
				NKE-13-5-3	100 (9/9)
	NKE-13	100	NKE-13-6	NKE-13-5-4	100 (10/10)
			NKE-13-5	NKE-13-5-5	100 (8/8)
	NKE-14	100	NKE-14-6	NKE-14-6-6	100 (12/12)
			NKE-14-8	NKE-14-6-6	100 (12/12)
				NKE-14-6-8	100 (12/12)
				NKE-14-6-12	100 (10/10)
				NKE-14-8-1	100 (8/8)
				NKE-14-8-3	100 (10/10)
				NKE-14-8-4	100 (10/10)
				NKE-14-8-5	100 (1/1)
				NKE-14-8-9	100 (10/10)

relative to the level of MDMV resistance. Plants which were 100% resistant had the Pa405 pattern for both enzymes. Probe *BNL6.29* was non-polymorphic for NKD-1, NKD-5 and NKC-2.

We have shown that near-isogenic lines can be used in conjunction with RFLPs to localize traits to a marker interval. Two independent near-isogenic conversions may be sufficient to localize a trait of interest and to eliminate regions from the nonrecurrent parent genomes unrelated to that trait. An advantage of the use of near-isogenic lines is that fewer lines are required for molecular marker analysis. A disadvantage is the number of generations required to construct near-isogenic lines by backcrossing. Near-isogenic lines are a powerful mapping tool in crops such as tomato and soybean where a large number of near-isogenic lines are available.

waxy PCR

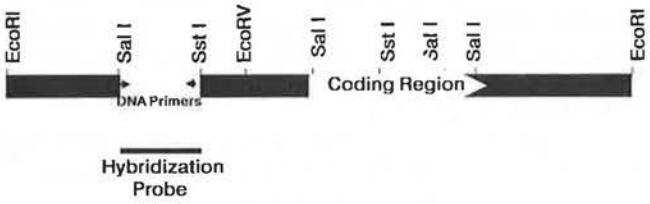
--David Farrar, Steve Larson and Ed Weck

The evaluation of individuals within a population is an essential aspect of any breeding program and the rapid identification of plants with desirable characterististics could save time and effort. In order to assess PCR (the Polymerase Chain Reaction) for application to plant breeding problems, a screen for important sources of the waxy gene has been developed.

The location of the waxy probe used for hybridization studies with six starchy inbreds and three waxy inbreds is shown in Figure 1. The band sizes were determined with a sonic digitizer from a Southern blot with Lambda DNA digested with *Hin*dIII as a marker. The waxy inbreds all have larger waxy fragments than the starchy inbreds.

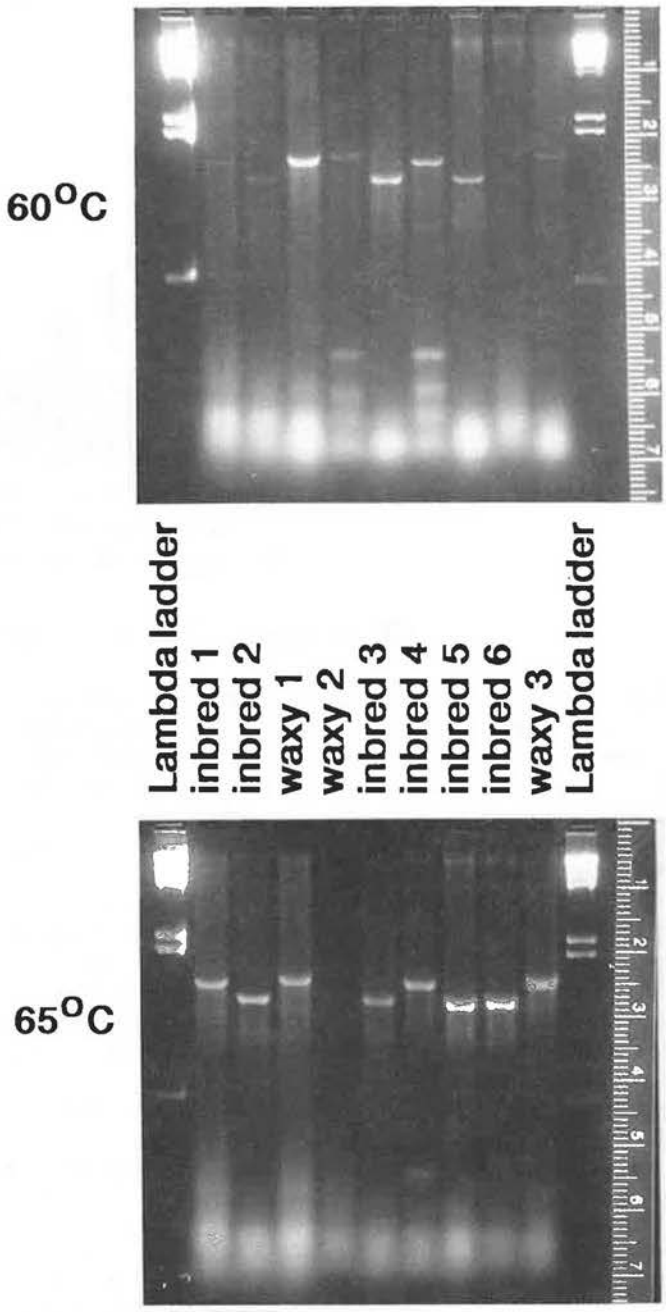
The waxy hybridization probes were sequenced terminally and DNA primers designed therefrom. DNA amplification was compared at two temperatures, 60 C and 65 C, using the corn lines listed in Figure 1. The fragment sizes generated with PCR agree with the RFLP results (Fig. 2) except for Inbred 5 which gives a 100 bp smaller band in PCR than in hybridization. The waxy 2 inbred appears to be a null allele when amplified at 65 C suggesting that one of the DNA primer binding sites is causally related

Figure 1., waxy Probe Location



Corn Line	size,bp
Inbred 1	1262
Inbred 2	1041
waxy 1	1324
waxy 2	1317
Inbred 3	1015
Inbred 4	1229
Inbred 5	1113
Inbred 6	1025
waxy 3	1317

Figure 2., waxy PCR

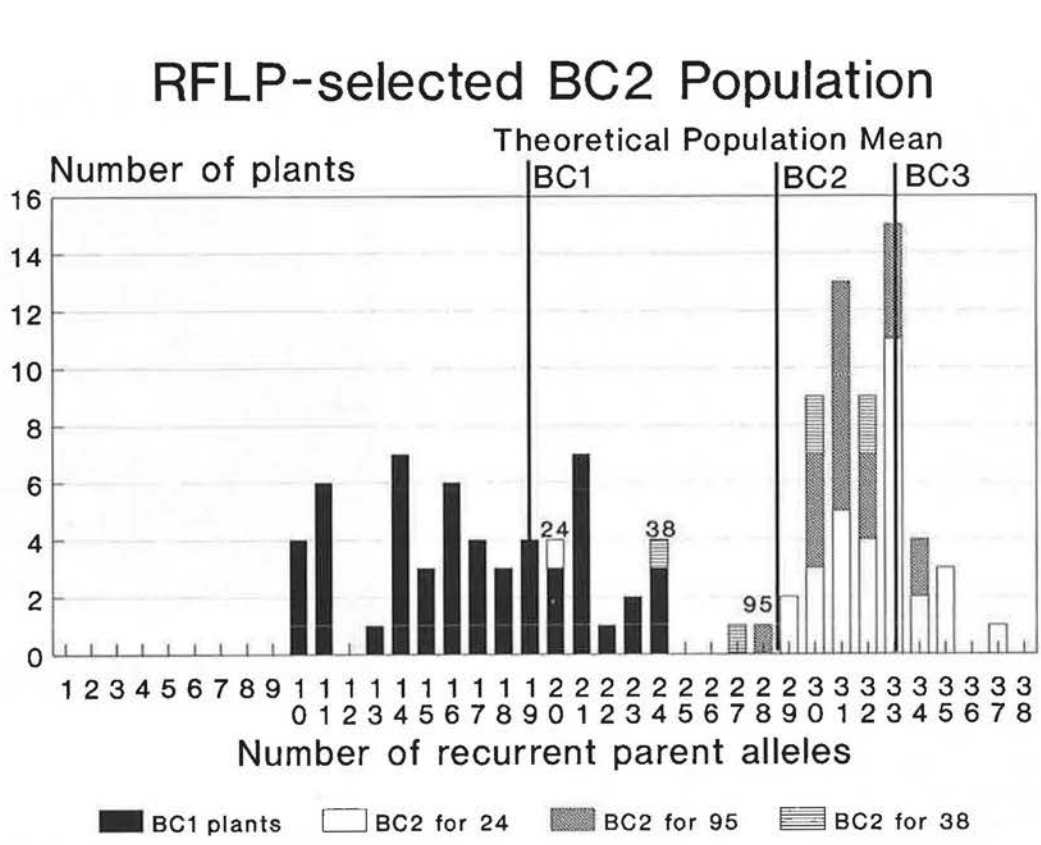


to the waxy mutation. PCR is more rapid than RFLP analysis and the resolution of small fragments on analytical gels is greater than on Southern blots.

RFLPs push BC2 selections ahead

--Ed Weck, Mary Bergstedt, David Farrar, Mike Kiefer, Carolynn Krumm, Mike Lundell, Doug Mead, Hope Sunderland, Roger Taylor and Carol Ann Wangen

RFLP-assisted selection for recurrent parent alleles should increase the generational gain over phenotypic se-



lection in backcross breeding. Computer simulations suggest that the recurrent parent genotype can be reconstructed in three generations of 30 individuals each (Tanksley et al. Bio/Technology 7:257, 1989). Here we compare RFLP-assisted selection of BC2 progeny with theoretical estimates of BC2 (backcross 2) and BC3 population means in the absence of selection.

A BC1 population of 125 plants was planted in the greenhouse and analyzed with probes for the single gene of interest. The 57 plants heterozygous for the trait were analyzed with up to 38 RFLP probes to estimate percentage recurrent parent. These 57 plants were backcrossed to the recurrent parent to create BC2 ears. BC2 seed (100) of the three BC1 plants with the highest percentage recurrent parent (plants 24, 95, and 38) were planted in the field. BC2 plants (58) heterozygous for the trait of interest were analyzed with probes heterozygous (non recurrent parent) in the BC1. Concurrent with the laboratory analysis the BC2 plants were backcrossed to create BC3 plants.

The recurrent parent allele number distribution for 57 BC1 plants is shown in solid bars in Figure 1. The three BC1 plants selected for further backcrossing (24,95, and 38) and the BC2 plants derived therefrom are shown, respectively, in: white bars; diagonal bars; and horizontal bars. The population mean of the RFLP-selected BC2 plants (32) is closer to the theoretical mean for a BC3 population (33.25) than a BC2 population (28.5). The attainment of 97.4% recurrent parent (37/38 probes; 58 plants) after two backcross generations suggests that two RFLP-assisted backcrosses may be sufficient to return an inbred to type.

6S mapping

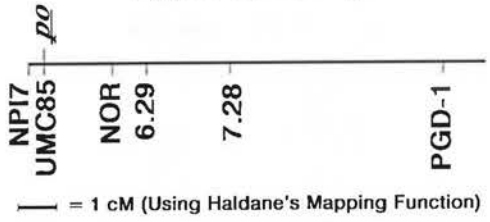
--Doug Mead, Russ Kwan, Mike Flaherty and Ed Weck

We have begun to create an integrated chromosome 6S map using morphological, isozymic and phenotypic marker data. As a preliminary step in establishing the most polymorphic cross, Stock Center lines (courtesy of E. B. Patterson) 601 A-G, 602 A-E, 603 B and 603 D were planted in Stanton in the summer of 1989 and analyzed with RFLP probes *NPI7*, *UMC85*, *BNL6.29*, *BNL7.28*, and NOR and the isozyme *Pgd1*. Line 601E (*ms6* x *ms6*/+; 78-599-4/-5) was polymorphic for the above mentioned RFLP and isozyme markers and an ear heterozygous for all markers was selected for further mapping studies.

117 plants from the selfed ear were sampled for *Pgd1* isozyme analysis and planted in the greenhouse. Although the plants showed early signs of stress, possibly due to tissue sampling for isozyme analysis, all plants could be scored for polymitotic. 116 plants (one plant gave bad DNA) were analyzed with the RFLP probes *NPI7*, *UMC85*, NOR, *BNL6.29*, *BNL7.28*.

Isozyme and RFLP data were analyzed with Mapmaker (Lander et al. Genomics 1:174, 1987). A program

Figure 1., 6S Map



was written in Fortran to compare *ms6* = *po* phenotypic data with RFLP and isozyme genotypic data using maximum likelihood equations from Allard, Hilgardia 24:235 (1956).

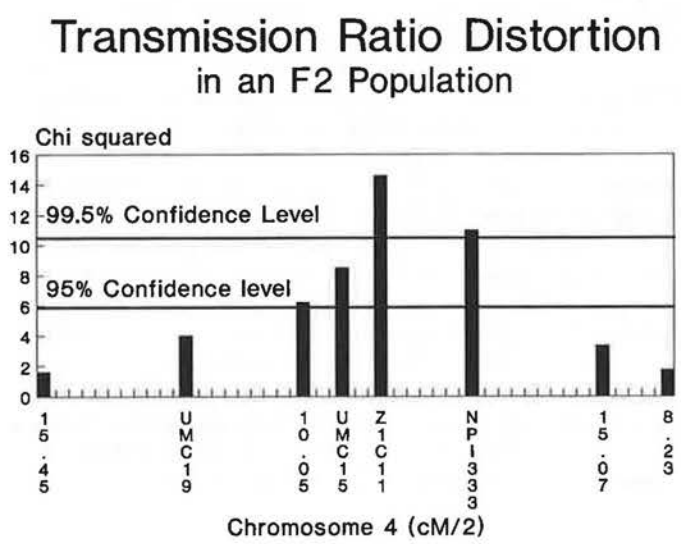
The 6S map in Figure 1 shows the distances between the 6S isozyme and RFLP markers as determined in Mapmaker. In this cross, the Fortran program shows the map position of *po* to be identical with that of *UMC85*. Additional crosses will be made in order to integrate ragged, wilted, and piebald leaves into the map.

Transmission ratio distortion

--Doug Mead, Mike Flaherty and Ed Weck

Preferential inheritance of one parental allele (hereafter, TRD, transmission ratio distortion) has been observed in interspecific crosses of mouse (see Biddle, Genome 29:389, 1987), tomato (Kinzer, et al. TAG 79:489, 1990), and other plant species (see Zamir et al., Bot.Gaz. 147:355, 1986). TRD has also been observed in maize crosses associated with *Ga* on chromosome 4S (Jones and Mangelsdorf, Anatomical Record 31:351, 1925). We report TRD on chromosome 4L in a cross of two elite inbred lines.

During the analysis of an F2 population created from two inbred lines, six of 108 loci gave ratios deviating from Mendelian expectations (Chi squared >5.99, 95% confidence level). Of particular interest was the long arm of chromosome 4, see Figure 1, where four of the six non-randomly segregating probes were located (Two of those probes exceeded Mendelian segregation expectations at the 99.5% confidence level; Chi squared > 10.60). In the other two cases (*UMC67*, 1L, Chi squared=8.70 and *BNL10.17*, 10S Chi squared=6.10), only a single probe per chromosome with a deviant Chi squared value was observed. These results are similar to those obtained by Kinzer et al. in tomato.



TRD in other plant species has been attributed to selection during one or more of the following phases of the plant's life cycle (Zamir et al.): 1) between meiosis and the formation of the mature gametophytes; 2) from pollination until fertilization of the embryo; 3) during seed development; and 4) during seed germination and plant growth. We have no indication of where in the maize life cycle TRD occurs in this F2 cross. Additional populations with these inbred parents will be analyzed for evidence of TRD on chromosome 4L.

TUCSON, ARIZONA
University of Arizona

Thoughts on future efforts for developing the maize genetics linkage map using RFLPs

--Tim Helentjaris

The development of molecular marker systems (such as isozymes, RFLPs, etc.) has given geneticists an exciting new tool and profoundly changed the way many of us now approach questions in both basic and applied genetic research. Those of us involved in the development of these tools over the last several years have been in discussion recently as to how this technology can best be advanced for the benefit of the general maize research community. Better correlation of these markers with previous marker types is one obvious direction and the Univ. of Mo. has been steadily working towards this goal with support from NSF. Better correlation of all of the available marker sets is also in progress utilizing the recombinant inbred lines developed by B. Burr.

Increased use of this technology is resulting in a rapid growth in the identification of the genomic positions of loci affecting plant phenotypes, particularly those that influence economically important traits. Meanwhile, interest has also mounted in utilizing this information to isolate the causative genes. A variation of chromosome walking has already been successfully used in arabidopsis to clone one such gene and these types of efforts will certainly continue in this species. However, one must recognize that the initial arabidopsis efforts have been much more challenging than originally anticipated and extrapolation of this approach into other species may be impractical. In maize, a tight positional linkage of 1cM would consist on average of greater than two million base pairs (5 X 10^6bp / 2300cM per genome), compared with a similar value for arabidopsis of about 140,000bp. The former represents an almost insurmountable distance to both traverse and search through for coding sequences. The presence of numerous repetitive elements in the maize genome will also severely complicate use of such a strategy.

The application of such approaches would require the development of a very high density marker map (approx. 1cM resolution) that is tightly correlated to the desired

phenotypic loci. Before embarking on any such development, I have questioned whether this is the only practical strategy or if others should also be considered. Instead of using random *Pst*I-generated genomic fragments as most of us have to date, I propose that it might be more productive in the long run to use randomly-selected cDNA clones as the source for marker sequences. Most of these are simple in their genomic organization, making them quite suitable for RFLP-mapping. The added benefit is that since they directly represent expressed genic sequences, every time one maps one of these sequences, one has also determined the genomic location of a gene which can be compared with the locations of loci affecting plant phenotypes. Where correlations are found, further efforts could in theory associate a cloned genic sequence with a phenotypic alteration. If this idea could be carried to the logical extreme, whereby one has mapped all of the expressed genic sequences and all of the loci that cause phenotypic alterations, this would represent a powerful tool for maize geneticists. Anyone interested in cloning a locus, whose genomic location could be established, could simply contact a stock center and request all of the expressed sequences within that region. Final confirmation to establish which of the candidate sequences actually represented the desired gene would necessitate the use of other strategies (i.e. DNA sequencing, anti-sense or complementation transformation) not based upon positional information. This confirmation would also be required of any chromosome walking strategy; what one gains here is not having to traverse a large segment of chromosome and subsequently searching through it for all of the expressed sequences.

Given such a scenario, the first question might be: is it practical? As this type of strategy is "processive" and not directed, it will require the development of both a "complete" and "normalized" cDNA library. I believe a relatively complete cDNA library can be prepared by using mRNA isolated from several tissues and subsequently combined. At least three normalization strategies have also been proposed that might be applicable in this case. Given some of the intrinsic properties of maize (very high polymorphism rates) and improvements in technology (such as PCR), it is already practical for a group to analyze 100-200 clones per week. By using 100 RIs or F2s as our mapping population, we could achieve an average resolution of less than half a centimorgan in the final map. This would represent an average resolution (enrichment) for any cDNA of about 5500-fold (i.e. 5456 "bins" are created in 100 RIs or F2s). I will use an estimate for the number of genes in a higher plant as approximately 25,000 (arabidopsis genome size of 0.7×10^8bp / an average-sized gene of 3000bp). At a rate of only 100 clones analyzed per week and 40 active weeks per year, one could have potentially mapped almost half of the genes in the maize genome in a four-year program. Pushing this only by a factor of two will mean that one could map a majority of the maize genes. There are a number of other considerations, such as duplicated chromosomal segments, non-polymorphic sequences, non-poly-A mRNAs, etc., that one should also recognize, but I do not believe they significantly burden the overall concept.

E. Coe, D. Weber, and myself are currently proposing to the USDA as part of the Plant Genome Mapping Initiative that this strategy be considered for maize. The Univ. of Az. would be responsible for most of the molecular mapping of random cDNA clones by recombinational methods. Illinois State Univ. would use cytogenetic landmarks to correlate the genetic and physical maps. Coe and Chao at the Univ. of Mo. would attempt to improve our information on the genomic positions of previously mapped Naked Eye Polymorphisms. Additionally the Univ. of Mo. will try to dramatically expand this number to several thousand loci using interval mapping strategies and the wealth of mutants collected and maintained by the maize research community, such as the one produced at Columbia by M. G. Neuffer totaling 6000 members. Our intention would be to release all biologicals and information to the general community as soon as we felt their analysis was satisfactory.

While this is certainly an ambitious goal, in many ways it simply represents a logical extension of previous efforts which have been enhanced by considering new ideas and technologies. Even if we fail in the final goal of mapping enough genic sequences to allow a researcher to possess a reasonable chance of finding their desired sequence amongst them, we should still obtain a very high density marker map (several thousand loci) that would be very amenable to other uses including chromosome walking. Meanwhile we should be able to include additional studies into the basic screening process that will reveal much more detail about the structure of the maize genome, the conservation of expressed sequences within the Gramineae, the regulation of individual sequences in different maize tissues or under different environmental conditions, etc. We have also committed ourselves to making a major effort to map maize sequences homologous to those isolated from and identified in other organisms. For instance the mapping of maize sequences homologous to Ti-tagged arabidopsis loci could provide a tremendous source of cloned genic sequences that could be associated with similar maize mutant phenotypes. Our reason for publicizing this idea now is to gain comments from the greater maize research community as to its practicality and utility. We also believe that there are many points in this process where other individuals could participate, by supplying either clones or mutants for analysis that would benefit them directly. Accordingly I would appreciate suggestions and criticisms on any of these topics.

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Brookhaven National Laboratory

Mapping new mutations using RFLPs

--Eileen C. Matz, Frances A. Burr and Benjamin Burr

The usual means of mapping a new mutation in maize employ either multiple marked testers, B-A translocations, or *wx* translocations. The latter two methods have been successfully used on a routine basis by a number of investigators. We have used B-A translocations ourselves to lo-

cate molecular markers to chromosome arms in initial stages of the construction of a molecular map. These stocks provide an elegant means of mapping a gene to a chromosome arm in one generation, providing the gene resides in the 85% of the genome covered by the available set of B-A translocations. We found that considerable time was spent in maintaining and assaying these stocks as well as in making the multiple crosses to mutant lines.

We thought that it would be interesting to explore the feasibility of mapping new mutations solely with molecular markers. We reasoned that if a modest number of well chosen molecular markers were used to screen a segregating population, the entire genome could be surveyed. Once linkage was detected, a fairly precise map location could be determined. Additionally, this work could be done at a time that would not conflict with field observations, genetic crosses, and data collection.

Linkage between two genes separated by 35% recombination in a small F2 or by 30% in a backcross can be detected at the 5% level of probability. This means that approximately 21 markers distributed over the genome are sufficient to detect linkage. The three examples presented here use backcross populations. In two cases we analyzed members of each phenotypic class and in the third case we examined only the homozygous mutants. A more efficient method, however, would be to examine only the homozygous mutant class in an F2 population.

Candidate RFLP markers were tested to see if they detected polymorphism between the two parents of the segregating population. Initially, parental DNA was digested with *Bam*HI, *Bgl*II, or *Eco*RI; however, we subsequently learned that screening for polymorphism was more efficient if *Hin*dIII was included in this group. Because of the extensive levels of polymorphism present in maize, most probes yield useful polymorphism. It must be emphasized that the selection of RFLP markers and restriction enzymes used to show polymorphism is specific to each population. Once polymorphism was detected for a specific RFLP marker, DNA from the segregating population was digested with the appropriate enzyme, prepared for Southern hybridization, and the distribution of alleles was scored from the resulting autoradiograms. Reprobing of filters reduces the number of times this entire procedure has to be repeated. The Chi-square method was used to test deviation of allele distributions from those expected by chance (indicating no linkage). Chi-square values that exceeded the 5% level of probability indicated that other markers in the same region of the genome required testing. These markers confirmed linkage and provided a clearer indication of the map position of the mutant.

Example 1: *Ufo1* is a dominant mutation giving rise to orange plant color (E. D. Styles, MNL 61:100, 1987). Derek Styles gave us homozygous mutant plants, a backcross population, and sibs of the normal backcross parent. Because of the heterozygosity present in the parental stocks, this was an unusual situation in that the + allele was undefined. Nevertheless, having the homozygous mutant allele allowed us to detect linkage.

Example 2: *rd3* is a plant with reduced stature that was obtained by screening segregating F2s from an EMS mutagenesis experiment designed to detect new mutations affecting plant color in a *B-Bolivia, pl, r-g* background. The original mutant plants were small and had reduced plant color. These were crossed to an unrelated *B-S, pl, R-g* stock and backcrossed to the mutant. Of the 22 plants obtained from the backcross, 10 had reduced stature. All of the normal plants were pigmented, but 5 of the small plants were nearly colorless. In this population we assayed only the reduced stature class for RFLP alleles. The colorless phenotype segregating in the reduced plants maps near *b1* on 2S. It appears that the recessive *rd3* genotype is a precondition for the expression of the colorless phenotype in the double mutant. Because *rd3* maps on 3L, we have made the cross to test allelism with *na1*, but the phenotypes are so dissimilar we do not expect them to be allelic.

Table 1. RFLP mapping of mutant phenotypes.

Mutant	Loci scored	Linked loci	Map position	Homozygous[a] class	Heterozygous[a] class	% Recomb
Ufo1	25	*pio200626*	10S-12	8/11	3/13	25
		bnl3.-04	10S07	9/9	3/13	14
		npi285	10S006	7/10	2/12	23
		npi264	10L038	7/10	3/13	26
rd3	25	*bnl6.06*	3L069	9/10		10
		vp1	3L076	9/10		10
		bnl5.37	3L084	9/10		10
		bnl10.24A	3L089	9/10		10
		bnl8.01	3L095	9/10		10
		bnl5.14	3L097	9/10		10
sh-459b*	25	*bnl6.22*	5S024	10/10	0/14	0
		bnl5.71	5L051	9/10	3/14	17

[a]Fraction that are homozygous for the marker allele present in the recessive parent.
[b]Allelic with *sh5*.

Example 3: *sh*-459* is our lab designation for what is actually a brittle kernel phenotype. The mutation is from an EMS treated population generated by Gerry Neuffer. His designation is *sh*-op1992*. As can be seen in Table 1, we mapped this mutant to 5S. Based on this information we tested for allelism with *sh5* and obtained a positive allelism test. The *sh*-459* allele has a more severe phenotype than the allele identified by George Sprague.

Table 1 indicates that 25 marker loci were scored for allele distribution in the case of each mutant. This number is probably on the high side because we did not detect linkage until most chromosome arms had been screened in all three cases. Several other marker loci were then scored to narrow the approximate map position. Given the small populations screened in all cases, the present results only indicate approximate map location.

Database for loci mapped in TxCM and COxTx RI families

--B. Burr, F.A. Burr and E.C. Matz

Ed Coe has asked us to reproduce our laboratory database that describes everything we know about the loci mapped in the recombinant inbred families. Because this database was never intended for publication, browsers will find it both idiosyncratic and cryptic. The fields are:

1) the name of the locus
2) estimated map position

LOCUS	POS.	INBRED LINE CM37	ENZYME T232	& MORPH SIZE Tx303	CO159	REMARKS
a1	3L149	Bgl2 ~9, Hin3 11	Bgl2 ~16, Hin3 7.3	Bgl2 ~6	Bgl2 ~16	probe is pAMu2 from Z. Schwarz-Sommer. 600 bp.
abp1	3L068					Auxin binding protein;data from Marian Lobler,probe = G900,saw SstI polymorphism. 10/18/90 combined with pKlamb+ of A Jones
aco1	4S059			1	4	isozyme
acp1	9L070	2	4	4	3	isozyme
acp4	1L177	5	4	4	5	isozyme
act1	8L025	Bgl2 6.0	3.9			probe is pMac1 from R. Meagher
adh1	1L128	Bam 12	Bam 16, 7.1	4	6	probe is pH2.3 from M. Freeling, isozyme in 2nd family
adh2	4S046	Hin3 11kb	5.4	Eco 7.2, Bam ~6	Eco 13.5 Bam ~5.5	probe is pZML841 from M. Sachs
agp1	6L109			BamHI 18.2 kb	8.3 kb	2.2 kb EcoRI cDNA clone.Embryo specific counterpart to sh2 from M.J. Giroux
agp2	2L106	EcoRI~24.5 kb	8.5 kb	EcoRI 13.5 kb	6.9 kb	1.8 kb EcoRI cDNA.Embryo specific counterpart to bt2.From M.J. Giroux.
agr-c94	4S021	Bam 1.5	Bam 5.8	Bam 1.5	Bam 8.3	cDNA probe from Agrigenetics; 0.83 kb fragment from a Pst digest, also a .35kb.
agr-r115	4S005	Bgl2 6.8	7.8	Bgl2 5.4	10.5	Blots probed by Yu Ma with a cDNA probe.
als1	4L087			Hin3 16-17	Hin3 2.5	acetolactate pyruvate-lyase from tobacco probed by N.S. Shepherd, 1st band
als2	5L049			Hin3 9.5	Hin3 3	2nd band probed with tobacco als by n.S. Shepherd
amp3	5S038	4	3	4	3	isozyme
ars1	2S051	BglII 8.0 kb (18.2 + 16.3 kb)	19.6 kb (10.6 kb)	BglII 8.0 kb (18 + 4.4 kb)	8.4 kb (5.45 + 4.7 kb)	800 bp PstI from Paul Sisco; Eileen thinks 680 bp.Should map close to b1. 10/16/90
b1	2S049	+	-	-	-	morphological
bnl1.297	3L126	Bam ~4.5	~5.5	Bam ~10	~15,7	same as 1.80, O.95 Eco cDNA
bnl1.326	1S016			Eco ~6.5	~7.5	0.54 Eco cDNA
bnl1.380	5S028	Eco ~4	~3.5	Eco ~4	~20	0.32 Eco cDNA
bnl1.45	8L015	Eco ~6.5	~5.5	Eco ~6.5	~5.5	0.44 Eco cDNA
bnl1.556	1S052	Eco ~13	~16	Eco ~18	~15	LINKED WITH 5.59, 0.44 Eco cDNA
bnl1.80	3L126	Bam ~4.5	~6	Bam ~12	~16	same as 1.297
bnl2.369	8L032	Eco >25	~14	Bgl2 ~14	6.6	0.70 kb Eco cDNA
bnl3.02	10S-07	Bam ~16	~6.5	Bam ~24	~10	Same as 3.04
bnl3.03	6L067	Bam 15	9	Bgl1 >25, 15.5	Bgl1 13, 4.4	2.5kb PstI genomic
bnl3.04	10S-07	Bam 22, 6.6	~10, 6	Bam 22, 6.6	~10. 6	LINKED WITH 5.40 AND 10.17 IN FIRST FAMILY; 2.2 kb Pst Genomic
bnl3.06	9S053	Bam ~5	~9, 12			2.35 kb Pst Genomic
bnl3.18	3L120			Bgl2 ~6	~8	2.1 kb Pst Genomic
bnl4.24	7L054	Bam ~6.8	~8	Hinc2 6.1	Hinc2 6.4	2.35 kb Pst genomic
bnl4.36	5S035	Bam ~17,15	~20	Bam ~16	~14	2.3 kb Pst genomic
bnl5.02	5S013	Eco ~24	~8	Eco ~10	~8	2.2 kb Pst genomic, NPI finds second locus on 1L near centromere.
bnl5.04	9L074	Bam ~18,7	~3	Bam ~7,(12)	~13,(9)	2.1 kb Pst genomic
bnl5.09	9L106	Bgl2 ~6.6	~16	Eco ~2	~18	2.3 kb Pst genomic
bnl5.10	9L064			Bgl2 ~5	~16	2.4 kb Pst genomic
bnl5.14	3L097			Eco 18	Eco 5.0	2.5 kb Pst Genomic
bnl5.21A	7L065	Bgl2 5.4	Bgl2 11.5	Bgl2 6.2	Bgl2 11.5	2.2 kb Pst Genomic, 1st band probed with 5.21
bnl5.21B	2L139	Bgl2 9.0	Bgl2 7.8	Bgl2 8.7	Bgl2 8.2	2.2 kb Pst genomic, 2nd band probed with 5.21
bnl5.24	5L112	Bam ~12	~20	Bam ~18,7	~15,9	2.5 kb Pst genomic; B-A possibly 5L
bnl5.27	5S018			Eco 9.0	Eco 7.5	2.0 kb Pst genomic
bnl5.33	3L123	Bgl2 6.6	Bgl2 5.7	Bgl2 5.4	Bgl2 5.8, 3.0	2.1 kb Pst genomic
bnl5.37	3L084	Bam ~9	6.6	Bam ~18 and 6.6	~12	2.3 kb Pst genomic
bnl5.40	5L067	Bam ~4.5	~5.5	Bgl2 >25	~22	LINKED WITH 10.12L; 2.25 kb Pst genomic
bnl5.46	4S054			Bam ~18	~8.5	2.25 kb Pst genomic
bnl5.47	6L088	Bgl1 1.7	Bgl1 4.0	Bam ~18, 4.8	~8, 6	2.25 kb Pst genomic
bnl5.59	1L062	Bam ~4.2	~4.0, 1.0	Bam ~4.0	~4.2	LINKED WITH 1.556; 2.25 kb Pst genomic; B-A not 1L, 1S, 4L, 7L, 9L, 6L false +
bnl5.61A	7L066	Bgl2 6.6, 5.5	Bgl2 12	Bgl2 6.4	Bgl2 11.5	2.2 kb Pst genomic, 1st band probed with 5.61
bnl5.61B	2L143	Bgl2 9.4	Bgl2 7.7	Bgl2 8.9	Bgl2 8.2	2.2 kb Pst genomic, 2nd band probed with 5.61
bnl5.62	1S-31	Bam ~9, 6.8	~10	Bam ~24, 10	~9.5, 7	2.1 kb Pst genomic; NPI finds a second locus on 2S between lg and b.
bnl5.67	4L141	Bgl2 ~1.8	~2.6	Bgl21.8	~2.6	LINKED WITH SSU. 10.05, 7.65; 2.5 kb Pst genomic; B-A positive for 4L, not 9L
bnl5.71	5L053	Bgl2 ~20	~5	Bam ~18	~4.5, 5.5	2.5 kb Pst genomic
bnl6.06	3L069	Bam ~6.5	~9.5,4.5	Bam ~9.5,4.5	~3	2.4 kb Pst genomic
bnl6.10	5S015			Eco ~25	~15	2.1 kb Pst genomic
bnl6.16	3L115	Bam ~5.5,(6.9)	~6.8	Bam ~6.8	~8,(6.6)	2.45 kb Pst genomic
bnl6.20	2L151	Bam ~6	~10	Bam ~9, 4.5	~10	Tightly linked w/ NPI298; 2.5 kb Pst genomic
bnl6.22	5S026	Eco ~20	~7			2.0 kb Pst genomic
bnl6.25	5S-25	Bam ~4.4	~4.8	Bam ~4.5	~12	linked with 8.33; 2.25 kb Pst Genomic; B-A not 1L, 3L, 7L, 9L, 10S, possibly 2L
bnl6.27	7L056	Bgl1 20, >25	Bgl1 18.5	Bgl1 17.5	Bgl1 >25	2.2 kb Pst genomic
bnl6.29	6S001	Eco 21	14			2.3kb PstI genomic; this clone originally called 6.24
bnl6.32	1L172	Bam two lower	~18	Bam 4.4	18	2.25 kb Pst genomic
bnl7.08	8L021	Bgl2 4.5	25	Bgl2 25	4.5	2.3kb PstI genomic; upon regrowing this plasmid found a second 2.4kb insert (?)
bnl7.13	9L075	Bgl2 ~5.5	~5.0	Bgl2 ~7	~5	2.1 kb Pst genomic
bnl7.20L	4L078	Eco 14	3.2, 2.3	Eco 4.1, 21.5	9.2, 3.1	2.4 kb Pst genomic, a higher Pst band in this clone was not a good probe
bnl7.21	1S042	Bam ~7	~2.4			2.5 kb Pst genomic
bnl7.24A	9L061			Bam 3.2	Bam 7.8	2.1 kb Pst genomic, 1st band probed with 7.24
bnl7.24B	5S-16			Bam 2.3	Bam ?	2.1 kb Pst genomic, 2nd band probed with 7.24
bnl7.25	1L138	Eco ~13	~7	Eco ~7.5	~13	2.5 kb Pst genomic
bnl7.26	3L165			Eco 17	20	2.4 kb Pst genomic
bnl7.28	6L009	Bam 20	8.9, 3.3	Bam 8.8	11.7	2.3 kb Pst genomic
bnl7.43	5S032	Bam 6.7	~18	Eco ~18	~6	2.4 kb Pst genomic
bnl7.49	10L080	Bam ~20	~16	Bam ~21(doublet)	~18	TIGHTLY LINKED WITH 17.02; 2.1 kb Pst genomic; B-A not 4L, not 6L
bnl7.50	9L091	Bam 22	3.6	Bam 5.8	22	2.3 kb Pst genomic
bnl7.56	5S010	Bam ~25	~9.5	Bam ~9.5	~25	2.2 kb Pst genomic
bnl7.57	9L110	Bam 7.7	Bam 6.0	Bam 7.1	Bam 6.5	2.5 kb Pst genomic
bnl7.61	7L077	Bam ~6.5	~8.5	Bam ~20	~9	1.8 kb Pst genomic
bnl7.65	4L106	Bam ~7	~12	Bam ~15	~8	LINKED WITH SSU, 10.05, 5.67; 2.2 kb Pst genomic; B-A probably 4L
bnl7.71	5S039	Bam >30	22	Bam ~15	~20	2.55 kb Pst genomic; B-A not 2L, 3L, 9L
bnl8.01	3L095	Bam ~20	~5	Bam ~6	~12	2.4 kb Pst genomic
bnl8.04	2S070			Bgl2 ~23	~25,20	TIGHTLY LINKED WITH 12.36; 2.5 kb Pst genomic
bnl8.05	1S-28	Bgl2 ~7.2,7.0	~6.8	Bgl2 ~6.8,3.0	~7.2,6.6	2.3 kb Pst genomic; linked w/ 5.62; B-A 1S confirmed, not 4S, 7L, 8L, 10L
bnl8.06A	8L017	EcoRI4.7 kb	13.0 kb	EcoRI 14.0 kb	18.5 kb	2.25 kb Pst I genomic
bnl8.06B	6L092	EcoRI 11.5 kb	2.9 kb	EcoRI		Minor band probed with 8.06; polymorphism in first family only.
bnl8.08	1L146	BamHI 2.7 kb	3.1 kb			2.15 kb Pst I genomic.Minor band.2F doesn't show parental polymorphism.
bnl8.10	1L112	Bgl1 2.5	Bgl1 7.7	Eco ~18	~15	2.5 kb Pst genomic
bnl8.15	3S010	Bgl ~5.5	~5	Hinc2 18.5	Hinc2 10.0	LINKED WITH E8; 2.1 kb Pst genomic
bnl8.17	9L086	Bam ~14	~20	Bam ~7	~20	2.2 kb Pst genomic
bnl8.21	7L079	Bam 10	23	Bam (20), 8	5	2.2 kb Pst genomic

bnl8.23	4L156	Eco 7.4	7.9	Eco 9.0	4.5	2.5kb PstI genomic
bnl8.26	8L037			Eco ~10	~9,5.5	2.4 kb Pst genomic
bnl8.29A	1L152	Hin3 11	Hin3 14	Eco ~4, Hin3 16	Eco ~9, Hin3 12.5	2.3 kb Pst genomic
bnl8.29B	5S-20	Hin3 6.5, 3.3	Hin3 8.0	Hin3 12	Hin3 3.0	2.3 kb genomic, 2nd band probed with 8.29
bnl8.32	7L070	Bam 10	2.3	Bam 10	2.4	2.4 kb Pst genomic
bnl8.33	5S-38	Bam 4.4	~5.5, 4.0	Bam ~4.8	4.3	linked with 6.25; 2.5kb PstI genomic
bnl8.35	3S054	Bgl2 23	4.5	Hinc2 18	Hinc2 2.0	2.2 kb Pst genomic
bnl8.37	7L078	Bgl2 ~25	~18	Bgl2 ~20	~15	2.1 kb Pst genomic
bnl8.39	7L087	Hin3 11.6	Hin3 22	Bam 4.4, 2.5	~5	2.2kb PstI genomic
bnl8.44	7L122	Bam 6.7	~15, 8	Bam ~10	~15, 7	TIGHTLY LINKED WITH 16.06; 2.2 kb Pst genomic
bnl8.45	2S010	Bam ~7.5	~6	Bam ~7	~7.5	linked with NPI 239; 2.1 kb Pst genomic
bnl9.07	9S006			BamHI 17.2 kb	21.5 kb	2.4 kb PstI genomic
bnl9.08	8L017	Bam ~15,8	~7,6	Bam ~18,9	~25,17	2.2 kb Pst genomic. Hybridize at 50c.
bnl9.11	8S-19	Bam 22	10	Eco ~25	~20,16	linked with 15.07, 13.05; 2.4 kb Pst genomic; B-A not 4L
bnl9.13	1S-01	BglII 7.1	2.1			2.0 kb PstI genomic
bnl9.44L	8L012	Bgl2 ~10	~12	Bgl2 ~12, 9	~10	0.5 kb Pst genomic
bnl10.05	4L104	Eco ~15, 7	~12	Eco ~18	~16	LINKED WITH SSU, 7.65, 5.67; 2.5 Pst genomic; B-A probably 4L, not 3L, not 10S
bnl10.06	5S030	Eco ~6	~18	Eco ~18	~6. (18)	2.3 Pst genomic
bnl10.12L	5L058	Eco ~9.5	~15	Bgl2 ~10	~4.8	2.2 kb PstI genomic
bnl10.12U	8L087	Bam ~4.6	~9.2	Bgl2 ~9	~13	2.5kb PstI genomic; TIGHTLY LINKED WITH 10.24 IN SECOND FAMILY; B-A not 4L
bnl10.13	10L059	Bam 2.9	4.4	Eco 9.2	22	2.2kb PstI genomic
bnl10.17	10S-17	Eco ~8.5	~9	Bcl 5.9	5.6	LINKED WITH 3.02 AND 3.04; 2.6 Pst genomic
bnl10.24A	3L089	Bgl2 ~8.5, Bam >25	Bgl2 ~5.5, Bam 19	Bam 23	Bam >25	29 Jan '87, poor; 2.2 Pst genomic (same as 10.24B)
bnl10.24B	8L081	Bam 5.1, 4.5	Bam 6.3	Eco ~3, Bam 6.8	Eco~2.9, 1.0, BAm 4.6	9 Dec '86; TIGHTLY LINKED WITH 10.12U IN 2ND; 2.2 Pst genomic (same as 10.24A)
bnl10.38	1S014			Eco ~5,3.5	~18	2.3kb PstI genomic; linked with 1.326
bnl10.39	8L008	Eco ~25	~16			2.0 Pst Genomic
bnl10.42	2S069	Bgl2 ~3	~6(double t)			tightly linked with 12.36; 2.6 Pst genomic
bnl12.06	1S031	Bam lower	upper	Bam >25, 20	24, 19	2.0 Pst genomic
bnl12.09	2L082	Bam 4.1, 1.5	2.0			1.3kb Pst genomic
bnl12.30A	8L048	Eco 9.9	15.3	Eco 15.3	6.3	1.6kb PstI genomic
bnl12.30B	3L150	EcoRI				Data from Paul Sisco. 12/14/90
bnl12.36	2S066	Eco ~13	~20	Eco ~4	~7	LINKED WITH 10.42 AND 8.44; 2.1 Pst genomic
bnl13.05A	8S-34	BglII 2.95 kb + 950 bp	1.33 kb (19.7 kb)	Bam 22.5 kb	17.5 kb	1.3kb PstI genomic; A second locus on 3L; B-A not 4S, 8L, 10L; 1F redone 10/16/90
bnl13.05B	3S063	BglII 13.5 kb	17.5 kb	Eco 23 kb	7.8 kb	2nd band probed with 13.05. 2nd family data from 20 Jan blot(Ben's); 1F redone 10/16/90
bnl13.05C	8L013	BglII 21.5 kb	22.5 kb			10/17/90
bnl13.24	7L059	Bam ~10	~20	Bam ~20	~18	1.45kb Pst Genomic
bnl14.07	7L076			Eco ~20	~6.8	1.2kb Pst genomic
bnl14.26	9L104	Bam ~15	12	Bam 9.5	~16	same as 14.28
bnl14.28	9L104	Bam ~14	~10	Bam 7	~15	1.55kb Pst genomic; B-A on 9L, not on 2L, 4L, 10S
bnl14.34	7L076			Eco ~20	~6.6	same as 14.07
bnl15.07	4L163	Bam 6.5	4.8	Bam 4.5, 2.4	6.8	Linked w/ 8.23;Loosely linked with 9.11, 13.03; B-A not 2L;1.95kb Pst genomic; 1/14/91
bnl15.18	1L123	Bam 9.5	3	Bam 7.5	2.8	1.4 kb Pst genomic
bnl15.20	3L112	Eco ~13	~8	Bam ~9	~7, 5	1.1 kb Pst genomic
bnl15.21	7L046	Bam ~22, (16)	~18, 12	Bam ~25, 30	~15	1.25 kb Pst genomic; B-A possibly 7L
bnl15.27	4L073	Bam 9.3	11.5			1.3kb Pst genomic; tightly linked with 15.45; B-A not 7L
bnl15.37	6L078	Bam ~25, (20)	~14	Bam ~18, 15, 12	~22	1.15 kb Pst genomic
bnl15.40	7S028	Bam ~10, 7, 5.5	~15	Bam ~6	~6.5	1.95 kb Pst Genomic
bnl15.45	4L073	Bam 15.5, 2.7	15.5. 14	Bam ~13, 8	~2.8	1.3 kb Pst genomic; B-A not uncovered by 4L
bnl16.06	7L114	Bam ~22	~9	Bam ~17	9.5	LINKED WITH 8.44; 1.8 kb Pst genomic; B-A positive for 6L
bnl17.01	8L055	Hin3 3.0	Hin3 2.2	Bgl2 15	8.6	This is a second band probed with the A1 clone pAMu2
bnl17.02	10L065	Bgl2 ~6	~1.8	Bgl2 ~4	~1.8	random genomic from B. Lowe and P. Chomet, 1.2 kb Eco
bnl17.03	2L166	Eco 9.3	5.3	Eco 3.3, Hin3 5.1	Eco 5.1, Hin3 5.7	This is a second band detected with the C2 cDNA probe; it may be Whp1
bnl17.04	1L124	Eco 8, 4	7	Eco 8, 7	3, 2,5	pUC9 -1 alpha tubulin probe from D.P. Weeks
bnl17.05	4L115	Bam 2.1	~1.8	Bam ~1.8	2.1	LINKED WITH 10.05, 7.65, 5.67; SSU probe is pC1 from R. Broglie
bnl17.06	1L099	Bam ~9	~12.5	Bam ~20, 15	~9	probe is p7.6-1, random genomic from K.C. Cone, 2 kb Acc, subcloned with Sal end
bnl17.07	10L060	Bam 8.4	4.2	Bam 4.2	12.4	probe is 1.85kb subgenomic in pBF245; B-A 10L confirmed
bnl17.09	4S069			Hinc2 9.0	Hinc2 5.8	This was called umc59, but is an unknown probe.
bnl17.12	6L095	EcoRI 4.9 kb	5.7 kb	EcoRI 5.6 kb	4.8 kb	cDNA subclone pBF263B (9.1.1L), 0.7 kb Eco insert.
bnl17.13A	7S022			BglII 20 kb + 11 kb	~25 kb + 9.9 kb + 7.2 kb	550 bp EcoRI insert. cDNA subclone pBF263F. Lambda clone 11.2.1AL. 10/16/90
bnl17.13B	4S035			BglII 5.8 kb	8.8 kb + 5.3 kb	550 bp EcoRI insert. cDNA subclone pBF263F. Lambda clone 11.2.1AL. 10/16/90
bnl17.13C	4S063			BglII no band	7.6 kb	550 bp EcoRI insert. cDNA subclone pBF263F. Lambda clone 11.2.1A. 10/16/90
bnl17.14	2L189	BglII 6.9 kb	6.0 kb	BamHI 18.2 kb	5.9 kb	1.25 kb PstI, Received as pio200075, but detected no linkage with 10S.
bnl17.15	1L082	BglII 5.4 kb	6.7 kb	BglII 6.8 kb	11.5 kb	1.65 kb Eco. Detected w/ bt2 cDNA from Hannah = adp1? 12/14/90
bnl17.16	8L019	BglII 2.0 kb	No band			Minor band probed by bt2. May be same as adp4. 1/22/91
bt1	5S042	BglII 9.8 kb	6.8 & 4.85 kb	BglII 10 kb	23.1 kb	1.85 kb EcoRI insert from cDNA clone from Tom Sullivan. 12/14/90
bt2	4S067			BglII 8.2 kb	5.6 kb	1.65 kb EcoRI. cDNA clone from Curt Hannah. 12/14/90
bz1	9S031	Eco ~5	Eco ~21	Eco	Eco	probe is pMBzPA from D. Furtek. Hybridize at 50c.
bz2	1L106	Bgl2 8.6	Bgl2 7.5	Bgl2 7.5	Bgl2 8.6	probe is pP300 from V. Walbot
c1	9S026	Bam 9.9	Bam 8.8			probe pEco1.0 from K.C. Cone
c2	4L117	Eco 10.2	15	Hin3 20	Hin3 9.2	Probe is 1.5kb maize cDNA clone from U. Wienand
dia1	2L109	8	12			isozyme
e8	3S020	4.5	4	4	5	isozyme
enp1	6L016			6	10	isozyme
gln1	10L095	Bam 15.5	9.6	Bam9.4	4.8	Probe is glutamine synthetase clone GS6.15:PBS+, 1350bp Eco insert from Messing.
glu1	10S026	7	2	7	6	isozyme
got2	5L096	2	4			isozyme
hcf106	2L092					Data from Rob Martienssen
hex2	6L062	1	2	2	1	isozyme
idh1	8L063			6	4	isozyme
idh2	6L105	6	4	6	4	isozyme
mah9	5S004	EcoRI 8.9 kb	5.9 kb	EcoRI 4.8 kb + 4.4 kb	6.0 kb	300 bp Pst cDNA frag. from M. Pages; 10/16/90
mdh1	8L002	6	1			isozyme
mdh2	6L107	3.5	6	3	6	isozyme

mdh3	3L146	16	18	18	16	Isozyme
mdh4	1L097			14.5	12	Isozyme
mdh5	5S017	N	15			Isozyme
me2	6L076					NADP-dependent malic enzyme, cytosolic; data of Beverly Rothermel (Yale).
mgs1	10L032	BamHI 5.6 kb	5.4 kb	BamHI 5.0 kb	5.5 kb	450 bp EcoRI + HindIII; this is zmc13 from Mascarenhas; Pl.Cell 1:173(1989).
mgs2	4L127	EcoRI 5.95 kb	8.0kb	EcoRI 5.95 kb	3.75 kb	Pectinalyase cDNA clone zmc58 from Mascarenhas.Pollen specific.
nabp1	7S026			Hin3 ~7.6kb	8.8	Data of Bill Cook, Missouri; nucleic acid binding protein, on top of pio200690B
ncr-nrA	1L076	HindIII		EcoRI		Data from Paul Sisco. 12/14/90
ncr-nrB	4L074	HindIII		EcoRI		Data from Paul Sisco. 12/14/90
ncrb32c3A	8L023					Data from Paul Sisco.B32 endosperm protein family probe from B. Boston.
ncrb32c3B	7L083					Data from Paul Sisco.B32 endosperm protein family probe from B. Boston.
ncrb70	5S031					Data from Paul Sisco.Probe from B. Boston.
niu1	8L010	Eco 2.3	2.7	Eco 3.05	2.25	Probe was 550 bp Sall-SphI fragment from Bs1; data from Rick Johns.
niu2	5S008	Eco 2.3	2.7	Eco 3.05	2.25	Probe is 550 bp Sal-Sph fragment from Bs1; data from Rick Johns.
nor	6S-06	Sst 6.6	Sst 11.5	Sst 4.2	Sst-	probe is pBF243
npi97	1S-13	Bgl2 12.3	Bgl22.0	Bcl1 3.0	Bcl1 8.0	1.1 kb HindIII fragment
npi98	6L036	Eco 2.9	Eco 2.3	Eco 2.85	Eco 2.2	1.2 kb insert cut out w/ H3; npi98 is minor band & npi225 is major band w/ probe. 1/14/91
npi105	10S027	EcoRI 9.1 kb	7.6 kb	BglII 12.2 kb	13.4 kb	780 bp PstI.This is most proximal probe on 10S.
npi109	1S-04					
npi113	7L111	Bgl2 19.5	12.5	Bgl2 22	19, 8	1.4 kb Hind III insert.
npi114	8S-55	HindIII		EcoRV		Data from Paul Sisco. 12/14/90
npi118	2L149	Bgl2 7.7	17	Bgl2 12	5.1	Second locus probed with npi113.
npi202	3L065					Data from Paul Sisco; near vp1.
npi208	4L098			Hin3 21.5, 12	Hin3 15.5, 6.5	0.91 kb fragment from a Pst+H3 digest; there is also a 0.75 kb frag.
npi209A	9L113	Bgl2 5.9	Bgl2 7.5	Eco 5.2	Eco6.0	1.6 kb Hind III insert; 1st locus probed by npi209
npi209B	1S-05	Bgl2 20	Bgl2 13	Eco 8.6	Eco 14.5	2nd band probed by npi209
npi220	8S-48	Bgl2 4.7	~13	Bgl1 6.9	7.7	<500 bp, HindIII + EcoRI
npi223	6L054	Bgl2 4.2	3.4	Bgl2 4.5	3.5	1.58 kb Hind III insert
npi224A	7S024	EcoRI no allele	20.5 kb			Probable Transposable Element. 10/16/90
npi224B	8L094	EcoRI no allele	15 kb			Probable Transposable Element. 10/16/90
npi224G	1L074			BamHI no allele	20 kb	Probable Transposable Element. 10/16/90
npi224I	3S032			BamHI no allele	7.9 kb	Probable Transposable Element. 10/16/90
npi225	1L127	Eco 6.4	Eco 11.5, 8.8	Eco 5.6	Eco 7.0	Major band probed with npi98, 1.2 kb H3 insert. 1/14/91
npi235	6S004	Hin3 9.5		Eco 18	24	0.71 kb Pst I insert
npi236	1L084	Eco 17kb	Eco 22kb			0.62 kb Pst insert.
npi238	1L147	EcoRI		EcoRV		data from Paul Sisco. 12/14/90
npi239	2S005	Bam 8.2	6.0	Eco 19.5	5.4	B-A 2S confirmed.0.86 kb Pst I insert.
npi242	2L097	BglII 23.1 kb	20.5 kb	EcoRI 12.0 kb	14.2 kb	1.15 kb PstI
npi253	9S016	Eco 6.0	19.5	Bam 4.8, 3.8	5.2	0.58 kb Pst I insert.
npi253B	4L097	BamHI		EcoRV		Data from Paul Sisco. 12/14/90
npi253C	5L084	BamHI				Data from Paul Sisco. 12/14/90
npi253D	6L060	BamHI				Data from Paul Sisco. 12/14/90
npi254	2S020	EcoRI		HindIII		Data from Paul Sisco. 12/14/90
npi263	7L068					Data from Paul Sisco. 12/14/90
npi264	10L038	Bam 9.1	16	Bam 8.4, 7.5	5.6	0.77 kb insert.
npi268	8L084	Bam 7.1	5.5	Bam 11	5.6	11 kb Bam is seg'g in COXTx, our version of Tx303 has a 7.2kb band; .71 Kb Pst
npi269A	2S060	Eco 10.6	17.5	Bam 21.5, Hinc2 19	Bam 14, Hinc2 7.0	One of two loci recognized by npi269.PstI 1.0 kb
npi269B	10L049	Eco ~25	3.3			This is a second band recognized by the probe used for NPI269A.PstI 1.0 kb.
npi271	2L094	Hin3 7.8, 4.8	Hin3 7.2, 3.7	Bgl2 10	2.8	B-A not uncovered on 2S.0.62 kb Pst I insert.
npi273	1S-02	BamHI 14.0 kb	13.0 kb	BglII 19.5 kb	16.0 kb	1.4 kb EcoRI + HindIII
npi283	7L061	Bgl2 16	9.4	Bgl2 16	13.7	B-A 6L confirmed.0.98 kb Pst I insert.
npi285	10S006	Eco 23	20	Bgl2 2.4	3.3	PstI 1.25 kb
npi288	5L094					Data from Paul Sisco.
npi291	9L115	Bgl2 3.3	Bgl2 3.1	Sst 8.8, 4.9	Sst 8.4, 4.5	1.15 kb PstI insert.
npi297	2L091	EcoRI >23.1 kb,14.8 kb	11.7 kb,7.6 kb			920 bp PstI
npi298	2L147	Bgl2 6.2	14	Bgl2 11.5	6.1	B-A 2L confirmed.0.66 kb Pst I insert.
npi306	10L073	Eco 7.9	9.4	Bgl1 11	Bgl1 24	Near 17.02, 0.84 kb insert cut out with EcoRI+HindIII. First family 16 June '87
npi340A	6S002	EcoRI 15.0 kb	17.5 kb	BglII 10.0 kb	6.0 kb	Supposed to be 2S (no linkage) & 2 other loci; 1.0 kb PstI; this data near umc85
npi350	10L098					Data from Paul Sisco.
npi361A	2L202	EcoRI no allele	13 kb			Probable Transposable Element. 10/17/90
npi361B	7S020	EcoRI 7.8 kb	no allele			Probable Transposable Element. 10/17/90
npi361C	6L038	EcoRI no allele	3.3 kb			Probable Transposable Element. 10/17/90
npi361D	2L169	EcoRI no allele	2.8 kb			Probable Transposable Element. 10/17/90
npi361E	7S025			EcoRI no allele	17.5 kb	Probable Transposable Element. 10/17/90
npi361F	8L022			EcoRI 15.6 kb	no allele	Probable Transposable Element. 10/17/90
npi361I	1L167			EcoRI 3.75 kb	no allele	Probable Transposable Element. 10/17/90
npi361J	4S016			EcoRI 3.45 kb	no allele	Probable Transposable Element. 10/17/90
npi361K	2L169			EcoRI 2.85 kb	no allele	Probable Transposable Element. 10/17/90
npi373	6L018	BamHI				Data from Paul Sisco. 12/14/90
npi400	7S012	Bgl2 13	9.6	Bgl2 9.6	10.5	
npi400B	2L196					Data from Paul Sisco; linked with "pio200075"(now bnl17.14) and pio200581B
npi403	9L112			Eco 7.7	Eco 12.5	0.75 kb PstI insert.
npi404	1S001			Eco 16	Eco 24	2nd locus probed by npi403
npi409	5S-30	Eco 6.7	5.2	Eco 5.75	5.3	0.71 kb Pst I insert.
npi411	1S005	Bam 2.5	7.2	Bam 4.4	7.3	0.98 kb Pst I insert; Veronique estimates 1.2 kb.
npi414	8L097			Bam 12	5.2, 4.4	0.87 kb Pst I insert.
npi421A	2S027	Hin3 6.0	Hin3 7.4	Bgl1 17.5 kb	Bgl13.4 kb	This is one of two bands recognized by this probe; B-A 2S confirmedPstI 500 bp
npi421B	10L085	Bgl2 ~11	2.2	Bgl11.98 kb	2.7kb	Second band probed w/npi421 insert. 2F don't have parents' polymorph.PstI 500 bp
npi425	3L158	Bam 3.7	3.45	Bgl2 5.7	8.6	1.0 kb Pst I insert.
npi427	9L096	Bam 4.8	Bam 4.5	Bam 15, 4.9	Bam 13, 6.6	0.66 kb PstI insert.
npi438	8L108	EcoRI 12.6 kb	9.0 kb	EcoRI9.1 kb	16.4 kb	910 bp PstI. 1/18/91
npi443	9L101	Bgl2 4.0, 2.3	Bgl2 3.4, 2.5			
npi445B	10S025	EcoRI 6.5 +/or 5.85 kb	4.0kb	BamHI 3.15 kb	2.75 kb	1.02 kb PstI, Supposed to be 5 near centromere.
npi446	3S054	BamHI		HindIII		Data from Paul Sisco. 1/14/91
npi451	4L161	Bgl2 5.6	6.8	Eco >25	19	B-A 4L confirmed.0.62 kb Pst I insert.
npi456	2L111	Bgl2 22.5	10.5	Bgl2 10.5	7.3	B-A 2L confirmed, not 2S.1.31 kb Pst I insert.
npi553	3L066					Data from Paul Sisco.Near 8.35.

nplB1	2S077	BglII 11.5 kb	4.8 kb	BglII 7.1 kb	5.3 kb	800 bp EcoRI + HindIII
o2	7S016	Eco 5.9	Eco 6.7	Bgl 7.0	Bgl 8.5	probe is pXho0.9 from R. Schmidt
orp1	4L076					Data from Karen Cone.trpB probe
orp2	10S024					Data from Karen Cone.trpB probe
p1	1S026	-	+	-	+	morphological
pcr1	1L103	EcoRI 7.2 kb	6.0 kb			1.32 kb Eco from OR1 cDNA for protochlorophyllide reductase from S. Kay. 12/14/90
pcr2	5S009	EcoRI 4.9 kb	4.2 kb	EcoRI 6.8 kb	5.0 kb	Probe is EcoRI cut OR1 (1.32 kb) protochlorophyllide reductase cDNA of Steve Kay. 12/14/90
pcr3	2S008	EcoRI >23.1 kb	21.5 kb	EcoRI 16.5 kb	23.1 & 17.6 kb	1.32 kb Eco from OR1 cDNA protochlorophyllide reductase of Steve Kay. 12/14/90
pcr4	7S021			EcoRI 14.3 + 13 kb	11 kb	Fourth locus probed by protochlorophyllide reductase clone, OR1 from Steve Kay. Dec. 90
pgd1	6L013			2	3.8	isozyme
pgd2	3L071	5	2.8			isozyme
pge2	8L004					Near mdh1; data from Brenda Lowe, transposed Spm.
pge3	7L072					Transposed Spm, data of Brenda Lowe, on top of pio200563
pge14.1	4L112	BamHI 14 kb + 10 kb	11 kb			Data from Brenda Lowe, transposed Spm, probe = 14.1
pge132	1S-24					Data from Brenda Lowe.Kn1 homeodomain homologous sequence. First family only.
pgeA4	8L042					Brenda Lowe: Kn1 homeodomain homologous sequence. Between 2.369 and 12.30.
pgeB5	5S005					Brenda Lowe: Kn1 homeodomain homologous sequence. Vicinity of nlu2 and bnl7.56.
pgeC2	1L127					Brenda Lowe: Kn1 homeodomain probed sequence.Same as Kn1?
pgm2	5S000	3	4	3	4	isozyme; supplimentary data from Oliver Nelson.
phi1	1L140	5	4	2	4	isozyme
php1	10S023					Chloroplast phosphoprotein polymorphic in second family.Data of J. Bennett
phy1	1L125	Bgl2 7.7	15	Bgl2 16	7.7	Probe is 1.5 kb Hind3-EcoR fragment from rice phytochrome cDNA pcPhy101, S.A.Kay
phy2	5S-04	Bgl2 4.5	4.9	Bam		same probe as phy1
pio060005	10S024	BglII 12.7 kb	20.5 kb	BglII 13 kb	21.8 kb	330 bp PstI
pio060007	6L023			Eco 7.8 or 10	Eco 11.5	350 bp PstI.Tx gave two different kinds of bands - same w/ Bgl2. 28 Nov 90, see pio book 9-5-89
pio100005	9S018	BglII 13.7 kb,5.45 kb	12.5 kb,7.2 kb	BglII 22 kb,7.55 kb	14.1 kb,5.8 kb	825 bp PstI
pio100017	5L102	EcoRI 23.1 kb	15.5 kb	EcoRI 9.4 kb	18 kb	465 bp PstI
pio100080	3L144	BamHI 16.7 kb,13.3 kb	8.8 kb	EcoRI 15 kb	18.5 kb	485 bp PstI
pio200026	3L101	BglII 15 kb	8.4 kb			12/14/90
pio200045	6L022	BglII 13.6 kb	11.2 kb			1.43 kb PstI. 1/18/91
pio200075A	10S-03	BglII 4.2 kb	4.6 kb	BglII 4.6 kb	7.4 kb	1.40 kb PstI. 1/18/91
pio200075B	9L060			BglII 2.6 kb	3.0 kb	1.40 kb PstI, Minor band.
pio200523	7L109	BglII 8.3 kb	11.5 kb	BglII 9.5 kb	8.6 kb	12/14/90
pio200563	7L072	BamHI 2.22 kb	5.5 kb	EcoRI 18.7 kb	13.5 kb	1.15 kb PstI, Dave Grant mapped to 4L terminal.
pio200569A	7L050			BglII 6.1 kb	6.6 kb	1.72 kb PstI, Major band, Supposed to be 7S; weak linkage to 4.24 on 7L.
pio200569B	2L130	BglII 5.25 kb	3.75 kb	BglII 5.1 kb	5.65 kb	1.72 kb PstI, Minor band, probed by pio200569.Weak linkage with umc5A on 2L.
pio200581A	7S000	EcoRI 6.3 kb	22.0 kb	EcoRI 16.7 kb	9.2 kb	1.40 kb PstI, Lower band
pio200581B	2L194	EcoRI 14.5 kb	>23.1 kb	EcoRI >23.1 kb	19.3 kb	1.40 kb PstI, Upper band, second band probed by pio200581; linked w/ "pio200075"
pio200599	6L111	BamHI 4.85 kb	4.2 kb	EcoRI 23.4 kb	14.5 kb	1.65 kb PstI
pio200626	10S-12	BglII 18.3 kb	8.5 kb	EcoRI >23.1 kb	18.7 kb	1.40 kb PstI
pio200690A	7L107	BamHI No band	18 kb	BamHI 5.05 kb	7.3 kb	1.43 kb PstI
pio200690B	7S026	BamHI 15 kb	No band	BamHI No band	15 kb	1.43 kb PstI
pio200713	4S031	BamHI 11.5 kb	4.15 kb	BamHI 16.7 kb	10.6kb,4.9 kb	1.47 kb PstI
pio200725	4S030	BamHI 11.5 kb	4.3 kb	BamHI 16.5 kb	11 kb,4.9 kb	1.74 PstI
pio200726	3L161	BglII >23.1 kb	20 kb,8 kb	BglII 7.5 kb	9.3 kb	1.20 kb PstI
pio200728	7L130	BglII 17.0 kb	13.1 kb	EcoRI 6.8 kb	7.2 kb	1.80 kb PstI
pKlamb+	3L068					Data from Paul Sisco.Combined with abp1 where consistant.
pl1	6L049	Hind III9.7 kb	18.0 kb	+	-	probe is H3-Sal O.6 from K.C. Cone, morphological in 2nd family
r1	10L061	Bgl II 10.3 kb	13.5 kb,8.4 kb			Probe is R5-4 frag.2.
rab17	6L074	EcoRI 11.72 kb	4.95 kb	EcoRI 9.1 kb	5.2 kb	400 bp Pst cDNA frag from M. Pages. Recognizes the same frag's as rny1. 10/16/90
rab30	1S-16	BglII 3.35 kb	2.5 kb	EcoRI 11.2 kb + 5.8 kb	4.7 kb	400 bp Hind3/EcoRI cDNA frag from M. Pages. Eileen thinks closer to 560 bp. 10/16/90
rny1	6L074	Sst 9.3	Sst 19.5	Bam 8.5	Bam 9.6	Probe is rice rab25 cDNA from J. Mundy.Homologous with rab17 (M. Pages)?
sh1	9S029	Hinc2 ~5	~3	Bgl1 ~14	~6	probe is Pst38 or Pvu55
sh2	3L149	BamHI 10.5 & 9.5 kb	11.0 & 8.0 kb	BamHI 8 & 5.6 kb	10.4 & 4.8 kb	1050 bp Eco. cDNA clone sh2 1050 from Curt Hannah. 1/22/91
stAc	10L030			BglII 5.85 kb	5.55 kb	600 bp H3-Xba fragment flanking Stabilized Ac, Paul Chomet
sus1	9L084			Bam ~11	~6	Same as Css1, probe is pshD13 from P. Chourey; B-A on 9L
tpi4	3S061	4	N			isozyme
trAc9705	1S-03					Data of Diane Burgess, DNAP.
umc3	3L119	HindIII		HindIII		Data from Paul Sisco. 12/14/90
umc5A	2L122					Data from Paul Sisco.Loosely linked with dia1.
umc5B	7L036					Data from Paul Sisco.
umc15	4L120			BglII 11.25 kb	9.5 kb	750 bp PstI. Tx alleles are 24.5 kb & 6.65 kb in RI Family. No poly in 2F. 1/14/91
umc19	4L137	BglII 11.7 kb	22.5 kb	EcoRI >23.1 kb	18.5 kb	670 bp
umc23	1L086					Data from Paul Sisco. 670 bp.
umc31A	4S040					Data from Paul Sisco. 550 bp.; A second locus on 2L.
umc31B	2L157					Data from Paul Sisco, near npi298
umc32A	3S011					data from Paul Sisco
umc32B	8L008					Data from Paul Sisco.
umc33	1L088					Data from Paul Sisco.
umc35	7L137			EcoRI 16.5 kb	13.9 kb	650 bp, PstI
umc37	1L103					Data from Paul Sisco.
umc39	3L121			EcoRV		Data from Paul Sisco. 12/14/90
umc42A	4S068			BamHI 7.0 kb	21.8 kb	640 bp.Data for first family probe a locus on 3L.
umc42B	3S062	BamHI 14.9 kb	10.5 kb			640 bp.Second locus probed with umc42A (4L). Poor data;linked with 6.06 & pdg2
umc43	5S016					Data from Paul Sisco
umc44A	10L055					Data from Paul Sisco.
umc44B	2S041					Data from Paul Sisco
umc47	4S070			BglII 5.9 kb	11.5 kb	740 bp
umc48	8L052					Data from Paul Sisco.
umc49	2L167					Data from Paul Sisco
umc50	3S058					Data from Paul Sisco.
umc51	5L075					Data form Paul Sisco.

umc52	4L113	HindIII				Data from Paul Sisco. 12/14/90
umc53	2S023					data from Paul Sisco
umc54	5L069					Data from Paul Sisco.
umc58	1L068	HindIII		HindIII		Data from Paul Sisco. 12/14/90
umc59	6L017	BamHI 12.8 kb	10.0 kb	BamHI 5.7 kb	10.7 kb	930 bp. Tx allele for progeny is 15.7 kb instead of 5.7 kb
umc60	3L073					Data from Pau Sisco.
umc61	2S050	BglII 7.3 kb	8.5 kb	BglII 9.4 kb	14 kb	Original data from Paul Sisco, near B1. 1.28 kb PstI.
umc64	10L029			BamHI		Data from Paul Sisco. 12/14/90
umc65L	6L042	Bgl2 4.1	Bgl2 7.7			0.65 kb PstI insert
umc66A	5S-13	EcoRI 5.85 kb	10 kb	EcoRI 9.0 kb	6.1 kb	1020 bp, Major band, supposed to be on 4S; loose linkage w/ 8.29B on 5S;.5kb2/88. 1F redone 10/16/90
umc66B	1L135	EcoRI 4.85 kb	5.1 kb	EcoRI 5.2 kb	5.6 kb	1020 bp, Minor band, probed by umc66.Loose linkage w/ phy1 on 1L in 2nd family. 1F redone 10/16/90
umc72A	5S-02	BamHI				Data from Paul Sisco. 12/14/90
umc72B	1L129	EcoRV				Data from Paul Sisco. 12/14/90
umc84	1L170			HindIII		Data from Paul Sisco. 12/14/90
umc85	6S001					Data from Paul Sisco.
umc95	9L089	BglII 2.9 kb	2.25 kb	EcoRI 19.5 kb	8.1 kb	680 bp
umc106	1L126	HindIII		HindIII,EcoRI,Bam		Data from Paul Sisco. 770 bp. 12/14/90
umc119	1L066	HindIII		HindIII		Data from Paul Sisco. 12/14/90
umc122	2L145					Data from Paul Sisco
umc128	1L102					Data from Paul Sisco.
umc130	10S021					Data from Paul Sisco.
umc135	2L108			BamHI		Data from Paul Sisco. 12/14/90
umc147A	5S-10			HindIII		Data from Paul Sisco. 12/14/90
umc147B	1L136			HindIII		Data from Paul Sisco. 12/14/90
umc153	9L072					Data from Paul Sisco. 12/14/90
umc154	3S057	EcoRV				Data from Paul Sisco. 12/14/90
umc155	10S023	EcoRI				Data from Paul Sisco. 12/14/90
umc156	4S071	EcoRI				Data from Paul Sisco. 12/14/90
umc157	1S-15	BamHI		EcoRV		Data from Paul Sisco. 12/14/90
umc158	4L109	EcoRV		EcoRV		Data from Paul Sisco. 12/14/90
umc175	3L066	EcoRV				Data from Paul Sisco. 12/14/90
vp1	3L076	Bgl2 4.3	Bgl2 4.75	Hinc2 4.5	Hinc2 5.1	probe is 0.7 kb Pst of pVPM1B from D. McCarty
wx1	9S056	Bam 5.0, 3.3	4.8	Bam 13, 4.7	4.9. 4.8	probe is pBF225
ynh-me1	3S040					NADP-dependent malic enzyme, chloroplastic leaf form; data of Beverly Rothermel
ynh20	1L120	Hin3 5.9	Hin3 9.4			probe is pBMP-TA69R 1 kb H3-Xho from J. Chen
ynh21	1S027	Bam>25	Bam 15, 6.7	Bam >25	Bam 15, 6.7	probe is pX.5-2 0.4 kb Sst from J. Chen
zp15	6L012	Bam9.4kb	12.5			probe is Eco-H3 insert from pGEMZ14 (Pederson, JBC 261, 6279)
zpB36	7L032	Eco ~12, 8	~11	Eco ~12	~16	zein probe
zpL1a	4S029	18	20.5			Zein IEF bands from C. Wilson.
zpL1b	4S031	36	29.5			Zein IEF bands from C Wilson.
zpL1c	4S031	32	31.5			Zein IEF bands from C. Wilson.
zpL1d	4S034	33.5 dark	33.5 light			Zein IEF bands from C. Wilson.
zpL1e	4S030			18, 60	17.5	Zein IEF bands from C. Wilson.; Same as zpL1g.
zpL1f	4S033			32, 36	35	Zein IEF bands from C. Wilson.
zpL2b	7S021			38	21.5, 53	Zein IEF bands from C. Wilson.

3) through 6) the restriction enzyme showing polymorphism and size of the fragment(s) detected segregating with that locus in each of the parental inbreds where known

7) comments on size of the insert, information about the clone and who contributed mapping information

Map positions have negative values where the loci appear to lie distal to 0 on the conventional map.

Experiments with *id1*

--B. Burr and Véronique Szabo

The mutation *id1* causes the maize plant to prolong its vegetative stage and, in the field, turns it into a short day plant. We were interested in the mutation because it behaved as one might expect a phytochrome mutation to act. In this investigation we compared three alleles, mapped the mutation relative to RFLP markers, and examined the effect of the mutation on light dependent plant pigmentation.

We obtained the *id1-R* allele from the Maize Co-op and two other putative alleles from Bob Brawn while he was associated with CIBA-GEIGY. The alleles from Brawn were *id-207* and *id-Compeigne*. Flowering can be prematurely induced in *id-R* homozygotes after only a week of 13 hour nights. Flowering could be induced in *id-207* after about one month of short days, but short day treatment did not induce premature flowering in *id-Compeigne* homozygotes. Plants heterozygous for either *id-207* or *id-Compeigne* were self-pollinated and crossed to induced *id-R* homozygotes. Selfed progeny and outcrossses from the same plant were then grown out to confirm that an *id* allele was present and test for allelism. Both *id-207* and *id-Compeigne* were allelic with *id1-R*.

id1 has been reported to map on *1L* in the vicinity of *bz2*. Since one of the phytochrome loci is nearby, we were interested to see if the two might map to the same locus. We obtained a segregating population from the Maize Co-op in which *id* had been crossed to inbred M14 and selfed. Three RFLP markers from this region, *bnl17.06, bnl15.18,* and *ynh20,* were tested on 24 plants in an F2 population. There were no recombinants with *bnl17.06* which we estimate to map on 1L at position 99 or about 26 map units from *phy1*.

id1-R homozygotes, which appeared to be *b, pl, r-g*, were crossed to a *B-S, pl, R-g* stock and the heterozygotes were selfed. We examined two large F2 populations obtained in this fashion. Fully 3/4 of the *id/id* plants were sun-red and these plants showed no reduction in pigmentation in comparison with their normal sibs. We conclude that *id1* has no effect on light dependent anthocyanin production.

Comparison of *bnl* probes redux

--B. Burr and F.A. Burr

Last year Rick Johns and colleagues (MNL 64:56, 1990) reported variable results with 68 *bnl* probes. They re-

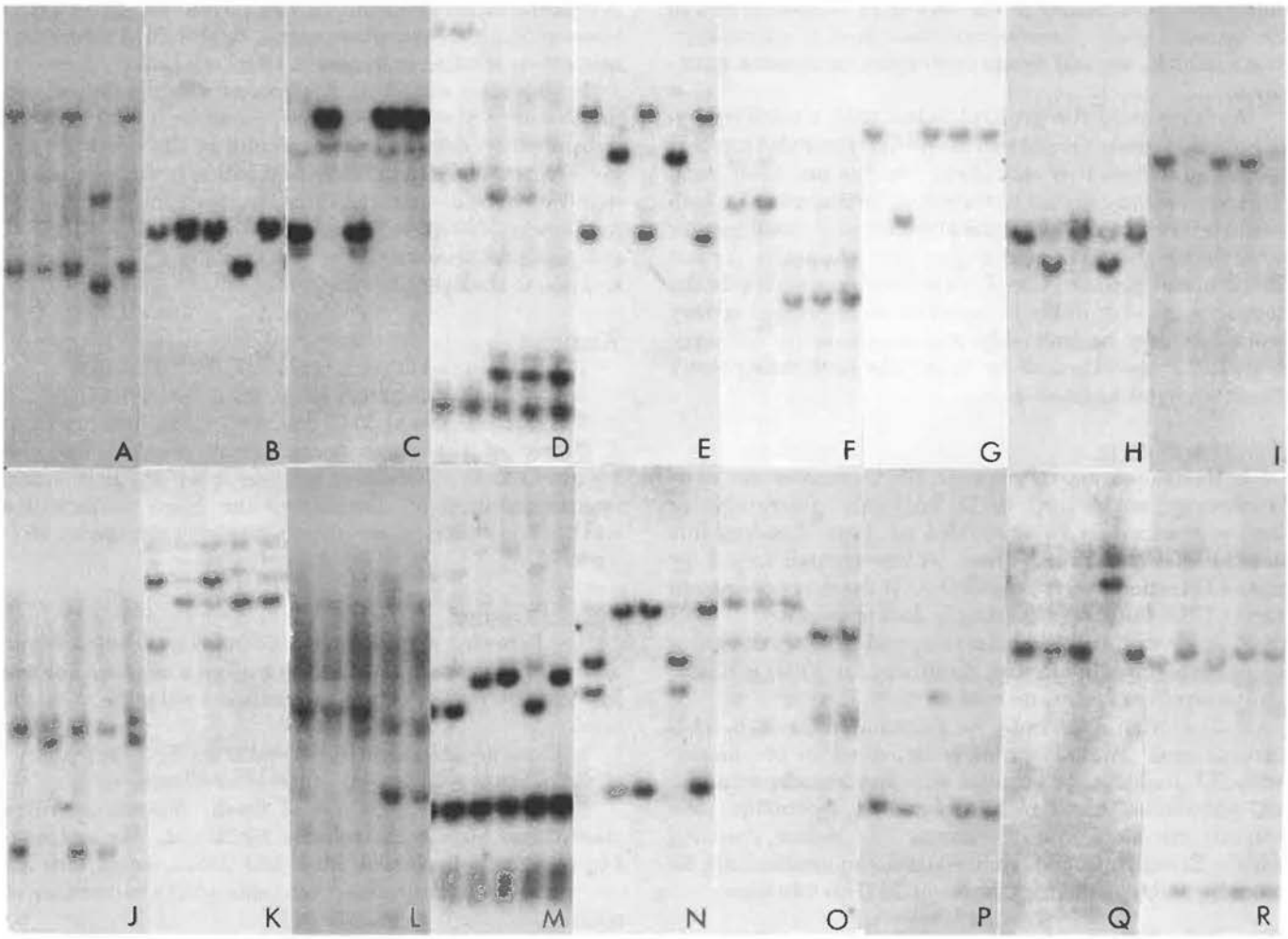


Figure 1. Portions of original, working autoradiograms used to map loci detected by BNL clones that performed poorly for Johns et al. For each probe, five lanes of a segregating family are shown. For each panel the name of the locus is followed by the restriction enzyme used and the recombinant inbred family. (A)*bnl3.04*, *Bam*HI, COxTx; (B)*bnl4.24*, *Bam*HI, TxCM; (C)*bnl5.02*, *Eco*RI, TxCM; (D)*bnl5.04*, *Bam*HI, TxCM; (E)*bnl5.37*, *Bam*HI, COxTx; (F)*bnl5.46*, *Bam*HI, COxTx; (G)*bnl6.10*, *Eco*RI, COxTx; (H)*bnl7.49*, *Bam*HI, TxCM; (I)*bnl8.04*, *Bgl*II, COxTx; (J)*bnl8.05*, *Bgl*II, COxTx; (K)*bnl8.10*, *Eco*RI, COxTx; (L)*bnl8.26*, *Eco*RI, COxTx; (M)*bnl8.33*, *Bam*HI, COxTx; (N)*bnl8.39*, *Bam*HI, COxTx; (O)*bnl9.08*, *Bam*HI, COxTx; (P)*bnl10.12U*, *Bam*HI, TxCM; (Q)*bnl10.13*, *Eco*RI, COxTx; (R)*bnl10.17*, *Eco*RI, TxCM.

ported that more than 25% gave too much background or were too faint to score. We were surprised by these results because one of the bases upon which we chose the probes was that they gave a strong signal. We went back to our original autoradiograms in which we had scored segregation in the TxCM or COxTx recombinant inbred families and found that all the probes in the Johns et al. worst category (D) gave respectable signals. Portions of these autoradiograms are reproduced here.

We published the specific conditions under which we used these probes in Burr et al. Genetics 118:519-526, 1988. The major differences in our protocols appear to be that we used nitrocellulose membranes and nick translated our inserts.

URBANA, ILLINOIS
University of Illinois

Chemiluminescent Southern detection of maize genomic single copy sequences

--Torbert Rocheford and *Nancy Wallace
* Present address: Dekalb Plant Genetics, Groton, CT

We have been increasingly working with nonisotopic methods of detecting maize single copy sequences on Southern blots for the past few months. We are pleased to report use of a digoxigenin based protocol that produces satisfactory results with UMC and BNL probes. All of our new RFLP work will use this method and we are in the process of phasing out our radioactive work as old membranes become used up through reprobing. The protocol we report here is a result of discussions with Frans Krens at Wageningen, Netherlands, and Joe Lanzillo at New England Medical Center, Boston. We have taken their protocols and slightly modified and integrated them so that

the procedure is suitable and repeatable for maize single copy detection work. The procedure still needs some refinements, particularly in the area of repeatability and in background levels. Since we are committed to nonradioactive methods, we will continue to optimize various conditions.

We have used the protocol below with a rotating hybridization oven (Robbins Scientific) for hybridization steps and a glass tray on a shaker for the detection steps. We have recently shifted to the hybridization oven for both the hybridization and detection steps because it will help us to scale up the procedure to the level we desire for our RFLP investigations. Use of glass trays on shakers for detection is quite suitable for qualitative molecular biology work but may be unwieldy and cumbersome for large scale RFLP operations or for large labs performing many Southern hybridizations a day.

Southern Blotting

1. Restricted maize genomic DNA samples are electrophoresed on 1% agarose 1X TAE gels. Currently, we load approximately 15 ug of DNA per lane. Loading this amount of DNA insures there will be enough target for initial detection and for reprobing. If there is not enough target DNA the procedure simply does not work.

2. After staining, visualization, and rinsing, the gel is depurinated for 15 minutes, denatured for 30-60 minutes, and neutralized for 30 minutes.

3. The nylon membrane we recommend is Hybond-N (Amersham). The membrane is saturated for 10 minutes with H_20 and then 10 minutes with the transfer solution: 1M ammonium acetate, 20 mM sodium hydroxide. We transfer our blots on a Stratagene Plosiblotter, crosslink with a Stratagene UV crosslinker for approximately 30 seconds, and bake the membrane at 80 C for two hours.

DNA Probe Labelling

We have labelled PCR amplified inserts as well as inserts cut out of gels. We are currently labelling 200 ng at a time since this gives high levels of incorporation of digoxigenin. We use Boehringer Mannheim DNA labelling kit (Cat. No. 1175 033). We will try labelling larger quantities in the future, however due to the ability to reuse the Dig-labelled probe, the need to label up very large quantities of probe is reduced.

1. Denature 200 ng of DNA in an H_2O volume of 15 ul by heating for 10 min. at 95 C.

2. On ice, add : 2 ul hexanucleotide mixture, 2 ul dNTP mixture. Mix and add: 1 ul of klenow. Mix and briefly spin down in microfuge.

3. Incubate 37 C overnight. Stop reaction by adding 2 ul of 0.2 M EDTA (pH 8.0). Add 20 ug of tRNA and mix. Precipitate with 1.8 ul of 3M sodium acetate and 3 volumes ethanol (71.4 ul). Leave at -70 for >30 minutes.

4. Centrifuge at 12,000 x g then wash with 50 ul ice-cold 70% ethanol. Dry thoroughly. Resuspend in 50 ul TE and 0.1% SDS. (5 ul of 1% SDS) at 37 C for 20 min with frequent vortexing.

Prehybridization and Hybridization

1. Prewet membrane with H_2O and then 2X SSC. Prehybridize membrane one hour or more at 65 C in hybridization oven with > 20 ml/100 cm^2 with the following prehybridization solution: 5X SSC, 1.0% Tropix blocking reagent, 0.1% N-laurylsarcosine, 0.02% SDS (store hybridization solution in freezer in 50 ml aliquots).

2. Hybridize with 3 ml of the above solution for a single membrane in a bottle. Denature the probe in 500 ul of the hybridization solution and then add to the other 2.5 ml, quickly mix, and add to the hybridization bottle. We use 12 ng/ml of hybridization solution, higher concentrations of probe may increase background. Hybridize at 65 C overnight (when we reuse probes, we simply boil the 3 ml and add to the hybridization bottle).

Washes

1. 2 X 5 minutes at room temp. 2X SSC; 0.1% SDS
2. 2 X 5 minutes at room temp. 0.2X SSC, 0.1% SDS
3. 2 X 15 minutes at 65 C 0.2X SSC, 0.1% SDS

(Some of the room temperature washes may be holdovers from radioactive protocols; we are in the process of reducing or eliminating the room temperature washes and changing simply to three high stringency 65 C washes).

Signal Detection

The following steps have the volumes used when signal detection is performed in a glass tray on a shaker. For the hybridization oven, we are currently reducing the volumes used.

1. Rinse membrane in 50-100 ml of buffer 1 for 5 min.
2. Incubate with 100 ml Buffer 2 for 30 min.
3. Incubate with 15 ml of fresh, filtered anti-Dig (Boehringer Mannheim) solution for 30 min. We add anti-Dig to 2-3 ml of solution, filter just that amount with .45 um millipore syringe filter, and add to the remainder of solution.
4. Wash 3X in 50-100 ml Buffer 2 for 10 min each.
5. Wash 3X in 50-100 ml Buffer 1 for 10 min each.
6. Incubate 2X 5 min in Tropix assay buffer.
7. Incubate in 15 ml of Tropix AMPPD solution for 10 min.
8. Drain excess solution and seal membrane in seal-a-meal bag. Do not allow membrane to dry at this time or until probe is stripped.
9. Expose film for 2-3 hours at room temperature. Develop film and determine length of subsequent exposure, if necessary. The blot can be put down the following day. Overnight exposures are usually too long and produce a lot of background. There is no need to worry about loss of signal. Sometimes the signal to noise ratio is better when the membrane is exposed a day or two later, but not always.

Solutions

Buffer 1: 0.1 M Tris-HCl (pH 7.5); 0.15 M NaCl

Buffer 2: Buffer 1 + 0.5% Tropix blocking reagent (Cat No. AB 100)

Anti-Dig: 3 ul Boehringer Mannheim anti-Dig solution (Cat no. 1093-274) per 15 ml Buffer 2. This gives a 1:5,000 dilution. (We have successfully used 1:10,000 and 1:15,000

dilutions. We are currently using 1:10,000 and we will continue to investigate modification of dilutions since they result in cost savings and higher dilutions may reduce background in the hybridization oven.

Assay Buffer: to make 250 ml - 2.4 ml diethanolamine, 0.5 g $MgCl_2$, .05g sodium azide, adjust pH to 10.0 with HCl.

AMPPD Substrate Solution: to make 15 ml - 165ul Tropix AMPPD (Cat. no. PD 100-B) added to 15 ml Assay Buffer.

Stripping Blots

Nonstripped membranes are stored in their seal-a-meal bags in the freezer.

1. Rinse membrane in 2X SSC.

2. Incubate for 10 min. at 37 C in 0.2 M NaOH, 0.1% SDS.

3. Rinse membrane in TE, membrane can then be stored at room temperature.

Comments

We have successfully been able to probe membranes 3-4 times and we have not exhausted the usefulness of these membranes. We will continue to reprobe these blots. The one major disadvantage of this system is that background can be a problem at times. Presently, we have less background problems than when we first started working with this type of detection system. Although background can be a problem, in general it does not prevent us from scoring the exposed films.

We are currently working on optimizing conditions for detection in bottles in the rotating hybridization oven. Our initial experiments revealed more background with detection in hybridization ovens than in glass trays on shakers, but the background has been reduced recently. We have increased the length of some of our detection incubations and washes and we are currently working with other reagent concentrations such as the anti-Dig concentration. The hybridization oven should enable scaling down the volumes used and probing more than one membrane per bottle, resulting in considerable time and cost savings. We have just started to probe two membranes per bottle.

A major advantage of this system is that reagents can be reused. We have not experimented with this very much since we were very concerned with reducing background levels upon reprobing. Since we have now reduced background levels, we are currently addressing reuse of solutions. This will also result in considerable cost savings. We have reused our Dig-labelled DNA probe hybridization solutions quite successfully. We greatly enjoy not having to use radioactive isotopes and I will be happy to discuss this procedure or send out updated modifications to this protocol to interested researchers. Finally, this technology has teaching attractiveness, for example undergraduate students can be integrated into lab operations more rapidly, calmly and safely than with radioactive procedures.

Identification of a putative globulin-specific protein processor by using anti-idiotypic antibodies

--Faith C. Belanger and Alan L. Kriz

The major storage protein of maize embryos is an Mr 63,000 vicilin-like globulin designated GLB1 which is encoded by the *Glb1* gene. GLB1 is synthesized as a preproproprotein which undergoes extensive post-translational processing (Kriz and Schwartz, Plant Physiol. 82:1069, 1986). The final processing step, controlled by the unlinked gene *Mep* (Schwartz, MGG 174:233, 1979), is a proteolytic cleavage near the amino terminus of the polypeptide (Belanger and Kriz, Plant Physiol. 91:636, 1989). Embryos homozygous for the recessive *mep* allele accumulate the processing intermediate proGLB1 (formerly designated GLB1'). It is not known if *Mep* encodes the protease which cleaves proGLB1 to form GLB1 or for a factor which regulates activity or specificity of a protease.

In an attempt to identify the *Mep* gene product, antibodies to a synthetic 30 amino acid peptide, designed to span the *Mep*-controlled cleavage site, were raised in rabbits. These antibodies were in turn used to raise anti-idiotype antibodies which should recognize proteins which interact with the peptide sequence used as the initial antigen. Immunoblot analysis of embryo protein body extracts indicated that the anti-idiotype antibodies recognized an Mr 25,000 polypeptide present in *Mep/Mep* embryos but absent in *mep/mep* embryos. A screen of a maize embryo cDNA expression library with the anti-idiotype antibodies resulted in the isolation of three cross-hybridizing clones, the longest of which was designated pcPOG1 (for *Processor Of Globulin*) and selected for further analysis. Northern blot analysis indicated that transcripts corresponding to pcPOG1 are present in developing embryos and in leaves of 7 day-old seedlings but not in mature embryos, etiolated shoots, roots, or developing endosperm. Transcripts corresponding to pcPOG1 are present, however, in developing *mep/mep* embryos.

Nucleotide sequence analysis of pcPOG1 revealed an open reading frame of 354 amino acids which is in the correct reading frame for the cDNA library expression vector. The predicted molecular weight for the deduced protein is 37,084 daltons. The deduced protein sequence contains several potential membrane-spanning domains and a consensus sequence for a flavin binding site. An extensive search of databases with either the pcPOG1 nucleotide or deduced protein sequence was unsuccessful in identifying sequences which exhibit significant degrees of homology. To obtain full-length clones corresponding to pcPOG1, the cDNA library was re-screened with a radiolabelled fragment from the 5' region of the original clone. Sequence analysis of this clone is underway.

Southern blot analysis of maize DNA indicates that pcPOG1 sequences represent a small gene family. The *Mep* gene is located on the long arm of chromosome 5 (near *Pr*), and localization of pcPOG1 to this region would strongly suggest that pcPOG1 corresponds to *Mep* . Two loci have been mapped by Paul Sisco (NC State, Raleigh) to chromosomes 6 and 3. A third locus remains to be localized.

We are currently attempting to determine the function of the protein encoded by pcPOG1. For this purpose, the pcPOG1 protein has been expressed in *E. coli* by using the pMAL-c vector (New England Biolabs) for subsequent purification and production of additional antibodies. We are

also planning in vitro transcription and translation experiments to determine if the pcPOG1 protein is capable of processing proGLB1 to GLB1.

ABA regulation of gene expression in embryos of viviparous mutants

--Renato Paiva and Alan L. Kriz

Studies on gene regulation during precocious germination of the viviparous mutants may provide important insights to processes of embryo development, maturation, and germination. GLB1 and GLB2, the major storage proteins of maize embryos which are respectively encoded by the *Glb1* and *Glb2* genes, accumulate to high levels during embryo development and are subsequently degraded with the initiation of seed germination. We previously reported that levels of *Glb1* transcripts in embryos of viviparous mutants differ from those of embryos undergoing normal germination, indicating that *vp/vp* embryos do not completely switch from development/maturation processes to germination processes (Kriz et al., Plant Physiol. 92:538, 1990). These studies also indicated that *Glb1* expression is positively regulated by ABA, and that homozygous *vp1* embryos are completely lacking in *Glb1* products, suggesting that a functional *Vp1* gene product is essential for *Glb1* expression. This is consistent with studies by McCarty et al. (Plant Cell 1:523, 1989) which indicate that the *Vp1* gene product is involved in ABA-mediated signal transduction processes in the developing embryo.

To further investigate the effect of ABA on the expression of *Glb1* and *Glb2*, we performed a series of "ABA-rescue" experiments of *vp* embryos. Sib normal and mutant embryos were obtained from 22 DAP ears segregating for each of the *vp* mutants and maintained for 4 days on growth medium in the presence or absence of 10 μM ABA. For all genotypes, absence of ABA in the medium resulted in germination while the presence of ABA suppressed germination except for the ABA-insensitive *vp1/vp1* embryos. Levels of *Glb1* and *Glb2* transcripts were subsequently evaluated by northern blot analysis. Results of this analysis for *Glb1* are shown in Figure 1; essentially identical results were obtained for *Glb2*. In the absence of ABA,

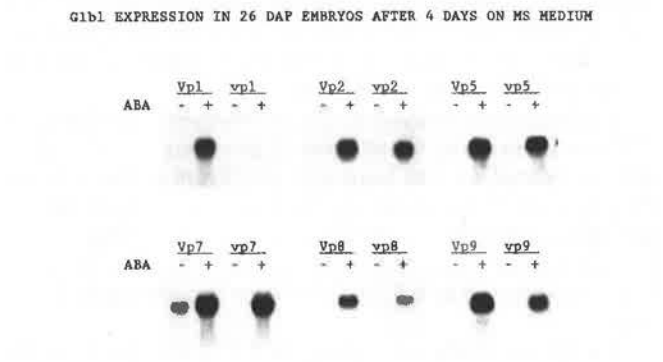


Figure 1. Northern blot analysis of *Glb1* transcripts in embryos after 4 days on Murashige-Skoog medium. Sib normal and *vp* embryos were collected at 22 DAP (except for *vp8*, which was collected at 30 DAP) and maintained on Murashige-Skoog medium for 4 d in the absence (-) or presence (+) of 10 μM ABA.

globulin transcripts are absent except for *Vp7* embryos, which apparently represents a special case. With the exception of *vp1/vp1* embryos, globulin transcripts are present in embryos of all genotypes maintained on medium supplemented with ABA. We therefore conclude that ABA plays a major role in regulation of the globulin genes, and that a functional *Vp1* gene product is required for globulin gene expression during embryo development.

A note of clarification concerning P3377 suspension culture

--David R. Duncan and Jack M. Widholm

Many research groups have obtained and are using the non-regenerable P3377 maize suspension culture developed in our lab. After attending several presentations concerning work done with this culture and after talking with the colleagues involved in the work presented, it has become apparent that some people think this culture is from an inbred. In fact, the culture was derived from a collection of embryos from a selfed Pioneer 3377 hybrid plant and thus is F2 material.

In physiological studies where an "average maize response" is desired, this suspension can be ideal. It is conceivably possible, however, that under some experimental circumstances clonal populations may be developed due to mere segregation within the F2 population. Consequently, we suggest that persons using the culture think through what the implications of using hybrid material might be for their research.

Breeding programs and the development of regenerable callus cultures

--David R. Duncan, David P. Deutscher and Jack M. Widholm

Several reports indicate that the ability of a maize genotype to produce regenerable callus cultures from immature embryos is genetically controlled (Tomes and Smith, Theor. Appl. Genet. 70:505-509, 1985; Williams et al., In Vitro 25:95-100; and others). These experiments used F1 hybrids, made from inbreds known to culture well and inbreds known to culture poorly, that were then backcrossed onto the parental materials. Also, there seems to be a general trend in maize tissue culture indicating that the more agronomically elite an inbred is, the more difficult it is to culture. Thus, A188 produces good cultures easily and B73 generally produces poor cultures or on rare occasions produces a good culture. Considering the genetic control of maize culturability, we thought that perhaps breeders might be selecting against culturability while selecting for useful agronomic traits. Such a scenario could explain the "trend" of agronomically elite lines producing poor cultures.

To test this hypothesis, we looked at culturability versus agronomic traits of Illinois Foundation Seed (IFS) inbreds developed from the same breeding population and in the final stages of the IFS breeding program. This experiment compared callus initiation of these inbreds with the agronomic production of these inbreds in test crosses. Two media and three field locations in the midwest were used.

The donor plants used in callus production were not grown in the three production fields.

Among the 21 lines tested, from 80% to less than 1% of the embryos cultured formed regenerable callus (Fig. 1). When this percent of callus induced was compared to yield, ear height, root lodging and stalk lodging no correlation could be detected (Fig. 2).

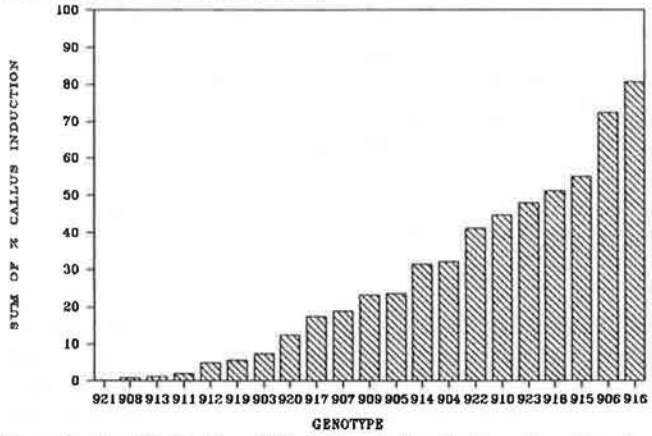


Figure 1. The distribution of Illinois Foundation Seed genotypes based on their production of regenerable callus from immature embryos. The y axis represents the summation of percent callus initiation from the different media.

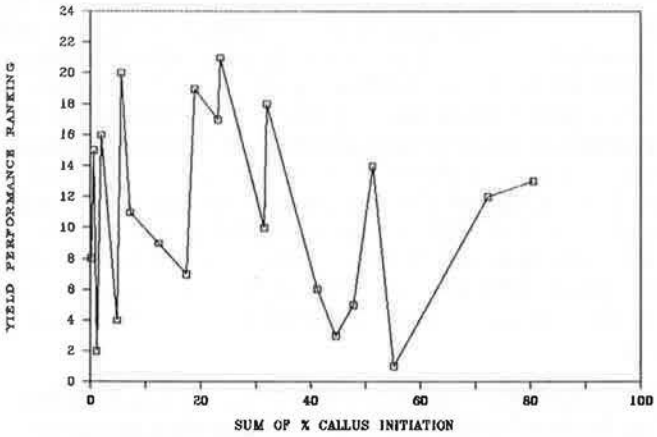


Figure 2. The relationship between percent callus initiation and yield performance (ranked highest to lowest) of 21 Illinois Foundation Seed genotypes. The x axis represents the summation of percent callus initiation from two different media.

These results indicate that using this breeding stock and these agronomic traits, culturability was not selected against in the IFS breeding program. These results also suggest that the loss of culturability with agronomic improvement of maize may best be explained by the incorporation into breeding programs of material initially devoid of gene(s) for culturability.

WALTHAM, MASSACHUSETTS
University of Massachusetts
Eastern Agricultural Center

The *ub* gene (unbranched tassel) for increasing productivity

--Walton C. Galinat

Productivity in corn has increased over the millenia by several structural changes including (1) an increase in the photosynthetic area above the ear; (2) an increase in the storage capacity of the ear as a whole; (3) an increase in femaleness including tassel reduction; (4) an increase in the precociousness of female development together with its consequence, the extent of husk enclosure.

Under the present system of high density planting of about 26,000 plants/acre, further increases in the photosynthetic area above the ear by increases in the number and size of leaves can be dangerous during years of drought stress. Losses from crop failure during one bad year would not be compensated for by an accumulation of small increases that only occur under ideal conditions.

Increases in the storage capacity of the ear may remain unfulfilled as barren cobs at high density planting. Increased productivity by means of increased femaleness is independent of the stress problems from high plant density. A shift in the reproductive investment of resources (photosynthate) from the tassel and pollen production into ear development has become at least an unconscious part of breeding programs for increased yields. But having recognized this, why not make use of the *ub* gene for unbranched or reduced tassels in order to partition more resources and productivity into the ear? The gene is simply inherited and it is usually expressed as a recessive gene showing some incomplete dominance in certain crosses. For maximum increases in ear productivity, the *ub* gene should probably occur in both parents of an F1 hybrid.

But there is a pollination problem in crossing fields with reduced or unbranched tassels in the male rows, especially when only 20% of the rows serve as the pollinators. Work is underway to determine if a certain recessive gene for a ramosa tassel (*ra-D*) on a plant with a normal ear can increase pollen shed from an otherwise genetically unbranched (*ub*) tassel.

Relationship between *ts2* alleles, femaleness and internode length

--Walton C. Galinat

Different *ts2* alleles in the presence of the *sk* (silkless) gene express themselves as different degrees of femaleness in the tassel and this in turn relates to different degrees of internode elongation in the main stalk (Table). The different *ts2* alleles originated, apparently, as a result of an *Ac* factor transposed from the nearby *P* locus because selection for solid red kernels in variegated stocks led to selection of the different degrees of femaleness in the tassels of *ts2 sk* combinations.

ts2 allele	Est. %	Av. hgt, cm	Av. internode length, cm
ts2	100	50	4.17
ts2-1	75	60	5.00
ts2-2	50	75	6.17
ts2-3	25	90	7.50
ts2-4	0	102	8.50

The relationship between the degree of femaleness in the floral phase and the amount of internode elongation in the vegetative phase below is the basis for the complete husk-enclosure of an all female ear. Apparently female development results in the feedback of a hormone-like substance that inhibits internode elongation in vegetative

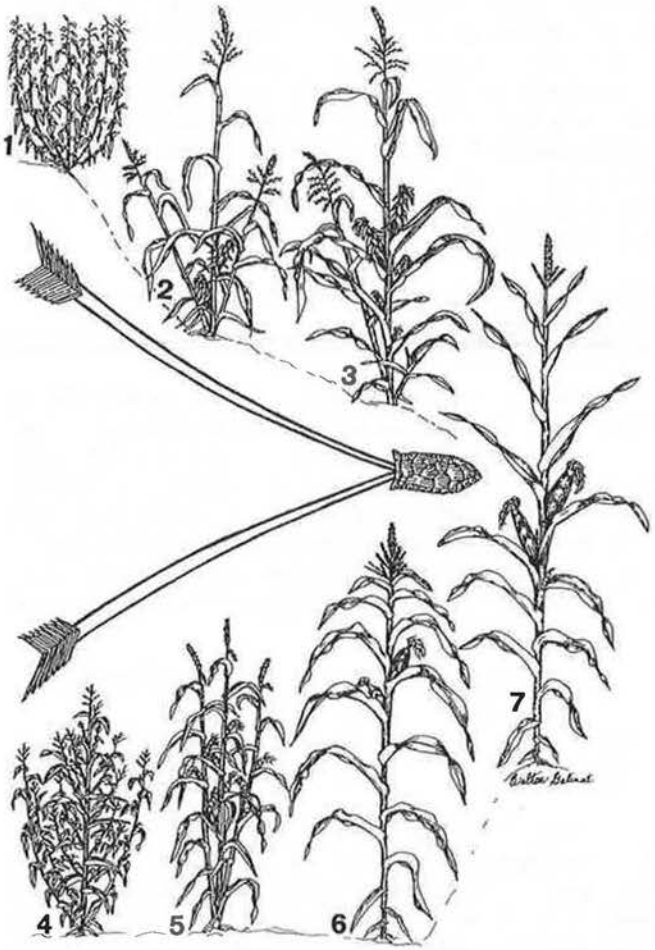
phase below which would otherwise continue simultaneously. The degree of precociousness of female development determines how soon internode elongation will be inhibited and, therefore, how complete the husk confinement of the ear.

A similar relationship may be observed in the tillers of most corn, especially the Northern Flints, in which tillers that are as long as the main stalk duplicate it in having an all male terminal tassel and all female lateral ears. But short tillers have tassels that are partly female and the degree of femaleness regulates the degree of internode elongation.

Growth patterns as additional evidence of a biphyletic domestication of teosinte in the origins of corn

--Walton C. Galinat

Previously I have described cob differences in cupule and rachilla development as they appear to relate to two different systems for kernel exposure by independent domestications of teosinte (Galinat, 1988). Additional evidence for this double origin of maize appears to come from two pathways in modes of branching and patterns of internode lengths leading to the northern flint and southern dent parents of the Corn Belt dent (as illustrated here in a line drawing adapted from my painting).



Plant Habit as Evidence for a Double Origin of Maize
(A line drawing adapted from a painting by Walton C. Galinat)

The plant habits from the two pathways of corn races based on cob morphology lend support for the double origin of corn. They form two series of plant habits that may be termed the basal branching type and the lateral branching type, both of which lead to the Corn Belt Dent.

1. Teosinte subspecies *parviglumis* race Balsas, under good growing conditions. Note the proliferation of tillers at the base of the plant.
2. The indigenous flour corn of the upper Missouri area is close to one type of the first corn, pre or proto-Chapalote. Its ear is near the ground where it comes from an elevated and condensed tiller.
3. The Northern Flint (Rhode Island Flint). The upper ear bearing node has elevated to a central position on the plant but a trail of its ascent remains.
4. Teosinte subspecies *mexicana* race Chalco. Note that the branching is lateral and each branch terminates in a tassel with ears borne below as on the main stalk of corn.
5. Palomero Toluqueno. This multi-eared popcorn has a barren zone both above and below the ear bearing region.
6. Southern Dent (Gourd Seed). The ear is high on the plant and tillers are suppressed.
7. The Corn Belt Dent. This hybrid between the Northern Flint and Southern Dent pathways shows extreme heterosis. It has productivity refinements indicated here as reduced tassels and erect upper leaves.

In each pathway there is a stepwise inhibition of vegetative branching that is related to increased feminization. In the Balsas or *parviglumis* pathway leading to the northern flints, the branching and its feminizing inhibition is basal. The *gt* (grassy tiller) gene in maize increases maleness and immediately restores the plant habit of Balsas teosinte characterized by basal branching. In the Chalco pathway leading to the southern dents, the branching and its inhibition by feminizing is lateral. The *tb* (teosinte branched) gene in maize increases maleness and immediately restores the plant habit of Chalco teosinte characterized by lateral branching. In each pathway, a polygenic trait of increased yield and branch inhibition has gradually evolved by way of increased levels of feminization only to have the whole sequence collapse from being undercut by either of two mutant genes (*gt* or *tb*) that increase the level of maleness and, thereby, promote internode elongation and branch or tiller development. These two genes differ only in timing of their action, which determines whether the branching will be basal (*gt*) or lateral (*tb*).

Interspace (*is*) and string cob (*Sg1*, *Sg2*) as stabilizing factors for the expression of key trait genes (*tr*, *pd*)

--Walton C. Galinat

The near failure of the internodes to elongate in the cob of most modern maize (except Coroico and related corn from the Bolivian lowland of South America and Confite Morocho related corn from the highland Andes) results in an unstable expression of single vs. paired female spikelets and two ranks of spikelets vs. many ranks. Apparently with the almost side by side juxtaposition of spikelet components, small fluctuations in the internal-external environment can easily result in either a proliferation or a reduction.

Thus, in developing key trait stocks for genetic and molecular analysis, it has become necessary to include the interspace and string cob traits in the genetic background for best results.

It is significant that while the *is* and *Sg1*, *Sg2* genes are now essentially absent in North American maize, they do occur in teosinte and thousands of years ago they were

characteristic of the oldest maize from Tehuacan, apparently as relic remains from its teosinte origin. At that time it was carried to South America where in isolation these genes survive to this day.

On the origin of Longfellow-Flint and bird control
--Walton C. Galinat

The early records of Longfellow's Flint are listed in the possessive as if it originated with the poet, Henry Wadsworth Longfellow. It is listed in this way in the seed catalogue of the W. Atlee Burpee & Co. of Philadelphia, 1886 where it says Longfellow's Flint is well adapted to Massachusetts where it produces 200 bushels of ears to the acre. It appears to me to be a yellow form of Rhode Island Flint. I have one ear of Longfellow's Flint grown in Byfield, Massachusetts on the farm of A. B. Forbes in 1883. The ear is 11 inches long with 8 rows of large yellow kernels. Byfield is near the coast of the north near the New Hampshire border with Massachusetts. Byfield is also the original family homestead of the Longfellows. Perhaps it was here Longfellow wrote his poem, "Blessing the Cornfields" in his *Song of Hiawatha* (1881:p.136, The Complete Works of Longfellow, Houghton, Miflin & Co. Boston). From my experience, crows are especially fond of Northern Flints and it was so then. The method of bird control used by Hiawatha is described in this poem as follows:

"They perceived no danger near them,
Till their claws became entangled,
Till they found themselves imprisoned
In the snares of Hiawatha.
From his place of ambush came he,
Striding terrible among them,
And so awful was his aspect
That the bravest quailed with terror.
Without mercy he destroyed them
Right and left, by tens and twenties,
And their wretched, lifeless bodies
Hung aloft on poles for scarecrows,
Round the consecrated cornfields,
As a signal of his vengeance,
As a warning to marauders.
Only Kahgahgee, the leader,
Kahgahgee, the King of Ravens,
He alone was spared among them
As a hostage for his people.
With his prisoner-string he bound him,
Led him captive to his wigwam,
Tied him fast with cords of elm bark,
To the ridge-pole of his wigwam.
"Kahgahgee, my raven!" said he,
"You the leader of the robbers,
You the plotter of this mischief,
The contriver of this outrage,
I will keep you, I will hold you,
As a hostage for your people,
As a pledge of good behavior!"

WINONA, MINNESOTA
St. Mary's College
ST. PAUL, MINNESOTA
University of Minnesota

Maternal effect leads to DNA replication pattern in maize endosperm
--R. V. Kowles, G. L. Yerk, R. L. Phillips, and F. Srienc

Within two to four hours after fertilization the endosperm nucleus begins rapid synchronous divisions resulting in a syncitial tissue. Cell walls are synthesized and laid down at about four days after fertilization leading to a cellular, uninucleate tissue. Mitotic activity in the endosperm peaks at 8 to 10 dap. DNA synthesis in the endosperm peaks at 16 to 18 dap and then declines. Starch and zein synthesis in the endosperm begin at about the same time as the increase in DNA synthesis occurs.

The endosperm may be divided into three regions with regard to the DNA increase: 1) a central region in which the DNA content per nucleus is elevated, 2) a peripheral region where normal mitotic activity occurs and the nuclei remain 3C, and 3) a transitional region between the central and peripheral regions where both types of cells occur. A number of molecular tests have indicated endoreduplication as the mode of DNA increase in endosperm cells; i.e. the entire genome undergoes replication during each round. These endoreduplicating cells have a cell cycle consisting of alternating G and S phases. DNA content may reach levels as high as 384 C per nucleus in inbred A188, with an average DNA content per nucleus of 90 to 100C in the central, endoreduplicating region.

Multiparametric flow cytometry has been used to further characterize the DNA content and replication patterns of a variety of maize endosperm samples including inbreds, F1s, F2s, and F3s. Nuclear preparations made from the entire endosperm of one kernel were stained with mithramycin A. Similar preparations were made from embryo tissue. The embryo nuclei serve as 2C and 4C reference cells for the endosperm nuclei of the same line.

The samples were analyzed using a Cytofluorograph IIs. Size of the nuclei was measured by small-angle light scattering and DNA content was determined by the magnitude of the fluorescence signal from each nucleus. DNA replication patterns were obtained from histograms of frequency vs. fluorescence intensity processed in area mode (Fig. 1). Peaks on such a plot correspond to cells in G phase with each peak representing a doubling in DNA content from the previous one (3C, 6C, 12C, etc.). The troughs between adjacent peaks correspond to cells in S-phase. The sharpness of these peaks and the percentages of cells in the trough areas vary among inbred lines.

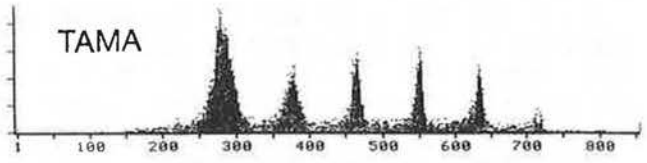


Figure 1. Number of nuclei vs. log DNA fluorescence in arbitrary units for self pollinated Tama Flint at 16 dap.

Analysis of pairs of inbred lines and their reciprocal hybrids revealed that the DNA replication pattern of the hybrid is the same as that of the female parent in the cross. Data from F2 kernels derived from reciprocal F1s gave DNA replication patterns among F2 kernels which were all the same regardless of the direction of the cross. This suggests the observed phenomenon is not due to either dosage effect or organellar genomes controlling the inheritance of the DNA replication patterns. If a dosage effect were present, differences among F2 kernels from an ear should be observed. In the case of organellar DNA controlling the patterns, no difference among F2 kernels from an ear would be seen but reciprocal F2 kernels derived from F1s should give different patterns.

The data described thus far suggest that the DNA replication patterns in maize endosperm nuclei could be controlled by maternal effects. Maternal effects arise due to protein(s) or transcript(s) encoded by the nuclear genome of the maternal parent that influence the development/performance of the progeny. If maternal effects are causing the observed patterns, the following outcomes would be predicted. First, the hybrid will always have the same DNA replication pattern as the maternal parent in the cross. Second, all F2 kernels will have the same pattern regardless of which direction the F1 hybrid they were derived from was made. This is because the nuclear genotype of the F1 is the same regardless of the direction of the cross. Third, provided kernels from enough different F3 ears are sampled, different patterns should be observed. This is because the F2 plants upon which the F3 seeds are growing have different genotypes.

Comparison of DNA replication patterns from F3 kernels demonstrated differences in the patterns among kernels. These findings support maternal effect as the basis of the observed DNA replication patterns.

Endoreduplication and mitotic activity in defective kernel mutants

--R. V. Kowles, G. Yerk, R. L. Phillips and F. Srienc

A group of 28 defective kernel (*dek*) mutants obtained from M. G. Neuffer, University of Missouri, Columbia, MO were studied with regard to DNA content per nucleus and cell number per endosperm. Defective kernels are phenotypically distinct from normal kernels. An obvious characterization is their pronounced smaller size on a segregating ear. The overall mean kernel weight for normal kernels was 215.6 mg compared to 49.2 mg for defective kernels constituting a 4.4-fold difference.

Developmentally normal and defective kernels were collected and fixed at 16 days after pollination (dap). The fixative was replaced with 70% ethanol after 24 hours, and the samples were stored at -20 C. Nuclear suspensions were made in such a way that each preparation essentially consisted of all of the nuclei of the endosperm from a single kernel. This was accomplished by carefully dissecting the endosperm from the pericarp, nucellus, and embryo tissues, and gently forcing it through a stainless steel screen with several successive washes of a grinding medium. By low-speed centrifugation, the grinding medium was replaced with mithramycin buffer which, in turn, was replaced with the fluorochrome mithramycin A. The number of nuclei in each endosperm was calculated by removing samples from the nuclear suspension for use with standard hemocytometric procedures. The nuclei were counted with a combination of fluorescence and low-illiumination phase contrast microscopy.

DNA content per nucleus and endoreduplication patterns were determined by flow cytometry. Nuclei were classified according to small angle light-scattering signals, indicative of the size of the nuclei, and according to fluorescence signals as a measure for the DNA content of the nuclei. Because of the large variation of the properties of the nuclei, the signals were collected on a logarithmic scale. Nuclei classified according to peak fluorescence and light-scattering signals appeared as clustered populations permitting convenient exclusion of debris by gating from further analysis. Gated signals were displayed as one-parameter histograms for analysis.

TABLE 1

CELL NUMBER AND DNA CONTENT PER NUCLEUS IN NORMAL AND DEFECTIVE KERNEL ENDOSPERM AT 16 DAP

dek Strain	Cell Number Per Endosperm		DNA Content Per Nucleus[1]	
	dek	Normal	*dek*	Normal
1288	43,000	139,000	381	446
1400	86,000	160,000	365	426
1519B	24,000	54,000	376	413
1384A	16,000	152,000	361	429
925A	44,000	80,000	364	414
1320B	45,000	178,000	386	476
918A	38,000	82,000	338	458
1368	76,000	222,000	406	458
1328A	20,000	166,000	401	502
1285	40,000	96,000	381	449
1387A	18,000	172,000	435	513
240B	80,000	142,000	383	460
1228	28,000	118,000	386	476
1112	51,000	123,000	428	516
1136B	31,000	230,000	435	491
1122A	88,000	144,000	364	490
1307A	42,000	136,000	395	499
1126A	25,000	92,000	402	469
1318	50,000	234,000	417	462
1009	47,000	158,000	511	512
1520B	38,000	226,000	381	447
1185A	64,000	195,000	446	491
1078A	83,000	189,000	434	472
802	26,000	170,000	418	440
1322A	38,000	194,000	394	449
399A	30,000	102,000	491	562
930	42,000	190,000	384	432
868A	82,000	342,000	367	434

1 Arbitrary units of fluorescence

DNA content per nucleus, indicative of the degree of endoreduplication, and the number of cells per endosperm, indicative of mitotic activity, are presented in Table 1. In all cases, a greatly reduced number of cells per endosperm was observed in the defective kernel versus the normal kernel from segregating ears. The mean cell number for the normal kernels in the 28 strains was 160,000 at 16 dap, and the mean for the defective kernels in these strains was only 46,000. Most defective kernels were also found to have considerably less DNA per nucleus than the normal kernels. The mean DNA content per nucleus in arbitrary units of fluorescence for the normal kernels in the 28 strains at 16 dap was 469 A.U., and the mean for the de-

fective kernels was 401 A.U.

In most of the strains, the defective kernels show a greater percentage of nuclei in the 3C and 6C levels, approximately the same percentage in the 12C level, and a much lesser percentage in the 24C, 48C, and 96C levels. Notable differences in DNA replication patterns, however, were noted among several *dek* strains. A comparable pattern between the defective and normal kernels is seen in strain *1009* (Fig. 1a, b) and almost a complete absence of endoreduplication in strain *868A* (Fig. 1c, d). One example in which fewer rounds of endoreduplication are apparent in the endosperm of the defective kernel is seen in strain *1387A* (Fig. 2a, b). In another example, the defective kernel undergoes the same number of rounds of endoreduplication but a lesser percentage of nuclei are found with the higher C levels of DNA (strain *1112*, Fig. 2c, d).

Strain *1009* proved to be an interesting exception to the other *dek* strains in that it shows a similar pattern of endoreduplication between the defective and normal kernels from a segregating ear. The normal kernel had a mean of

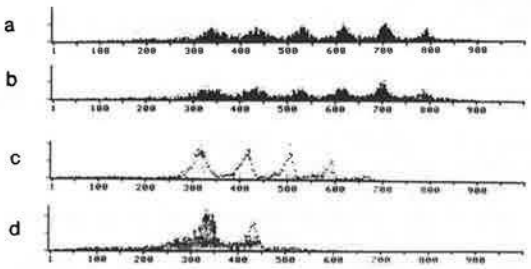


Figure 1. Endoreduplication patterns displayed as histograms in which the frequency (Y axis) of nuclei are recorded relative to their fluorescence intensity (X axis). The fluorescence intensity, in turn, is indicative of the DNA content of the nucleus. (a) Strain *1009* normal kernel; (b) Strain *1009* defective kernel; (c) Strain *868A* normal kernel; (d) Strain *868A* defective kernel.

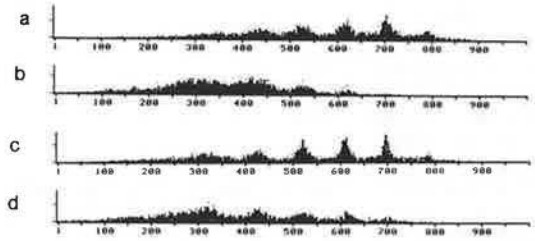


Figure 2. Endoreduplication patterns of (a) Strain *1387A* normal kernel; (b) Strain *1387A* defective kernel; (c) Strain *1112* normal kernel; (d) Strain *1112* defective kernel.

512 A.U. of DNA per nucleus, and the defective kernel had a mean of 511 A.U. A comparison of their histograms, which reveal the distribution of nuclei in each of the C level regions, also shows a striking similarity. The cell number in the normal endosperm, however, is 158,000 to only 47,000 in the defective kernel endosperm. Apparently, this mutation affects mitotic activity without having an effect upon the endoreduplication process. This strain may be useful for other studies of endosperm development and cellular kinetics.

This is a summary of selected genetic research information (e.g., new factors; mapping; cloning) reported in recent literature and in this News Letter ("r" refers to numbered references in the Recent Maize Publications section). The Symbol Index refers by number to all current published research involving genetic materials. Comments or suggestions on these research aids would be welcome.
BS = Base Sequence; BSH = Broad Sense Heritability; GCA, SCA = general and specific combining ability; GxE = Genotype x environment interactions; NSH = Narrow Sense Heritability; PCR = Polymerase Chain Reaction; QC = Quantitative Character; QTL = Quantitative Trait Loci; RFLP = Restriction Fragment Length Polymorphisms; RI = Recombinant Inbreds; RM = Restriction Map; unc. = uncovered
* in symbols identifies loci needing allelism tests, documentation, or standardization of the symbol.

CHROMOSOME 1
Phy1 genomic ("*phyA1* gene") RM BS --r121
Adh1: ARF-BS factor binding to region of anaerobic response element --r210
Ht1 near *UMC122* --r289
Ac-like sequences in CM37 vs. T232 & CO159 vs. Tx303 --r311
Tub1 & *Tub**, alpha-tubulin family; Tub alpha1 & 2 in tandem; cDNA & genomic BS RM --r456 r457
bz2 genomic RM BS --r476
P1-vv, *P1-ovov* RM, *Ac* inserts --r525
GII-I96 right of TB-1La --r575
NPI238 BS & variants in cultivars --r621
Kn1-O tandem duplication; genomic, BS RM; *Mu* insertion locations --r704
Adh1 methylation --r714
chromomeres --r753
dek-2045* -32- T1-9(8918) *wx1*; , *dek*-1568* -10- *bz2* - *Kn1* - *bm2*; *dek*-2115*, *dek*-8319*, *dek*-6214* on 1 --65:11
idd-2286A* allelic to *id1* and *an1* (of *an1-6923*) but not to *bz2*; conclude order *id1* - *an1* - *bz2*; *UMC83* - *id1* - *bz2* --65:52
RFLP core markers --65:55
RFLP & protein markers associated with QCs --65:66
markers, loci, polymorphisms (RFLPs & others) --65:105
id1 -0- *BNL17.06* among 24 F2 plants --65:110

CHROMOSOME 2
B1 genomic, RM --r112
Ac-like sequences in CM37 vs. T232 & CO159 vs. Tx303 --r311
Hrg1, hydroxyproline-rich glycoprotein cDNA, genomic, BS; *UMC145-HRG1* --r408 r654
hcf106, *Mu1* modification --r429
B1-Peru and *Mu* inserts; RM --r515
chromomeres --r753
PIO1012 -9- *dek*-1047* -14- *UMC131*; *dek*-1047* -18- T2-9d *wx1*; *dek*-2159* -18- T2-9d *wx1*; *dek*-4160*, *dek*-2444*, *dek*-1365-6*, *dek*-1047* on 2 --65:11
regulator of *Mu* linked to T2-9d *wx1* --65:12
os1, opaque endosperm-small germ, weak but viable plants; unc. by TB-3La-2S(6270) but not by TB-3La, linkage with T2-9b *wx1* --65:23
whp1 genomic, cDNA --65:51
RFLP core markers --65:55
MDMV R associated with markers on 2L --65:100
markers, loci, polymorphisms (RFLPs & others) --65:105

CHROMOSOME 3
Sh2 cDNA RM BS; ADPG pyrophosphorylase requires *Sh2* and *Bt2* subunits --r58 r540
preferential pairing: zygomeres --r183
Ac-like sequences in CM37 vs. T232 & CO159 vs. Tx303 --r311
abp1, auxin binding protein (replaces *axr1*) between *BNL6.06* and *Pgd2* --r400
GII-A30 right of TB-3La --r575
chromomeres --r753
te1 unc. by TB-3La; allele *te1-sn* from *Mu* screening linked with a *Mu8* element --65:2
transposed *En* linkages with *A1* --65:10
dek-1185* -30- *cl1*; *dek*-1364* on 1; *dek*-33* & *dek*-2525* alleles of *dek5*; *dek*-507* & *dek*-3328* & *dek*-2424* & *dek*-24* & *dek*-2320* & *dek*-2352* & *dek*-2457* & *dek*-216* & *dek*-27* & *dek*-34* alleles of *et1* --65:11
RFLP core markers --65:55
MDMV R associated with markers on 3L --65:100
rd3, reduced plant; anthocyanin interaction? One recombination with *BNL6.06*, *vp1*, *BNL5.37*, *BNL10.24A*, *BNL8.01*, & *BNL5.14* in 10 recessive F2 plants --65:105
markers, loci, polymorphisms (RFLPs & others) --65:105

CHROMOSOME 4
association of *su1* with head smut R --r6
Bt2 cDNA BS; ADPG pyrophosphorylase requires *Bt2* and *Sh2* subunits --r28 r540
Zpr10/(22) -7±4- *Ga1*; *Zpr10/(22)* -22±2- *fl2* --r46
centr. - *PGE1* -5- *C2*; *PGE1* = a PCR-amplified *Spm* sequence --r189
*CP2**, Central-Plateau, cross-incompatibility; *Ga1* - *Ts5* -17.9- *CP2** -4.2- *su1* -r301
Ac-like sequences in CM37 vs. T232 & CO159 vs. Tx303 --r311
GII-F5, *GII-A95*, *GII-O40* left of TB-4Sa --r575
NPI451 BS & variants in cultivars --r621
chromomeres --r753
dek-2058* on 4; *dek*-2410* & *dek*-1566* alleles of *dek25*; *dek*-2608* allele of *dek7*; *dek*-2689* allele of *dek31* --65:11
su1 -10.5- *lw4* -10.5- *gl4* --65:18
UMC111 morphs in B86 different from morphs in B52 and Oh43 --65:21
o1 -2.6±0.3- *Tu1* -8.9±0.5- *gl3* --65:37
C2-Idf genomic, cDNA --65:51
RFLP core markers --65:55
TB-4Lh unc. *c2*, *dp1*; TB-4L13474 apparently a compound (i.e., TB-6Se-4L003-16) unc. *c2*, *dp1* --65:57
zein protein polymorphisms, *ZpL1a*, *ZpL1b*, *ZpL1c*, *ZpL1d*, *ZpL1e*, *ZpL1f* mapped on 4S, *ZpL2a* & *ZpL3a* mapped on 4L with RIs -- Wilson, TAG 77:217, 1989; 65:91
transmission ratio distortion (maximum at *) *BNL15.45* -11- *UMC19* -9- *BNL10.05* -3- **UMC15* -3- **Z1C11* -7- **NPI333* -10- *BNL15.07* -5- *BNL8.23* --65:103
markers, loci, polymorphisms (RFLPs & others) --65:105

CHROMOSOME 5
Phy2 genomic ("*phyA2* gene") RM BS --r121
a2 cDNA. genomic BS --r444
Pgm2-null on 5S by RI linkages --r504
GII-A52 right of TB-5La --r575
chromomeres --r753
transposed *En* 12 cM from *A2* --65:3
UMC51 -8- *dek*-807* -12- *UMC68*; *dek*-8186* -13- *ae1*; *dek*-8186* -21- T5-9a *wx1*; *Dap1* -4- T5-9a *wx1*; *dek*-2146*, *dek*-1182*, *dek*-5133* on 5 --65:11
lw3 unc. by TB-5La --65:18
BNL6.22 morphs in B86 different from morphs in B52 and Oh43 --65:21
pr1 -24.8- *Hsf1* -26.4- *zb3* --65:30
Nl2 -10- *a2* -3- *bt1*; *D9* -32- *a2* -5- *bt1*; *D9* (was *D*-2319*)
RFLP core markers --65:55
TB-5Sc dosage affects QC --65:58
MDMV R associated with markers on 5S --65:100
sh5 -0- *BNL6.22* --65:105
markers, loci, polymorphisms (RFLPs & others) --65:105

CHROMOSOME 6
Y1 genomic via *Mu3* in *y1-mum2053*; RM --r91
Pdk1 (or *Pdk2*) genomic & cDNA RM --r242
Dhn1, dehydrin, dehydration-induced proteins, cDNA maps polymorphisms to 6 --r126
Ac-like sequences in CM37 vs. T232 & CO159 vs. Tx303 --r311
rDNA18S, rDNA26S, rDNA5.8S, methylation --r315
Pdk1 linkage to *NPI616*, *NPI252*; RM BS --r434
GII-N19 right of TB-6Lc --r575

MDMV R (*Rmd1* = *Mdm1*) - *y1* - *su2* --r578
rDNA intergenic spacer-length variation --r584
NPI288 BS & variants in cultivars --r621
Rab17 protein, ABA-induced; cDNA & genomic BS --r708 65:109
chromomeres --r753
dek-1184* close to *y1*; *dek*-1104* on 6 --65:11
mn3 (was *de*-1184*) not unc. by TB-6Lc or TB-6Sa; *mn3* -4.4- *w15* - 2.5- *y1*; *mn3* -2.1- *y1* - 7.7- *l15*; conclude near *Mdm1* --65:16
ts8 (Ames stock) allelic to *si1* --65:18
BNL5.47 morphs in B86 different from morphs in B52 and Oh43 --65:21
RFLP core markers --65:55
Mdm1: near *NPI7* & *UMC85*; other regions associated in isogenic lines --65:99
NPI7 -0.5- (*UMC85*, *po1*) -2- NOR -1- *BNL6.29* -2- *BNL7.28* -5.5- *Pgd1* --65:102
markers, loci, polymorphisms (RFLPs & others) --65:105

CHROMOSOME 7
Ask1, aspartate kinase, lysine-threonine R (was *Ltr*-1*, Lt1a, LT19) -10.6±1.3- *o2* --r27 r179
Ac-like sequences in CM37 vs. T232 & CO159 vs. Tx303 --r311
o2 cDNA BS --r611
chromomeres --r753
dek-2082* -35- T7-9(4363) *wx1*; d*ek*-NS95* & *dek*-NS326* allelic & on 5; *dek*-3193*, *dek*-5153* on 7 --65:11
RFLP core markers --65:55
ij1 -4.8- (*BNL4.24*, *BNL13.24*, *UMC110*) -7.4- (*NPI435*, *UMC56*) -2.3- *NPI283* -4.8- *UMC125B* --65:56
zein band B9/21 closely linked (in repulsion) to B9/10 and B9/22 --65:91
zein protein polymorphisms, *ZpL2b*, mapped on 7 --Wilson, TAG 77:217, 1989; 65:91
markers, loci, polymorphisms (RFLPs & others) --65:105

CHROMOSOME 8
Pdk2 (or *Pdk1*) genomic & cDNA RM --r242
b-32 (*pro1*) protein family, genomic & cDNA BS --r273 r465
Pdk2 linkage --r434
chromomeres --r753
dek-5132* on 8 -65:11
Lg4, linked to T8-9d *wx1* and T8-9(6673) *wx1*; *UMC120* -22- *Lg4* -3.7- *UMC89* --65:31
RFLP core markers --65:55
RFLP and protein markers on 8L associated with QC --65:66
markers, loci, polymorphisms (RFLPs & others) --65:105

CHROMOSOME 9
Zps10/(22) -0- *Wx1*; *Zps10/(22)* -15±0.04- *BNL3.06*; *Zps10/(22)* - 3±2- *UMC81* --r46
bz1 RM; *Ds* and *Mu1* insertions --r177
bz1-m4D6856 and *Bz'* revertants: RM --r182
Bz1 dSpm insertions, maps --r341
Sh1 promoter BS --r417
C1-I cDNAs, genomic, RM BS --r517
bz1-m2(Ac), *bz1-s:2094(Ac)* RM --r555
Sh1 methylation --r649
wx1-B1, *wx1-C4*, *wx1-W23*, *wx1-B6*, *wx1-B*, *bz1-R* BS --r728
chromomeres --r753
yg2 -4.3- *UMC109* -11.0- *NPI203* -21.0- *C1* -2.3- *UMC113* -3.6- *sh1* - 36.1- *wx1* -3.0- *BNL3.06* -6.4- *UMC127* -2.2- *d3* -4.5- *BNL5.10* -2.8- *UMC81* -1.8- *gl15* -0.9- *UMC20* -3.6- *UMC114* -5.6- *BNL5.04* -12.3- *BNL7.13* -11.0- *bk2* -1.7- *UMC95* -22.9- *BNL5.09* -3.5- *BNL14.28* - 4.5- *NPI209* - (*NPI97*, *Wc1*, *Bf1*, *bm4*) --65:52
RFLP core markers --65:55
v28 unc. by *wd1*, not allelic to *yg2*; *v28* -20- *sh1*; *v31* (was *v*-828*, gry-wlv) unc. by *wd1*, not allelic to *yg2*; *v31* -23- *sh1*; *d*-660B* allelic to *d3*; *bz1* -12- *Zb8*; *Cb*-1456* -3- *wx1* --65:86
wx1 RM, PCR --65:101
markers, loci, polymorphisms (RFLPs & others) --65:105

CHROMOSOME 10
Rp1: unequal crossing over; *BNL3.04* -3- (*Rp1-G* - *Rp5* - *Rp1-F* - *Rp1-D* - *Rp1-E* - *Rp1-B*) -0.6- *NPI422* -2- *NPI285*; *BNL3.04* -3- (*Rp1-G*, *Rp5* -3- *Rp1-F*, *Rp1-D* -0.1- *Rp1-E* -0.1- *Rp1-B*) -0.6- *NPI285*; *Rp1-G* possibly an allele of *Rp5* --r47
Mgs1, male gametophyte specific: pollen-specific cDNA Zmc13, genomic clone Zmg13 RM BS; RFLPs map near *Glu1* and *stAc* --r267
Ac-like sequences in CM37 vs. T232 & CO159 vs. Tx303 --r311
Rf Ds insertions, maps, interlocus recombination --r330
GII-A15 left of TB-10Sc --r575
NPI445 BS & variants in cultivars --r621
chromomeres --r753
dek-1339* -32- *o7*; *dek*-1339* -31- T9-10b *wx1*; *dek*-2424-9*, *dek*-2181*, *dek*-8627* on 10 --65:11
cr4, crinkly-leaf aleurone mosaic (was *dap*-6143*), unc. by TB-10Sc --65:17
dek21 allelic to *w2* --65:19
RFLP core markers --65:55
wx1, intragenic recombination vs. physical distance --65:84
Ufo1 -14- *BNL3.04*, linked with *PIO200626*, *NPI285*, *NPI264* --65:105
markers, loci, polymorphisms (RFLPs & others) --65:105

UNPLACED
imidazolinone and sulfonylurea R selected in culture (line XA17) semidominant; decreased sensitivity of acetohydroxy acid synthase (AHAS). Line QJ22 R to imidazolinone only, dominant --r14
Mct1, modifier of mitochondrial *cox2* transcripts --r128
zein27kD gene: somatic rearrangements --r143
Ask2, aspartate kinase; lysine-threonine R (was LT20) --r163 r179
Atp2: F1-ATPase subunit 2, BS --r191 r747
WF9 vs. M825: mtDNA organization changes --r195
luciferase (LUC), acetolactate synthase (ALS) transformants: inheritance --r221
phosphinothricin acetyltransferase (PAT) transformants: inheritance --r248
primisulfuron sensitivity: recessive; slower metabolizing of herbicide --r272
seed storage susceptibility, recessive; polyamine (spermidine, putrescine) content correlated --r405 r406
pollen-specific cDNA Zmc58, homologous to pectate lyases --r431
Acc1, partially dominant R to cyclohexanedione (sethoxydim) and aryloxyphenoxy propionate (haloxyfop) herbicides selected in tissue cultures (callus S2); altered sensitivity and activity of acetyl-coA carboxylase (EC6.4.1.2) --r509 65:94
zein-2 component Zc1, 16kD, cDNA & genomic, BS; glutelin-2 component Zc2, 28kD, genomic, BS --r570 r571
dek-1047*, *dek*-NS807*, *dek*-NS326* (and its allele *dek*-NS95*), *Mu1*-generated; inserts, RM --65:10
Kn2, *Rs1*, *Rs*-1025* closely linked or allelic --65:29

B CHROMOSOME

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Ac: excision-specific sequences --r130
Mu1 BS, transcript initiation sites --r151
Ac methylation --r153
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Spm methylation --r203
Ds of *wx1-B4*, RM BS --r702
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zein19kD (pMS1 clone) P1 and P2 promoters, BS --r86 r252 r422
snRNA-U2-27 genomic RM BS --r87
Mu1.4-B37 RM; methylation --r111
histone-3 (H3) family , histone-4 (H4) family: genomic clones H3C2, H3C3, H3C4, H4C7, H4C13, H4C14 --r121
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hypervariable sequences, phage M13-specific --r240
Pdk1, *Pdk2* genomic & cDNA RM --r242
b-32 (incl. *pro1*) protein family, genomic & cDNA BS --r273 r465

PEPcase cDNA from root differs from photosynthetic-PEPcase clone; RM BS --r328
alpha-tubulin family; Tub alpha1 & 2 (*Tub1*, *Tub2*) in tandem; Tub alpha3; cDNA & genomic BS RM --r456 r457
HSP26 cDNA BS; chloroplast-imported & processed --r485
oleosin18kD cDNA RM BS --r548
Sod3 cDNA --r608
ocs-element binding protein (OCSBF-1) cDNA, BS --r630
Cin2 BS --r705
U6 sequence, BS --r707
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thrip R, *Anaphothrips obscurus* Mueller, *Frankliniella fusca* Hinds, *F. tenuicornis* Uzel, GCA SCA --r64
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heat-stress tolerance via gamete selection --r526
phytic acid content --r551 r552
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ECB R, altitudes of origin, DIMBOA; stalk rot R, *Gibberella zeae*; smut R, *Ustilago maydis* --r568 r569
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adaptation & QC in temperate x tropical sweet --r590
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Goss' wilt R, *Corynebacterium michiganense* ssp. *nebraskense* Schuster &, GCA SCA --r682 r683
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TB-5Sc dosage affects pollen-silk interval, rind puncture resistance, leaf length & width, plant & ear height, tassel branch number, leaf number above ear --65:58
QCs for heat units, pollen-silk interval, percent moisture, staygreen, ear height, plant height, test weight, cob diameter, ear diameter, row length, row number, kernels per row, yield, 100 kernel weight associated with protein and RFLP markers --65:66
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MDMV R associated with markers on 2L, 3L, 5S, 6S --65:100
culturability & regenerability vs. yield --65:115

--Assembled unrestricted by Prof. Ligate

IV. MAIZE GENETICS COOPERATION STOCK CENTER

During calendar 1990, 2656 seed samples were supplied in response to 202 requests. Included were requests from 17 foreign countries. In addition, advice and information were provided particularly with regard to the classification and use of genetic stocks and relative to pedigree lineages and genetic backgrounds. Each year a substantial number of misdirected requests are received, and an effort is made to refer these to appropriate persons.

Because of a record high rainfall total during the May-June period, there were problems of seedling emergence in the early May planting and later plantings were much delayed. A late season drouth in August and September resulted in poor kernel fill in late plantings. Seed increase efforts met with varied success, largely dependent on the vigor and agronomic performance of individual stocks. In many instances, seed increases were too limited to constitute a long-term seed supply and insufficient for catalog listing prior to further increases.

Field and greenhouse plantings of the past year included the following:

(1) Stocks of miscellaneous dominant or recessive symbolized genes newly available in recent years.
(2) Extensive plantings of chromosome 1 and chromosome 4 stocks.
(3) New genes or combinations received during the past year from Cooperators.
(4) A traditional series of B-A translocations, supplemented by additional interchanges mostly located in 3L, 4L or 10L.
(5) Stocks of *wx*-marked translocations received from D. S. Robertson.
(6) Tetraploid stocks.
(7) A selfing block of mutant alleles expressed as kernel or seedling traits.
(8) Observation plantings for field classification.
(9) Greenhouse sandbench plantings to determine or confirm genotypes relative to seedling traits.

We are hopeful that within the next year or two supplemental cold storage facilities may become available. In that event, we plan to transfer seed samples of less-frequently requested stocks to such facilities, freeing space in our current cold room to receive additional items. Included in such transfer will be numerous multiple gene chromosome tester combinations that are occasionally requested but that no longer represent the best available tools for location of genes to chromosome and that span too long a chromosome region for effective use in detailed mapping.

Listings of catalog stock items change from year to year. Requests should be based on the most recent listing. In making written requests, you should indicate both the code number of a particular stock and its genotype. In some cases, this information allows us to recognize typographical errors directly, and in other instances non-correspondence between these two categories of information alerts us to seek clarification of your intent.

It is sometimes necessary to discontinue providing samples of particular listed items because of inadequate seed supplies or because of errors detected in pedigrees. On these occasions, in filling requests we attempt to substitute stocks with closely similar genotypes.

As you cease active work with particular genetic stocks, and while their viability is still good, we urge you to give timely consideration to whether the materials are sufficiently valuable to merit sending them to the Stock Center for continued preservation. If they involve traits that require special techniques, facilities or skills for classification (e.g., isozyme variants, resistance to pathogens), under our current capabilities it is virtually essential that the stocks be homozygous for the designated alleles so they may be propagated by selfing, sibbing or intercrossing without the necessity for classifying segregating progenies.

E. B. Patterson
S-118 Turner Hall
Agronomy Department
University of Illinois
1102 S. Goodwin Avenue
Urbana, IL 61801
Phone (217)-333-6631 (Lab)

CATALOG OF STOCKS

CHROMOSOME 1

101A *sr1 zb4 P1-WW*
101B *sr1 P1-WR*
101C *sr1 P1-WW*
101D *sr1 P1-RR*
101F *sr1 ts2 P1-RR*
102B *sr1 P1-WR an1 bm2*
102C *sr1 P1-RW ad1 bm2*
102D *sr1 P1-RR ad1 bm2*
103C *sr1 P1-WR bm2*
103D *vp5*
103E *zb4 ms17 P1-WW*
103G *sr1 P1-RR bm2*
104B *zb4 ts2 P1-WW bm2*
105A *zb4 P1-WW*
105E *ms17 P1-WR*
105F *ms17 P1-WW*
106A *zb4 P1-WW bm2*
106B *ts2 P1-RR*
106C *ts2 P1-WW bm2*
107A *P1-CR*
107B *P1-RR*
107C *P1-RW*
107D *P1-CW*
107E *P1-MO*
107F *P1-VV*
107G *P1-OR*
107H *P1-WW*
108C *P1-RR br1 f1 an1 gs1 bm2*
109A *P1-RR an1 ad1 bm2*
109B *P1-RR an1 gs1 bm2*
109D *P1-RR ad1 bm2*
109E *P1-WR br1 f1*
110B *P1-WR an1 Kn1*
110C *P1-WR an1 ad1 bm2*
110D *P1-WR an1 bm2*
110E *P1-WR ad1 bm2*
110F *P1-WR br1 Vg1*
110G *P1-WR br1 f1 gs1 bm2*
110K *P1-WR br1*
111A *P1-WW rs2*
111D *P1-WW hm1 br1 f1*
112B *P1-WW br1 f1 bm2*
112E *as1*
112H *P1-WW br1*
113A *as1 br2*
113B *rd1*
113C *br1 f1*
113E *br1 f1 Kn1*
113K *hm1; hm2*
113L *Hm1; hm2*
114B *br1 f1 Kn1 bm2*
114D *Vg1*
114E *br1 Vg1 f1*
114F *hm1 br2*
115B *Vg1 br2 bm2*
115C *v22*
115D *bz2-m; m A1 A2 C1 Pr1*
115E *br2 Vg1*
116A *bz2-m; M A1 A2 C1 R1 Pr1*
116C *an1 bm2*
116D *an1-bz2-6923* (Df)
116I *bz2 gs1 Ts6 bm2*
117A *br2*
117B *br2 bm2*
117D *tb1 (tb*-8963)*
117E *Kn1*
118A *Kn1 Ts6*
118B *Kn1 bm2*
118C *lw1*
119B *vp8*
119C *gs1*
119D *gs1 bm2*
119E *Ts6*
119F *bm2*
120A *id1*
120B *nec2*
120C *ms9*
120D *ms12*
120F *Mpl1*
121A *ms14*
121C *D8*
121D *Lls1*
121J *ms14 br2*
122A TB-1La
122B TB-1Sb
124 *v*-5688*
124 *j*-5828*
124 *w*-8345*
124 *v*-5588*
124 *w*-018-3*
124 *w*-4791*
124 *w*-6577*
124 *w*-8054*
124 *v*-032-3*
124 *v*-8943*
124 *yg*-8574*

125A *Les2*
127 *bz2 zb7 bm2*
127 *dek1*
127 *dek2*
127 *dek22*
127 *f1*
127 *Msc1*
127 *Tlr1*
128 *ij2*
128 *l16*
128 *l17*
128 *pg15*
128 *pg16*
128 *v25*
128 *w18*
128 *wlu5*
129 *o10*

CHROMOSOME 2

201F *ws3 lg1 gl2 b1*
203B *al1 = y3*
205B *lg1*
205C *lg1 gl2*
206A *lg1 gl2 B1*
206B *lg1 gl2 B1 gs2*
208B *lg1 gl2 B1 sk1*
208D *lg1 gl2 B1 v4*
208E *lg1 gl2 b1*
208F *lg1 gl2 b1 gs2*
208H *gl2*
209B *lg1 gl2 b1 gs2 v4*
209E *lg1 gl2 b1 sk1*
209F *lg1 gl2 b1 sk1 fl1*
210A *lg1 gl2 b1 sk1 v4*
211A *lg1 gl2 b1 fl1*
211D *gl2 b1 wt1*
212B *lg1 gl2 b1 fl1 v4*
212D *lg1 gl2 b1 v4*
212E *lg1 gl2 b1 v4 Ch1*
213A *lg1 gl2 mn1 v4*
213B *lg1 gl2 wt1*
213C *lg1 gl2 w3*
213D *lg1 gl2 w3 Ch1*
213E *lg1 gl2 b1 Ch1*
213F *lg1 B1-V Ch1*
213G *lg1 Ch1*
214C *d5*
214D *B1 gl11*
214E *B1 ts1*
214F *gl2 v4 Ch1*
214G *lg1 gs2 v4*
215B *gl11*
215C *wt1*
215E *fl1*
215G *fl1 v4*
216A *fl1 v4 Ch1*
216D *fl1 w3*
216E *fl1 v4 w3*
216F *fl1 w3 Ch1*
217A *ts1*
217B *v4*
217E *w3 Ht1 Ch1*
217H *ba2 v4*
218A *w3*
218C *w3 Ch1*
218D *Ht1* (source *A1* and *B1*)
218E *ba2*
218F *B1 ba2*
219B *b1("r2"); r1-g A1 A2 C1*
219C *Ch1*
220A *Les1*
220B 2 2T T2/*ws3 lg1 gl2*
(T=*Tripsacum*)
221A *gs2*
221B *B1 gs2*
222A TB-1Sb-2L4464
222B TB-3La-2S6270
223A Primary trisomic 2
224A *w*-4670*
224B *v*-5537*
224F *w*-062-3*
224G *yel*-8630*
224H *whp1; A1 A2 C1 c2 R1*
224J *ij-mos*-7335*
224K *gl-nec*-8495*
227 *dek3*
227 *dek4*
227 *dek16*
227 *dek23*
227 *Les4*
228 *l18*
228 *spt1*
228 *v26*
229 *rf3 Ch1*
229 *v24*

CHROMOSOME 3

301A *cr1*
302A *d1 = d1-6016* (rosette)
302E *d1-tall*
303A *d1 rt1 Lg3*
303B *d1 Rf1 lg2*
303F *g2 = v19 = pg14 = g5*
303G *g2 d1*
304A *d1 ys3*
304B *d1 ys3 Rg1*
304G *Lg3 Rg1*
305A *d1 Lg3*
305D *d1 Rg1*
305K *d1 cl1; Clm4*
307C *pm1*
308A *d1 ts4 lg2 a1-m; A2 C1 R1 Dt1*
308B *d1 ts4*
308C *d1 lg2 a1-m; A2 C1 R1 Dt1*
308E *ra2*
308G *d1 ts4 a1-m; A2 C1 R1 Dt1*
309D *ra2 Rg1 lg2*
309E *ra2 pm1 lg2*
310A *ra2 ts4*
310C *ra2 lg2*
310D *Cg1*
310G *ra2 y10*
310I *Cg1 Lg3*
311A *cl1*
311C *cl1; Clm3*
311D *cl1-p; Clm4*
311E *rt1*
311F *ys3*
311G *Lg3 ys3*
312C *ys3 ts4 lg2*
312D *Lg3*
313A *gl6*
313C *gl6 Lg3 Rg1*
313E *gl6 Lg3*
314F *gl6 Rg1 lg2*
314G *gl6 lg2*
315B *Rg1 gl6*
315D *A1-b(P415)*
316A *ts4*
318A *ig1*
318B *ba1*
318C *w*-7748 = y10*
319C *lg2 a1-m et1; A2 C1 R1 dt1*
319D *lg2 a1-m et1; A2 C1 R1 Dt1*
319F *lg2 a1-st et1; A2 C1 R1 Dt1*
320A *lg2*
320D *A1 sh2; A2 C1 R1 B1 Pl1 dt*
320F *A1 sh2; A2 C1 R1 b1 pl1*
320I *A1 sh2; A2 C1 R1*
321A *A1-d31; A2 C1 R1*
322A *A1-d31 sh2; A2 C1 R1 dt*
322B *A1-d31 sh2; A2 C1 R1 Dt1*
322D *a1; A2 C1 R1 B1 Pl1*
322E *a1-m; A2 C1 R1 B1 Pl1 dt1*
322F *a1-m; A2 C1 R1 b1 pl1 dt1*
322G *a1; A2 C1 C2 R1*
323A *a1-m; A2 C1 R1 Dt1*
323B *a1-m; A2 C1 R1 B1 Pl1 Dt1*
323C *a1-m sh2; A2 C1 R1 B1 Pl1 dt1*
324A *a1-st; A2 C1 R1 Dt1*
324B *a1-st sh2; A2 C1 R1 Dt1*
324E *a1-st et1; A2 C1 R1 Dt1*
324G *a1-st; A2 C1 R1 dt1*
325A *a1-p et1; A2 C1 R1 dt1*
325B *a1-p et1; A2 C1 R1 B1 Pl1 Dt1*
325C *a1-x1*
325D *a1-x3*
325G *a3*
325J *a1-p; A2 C1 R1 Pr1*
326A *sh2*
326B *vp1*
326C *Rp3*
327A TB-3La
327B TB-3Sb
327C TB-3Lc
327D TB-3Ld
328A Primary trisomic 3
329 *v*-9003*
329 *v*-8623*
329 *w*-022-15*
329 *yd2*
329 *w*-8336*
330A *h1*
331A TB-1La-3L5267
331B TB-1La-3L4759-3
331E TB-3Lf
331F TB-3Lg
331H TB-3Li
331I TB-3Lj
331J TB-3Lk
331K TB-3Ll
332 *d2*
332 *dek5*
332 *dek24*
332 *Wrk1*
332 *gl9*
332 *gl19*
332 *dek6*
332 *dek17*
332 *Lxm1*
332 *ms23*

CHROMOSOME 4

401A *Rp4*
401B *Ga1*
401C *Ga1 su1*
401D *Ga1-S*
402A *st1*
402C *st1 fl2*
402D *Ts5*
403A *Ts5 fl2*
404A *Ts5 su1 zb6*
405B *la1*
405D *la1 su1 gl3*
405G *la1 su1 gl4*
406C *fl2*
406D *fl2 su1*
407B *fl2 su1 bm3*
407D *su1*
407E *su1-am*
408B *su1 bm3*
408E *bm3*
408K *su1 se1*
409A *su1 zb6 Tu1*
410D *su1 zb6 gl3*
412C *su1 gl3*
412E *su1 j2 gl3*
413B *su1 gl4*
414A *bt2*
414B *gl4*
414C *gl4 o1*
414E *de** (on Chr. 4 = *de16*?)
415A *j2*
415C *j2 C2; A1 A2 C1 R1*
416A *Tu1*
416B *Tu1-l* 1st
416C *Tu1-l* 2nd
416D *Tu1-d*
416E *Tu1-md*
417A *j2 gl3*
417B *v8*
417C *gl3*
417D *gl3 o1*
418A *gl3 dp1*
418B *c2; A1 A2 C1 R1*
418C *C2; A1 A2 C1 R1*
418D *C2-Idf1* (Active-1); *A1 A2 C1 R1*
418E *dp1*
418F *o1*
418G *v17*
419B *su1 gl3 ra3*
419F *Dt6 gl3; a1-m A2 C1 R1*
420A *Dt4 su1; a1-m A2 C1 R1*
420B TB-9Sb-4L6504
420C *nec*-rd*
420D *yel*-8457*
420I TB-9Sb-4L6222
421A TB-4Sa
421B TB-lLa-4L4692
421C TB-7Lb-4L4698
422A Primary trisomic 4
423A TB-4Lb
423B TB-4Lc
423C TB-4Ld
423D TB-4Le
423E TB-4Lf
427 *dek7*
427 *dek25*
427 *Ysk1*
427 *orp1; orp2*
427 *dek8*
427 *dek10*
427 *Ms41*
427 *dek31*
428 *gl5; gl20*
428 *lw4; lw3*
428 *nec5*
428 *spt2*
428 *wt2*

CHROMOSOME 5

501A *am1 a2; A1 C1 R1*
501B *lu1*
501C *lu1 sh4*
501D *ms13*
501E *gl17*
501H *gl17 a2 bt1; A1 C1 R1*
501I *am1*
502A *gl17 a2 bt1 v2; A1 C1 R1*
502B *A2 vp7 = ps1 pr1; A1 C1 R1*
502D *A2 bm1 pr1; A1 C1 R1*
503A *A2 bm1 pr1 ys1; A1 C1 R1*
503D *A2 bt1 v3 pr1; A1 C1 R1*
504A *A2 bt1 pr1; A1 C1 R1*
504B *A2 bm1 pr1 ys1 v2; A1 C1 R1*
504C *A2 bm1 pr1 zb3; A1 C1 R1*
505B *A2 pr1 ys1; A1 C1 R1*
505C *A2 bt1 ga2 pr1; A1 C1 R1*
505E *A2 v3 pr1 ys1; A1 C1 R1*
506A *A2 v3 pr1; A1 C1 R1*
506B *A2 pr1; A1 C1 R1*
506C *A2 pr1 v2; A1 C1 R1*
506D *A2 na2 pr1; A1 C1 R1*
506F *A2 pr1 v12; A1 C1 R1*
506L *A2 br3 pr1; A1 C1 R1*
507A *a2; A1 C1 R1*
508B *a2 bm1 bt1 pr1 ys1; A1 C1 R1*
508C *a2 bt1 bv1 pr1; A1 C1 R1*
508F *a2 bm1 pr1 ys1; A1 C1 R1*
510A *a2 bm1 pr1 v2; A1 C1 R1*
510B *A2 bm1 pr1 eg1; A1 C1 R1*
510G *a2 bm1 pr1 eg1; A1 C1 R1*
511A *a2 bt1 v3 pr1; A1 C1 R1*
511C *a2 bt1 pr1; A1 C1 R1*
512A *a2 bt1 v2; A1 C1 R1*
512B *a2 v3 pr1; A1 C1 R1*
512C *a2 bt1 ga2 pr1; A1 C1 R1*
513A *a2 pr1; A1 C1 R1*
513C *a2 pr1 v2; A1 C1 R1*
513E *a2 pr1 v12; A1 C1 R1*
513G *a2; A1 C1 R1*
515A *vp2*
515C *vp7 = ps1*
515D *bm1*
516A *bm1 yg1; Ch1*
516B *bt1*
516C *ms5*
516D *td1 ae1*
516G *A2 bm1 pr1 yg1; A1 C1 R1*
516I *td1; Rp1*
517A *v3*
517B *ae1*
518A *sh4*
518B *gl8*
518C *na2*
518D *lw2*
518F *sh4 v2*
518H *gl8 v2*
519A *ys1*
519B *eg1*
519C *v2*
519D *yg1*
519E *A2 pr1 yg1; A1 C1 R1*
519F *A2 pr1 gl8; A1 C1 R1*
520B *v12*
520C *br3*
521A *nec3*
521C *nec*-8624*
521D *nec*-5-9(5614)*
521E *nec*-7476*
521F *nec*-6853*
521G *nec*-7281*
521H *nec*-8376*
521I *v*-6373*
521K *lw3; lw4*
521L *w*-021-7*
522A TB-5La
522B TB-5Lb
522C TB-5Sc
523A Primary trisomic 5
527 *dek18*
527 *dek9*
527 *dek26*
527 *dek27*
527 *grt1*
527 *nec7*
527 *pr1 sh5*

CHROMOSOME 6

601C *rgd1 y1*
601D *rgd1 Y1*
601E *po1 = ms6*
601F *po1 y1 pl1*
601G *po1 y1 Pl1*
602A *po1 y1 wi1*
602K *y1-gbl*
603A *y1 l10*
603B *y1 l11*
603C *y1 l12*
603D *y1 w15*
604A *y1 pb4 pl1*
604B *y1 pb4 Pl1*
604F *y1 ms1-si*
604H *y1 ms1*
604I *Y1 ms1*
605A *y1 wi1 Pl1*
605F *Y1 wi1 pl1*
606A *Y1 pg11; Wx1 pg12*
606B *y1 pg11; wx1 pg12*
606C *Y1 pg11; wx1 pg12*
606D *y1 pg11; Wx1 pg12*
606E *y1 pl1*
606F *y1 Pl1*
607A *y1 Pl1 Bh1; c1 sh1 wx1 A1 A2 R1*
607B *y1 pl1 Bh1; c1 sh1 wx1 A1 A2 R1*
607C *y1 su2*
607D *y1 pl1 su2*
607F *y1 Pl1 su2*
608A *y1 l10*
608G *Y1 l11*
609B *Y1 wi1 pl1*
609C *Y1 wi1 Pl1*
609D *Y1 su2*
609E *ms1-si*
610B *Pl1 Dt2; a1-m A2 C1 R1*
610C *pl1 sm1; P1-RR*
610H *Y1 Dt2 pl1; a1-m A2 C1 R1*
611A *Pl1 sm1; P1-RR*
611D *Pt1*
611E *wl*
611F *Pl1 sm1 Pt1; P1-RR*
611H *py1*
612A *w14*
612B *ms6*
612C *l*-4923*
612D *oro1*
613A 2NOR; *a2 bm1 pr1 v2; A1 C1 R1*
613F *whs*-8613*
613L *w*-8954*
613M *yel*-039-13*
613R *wh*-8889*
613T *pg*-6656* (= *pg11 pg12*)
613U *wh*-8624*
614A TB-6Lb
614B TB-6Sa
614C TB-6Lc
615A Primary trisomic 6
627 *dek28*
627 *dek19*
627 *vp*-5111*

CHROMOSOME 7

701B *In1-D*
701D *a2*
702B *o2 v5 ra1 gl1*
702E *o2 v5 ra1 gl1 ij1*
703A *o2 v5 gl1*
704B *o2 ra1 gl1 sl1*
704C *o2 v5 gl1 sl1*
705A *o2 gl1*
705B *o2 gl1 sl1*
705C *o2 ij1*
705D *o2 bd1*
706E *v5 gl1 Tp1*
707A *y8 v5 gl1*
707B *in1; A2 pr1 A1 C1 R1*
707D *v5*
707E *vp9*
707F *y8 gl1*
708A *ra1*
708G *y8*
709A *gl1*
710H *ms7 gl1 Tp1*
711A *Tp1*
711B *ij1*
712A *ms7*
713A *Bn1*
713B *bd1*
714B *a5*
714D *va1*
715A *Dt3; a1-m A2 C1 R1*
715C *gl1 Dt3; a1-m A2 C1 R1*
716A *v*-8647*
716B *yel*-7748*
716F *Les9*
716G *y8*
717A TB-7Lb
718A Primary trisomic 7
727 *dek11*
727 *wlu2*

CHROMOSOME 8

801A *gl18*
801B *v16*
801C *v16 j1*
801D *v16 ms8 j1*
801G *v16 gl18*
803A *ms8*
803B *nec1*
803D *ms8 gl18*
804A *v21*
804D *wh*-053-4*
804E *w*-017-4*
804F *w*-034-16*
804G *w*-8635*
804H *w*-8963*
805A *fl3*
805C *gl18 v21*
805D *fl3 ms8 j1*
805E *el1*
805F *gl18 v16 ms8*
806A TB-8La
806B TB-8Lb
808 *ct1*
809A TB-8Lc
827 *dek20*
827 *dek29*
827 *Bif1*
827 *Sdw1*
827 *Clt1*

CHROMOSOME 9

901D *yg2 C1-I sh1 bz1 wx1; A1 A2 R1*
901E *yg2 C1 bz1 wx1; A1 A2 R1*
902A *yg2 c1 sh1 bz1 wx1; A1 A2 R1*
902B *yg2 c1 sh1 wx1; A1 A2 R1*
902C *yg2 c1 sh1 wx1 gl15; A1 A2 R1*
902D *K-S9 yg2 c1 sh1 wx1 gl15; A1 A2 R1*
902E *yg2 c1 bz1 wx1; A1 A2 R1*
903A *C1 sh1 bz1; A1 A2 R1*
903B *C1 sh1 bz1 wx1; A1 A2 R1*
903D *C1-I sh1 bz1 wx1; A1 A2 R1*
904B *C1 sh1; A1 A2 R1*
904C *C1 sh1 wx1; A1 A2 R1*
904D *C1 wx1 ar1; A1 A2 R1*
905A *K-L9 C1 sh1 wx1; A1 A2 R1*
905B *C1 sh1 ms2; A1 A2 R1*
905C *C1 bz1 Wx1; A1 A2 R1*
905D *K-L9 C1 sh1 wx1; K10 A1 A2 R1*
905E *C1 sh1 wx1 v1; A1 A2 R1*
906A *C1 Ds wx1; A1 A2 R1 Pr1 y1*
906B *C1 Ds wx1; A1 A2 R1 pr1 Y1*
906C *C1-I Ds Wx1; A1 A2 R1*
906D *C1-I; A1 A2 R1*
906G *C1-I Ds wx1; A1 A2 R1*
907A *C1 wx1; A1 A2 R1*
907D *C1 wx1; A1 A2 R1 B1 pl1*
907E *C1-I wx1; A1 A2 R1 y1*
907G *C1-I(p); A1 A2 R1 B1-b pl1*
907H *C1-I(m); A1 A2 R1 b1 pl1*
908B *C1 wx1 v1; A1 A2 R1*
908D *C1 wx1 gl15; A1 A2 R1*
908E *C1 wx1 gl15; A1 A2 R1 pr1*
908F *C1 wx1 da1; A1 A2 R1*
908H *C1 wx1; y1 A1 A2 R1*
909A *C1 wx1 Bf1; A1 A2 R1*
909B *c1 bz1 wx1; A1 A2 R1*
909C *c1 sh1 bz1 wx1; A1 A2 R1 y1*
909D *c1 sh1 wx1; A1 A2 R1*
909E *c1 sh1 wx1 v1; A1 A2 R1*
909F *c1 sh1 wx1 gl15; A1 A2 R1*
910B *c1 sh1 wx1 gl15 Bf1; A1 A2 R1*
910C *c1 sh1 wx1 bk2; A1 A2 R1*
910D *c1; A1 A2 R1*
910G *sh1-bz1-x2 Wx1; A1 A2 C1 R1*
911A *c1 wx1; A1 A2 R1 y1*
911B *c1 wx1 v1; A1 A2 R1*
911C *c1 wx1 gl15; A1 A2 R1*
911D *c1 wx1 Bf1; A1 A2 R1*
912A *sh1*

912B *sh1 wx1 v1*
913A *sh1 wx1*
913C *sh1 l7*
914A *wx1 d3*
914E *Wx1 pg12; y1 pg11*
914F *wx1 pg12; y1 pg11*
914G *Wx1 pg12; Y1 pg11*
914H *wx1 pg12; Y1 pg11*
915A *wx1* (Other alleles from O. Nelson avail.)
915B *wx1-a*
915C *wl1*
916A *wx1 v1*
916C *wx1 bk2*
917A *wx1 Bf1*
917C *v1*
917D *ms2*
917E *gl15*
917F *d3*
918A *gl15 Bf1*
918D *Wc1*
918E *Wx1 bk2 bm4*
918F *Wx1 Bf1*
918G *Wc1-Wh Bf1 bm4*
918H *Wc1 bm4*
918I *Wx1 bk2*
919A *bm4*
919B *Bf1 bm4*
919C *l6*
919D *l7*
920A *yel*-034-16*
920B *w*-4889*
920C *w*-8889*
920E *w*-8950*
920F *w*-9000*
920G Tp9 N9 N3 Df3
920L *yg-zb*-5588*
920M *w-nl*-034-5*
921A TB-9La
921B TB-9Sb
921C TB-9Lc
921D TB-9Sd
922A Primary trisomic 9
924A *wd1* and Ring9 *C1-I; A1 A2 R1*
927 *dek12*
927 *dek13*
927 *dek30*
927 *Les8*
927 *Zb8*
927 *Dt7; a1-r; A2 C1 R1*
928 *v28*
928 *wlu4*
928 *C1 wx1 Bf1; A1 A2 r1*
930C *wx1 ms2 Bf1; A1 A2 r1*

CHROMOSOME 10

X01A *oy1*
X01B *oy1 R1; A1 A2 C1*
X01C *oy1 bf2*
X01E *oy1 bf2 R1; A1 A2 C1*
X02G *oy1 zn1*
X02I *oy1 bf2 ms10*
X02K *oy1 zn1*
X03A *sr3*
X03B *Og1*
X04A *Og1 du1 R1; A1 A2 C1*
X04B *ms11*
X04C *ms11 bf2*
X04D *bf2*
X05A *bf2 zn1*
X05E *bf2 sr2*
X06C *nl1 g1 R1; A1 A2 C1*
X07C *y9*
X07D *nl1*
X09B *li1 g1 R1; A1 A2 C1*
X09F *ms10*
X10A *du1*
X10D *du1 g1 r1; A1 A2 C1*
X10F *zn1*
X10G *du1 v18*
X11A *zn1 g1*
X11F *g1 r1; A1 A2 C1*
X11H *zn1 R1-r; A1 A2 C1*
X12A *g1 r1 sr2*
X12E *g1 R1; A1 A2 C1*
X13D *g1 r1-r sr2; A1 A2 C1*
X13H *r1-g wx1; A1 A2 C1 y1*
X13I *r1-g Wx1; A1 A2 C1 y1*
X14A *Isr1 r1-r; A1 A2 C1*
X14F *v18 r1; A1 A2 C1*
X14G *v18 r1 sr2; A1 A2 C1*
X15C *R1-g; A1 A2 C1*
X15D *r1-ch; Pl1 A1 A2 C1*
X16B *r1 K10; A1 A2 C1*
X16C *R1-ch; A1 A2 C1 B1 pl1*
X16D *r1 sr2; A1 A2 C1*
X17A *r1-g; A1 A2 C1*
X17B *r1-r; A1 A2 C1*
X17C *R1-mb; A1 A2 C1*
X17D *R1-nj; A1 A2 C1*
X17E *R1-r; A1 A2 C1*
X17F *R1-nj* purple embryo Chase; *A1 A2 C1*
X18A *R1-lsk; A1 A2 C1*
X18C *R1-st; A1 A2 C1*
X18D *R1-sk; A1 A2 C1*
X18E *R1-st Mst1*
X18G *R1-scm2; bz2 A1 A2 C1 C2*
X18H *R1-nj; A1 A2 C1 bz2*
X19A *Lc1*
X19B *w2*
X19C *w2 l1*
X19D *o7*
X20B *l1*
X20C *v18*
X20F *yel*-8721*
X21A TB-10La
X21B TB-10L19
X21C TB-10Lb
X22A TB-10Sc
X23A Primary trisomic 10
X24A *cm1*
X24 *nec*-4889*
X24 *nec*-5876*
X24 *wh*-7165*
X24 *yel-gr*-8631*
X24 *wh*-8129*
X25A *R1-scm2; a1-st A2 C1 C2*
X25B *R1-scm2; c2 A1 A2 C1*
X25C *R1-scm122; pr1 A1 A2 C1 C2*
X25D *R1-scm2; a2 A1 C1 C2*
X25E *R1-scm2; c1 A1 A2 C2*
X26A *r1-x1; A1 A2 C1*
X27 *dek14*
X27 *dek15*
X27 *dek21*
X27 *Les6*
X27 *gl21*
X27 *Vsr1*
X27 *Oy*-700*
X27 *orp2; orp1*
X27 *l19*

UNPLACED GENES

U140 *dv1*
U140 *dy1*
U140 *l4*
U141 *ms22*
U141 *ms24*
U141 *o9*
U141 *o11*
U142 *o12*
U142 *o13*
U142 *rd3*
U142 *ts*-br*
U142 *ub1*
U142 *y11*
U142 *y12*
U240 *Les7*

MULTIPLE GENE STOCKS

M141A *A1 A2 C1 C2 R1-g Pr1 B1 Pl1*
M141B *A1 A2 C1 C2 R1-g Pr1 B1 pl1*
M141C *A1 A2 C1 C2 R1-g b1 Pl1*
M141D *A1 A2 C1 C2 R1-g b1 pl1*
M241A *A1 A2 C1 C2 r1-g Pr1 B1 Pl1*
MX17A *A1 A2 C1 C2 r1-g Pr1 b1 pl1*
M241B *A1 A2 C1 C2 r1-g Pr1 B1 pl1*
M340A *A1 A2 c1 C2 R1-g Pr1 B1 pl1*
M241C *A1 A2 C1 C2 R1-r Pr1 B1 Pl1*
M341B *A1 A2 C1 C2 R1-r Pr1 B1 pl1*
M341C *A1 A2 C1 C2 R1-r Pr1 b1 Pl1*
M341F *A1 A2 C1 C2 R1-r Pr1 b1 pl1*
M441A *A1 A2 C1 C2 R1-r Pr1 B1 Pl1 wx1*
M441B *A1 A2 C1 C2 R1-r Pr1 B1 pl1 wx1*
M441F *A1 A2 C1 C2 R1-g Pr1 b1 pl1 wx1*
M541 *A1 A2 C1 C2 R1 Pr1*
M641A *A2 C1 C2 R1 Pr1 wx1*
M641D *A1 A2 C1 C2 r1 Pr1 y1 wx1*
MX41A *A1 A2 C1 C2 R1 pr1 y1 wx1 gl1*
M941A *A1 A2 c1 C2 R1 Pr1 y1 wx1*
M341D *A1 A2 c1 C2 R1-r Pr1 B1 Pl1*
M341E *A1 A2 c1 C2 R1-g Pr1 b1 pl1*
M441D *A1 A2 C1 C2 r1-r Pr1 B1 Pl1*
M441E *A1 A2 c1 C2 r1-r Pr1 B1 Pl1*
MX41B *su1 pr1 y1 gl1 wx1; A1 A2 C1 C2 R1*
M841A *A1 su1 pr1; A2 C1 C2 R1*
MX41C *bz2 a1 c2 a2 pr1 Y1/y1 c1 bz1 wx1 r1*
M841B *a1 su1 A2 C1 C2 R1*
MX40A *bm2 lg1 a1 su1 pr1 y1 gl1 j1 wx1 g1*
M841C colored scutellum; *A1 A2 C1 C2 R1 Pr1*
M841E colored scutellum; *A1 A2 C1 C2 R1 pr1*
MX41D *a1 su1 pr1 y1 gl1 wx1 A2 C1 C2 R1*
M741C Stock 6 : Hi-haploid *R1-r B1 Pl1*
M741 Stock 6: Hi-haploid *A1 C1 R1-g* colored scutellum
M741 Stock 6: Hi-haploid *y1 C1-I wx1 A1 R1-g*

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
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
E642B Gourdseed
E642C Knobless Wilbur's Flint
E742A Maiz Chapalote
E742B Papago Flour Corn
E742C Parker's Flint
E842A Tama Flint
E842B Zapalote Chico
E942A Winnebago Flint
E942B Missouri Cob Corn

TETRAPLOID STOCKS
N103A *P1-RR*
N103D *P1-WR*
N104B *pr1; A1 A2 C1 R1*
N107C Synthetic B
N107B W23 conversion
N104C *su1 wx1*
N106D *sh1 Wx1 ; Y1*
N106E *sh1 wx1 ; y1*

CYTOPLASMIC TRAITS

C337 NCS2
C337 NCS3

CYTOPLASMIC STERILES AND RESTORERS

C836A WF9-(T) *rf1 rf2*
C836B WF9 *rf1 rf2*
C736A R213 *Rf1 rf2*
C736C B37 *rf1 Rf2*
C736D N6 *rf1 Rf2*
C736B Ky21 *Rf1 Rf2*

WAXY RECIPROCAL TRANSLOCATIONS*

wx01A *wx1* T1-9c (1S.48; 9L.22)
wx01B *wx1* T1-9(5622) (1L.10; 9L.12)
wx03A *wx1* T1-9(8389) (1L.74; 9L.13)
wx04A *wx1* T2-9c (2S.49; 9S.33)
wx05A *wx1* T2-9b (2S.18; 9L.22)
wx06A *wx1* T2-9d (2L.83; 9L.27)
wx07A *wx1* T3-9(8447) (3S.44; 9L.14)
wx08A *wx1* T3-9c (3L.09; 9L.12)
wx10A *wx1* T4-9e (4S.53; 9L.26)
wx11A *wx1* T4-9g (4S.27; 9L.27)
wx12A *wx1* T4-9(5657) (4L.33; 9S.25)
wx13A *wx1* T4-9b (4L.90; 9L.29)
wx15A *wx1* T5-9(4817) (5L.06; 9S.07)
wx16A *wx1* T5-9d (5L.14; 9L.10)
wx17A *wx1* T5-9a (5L.69; 9S.17)
wx18A *wx1* T6-9(4778) (6S.80; 9L.30)
wx20A *wx1 y1* T6-9b (6L.10; 9S.37)
wx21A *wx1* T6-9(4505) (6L.13; 9ctr.)
wx22A *wx1* T7-9(4363) (7ctr.; 9ctr.)
wx23A *wx1* T7-9a (7L.63; 9S.07)
wx24A *wx1* T8-9d (8L.09; 9L.16)
wx25A *wx1* T8-9(6673) (8L.35; 9S.31)
wx26A *wx1* T9-10(8630) (9S.28; 10L.37)
wx27A *wx1* T9-10b (9S.13; 10S.40)
wx28A *wx1* T5-9(8386) (5L.87; 9S.13)

NON-WAXY RECIPROCAL TRANSLOCATIONS*

Wx30A *Wx1* T1-9c (1S.48; 9L.22)
Wx30B *Wx1* T1-9(4995) (1L.19; 9S.20)
Wx30C *Wx1* T1-9(8389) (1L.74; 9L.13)
Wx31A *Wx1* T2-9c (2L.49; 9S.33)
Wx31B *Wx1* T2-9b (2S.18; 9L.22)
Wx32A *Wx1* T3-9(8447) (3S.44; 9L.14)
Wx32B *Wx1* T3-9(8562) (3L.65; 9L.22)
Wx32C *Wx1* T3-9c (3L.09; 9L.12)
Wx33A *Wx1* T4-9e (4S.53; 9L.26)
Wx33B *Wx1* T4-9(5657) (4L.33; 9S.25)
Wx33C *Wx1* T4-9g (4S.27; 9L.27)
Wx34A *Wx1* T5-9c (5S.07; 9L.10)
Wx34B *Wx1* T5-9(4817) (5L.06; 9S.07)
Wx34C *Wx1* T4-9b (4L.90; 9L.29)
Wx35A *Wx1* T5-9(8386) (5L.87; 9S.13)
Wx35B *Wx1* T5-9a (5L.69; 9S.17)
Wx35C *Wx1* T5-9d (5L.14; 9L.10)
Wx36A *Wx1* T6-9(4778) (6S.80; 9L.30)
Wx37A *Wx1* T6-9(8768) (6L.89; 9S.61)
Wx37B *Wx1* T7-9(4363) (7ctr.; 9ctr.)
Wx37C *Wx1* T6-9(4505) (6L.13; 9ctr.)
Wx38A *Wx1* T7-9a (7L.63; 9S.07)
Wx38B *Wx1* T8-9d (8L.09; 9L.16)
Wx38C *Wx1* T8-9(6673) (8L.35; 9S.31)
Wx39A *Wx1* T9-10(8630) (9S.28; 10L.37)
Wx39B *Wx1* T9-10b (9S.13; 10S.40)

* = Single cross of homozygotes between M14 and W23 versions will be supplied if available

INVERSIONS

I143A Inv1a (1S.86-L.50)
I143B Inv1c (1S.35-L.01)
I143C Inv1d (1L.55-L.92)
I143D Inv1(5131-10) (1L.46-L.82)
I444A Inv2a (2S.70-L.80)
I243A Inv2(8865) (2S.06-L.05)
I243B Inv2(5392-4) (2L.13-L.51)
I343A Inv3a (3L.38-L.95)
I343B Inv3L (3L.19-L.72)
I343C Inv3(3716) (3L.09-L.81)
I443A Inv4b (4L.40-L.96)
I443B Inv4c (4S.86-L.62)
I543A Inv4e (4L.16-L.81)
I743A Inv5(8623) (5S.67-L.69)
I743B Inv6(8452) (6S.77-L.33)
I843A Inv6(8604) (6S.85-L.32)
I743C Inv6(3712) (6S.76-L.63)
I943A Inv7(5803) (7L.17-L.61)
I943B Inv7(8540) (7L.12-L.92)
I943C Inv7(3717) (7S.32-L.30)
IX43A Inv8a (8S.38-S.15)
I344A Inv9a (9S.70-L.90)
IX43B Inv9b (9S.05-L.87)
IX43C Inv9c (9S.10-L.67)

Cooperators (that means you) need the Stock Center.

The Stock Center needs Cooperators (this means you) to:

(1) Send stocks of new factors that you have reported in this News Letter or in publications, and stocks of new combinations, to the collection.

(2) Inform the Stock Center on your experience with materials received from the collection.

(3) Acknowledge the source of the stocks for research when you publish, and advice or help you have received in development of your research project.

V. GENE LIST AND WORKING MAPS

A list of defined genes for maize follows. The table includes the symbol for the locus, the chromosome (L=long arm, S=short arm) and map location, name and phenotype, availability from the Stock Center (S), photograph (P) in Mutants of Maize (Neuffer et al. 1968), and references to original descriptions. Stocks may be obtained from the Maize Genetics Stock Center (see the preceding section); in many instances variants (e.g., isozymes) exist inherently among generally available strains.

Following the gene list is a new table of mapped RFLP loci. The locations given ARE NOT DIRECTLY CROSS-REFERRABLE between maps. Locations were taken from the BNL and UMC maps beginning with 0 at the most distal marker on the short arm and summing shortest intervals; for others, recent available maps were measured or approximated and subjective judgments were applied when necessary (probe availability was not considered). The BNL and NPI locations were derived with data from one set of 89 recombinant inbreds (RIs) from 2 pedigrees (49 from CM37/T202, 41 from Tx303/CO159; see MNL65 article by Burr et al.); UMC '89 locations with 46 mortal F2 individuals from Tx303/CO159; UMC'91 locations with an Immortal F2 (IF2) of 56 individuals from Tx303/CO159 (see MNL65 article by Gardiner et al.); PIO locations with combined F2s from four hybrids; AGR locations with F2s from A619/Mangelsdorf's Tester. This table was prepared on a short time scale (with little consultation) to simplify "lookups" of potential markers by us and other Cooperators, and to help us in compiling the integrated maps of genes; please treat it as TENTATIVE, pending availability of explicit supporting data.

NOMENCLATURE: New definitions of standards and criteria are needed, but meanwhile every attempt has been made to avoid ambiguities, to honor the Rules of Nomenclature (MNL 49:3-4; see also MNL 61:49), and to retain the distinction between genes defined by genetic vs.molecular criteria. Please inform us of any errors, inconsistencies, or potential flaws in the specific symbols or the standards applied. The terms "homolog" or "candidate" are used whenever a conservative designation appears appropriate.

The working maps follow the lists. The traditional linkage map, based on recombinational analyses of Mendelizing variations in an expression or a gene product, is in the center. Each map represents the order and distances in centimorgans (1% recombination = 1 cM), for loci for which sufficient information is available to make a reasonable judgment of location. Each chromosome begins at the top with the most distal locus known 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, immediately 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 B-A translocations, which generate hemizygous segments, are shown as TB-..., and A-A translocations as T with chromosome numbers and identifiers (see MNL 52:129ff., 55:140ff., 59:159ff., 60:149ff.); placement on the physical map is in accordance with observed breakpoints; placement on the linkage map is in relation to cytogenetic mapping data. 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 spanning one or more genes may or may not uncover the indicated gene or genes. To the right of the linkage map are shown genes (alphabetically in groups) for which a "rough" placement has been defined, either near a gene already on the map or to a region of the map. Furthest to the right are shown genes placed only to chromosome (vertical line with arrows at both ends) or to one arm (vertical line from near the centromere to the end of the arm).

To the left of each physical map are the RFLP maps developed at Brookhaven (left) and at Missouri (right). Preliminary localizations of conventional markers are shown adjacent to the RFLP maps; horizontal ticks indicate the RFLP loci used in mapping these genes.

The Integrated Mapping Project, developed under the encouragement, advice and efforts of the maize community, is in the third year of support from NSF. The priorities under this support are (1) to set a universally usable framework of RFLP markers (see the Core Map in MNL65); (2) to define physical locations with translocations; and (3) to map a selected group of markers (see notes in MNL 64). We emphasize that this effort in no way decreases the need for others to map (either traditionally or with RFLPs). This project provides a means by which data can be assembled and distributed to all interested research workers.

The importance of placing loci defined by probes of known function cannot be overstressed. In a number of cases these give very accurate ties to the conventional map and, in the very least, provide functional significance to a particular region of the genome that will be important as further additional studies (particularly in the area of quantitative genetics) progress. Therefore, if you have a clone for a known function and know or believe that it hybridizes to a maize genomic sequence, please attempt to map the locus (or loci). This can be accomplished in a couple of ways (and we recommend doing both). First, the set of recombinant inbreds should be probed and the data sent to Ben Burr for analysis. Second, it would be appreciated if the probe could be sent to Missouri for mapping in the Immortal F2 population. We would also use the probe in correlation to physical and conventional markers. We have included in this Newsletter a sample form of the desired information for each clone you provide. If you have any questions regarding mapping of RFLP loci (both old and new), please call or write.

As usual, any comments and/or changes to the maps and lists are greatly appreciated.

Ed Coe, Gerry Neuffer, Dave Hoisington and Shiaoman Chao

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
a1	3L-149.0	anthocyaninless: colorless aleurone, green or brown plant, brown pericarp with *P1-RR*; for alleles and interactions, see Coe et al., 1988; dihydroflavonol reductase; *BNL#-A1*(pAmu2), *NPI51-A1*(), *NPI467-A1*()	S	P	93
A1(2)	1L	anthocyanin homolog: *NPI482-A1*()			422
A1(3)	2L	anthocyanin homolog: *NPI468-A1*()			422
A1(4)	5L	anthocyanin homolog: *NPI469-A1*()			422
A1(5)	7S	anthocyanin homolog: *NPI470-A1*()			422
A1(6)	8L	anthocyanin homolog: *UMC189A-A1*()			122
a2	5S-35	anthocyaninless: like *a1*, but red pericarp with *P1-RR*; flavanone-3-hydroxylase or a dehydrase/oxidase	S	P	170
a3	3L-132	anthocyanin: recessive intensifier of expression of *R1* and *B1* in plant tissues	S	P	215
abp1	3L-near *Rg1*	auxin receptor binding protein candidate (was *axr1*): *BNL#-ABP*(cDNA probeG100)			400
Ac		activator: designator for autonomous transposable elements; regulates *Ds* transposition and dissociation; ex. *Ac9* designates element isolated from *wx1-m9*		P	239
Ac2		activator: similar to *Ac*			75

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Acc1	?	acetyl-coenzyme A carboxylase (EC6.4.1.2): tissue-culture selected resistance to cyclohexanedione (e.g., sethoxydim) and aryloxy phenoxypropionate (e.g., haloxyfop) herbicides; ACCase altered			300
Aco1	4S	aconitase: electrophoretic mobility; monomeric			412
Aco2	?	aconitase: electrophoretic mobility			412
Aco3	?	aconitase: electrophoretic mobility			412
Aco4	?	aconitase: electrophoretic mobility; monomeric			412
Acp1	9L	acid phosphatase (was *Ap1*, *Acph1*, *Phos*): electrophoretic mobility; cytosolic; dimeric			88 140
Acp2	?	acid phosphatase (was *Ap2*): electrophoretic mobility; dimeric			88 140 182
Acp4	1L-176	acid phosphatase: electrophoretic mobility; monomeric			182
Act1	8	actin family: *BNL#-ACT*(pMAcI), *NPI368-ACT*()			43 357
ad1	1L-108	adherent: seedling leaves, tassel branches, and occasionally top leaves adhere	S	P	185
Adh1	1L-128	alcohol dehydrogenase: electrophoretic mobility; null allele is known; dimeric; intra/interlocus hybrid bands occur; *BNL#-ADH1*(pH2.3), *NPI21-ADH*()			356
Adh2	4S-46	alcohol dehydrogenase: electrophoretic mobility; null allele is known; dimeric; intra/interlocus hybrid bands occur; *BNL#-ADH2*(pZML841), *NPI228-ADH2*()			354
Adk1	6S-0	adenylate kinase: electrophoretic mobility; plastidial			413
Adp1	1L-near *f1*	ADP glucose pyrophosphorylase candidate: *BNL17.15-BT2*(bt2), *NPI309-ADP*()			422
Adp2		(= *Agp2*)			
Adp3		(see *Bt2*)			
Adr1	?	alcohol dehydrogenase regulator			197
ae1	5L-57	amylose extender: glassy, tarnished endosperm; high amylose content; endosperm-specific starch branching enzyme IIb	S	P	409
afd1	?	absence of first division: male and female sterility; anaphase I equatorial			136
Agp1	6L-near *w14*	ADPG pyrophosphorylase: embryo-specific; *BNL#-AGP1*(cDNA probe pBS.ABP)			44
Agp2	2L-near *v4*	ADPG pyrophosphorylase: embryo-specific; *BNL#-AGP2*(), *NPI310-ADP*(cDNA probe pBS.ABP)			44
AGR		Agrigenetics: designator for loci defined by restriction fragment polymorphisms			
agt1	?	agravitropic: primary root unresponsive to gravity			83
al1	2S-4	albescent plant: variably cross-banded to white leaves; pale yellow endosperm, some alleles viviparous (see Coe et al., 1988)	S	P	309
alh1	1L-near *bm2*	histone Ia (was H1a): electrophoretic mobility			392
Alpha		*A1* locus component (see *Beta*): determines reduced aleurone and plant color, brown pericarp			203
Alr1	2L	aleurain homolog: *NPI348-ALR*()			423
Alr2	7L	aleurain homolog: *NPI349-ALR*()			423
Als1	4L-near *orp1*	acetolactate pyruvate lyase candidate: *BNL#-ALS*()			44
Als2	5L-near *bv1*	acetolactate pyruvate lyase candidate: *BNL#-ALS*()			44
am1	5S-20	ameiotic: male and female sterility; anaphase I equatorial	S	P	298 325
Amp1	1L-near *f1*	aminopeptidase: electrophoretic mobility; cytosolic; monomeric			296
Amp2	1-near *hm1*	aminopeptidase: electrophoretic mobility; monomeric			296
Amp3	5S-near *a2*	aminopeptidase: electrophoretic mobility; monomeric			296
Amp4	?	aminopeptidase: electrophoretic mobility; monomeric			296
Amy1	?	alpha amylase: electrophoretic mobility; monomeric			49
Amy2	5S-near *Mdh5*	beta amylase: electrophoretic mobility; monomeric			48
an1	1L-104	anther ear: andromonoecious dwarf, intermediate stature; few tassel branches; responds to gibberellins; *an1-6923* includes deletion of *Bz2*	S	P	90 100
anl1	5S-near *lu1*	anthocyaninless lethal: colorless aleurone; small kernels; embryo lethal			56
Ant1	5L	adenine nucleotide translocator candidate: *UMC142-ANT*(pZmc-ATP-01)			12
Ant2	?	adenine nucleotide translocator candidate (cDNA probe)			13
aph1	?	aphid resistance			47
ar1	9L-62	argentia: virescent seedling, greens rapidly; husk leaf tips striped	S	P	104
ARS		designator for autonomously replicating sequences			
as1	1-56	asynaptic: synaptic failure in male and female	S	P	20
Ask1	-near *o2*	aspartate kinase (was LT19, *Ltr*-1*, Lt1a): lysine-threonine resistance in cultures and seedlings, increased threonine in kernels			76
Ask2	?	aspartate kinase (was LT20): lysine-threonine resistance			76
Asr1	4S-19	absence of seminal roots			253
Atc1		(see *Zb8*)			
atn1	?	anaerobic tolerant null: enhances survival of ADH-null under anoxia			204
Atp2	?	ATPase candidate: F1 ATPase subunit 2 (cDNA probe)			87
ats1	8	atrazine susceptible: lacks glutathione S-transferase			144
B chr		B chromosome: supernumerary chromosome	S	P	319
b-32		(see *pro1*)			
B1	2S-49	colored plant: anthocyanin in major plant tissues; some alleles affect aleurone and embryo color (for alleles, see Coe et al., 1988); regulates flavonoid enzymes; *NPI#-B1*()	S	P	96
ba1	3L-102	barren stalk: ear shoots and most tassel branches and spikelets absent	S	P	154
ba2	2-near *ts1*	barren stalk: like *ba1*, but tassel more normal	S		154
ba3	?	barren stalk			299
baf1	9S-near *w11*	barren stalk fastigiate (was *ba*-s*): ear shoots often absent; tassel branches erect			57
bd1	7L-109	branched silkless: ear silkless, branched at base; tassel proliferated, bushy	S	P	188
beta		*A1* locus component (see *alpha*): determines aleurone and plant color, red pericarp			203
Bf1	9L-137	blue fluorescent: homozygous seedlings (homozygous or heterozygous anthers) fluoresce blue under ultraviolet; anthranilic acid present	S	P	399
bf2	10L-33	blue fluorescent: similar to *Bf1* in expression; shows earlier, stronger seedling fluorescence	S		5
Bg		Bergamo: regulatory element mediating *o2-mr*			340
Bh1	6L-50	blotched: colored patches on colorless (*c1*) aleurone	S	P	95
Bif1	8	barren inflorescence (was *Bif*-1440*): ear and tassel have many fewer spikelets, bare rachis appendages	S		284
bk2	9L-82	brittle stalk: brittle plant parts after 4-leaf stage	S	P	200
Blh1	1S	bleached (was Bleached-1593): pale green midveins and base in upper leaves			280
bm1	5S-41	brown midrib: brown pigment over vascular bundles of leaf sheath, midrib, and blade	S	P	103
bm2	1L-161	brown midrib: like *bm1*	S		42
bm3	4-near *bt2*	brown midrib: like *bm1* (C.R. Burnham, 1935, unpublished data)	S		101 194

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
bm4	9L-141	brown midrib: like *bm1*	S		39
Bn1	7L-71	brown aleurone: yellowish brown aleurone color	S		196
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	184 187
br2	1L-near *hm1*	brachytic: like *br1*	S		206
br3	5	brachytic: like *br1*	S		369
brn1	3S-19	brown aleurone: brown kernel, brown embryo; seedling lethal			337
Bs-1		barley stripe: transposable element, retrovirus-like; 1-5 copies in genome			175
bs1	?	barren sterile			245
bt1	5L-42	brittle endosperm: mature kernel collapsed, angular, often translucent and brittle; affects starch-granule-bound phospho-oligosaccharide synthase; *BNL#-BT*(cDNA)	S	P	226 416
bt2	4S-67	brittle endosperm: like *bt1*; endosperm ADP glucose pyrophosphorylase subunit; (compare *sh2*) (G.F. Sprague, 1935, unpublished data); *BNL#-BT2*(cDNA), *NPI314-ADP*	S		101 398
btn1	?	brittle node			183
bu1	?	leaf burn: leaves show burning, sometimes horizontal bands, accentuated by high temperature			121
bv1	5L-47	brevis plant: short internodes, short plant	S		208
bv2	?	brevis plant: plant height 30-50% of normal			310
bx1	4S	benzoxazinless: absence of cyclic hydroxamates (blue color in crushed root tip with FeCl3), which inhibit *Ostrinia nubilalis* and *Helminthosporium turcicum*			67
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*; *AGR#-BZ1*(), *BNL#-BZ1*(pMBzPA), *NPI8-BZ1*(), *UMC192-BZ1*(pbz)	S	P	324
bz2	1L-106	bronze: like *bz1*; anthers not fluorescent; *an1-6923* mutation includes deletion for *Bz2*; potential function flavonoid acylation, glycosylation, transport, or deposition; *BNL#-BZ2*(pP300), *UMC181-BZ2*(pP300)	S	P	294
C-bands		brightly stained regions (bands) on chromosomes with Giemsa stain; correspondence to knobs (see K)			1 410
C1	9S-26	colored aleurone: *c1* colorless; *C1-I* dominant colorless; *c1-p* pigment inducible by light (see Coe et al., 1988); regulates flavonoid enzymes; *BNL#-C1*(pEco1.0)	S	P	85
c2	4L-117	colorless: colorless aleurone, reduced plant color; chalcone synthase; *C2-Idf* dominant inhibitor (see Coe et al., 1988); duplicate factor with *whp1* for pollen color and for anthocyanins; *BNL#-C2*(pC2-c46)	S	P	143
Cab1	3L-near *Mdh3*	chlorophyll a/b binding protein candidate: *NPI477-CAB*()			422
Cab2	7L	chlorophyll a/b binding protein candidate: *NPI478-CAB*()			422
Cab3	7L	chlorophyll a/b binding protein candidate: *NPI478-CAB*()			422
Cab4	8L	chlorophyll a/b binding protein candidate: *NPI479-CAB*(); *UMC74-CABM2*(8-22)			422
Car1	1S	catalase regulator: enzyme activity level increased			346
Cat1	5S-near *Mdh5*	catalase: electrophoretic mobility; cytosolic/glyoxysomal; tetrameric; intra/interlocus hybrid bands occur			23
Cat2	1S	catalase: electrophoretic mobility; null allele is known; cytosolic/glyoxysomal; tetrameric; intra/interlocus hybrid bands occur			343
Cat3	4S	catalase: electrophoretic mobility; null allele is known; mitochondrial; tetrameric; intralocus hybrid bands occur			345
Cdh1	?	cinnamyl alcohol dehydrogenase: electrophoretic mobility			113
Ce1	?	curled entangled: rolled leaves tend to be entangled			50
cfl2	?	complementary to *fl2*			297
cfr1	1S	coupling factor reduction: chloroplast ATP synthase affected			86
Cg1	3S-35	corngrass: narrow leaves, extreme tillering	S	P	368
Cg2	?	corngrass: like *Cg1*; mutable			220
Cgl1	?	*Colletotrichum graminicola* resistance			11
Ch1	2L-155	chocolate pericarp: dark brown pericarp	S	P	7
Cin		Cinteotl corn insert: repetitive sequences dispersed in the genome			360
cl1	3S-60	chlorophyll: white to green seedlings, depending upon *Clm1*; pale yellow endosperm	S		102
clh1	?	histone Ic: electrophoretic mobility			392
Clm1	8	modifier of *cl1*: greens *cl1* seedlings; does not restore endosperm carotenoids	S		102
Clt1	8	clumped tassel (was *Clt*-985*): variable dwarfing, developmental anomalies	S		125 282
cm1	10L-near *R1*	chloroplast mutator: white or yellow stripes on leaves (compare *ij1*); conditions chloroplast modifications that are maternally inherited	S		394
cms-C		cytoplasmic male sterility: female-transmitted male sterility, C type; restored by *Rf4*			21
cms-S		cytoplasmic male sterility: female-transmitted male sterility, S type; restored by *Rf3*			178 180
cms-T		cytoplasmic male sterility: female-transmitted male sterility, Texas type; restored by *Rf1 Rf2*			178 180
cp1	7S-near *vp9*	collapsed: endosperm collapsed and partially defective			218
cp2	7S-near *vp9*	collapsed: endosperm rough, collapsed, partially defective; seedling very light green with darker streaks; lethal			289
cpDNA		chloroplast DNA (= ctDNA): sequences or loci in chloroplast genome			
cr1	3S-26	crinkly leaves: plant short; leaves broad, crinkled, foreshortened	S	P	97
Css1	9L-near *gl15*	sucrose synthase (= *Sus2*, SS2): sucrose synthase-2 of embryo and other tissues; (compare *sh1*); *BNL-CSS*(pshD13), *NPI121-CSS*()			236
ct1	8	compact plant: semi-dwarf plant, ear furcated	S		270
ct2	1S	compact plant: semi-dwarf plant with club tassel			128
ctDNA		chloroplast DNA (= cpDNA): sequences or loci in chloroplast genome			
cto1	?	cob turned out: ear inverted to a sheet or tube, kernels internally placed; variable expression			406
Cx1	10L-near bf2	catechol oxidase: electrophoretic mobility; null allele is known; monomeric; no hybrid bands			317
Cy	5L-near *pr1*	Cycler: regulatory element mediating *bz1-rcy*			348
d1	3S-44	dwarf plant: andromonoecious, short, compact plants; responds to gibberellins; *d1-t* intermediate in height	S	P	90
d2	3	dwarf plant: like *d1*	S		396
d3	9S-59	dwarf plant: like *d1*	S		72
d5	2S-34	dwarf plant: like *d1*	S		396
D8	1L-133	dwarf plant: dominant, resembles *d1*; not responsive to gibberellins; (compare *Mpl1*, possible allele)	S	P	307

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
D9	5S-6	dwarf plant (was *D*-2319*): dominant semidwarf with broad, dark green leaves; not andromonoecious, not responsive to gibberellins			275 276
da1	9	dilute aleurone: aleurone color diluted	S		108
Dap1	5L-near *Got2*	dappled aleurone: patches of normal and abnormal aleurone cells			391
db1	?	dichotomously branching plants (= dib): variable location of dichotomy, usually at 4-8th node (possible association with aneuploidy)			244 245
dek1	1S-27	defective kernel (was *clf, gay, clf*-792*): germless; floury endosperm; anthocyanins and carotenoids absent; cultured embryos not obtained	S		287 288
dek2	1L	defective kernel (was *dsc*-1315A*): discolored, scarred endosperm; lethal; cultured embryos green	S		287 288
dek3	2S	defective kernel (was *gm*-1289*): germless; cultured embryos white with green stripe	S		287 288
dek4	2L	defective kernel (was *clf*-1024A*): germless; floury endosperm; cultured embryos green, narrow leaved	S		287 288
dek5	3S	defective kernel (was *sh*-874A*): shrunken endosperm; white seedling with green stripes	S		287 288
dek6	3L	defective kernel (was *sh*-627D*): shrunken endosperm; lethal; cultured embryos normal	S		287 288
dek7	4S-near *Ts5*	defective kernel (was *su*-211C*): shrunken sugary endosperm; white seedling with green stripes	S		287 288
dek8	4L	defective kernel (was *sh*-1156A*): shrunken endosperm; lethal; cultured embryos green, small	S		287 288
dek9	5L	defective kernel (was *crp*-1365*): crumpled endosperm; lethal; anthocyanins and carotenoids reduced; cultured embryos not obtained	S		287 288
dek10	4L	defective kernel (was *cp*-1176A*): collapsed endosperm; lethal; cultured embryos green, curled, stubby	S		287 288
dek11	7L	defective kernel (was *et*-788*): etched endosperm; lethal; cultured embryos white with green stripes	S		287 288
dek12	9S	defective kernel (was *cp*-873*): collapsed endosperm; lethal; cultured embryos green, narrow-leaved, curled	S		287 288
dek13	9L	defective kernel (was *o*-744*): defective opaque endosperm; lethal; cultured embryos pale green with green stripes	S		287 288
dek14	10S	defective kernel (was *cp*-1435*): collapsed endosperm; lethal; cultured embryos yellow-green	S		287 288
dek15	10L	defective kernel (was *cp*-1427A*): collapsed floury endosperm; lethal; cultured embryos green	S		287 288
dek16	2L	defective kernel (was *fl*-1414*): floury endosperm; lethal; cultured embryos normal	S		362
dek17	3L	defective kernel (was *cp*-330D*): collapsed endosperm; lethal; cultured embryos not obtained	S		362
dek18	5S	defective kernel (was *cp*-931A*): collapsed endosperm; lethal; cultured embryos green, narrow-leaved	S		362
dek19	6L	defective kernel (was *o*-1296A*): collapsed opaque endosperm; lethal; cultured embryos green	S		362
dek20	8L	defective kernel (was *cp*-1392A*): collapsed endosperm; lethal; cultured embryos green	S		362
dek21		(= *w2*)	S		
dek22	1L	defective kernel (was *cp*-1113A*): collapsed endosperm; lethal; cultured embryos not obtained	S		52 363
dek23	2L	defective kernel (was *dcr*-1428*): defective crown; lethal; cultured embryos not obtained	S		52 363
dek24	3S	defective kernel (was *cp*-1283*): collapsed endosperm; lethal; cultured embryos normal	S		363
dek25	4S	defective kernel (was *sh*-1167A*): shrunken endosperm; lethal; cultured embryos normal	S		363
dek26	5L	defective kernel (was *cp*-1331*): collapsed endosperm; lethal; cultured embryos normal	S		363
dek27	5L	defective kernel (was *cp*-1380A*): collapsed endosperm; lethal; cultured embryos green	S		363
dek28	6S	defective kernel (was *o*-1307A*): opaque endosperm	S		363
dek29	8L	defective kernel (was *cp*-1387A*): collapsed endosperm; viable; cultured embryos green, narrow-leaved	S		363
dek30	9L	defective kernel (was *fl*-139*): floury endosperm; lethal; cultured embryos green, narrow-leaved	S		363
dek31	4L-near *Tu1*	defective kernel (was *ptd*-1130*): pitted endosperm; lethal	S		361
dep1	6	defective pistils			246
Df		deficiency: general symbol for loss of segments of chromosome			
Dhn1	6L-near *py1*	dehydrin candidate: dehydration-induced mRNA & protein; *UMC170-DHN*(pM3-4)			53
Dia1	2-near *v4*	diaphorase: electrophoretic mobility; cytosolic; monomeric			412
Dia2	1L-near *bm2*	diaphorase: electrophoretic mobility; cytosolic; dimeric			412
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	239
dSpm		defective suppressor-mutator: designator for transposable factors regulated by *Spm*			347
dsy1	?	desynaptic: male and female sterility; synaptic failure			132
dsy2	?	desynaptic: like *dsy1*			135
dsy3	?	desynaptic: like *dsy1*			130
dsy4	?	desynaptic: like *dsy1*			130
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	320
Dt2	6L-44	dotted: like *Dt1*	S		295
Dt3	7L	dotted: like *Dt1*, but expression variable	S		295
Dt4	4	dotted: like *Dt1*, but dots chiefly on crown of kernel	S		77
Dt5	9S-near *yg2*	dotted: like *Dt1*			77
Dt6	4-near *su1*	dotted: like *Dt1*	S		380
du1	10L-31	dull endosperm: glassy, tarnished endosperm; affects soluble starch synthase and branching enzyme IIa (P.C. Mangelsdorf, 1935, unpublished data)	S		101 227
dv1	?	divergent spindle: chromosomes unoriented at metaphase I; partial male and female sterility	S		51
dy1	?	desynaptic: chromosomes unpaired in microsporocytes; partial male and female sterility	S		272
E1	7L	esterase: electrophoretic mobility; null allele is known; dimeric; intralocus hybrid bands occur			351
E2	?	esterase: presence-absence			353
E3	3S	esterase: electrophoretic mobility; dimeric; intralocus hybrid bands occur			352
E4	3S-near *cl1*	esterase (was *Est4*): electrophoretic mobility; null allele is known; monomeric			149
E5(I)	?	esterase (duplicate factor with *E5(II)*): electrophoretic mobility			222
E5(II)	?	esterase (duplicate factor with *E5-(I)*): electrophoretic mobility			222
E6	?	esterase: presence-absence			222
E7	?	esterase: presence-absence			222
E8	3S-14	esterase: electrophoretic mobility; null allele is known; dimeric; intralocus hybrid bands occur			222
E9	?	esterase: electrophoretic mobility; null allele is known			222
E10	?	esterase: electrophoretic mobility			222
eg1	5L	expanded glumes: glumes open at right angle	S		40

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Ej1		(= *Isr1*)			
el1	8L	elongate: chromosomes uncoiled during meiotic metaphase and anaphase in male and female; frequent unreduced gametes	S	P	325
Emu1	2	endogenous *Mu*: *NPI347-EMU*()			423
En		enhancer: transposable element (equivalent to *Spm*); autonomous, regulates *I* transposition (e.g. at *g2-m* = *pg-m* = *pg14-m*)		P	305
Enp1	6L-near *y1*	endopeptidase: electrophoretic mobility; null allele is known; monomeric			243
Est		(see *E*)			
et1	3L-161	etched: pitted, scarred endosperm, virescent seedling; plastid membranes altered	S	P	385
fl	1L-86	fine stripe: virescent seedling, fine white stripes on base and margin of older leaves	S	P	212
fae1	?	fasciated ear: small, rounded ears branched at their tips			361
Fas1	?	fascicled ear: ears and tassels branch dichotomously, may fasciate			313
Fbr1	?	few-branched (was *Fbr*-1602*): tassel reduced to 0-3 branches; bract replaces next-to-bottom branch			280
Fcu		factor Cuna: controlling element of *r1-cu*			138
fl1	2S-68	floury endosperm (= *o4*): endosperm opaque, soft; dosage effect with *fl1-ref* (but *o4* is recessive)	S	P	151
fl2	4S-58	floury: endosperm opaque, soft; dosage effect (W.J. Mumm, 1935, unpublished data)	S		101 274
fl3	8L-0	floury: endosperm opaque, soft; dosage effect	S		267
Flt		flint: designator for factors determining flint endosperm type			257
g1	10L-50	golden plant: seedling and plant with distinct yellow cast	S	P	90 92
g2	3S-0	golden plant (= *g5* = *pg14* = *v19*): like *g*, but more extreme; sheaths whitish yellow-green	S		166
g5		(=*g2*)			
G6	9S-near *l7*	golden plant (was *G*-1585*): like *g1*; lighter yellowish sheaths			290
Ga1	4S-32	gametophyte factor (= *ga9*): *Ga1* pollen grains competitively superior to *ga1* on *Ga1* silks; *Ga1-S* super-gametophyte	S		179
ga2	5L-55	gametophyte factor: *Ga2* pollen grains competitively superior to *ga2*	S		38
ga7	3L-167	gametophyte factor: *ga7* pollen from heterozygotes 10-15% functional regardless of silk genotype			322
ga8	9S-near *lo2*	gametophyte factor: *Ga8* pollen grains competitively superior to *ga8* on *Ga8* silks			350
ga9		(= *ga1*)			
ga10	5	gametophyte factor			137
Gdh1	1L-near *vp8*	glutamic dehydrogenase: electrophoretic mobility; null allele is known (cold sensitivity); intra/interlocus hybrid bands occur			315
Gdh2	10	glutamic dehydrogenase: electrophoretic mobility; intralocus hybrid bands occur			139
Ger		glucoside earworm resistance: designator for earworm resistance factors from Cateto Palha Roxa			257
Gll		Genetics Inst. Inc.: designator for loci defined by restriction fragment polymorphisms			
gl1	7L-36	glossy: cuticle wax altered; leaf surface bright, water adheres	S	P	196
gl2	2S-30	glossy: like *gl1*	S	P	152
gl3	4L-112	glossy: like *gl1*	S		152
gl4	4L-81	glossy (= *gl16*): like *gl1* (G.F. Sprague, unpublished)	S		
gl5	4-near *su1*	glossy (was *gl5-1*, duplicate factor with *gl20*): like *gl1* (G.F. Sprague, 1935, unpublished data)	S		101 381
gl6	3L-69	glossy: like *gl 1* (G.F. Sprague, 1935, unpublished data)	S		101
gl7	?	glossy (= *gl12*): like *gl1* (G.F. Sprague, 1935, unpublished data)			101
gl8	5L-68	glossy (= *gl10*): like *gl1* (G.F. Sprague, 1935, unpublished data)	S		101
gl9	3L	glossy: expression poor (G.F. Sprague, 1935, unpublished data)	S		101
gl10		(= *gl8*)			
gl11	2S-near *B1*	glossy: like *gl* ; abnormal seedling morphology	S		378
gl12		(= *gl7*)			
gl14	2	glossy (duplicate factor with *gl24*): like *gl1*			6
gl15	9L-66	glossy: like *gl1*; expressed after 3rd leaf	S	P	6
gl16		(= *gl4*)			
gl17	5S-34	glossy: like *gl1*, but semi-dwarf with necrotic crossbands on leaves	S		326
gl18	8L-near *fl3*	glossy: like *gl1*; expression poor	S		6
gl19	3S	glossy (was *gl*-169*): like *gl1*; lethal	S		283
gl20	?	glossy (was *gl5-2*, duplicate factor with *gl5*): like *gl1*	S		381
gl21	10S	glossy (was *gl*-478B*, duplicate factor with *gl22*): like *gl1*	S		283
gl22	?	glossy (was *gl*-478C*, duplicate factor with *gl21*): like *gl1*			279
gl23	?	glossy (was *gl*-PI262490*): like *gl1*			383
gl24	?	glossy (duplicate factor with *gl14*): like *gl1*			383
Glb1	1L-121	globulin (was *Pro*, *Prot1*): Mr 63,000, electrophoretic mobility; null allele is known; embryo protein			193 355
Glb2	?	globulin: Mr45,000, presence-absence			193
Gln1	10L-near *sr2*	glutamine synthetase candidate: *BNL#-GLN*(GS6.15)			44
Glu1	10L-near *bf2*	beta glucosidase: electrophoretic mobility; cytosolic; dimeric; intralocus hybrid bands occur			316
Got1	3L-120	glutamate-oxaloacetate transaminase (possibly = *Ta1*): electrophoretic mobility; null allele is known; glyoxysomal; dimeric; intralocus hybrid bands occur			344
Got2	5L-96	glutamate-oxaloacetate transaminase: electrophoretic mobility; null allele is known; plastidial; dimeric; intralocus hybrid bands occur			141
Got3	5S-near *a2*	glutamate-oxaloacetic transaminase: electrophoretic mobility; null allele is known; mitochondrial; dimeric; intralocus hybrid bands occur			141
Gpa1	10L-near *g1*	glyceraldehyde-3-phosphate dehydrogenase, chloroplastic, A subunit; *UMC188-GPA*(cDNA probe pZm57)			34
Gpc1	4-near *orp1*	glyceraldehyde-3-phosphate dehydrogenase: cytosolic, C subunit; *UMC191-GPC*(cDNA probe pZm9)			34 229 338
Gpc2	?	glyceraldehyde-3-phosphate dehydrogenase, cytosolic, C subunit (cDNA probe pGAPC2)			338
Gpc3	?	glyceraldehyde-3-phosphate dehydrogenase, cytosolic, C subunit (cDNA probe pGAPC3)			338
grt1	5L	green tip (was *grt*-1308B*): pale yellow seedling with green first leaf tip; lethal	S		283
gs1	1L-135	green stripe: grayish green stripes between vascular bundles on leaves; tissue wilts	S	P	95 251
gs2	2S-54	green stripe: like *gs1*, but pale green stripes; no wilting (G.F. Sprague, 1935, unpublished data)	S	P	101
gs3	6L	green stripe (was *gs*-268*): like *gs2*			283
Gs4	10	green stripe (was *Gs*-1439*): like *gs1*			281
gt1	1	grassy tillers: numerous basal branches; vegetatively totipotent in combination with *id1* and *pe1*			359
h1	3	soft starch: endosperm soft, opaque	S		265
H3		histone H3: designator for histone H3 family			

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
H4		histone H4: designator for histone H4 family			
hcf1	2L	high chlorophyll fluorescence: affects NADP+ oxidoreductase; green seedling			248
hcf2	1L	high chlorophyll fluorescence: missing cytochrome f/b6 complex; yellow-green seedling			248
hcf3	1S	high chlorophyll fluorescence (= *hcf9*): missing PSII thylakoid membrane core complex; green seedling			248
hcf4	1L	high chlorophyll fluorescence: affects CO2 fixation; green seedling			249
hcf5	6S	high chlorophyll fluorescence: affects PSII reaction; green seedling			250
hcf6	1S	high chlorophyll fluorescence: missing cytochrome f/b6 complex; green seedling			207
hcf9		(= *hcf3*)			
hcf12	1L	high chlorophyll fluorescence			207
hcf13	1L	high chlorophyll fluorescence: affects CO2 fixation; green seedling			207 249
hcf15	2L	high chlorophyll fluorescence: affects photophosphorylation; yellow-green seedling, may survive			207
hcf18	5L-near *pr1*	high chlorophyll fluorescence (= *hcf43*): major loss of PSI; other thylakoid complexes reduced; yellow-green seedling			249
hcf19	3L	high chlorophyll fluorescence: affects PSII thylakoid membrane core complex; green/yellow-green seedling			207 249
hcf21	5L	high chlorophyll fluorescence: affects CO2 fixation, Rubisco; green seedling			249
hcf23	4S	high chlorophyll fluorescence: affects photophosphorylation; yellow-green seedling, may survive			207 249
hcf26	6S	high chlorophyll fluorescence: affects electron transport; yellow-green, viable seedling			207 249
hcf28	10L	high chlorophyll fluorescence: affects CO2 fixation; green seedling			250
hcf31	1S	high chlorophyll fluorescence: missing chlorophyll a/b binding protein; yellow-green seedling			250
hcf34	6L	high chlorophyll fluorescence: affects photophosphorylation; yellow-green seedling			207 249
hcf36	6L	high chlorophyll fluorescence: affects electron transport; green seedling			250
hcf38	5L	high chlorophyll fluorescence: affects cytochrome f/b6 complex, alpha and beta components of CF1; green seedling			207
hcf41	1L	high chlorophyll fluorescence: affects PSII thylakoid membrane core complex; green seedling			207 249
hcf42	9L	high chlorophyll fluorescence: affects Rubisco; green/yellow-green seedling			249
hcf43		(= *hcf18*)			
hcf44	1L	high chlorophyll fluorescence: affects PSI membrane core complex; pale-green seedling			249
hcf46	3L	high chlorophyll fluorescence			207
hcf47	10S	high chlorophyll fluorescence: affects cytochromes; yellow-green seedling			250
hcf48	6L	high chlorophyll fluorescence: affects electron transport; yellow-green seedling			250
hcf50	1L	high chlorophyll fluorescence: missing PSI thylakoid membrane core complex; green seedling			249
hcf101	7L	high chlorophyll fluorescence (was *Mu-5**): affects PSI thylakoid membrane core complex			250
hcf102	8L	high chlorophyll fluorescence: affects cytochrome f/b6 complex (D. Miles, unpublished)			
hcf103	7L	high chlorophyll fluorescence: affects PSII			64
hcf104	7L	high chlorophyll fluorescence: photosystem I-deficient			64
hcf106	2-near *ts1*	high chlorophyll fluorescence: affects PSI, PSII, cytochrome f/b6; *BNL#-HCF106*			228
hcf108	5	high chlorophyll fluorescence: ATPase-deficient			64
hcf111	7L	high chlorophyll fluorescence: cytochrome b/f-deficient			64
hcf113	9S	high chlorophyll fluorescence: multiple effects; yellow-green seedlings			63
hcf114		(= *hcf103*)			64
hcf316	10S	high chlorophyll fluorescence: affects chlorophyll a/b binding protein; yellow-green seedling			250
hcf323	6S	high chlorophyll fluorescence: affects photophosphorylation, coupling factor; green seedling			250
hcf408	6L	high chlorophyll fluorescence: affects chlorophyll a/b binding protein; yellow-green seedling			250
Hex1	3S-near *Cg1*	hexokinase: electrophoretic mobility; null allele is known; cytosolic; monomeric			414
Hex2	6L-near *Pt1*	hexokinase: electrophoretic mobility; null allele is known; cytosolic; monomeric			414
hm1	1L-64	*Helminthosporium carbonum* susceptibility: disease lesions vs. yellowish flecks (resistant) on leaves with race 1	S	P	405
hm2	9L-near *bk2*	*Helminthosporium carbonum* susceptibility: like *hm1*; masked by *Hm1*			271
Hrg1	2-near *wt1*	hydroxyproline-rich glycoprotein candidate (cDNA probe): *UMC145-HRG*(pMC56)			388
Hs1	7S-0	hairy sheath: abundant hairs on leaf sheath	S	P	397
Hsf1	5	hairy sheath frayed (was *Hsf*-1595*): pubescent sheaths and leaf margins; liguled enations at leaf margins			26
Hsp1	8L	heat shock protein (70kD) candidate: *NPI119-HSP70*()			423
Ht1	2L-121	*Helminthosporium turcicum* resistance	S		158
Ht2	?	*Helminthosporium turcicum* resistance			159
Ht3	?	*Helminthosporium turcicum* resistance: (from *Tripsacum floridanum*)			160
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*; (see also Inv)			
id1	1L-near *an1*	indeterminate growth: requires extended growth and short days for flowering; vegetatively totipotent with *gt1* and *pe1*	S		367
Idh1	8L	isocitrate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			141
Idh2	6L-near *w14*	isocitrate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			141
ig1	3L-90	indeterminate gametophyte: low male fertility, polyembryony, heterofertilization, polyploidy, androgenesis (male and female affected)	S		189
ij1	7L-52	iojap striping: many variable white stripes and margin patterns on leaves (compare *cm1*); conditions chloroplast defects that are cytoplasmically inherited	S	P	165
ij2	1L	iojap striping: like *ij1*; chloroplast inheritance unknown			283
in1	7S-20	intensifier: intensifies aleurone anthocyanin pigments; *In1-D* dominant dilute	S	P	111
Inv		Inversion: general symbol for inversion of a segment of chromosome	S	P	
is1	?	cupulate interspace			118
Isr1	10L-near *R1*	inhibitor of striate (was *Ej1*): reduces expression of *sr2* and other leaf-striping factors	S		190
j1	8L-42	japonica striping: white stripes on leaf and sheath; not expressed in seedling	S	P	92
j2	4L-106	japonica striping: extreme white striping of leaves, etc. (R.A. Emerson, 1935, unpublished data)	S	P	101
K		knob: general symbol for heterochromatic structures that are heritably polymorphic in size and are found at characteristic positions on the chromosomes; homology with 185bp probe			301
K3L	3L-115	knob: heterochromatic structure (see K)			74
K10	10L-near *sr2*	abnormal-10: heterochromatic appendage on long arm of chromosome 10; neocentric activity distorts segregation of linked genes	S	P	217

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Kn1	1L-near *Adh1*	knotted: localized proliferation of tissue at vascular bundles on leaf	S	P	36
Kn2	?	knotted: finger-like projections of leaf at the ligule			114
Krn		kernel row number: designator for factors determining kernel row number			257
l1	10L-near *R1*	luteus: yellow pigment in white tissue of specific chlorophyll mutants *w1*, *w2*, *j1*, *ij1*, others	S	P	210 211
l3	?	luteus: lethal yellow seedling	S		173
l4	?	luteus: lethal yellow seedling	S	P	172
l6	9S-near *bz1*	luteus: like *l4* (W.H. Eyster, 1935, unpublished data)	S		101
l7	9S-42	luteus: yellow seedling and plant; lethal	S		108
l10	6L-19	luteus: like *l4*; fails to convert protochlorophyllide to chlorophyllide	S		332
l11	6S	luteus (was *l*-4120*): yellow seedling with green leaf tips; lethal	S		10
l12	6L-16	luteus (was *l*-4920*): like *l11*	S		68
l13	10L-94	luteus (was *l*-59A*, *l*-Neuffer2*): dark yellow, lethal seedling; fails to convert protoporphyrin IX to Mg-protoporphyrin			230 283
l15	6L-30	luteus (was *l*-Blandy3*, *l*-Brawn*): like *l4*			336
l16	1S	luteus (was *l*-515*): like *l4*; leaves bleach to paler yellow in patches			283
l17	1L	luteus (was *l*-544*): like *l4*; leaves with lighter yellow crossbands			283
l18	2L	luteus (was *l*-1940*): *like l4*			283
l19	10S	luteus (was *l*-425*): like *l4*			283
la1	4S-55	lazy plant: prostrate growth habit	S	P	171
lbl1	?	leaf bladeless: leaf blade reduced to absent; low temperature enhances expression			247
Lc1	10L-68	red leaf color: anthocyanin in coleoptile, nodes, auricle, leaf blade, etc.; (compare *Sn1*)	S		82
Lcs1	?	thylakoid membrane polypeptide: electrophoretic mobility			260
Lct1	?	thylakoid membrane polypeptide: electrophoretic mobility			260
lct2	?	thylakoid membrane polypeptide: presence-absence			260
Les1	2S-58	lesion (was *Les*-843*): large necrotic lesions resembling disease lesions formed by fungal infections on susceptible lines	S		286
Les2	1S-near *sr1*	lesion (was *Les*-845A*): small white lesions resembling disease lesions formed by fungal infections on resistant lines	S		286
Les3	10	lesion: like *Les1*			9
Les4	2L	lesion (was *Les*-1375*): late expression of large necrotic lesions	S		155
Les5	1S	lesion (was *Les*-1449*): like *Les2*			155
Les6	10S	lesion (was *Les*-1451*): like *Les4*	S		155
Les7	?	lesion (was *Les*-1461*): late expression of small chlorotic lesions	S		155
Les8	9S-near *lo2*	lesion (was *Les*-2005*): late expression of small, pale green lesions	S		155
Les9	7L-near *ra1*	lesion (was *Les*-2008*): late expression of small necrotic lesions	S		155
Les10	2-near *v4*	lesion (was *Les*-A607*): like *Les1*			156
Lfy1	?	leafy: increased number of leaves			358
lg1	2S-11	liguleless: ligule and auricle missing; leaves upright, enveloping	S	P	90 91
lg2	3L-101	liguleless: like *lg1*, less extreme	S	P	30
Lg3	3-65	liguleless: dominant, no ligule; leaves upright, broad, often concave and pleated	S	P	302
Lg4	8L	liguleless (was *Lg*-403*): dominant, no ligule or auricle but vestiges sporadically in blade			110
li1	10L-near *bf2*	lineate leaves: fine, white striations on basal half of mature leaves	S	P	62
lls1	1S	lethal leaf spot: chlorotic-necrotic lesions resembling *Helminthosporium carbonum* infection	S		404
ln1	6	linoleic acid: lower ratio of oleate to linoleate in kernel			69
lo2	9S-50	lethal ovule: ovules containing *lo2* gametophyte abort			272
loc1	?	low oil content in kernel: associated with albino seedlings			311
lp1	4	lethal pollen: *lp1* pollen fails in competition with *Lp1*			269
lte1	2	latente: drought, heat, aluminum tolerance; frost resistance; from Michoacan 21; dominance varies			254
Lte2	10L-near *g1*	latente: drought, heat, aluminum tolerance; from Cateto; epistatic to *lte1*			255
lty1	?	light yellow endosperm			79
lty2	?	light yellow endosperm			79
lu1	5S-29	lutescent: pale yellow green leaves	S		365
lw1	1L-near *Adh1*	lemon white: white seedling, pale yellow endosperm	S		402
lw2	5L-near *pr1*	lemon white: like *lw1*	S	P	402
lw3	5L-near *v2*	lemon white (duplicate factor with *lw4*): like *lw1*	S		402
lw4	4-near *zb6*	lemon white (duplicate factor with *lw3*): like *lw1*	S		402
Lxm1	3	lax midrib (was *Lxm*-1600*): leaves with wide, flat, flexible midrib	S		278
lyc1		(= *ps1-lyc*)			
mal1	9	multiple aleurone layering: recessive interacts with two complementary dominants *Mal2* and an unnamed factor, giving multiple cell layers			252
Mal2	4	multiple aleurone layering: (see *mal1*)			252
Mc1	?	mucronate: opaque endosperm			341
Mch1	?	maize CRY1 homolog: ribosomal protein gene family (cDNA probe)			201
Mch2	?	maize CRY1 homolog: ribosomal protein gene family (cDNA probe)			201
Mct1	?	modifier of *cox2* transcripts: changes transcripts of mitochondrial gene			65
Mdh1	8	malate dehydrogenase: electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur			291
Mdh2	6L-near *w14*	malate dehydrogenase: electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur			291
Mdh3	3L-146	malate dehydrogenase: electrophoretic mobility; null allele is known; mitochondrial; dimeric; intra/interlocus hybrid bands occur			291
Mdh4	1L-near *an1*	malate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			291
Mdh5	5S-17	malate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			291
Mdm1	6-near *w15*	maize dwarf mosaic virus resistance			242
Me1	3L-125	NADP malic enzyme: electrophoretic mobility; null allele is known; tetrameric; *NPI231-ME*(),*YNH#-ME*() (may be ambiguous)			141
Me2	6L	NADP malic enzyme candidate: *NPI330-ME*()			423
Mei1	?	meiosis: chromosomes sticky in metaphase I; male sterile			131 134
mep1	5L	modifier of embryo protein: affects quantities of *Glb* (was *Pro*) protein forms			355

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Mer		Maya earworm resistance: designator for earworm resistance factors from IAC Maya			256
mg1	?	miniature germ (replaces *mg* of Wentz): germ 1/4 to 1/3 of normal; viable			198
mgs1	10-near *bf2*	male-gametophyte specific: cDNA expressed in pollen; *BNL#-MGS1*(Zmc13)			146
Mgs2	4L-near *c2*	male gametophyte-specific: cDNA with pectin lyase homology; *BNL#-MGS2*(cDNA probe Zmc58)			44
mi1	1	midget plant: small plant (H.S. Perry, 1935, unpublished data)			101
mmm1	1L-near *an1*	modifier of mitochondrial malate dehydrogenases: mobilities			291
mn1	2-near *fl1*	miniature seed: small, somewhat defective kernel, fully viable; invertase reduced	S	P	219
mn2	7	miniature seed: small kernel, loose pericarp; extremely defective but will germinate (R.J. Lambert, unpublished)			
mn3	6-near *Mdm1*	miniature seed (was *de*-1184*): small kernel, etched/pitted endosperm; viable			390
Mod		modifier: inactive *Spm* element, enhances excisions elicited by active *Spm*			241
Mp		modulator of pericarp: transposable factor affecting *P1* locus; parallel to *Ac-Ds*			33
Mpi1		transposable element: 10-15 copies in the genome			418
Mpl1	1L-near *Adh1*	miniplant: dominant, andromonoecious, intermediate dwarf (compare *D8*, possible allele); not responsive to gibberellins	S		147
Mr	9S-near *l7*	mutator of *R1-m*: transposable factor, regulates *R1-m* mutation		P	46
Mrh	5	mutator: controlling element of *a1-m-rh*			327
ms1	6L-near *si1*	male sterile: anthers shriveled, not usually exserted; affected at microspore vacuolation	S		370
ms2	9L-64	male sterile: like *ms1*; affected between vacuolation and pore formation	S		106 108
ms3	3	male sterile: anthers shrivelled; not usually exserted			106 108
ms4		(= *po1*)			
ms5	5-near *v3*	male sterile: anthers not exserted; affected at microspore mitosis	S		18
ms6		(= *po1*)			
ms7	7L-near *ra1*	male sterile: like *ms2*	S		18
ms8	8L-28	male sterile: like *ms5*; affected in meiosis	S	P	18
ms9	1S-near *P1*	male sterile: like *ms5*; affected in meiosis	S		18
ms10	10L-near *bf2*	male sterile: like *ms5*; affected at microspore vacuolation	S		18
ms11	10	male sterile: like *ms5*; affected at microspore mitosis	S		18
ms12	1	male sterile: like *ms1*; affected at microspore vacuolation	S		18
ms13	5S	male sterile: like *ms5*; affected at microspore vacuolation	S		18
ms14	1-near *as1*	male sterile: like *ms5*; affected at microspore mitosis	S		18
ms17	1S-23	male sterile: like *ms1*; affected variably in meiosis	S		98
ms20	?	male sterile			108
Ms21	6	male sterile: pollen grains developing in presence of *Ms21* are defective and nonfunctional if *sks1*, normal if *Sks1*			205 349
ms22	?	male sterile: affected in meiosis	S		417
ms23	3L	male sterile (allelic to *ms*-Bear7*): affected in meiosis	S		417
ms24	?	male sterile: like *ms*1; affected in microspore mitosis	S		417
ms28	?	male sterile: anaphase I disturbed, spindle persists			134
Ms41	4L	male sterile (was *Ms*-1995*)	S		290
ms43	8L	male sterile: anaphase I impaired			131 134
Ms44	4L	male sterile (was *Ms*-7255*)			2
Msc1	1L	mosaic (was *Msc*-791A*): aleurone mosaic for anthocyanin color	S		290
Msc2	5S	mosaic (was *Msc*-1124B*): aleurone mosaic for anthocyanin color			290
Mst1	10L-70	modifier of *R-st*: affects expression of *R1-st*	S		8
mtDNA		mitochondrial DNA: sequences or loci in the mitochondrial genome			
Mu		mutator: freely transposable element; *Mu1* designates element isolated from *Adh1-S3034*			335
Mut	2S-near *gl2*	mutator: controlling element for *bz1-m-rh*			327
Mv1	?	resistance to maize mosaic virus I ("corn stripe")			28
na1	3L-113	nana plant: short, erect dwarf; no response to gibberellins		P	164 209
na2	5S-near *bt1*	nana plant: like *na1* (H.S. Perry, unpublished)	S		
Nabp1	7S-near *vp9*	nucleic acid binding protein			44
NCR		North Carolina, Raleigh: designator for loci defined by restriction fragment polymorphisms			
NCS1		nonchromosomal stripe: maternally inherited light green leaf striping			366
NCS2		nonchromosomal stripe: maternally inherited pale green and depressed striping; mitochondrial	S		55
NCS3		nonchromosomal stripe: maternally inherited striations, distorted plants; mitochondrial	S		55
NCS5		nonchromosomal stripe: maternally inherited stunted growth, yellow stripes, aborted kernels; mitochondrial cytochrome oxidase subunit 2 (*cox2*) alteration			292
NCS6		nonchromosomal stripe: maternally inherited stunted growth, yellow stripes, aborted kernels; mitochondrial cytochrome oxidase subunit 2 (*cox2*) alteration			202
nec1	8L-near *fl3*	necrotic (was *nec*-669*, *sienna*-7748*): chlorotic seedling that stays rolled, wilts and dies	S		231
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, 1952, unpublished data)	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		277
nec4	2S-near *d5*	necrotic (was *nec*-516B*): seedling yellow, leaf tips necrotic; lethal	S		157
nec5	4L	necrotic (was *nec*-642A*): pale green seedling becoming necrotic; dark brown exudate; lethal	S		283
nec6	5S-near *a2*	necrotic (was *nec*-493*): like *nec3*	S		283
nec7	5L	necrotic (was *nec*-756B*): seedling becoming necrotic in crossbands; lethal			283
NIU		Northern Illinois University: designator for loci defined by restriction fragment polymorphisms			
nl1	10L-near *bf2*	narrow leaf: leaf blade narrow, some white streaks (R.A. Emerson, 1935, unpublished data)	S	P	101
Nl2	5S-25	narrow leaf (was *Rgd2*, *Rgd*-1445*): leaves narrow and distorted; tillering			284
NOR	6S	nucleolus organizer: codes for ribosomal RNA; *BNL#-NOR*(pBF243)	S		237
NPI		Native Plants, Inc.: designator for loci defined by restriction fragment polymorphisms			
o1	4L-98	opaque endosperm: endosperm starch soft, opaque (W.R. Singleton and D.F. Jones, 1935, unpublished data)	S		101 274
o2	7S-16	opaque endosperm: like *o1* (W.R. Singleton and D.F. Jones, 1935, unpublished data); high lysine content; regulates b-32 protein (see *pro1*); reduced lysine degradation (lysine-ketoglutaric reductase); *BNL#-O2*(pXho0.9), *NPI480-O2*()	S	P	101 274
o4		(= *fl1*)			
o5	7L-near *ra1*	opaque endosperm: like *o1*; virescent to yellow or white seedlings	S		331

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
o6		(= *pro1*)			
o7	10L-90	opaque: like *o* ; high lysine content	S		258
o9	?	opaque endosperm (was *ox*-74120*): crown opaque and light in color, frequently with a cavity; base or abgerminal side of kernel often corneous	S		268
o10	1L	opaque endosperm (was *o*-E1356*, *ox*-7747*): like *o1*	S		268
o11	?	opaque endosperm (was *ox*-7455*): thin, opaque, somewhat shrunken kernels with greyish cast	S		268
o12	?	opaque endosperm (was *ox*-7638*): thin, etched or scarred kernels, variable in size; plants chlorophyll deficient and small, with pollen but few ears	S		268
o13	?	opaque endosperm (was *ox*-7729*): opaque, etched kernels with rim of corneous starch on abgerminal side	S		268
Oec1	4	oxygen-evolving complex protein candidate: *NPI472-OEC*(), *UMC171A-OEC23*(8-18)			422
Oec2	2S-near *B1*	oxygen-evolving complex protein candidate: *NPI473-OEC*(), *UMC171B-OEC23*(8-18)			422
Oec3	5S-near *Pgm2*	oxygen-evolving complex protein candidate: *NPI474-OEC*()			422
Oec4	7L	oxygen-evolving complex protein candidate: *NPI475-OEC*()			422
Oec5	8	oxygen-evolving complex protein candidate: *NPI476-OEC*()			422
Og1	10S-19	old gold stripe: variable bright yellow stripes on leaf blade	S	P	215
ora2	?	orange endosperm			78
ora3	?	orange endosperm			79
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		230
oro2	?	orobanche: like *oro1*			230
Orom1	?	orobanche modifier: partially corrects chlorophyll loss in *oro1*			230
orp1	4S-near *su1*	orange pericarp (duplicate factor with *orp2*): pericarp orange over *orp1 orp2* kernels; lethal, tryptophan auxotroph; tryptophan synthase B; *BNL#-ORP*(trpB), *UMC193A-ORP*(trpB)	S		285
orp2	10S-near *y9*	orange pericarp (duplicate factor with *orp1*): *BNL#-ORP*(trpB); *UMC193B-ORP*(trpB)	S		285
os1	2S	opaque-endosperm, small germ: opaque crown; kernel larger, lighter color; viable; reduced oil content			374
oy1	10S-15	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 (see Coe et al., 1988)	S	P	107
P		plant color component at *R1* : anthocyanin pigmentation in seedling leaf tip, coleoptile, anthers			81 386 387
P1	1S-26	pericarp color: red pigment in cob and pericarp (for alleles, see Coe et al., 1988); *NPI370-P1*()	S	P	89 216
pam1	?	plural abnormalities of meiosis: desynchronized meiotic divisions and premeiotic mitosis; male sterile, incompletely female sterile			133
pam2	?	plural abnormalities of meiosis: like *pam1*			135
pb1	6L-near *y1*	piebald leaves: very light, irregular green bands on leaf		P	73
pb4	6L-near *y1*	piebald leaves: like *pb1*	S		73
Pcr1	1L-near *bz2*	protochlorophyllide reductase candidate: *BNL#-PCR*(OR1)			44
Pcr2	5S-near *Pgm*	protochlorophyllide reductase candidate: *BNL#-PCR*(OR1)			44
Pcr3	2S-near *al1*	protochlorophyllide reductase candidate: *BNL#-PCR*(OR1)			44
Pcr4	7S-near *in1*	protochlorophyllide reductase candidate: *BNL#-PCR*(OR1)			44
pd1	3	paired rows: single vs. paired pistillate spikelets; quantitative, one of a family of loci differentiating maize vs. teosinte			199
Pdf1	?	thylakoid membrane polypeptide: dominant increase in electrophoretic mobility			261
Pdk1	6L	pyruvate, Pi dikinase candidate: *UMC173-PDK*(p1-9), *NPI229-PDK*()			233
Pdk2	8L	pyruvate, Pi dikinase candidate: *NPI230-PDK2*()			233
pe1	?	perennialism: vegetatively totipotent in combinations with *gt1* and *id1*			359
Pep1	9-near *pg12*	phosphoenol pyruvate carboxylase candidate: *NPI332-PEP1*()			423
Pep2	5-near *a2*	phosphoenolpyruvate carboxylase candidate: *NPI#-PEP*()			411
Pep3	4L	phosphoenolpyruvate carboxylase candidate: *NPI#-PEP*()			411
pg11	6L-38	pale green (duplicate factor with *pg12*): seedling light yellowish green; mature plant pale and vigorous	S	P	323
pg12	9-61	pale green (duplicate factor with *pg11*)	S		323
pg13	?	pale green: seedling light yellowish green; stunted growth			364
pg14		(= *g2*)	S	P	305
pg15	1S	pale green (was *ppg*-340B*): seedling light yellowish green; bleaches to near white in patches; lethal	S		283
pg16	1L	pale green (was *pg*-219*): seedling light yellowish green	S		283
Pgd1	6-near *rgd1*	6-phosphogluconate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			141
Pgd2	3L-near *Rg1*	6-phosphogluconate dehydrogenase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intra/interlocus hybrid bands occur			141
PGE		Plant Gene Expression Center: designator for loci defined by restriction fragment polymorphisms			
Pgm1	1L-near *Glb1*	phosphoglucomutase: electrophoretic mobility; null allele is known; cytosolic; monomeric			141
Pgm2	5S-0	phosphoglucomutase: electrophoretic mobility; null allele is known; cytosolic; monomeric			141
Ph1	4S-0	pith abscission: cob disarticulation; quantitative, one of a family of loci differentiating maize vs. teosinte			119
Phi1	1L-149	phosphohexose isomerase: electrophoretic mobility; null allele is known; cytosolic; dimeric; intralocus hybrid bands occur			141
Php1	10S-near *orp2*	chloroplast phosphoprotein: isozyme			44
Phy1	1L-near *Adh1*	phytochrome phyA1: *BNL#-PHY*(pcPhy101), *NPI251-PHY*()			423
Phy2	5S-near *Pgm2*	phytochrome phyA2: *BNL#-PHY*(pcPhy101), *NPI369-PHY*()			423
pi1	?	pistillate florets (duplicate factor with *pi2*): secondary florets develop ("Country Gentlemen" or "Shoe Peg" expression) in *pi1 pi2* ears; quantitative character			162
pi2	?	pistillate florets (duplicate factor with *pi1*)			162
PIO		Pioneer Hi-Bred, International: designator for loci defined by restriction fragment polymorphisms			
Pl1	6L-49	purple plant: sunlight-independent purple pigment in plant; regulates flavonoid enzymes; *BNL#-PL1*(pH3-Sal0.6)	S	P	96
pm1	3L-near *ts4*	pale midrib: midrib and adjacent tissue lighter green; reduced plant vigor	S	P	31

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
Pn1	7L-112	papyrescent glumes: long, thin papery glumes on ear and tassel		P	120
po1	6S-4	polymitotic (= *ms4*): repeats 2nd meiotic division in male and female	S	P	16
ppg1	5L	pale pale green (was *cb*-199A*): white seedling with faint green; white necrotic crossbands; lethal			283
pr1	5L-67	red aleurone: changes purple aleurone to red; flavonoid 3'-hydroxylase	S	P	85
pro1	8L-near *fl3*	proline requiring (= *o6*): crumpled opaque kernel; b32 protein isoforms and null; green-striped lethal seedling			123
Prot1		(= *Glb1*)			
Prx		(see *Px*)			
ps1	5S-39	pink scutellum (= *vp7*): viviparous; endosperm and scutellum pink, seedling white with pink flush; *ps1-lyc* not viviparous	S	P	377
Pt1	6L-60	polytypic ear: proliferation produces irregular growth on ear and tassel	S	P	273
Px1	2L	peroxidase: electrophoretic mobility; null allele is known; monomeric			145
Px2	?	peroxidase: electrophoretic mobility; monomeric			221
Px3	7L-near *Pn1*	peroxidase: electrophoretic mobility; monomeric			221
Px4	?	peroxidase: electrophoretic mobility; null allele is known; monomeric			221
Px5	?	peroxidase: presence-absence			221
Px6	?	peroxidase: presence-absence			221
Px7	?	peroxidase: electrophoretic mobility; null allele is known; monomeric			221
Px8	?	peroxidase: electrophoretic mobility; monomeric			29
Px9	?	peroxidase: electrophoretic mobility; null allele is known; monomeric			29
py1	6L-69	pigmy plant: leaves short, pointed; fine white streaks	S	P	396
py2	1L	pigmy: like *py1*			283
pyd1	9S-near *yg2*	pale yellow deficiency: pale yellow seedling; deficiency for short terminal segment of chromosome arm; lethal; (for alleles, see Coe et al., 1988)			238
R1	10L-64	colored: red or purple color in aleurone and/or anthers, leaf tip, brace roots, etc. (for alleles, see Coe et al., 1988); regulates flavonoid enzymes; *BNL#-R1*(R5-4frag2), *NPI308-R1*()	S	P	85
ra1	7L-32	ramosa: ear and tassel many-branched; tassel branches taper to tip	S	P	18 126
ra2	3S-49	ramosa: irregular kernel placement; tassel many-branched, upright (R.A. Brink, 1935, unpublished data)	S	P	101 293
ra3	4	ramosa: (H.S. Perry, 1954, unpublished data)	S		
Rab17	6L-near *py1*	responsive to abscisic acid (was *RNY1*): *BNL#-RAB17*(cDNA probe)			408
Rab30	1S-near *sr1*	responsive to abscisic acid: *BNL#-RAB30*(cDNA probe)			44
rbcS		(= *Ssu1*)			
rBg		receptor of *Bg*			340
Rcm1	7-near *vp9*	rectifier: restores miniature seed of teosinte cytoplasm to normal			3
Rcm2	?	rectifier: weakly restores miniature seed of teosinte cytoplasm to normal			3
Rcm3	?	rectifier: restores miniature seed of teosinte cytoplasm to normal; from *Z. diploperennis*			3
rcu		receptor of *Fcu*			138
rcy		receptor of *Cy*			348
rd1	1L-near *Adh1*	reduced plant: semi-dwarf plant	S		270
rd2	6L	reduced plant: like *rd1*, but not as extreme			129
rd3	3L-near *vp1*	reduced plant: like *rd1*; anthocyanin interactions			235
rDNA		ribosomal DNA: rDNA5.8S, rDNA18S and rDNA25S located in NOR on 6S; rDNA5S on 2L near *Ht1*			
rDt		receptor of Dotted			375
Rf1	3S-near *Wrk1*	fertility restorer: restores fertility to cms-T; complementary to *Rf2*	S		177
Rf2	9-near *wx1*	fertility restorer: see *Rf1*	S		84
Rf3	2L	fertility restorer: restores fertility to cms-S	S		37
Rf4	8	fertility restorer (complementary with *Rf5* and *Rf6*): restores fertility to cms-C			142
Rf5	?	fertility restorer (complementary with *Rf4* and *Rf6*): restores fertility to cms-C			181 407
Rf6	?	fertility restorer (complementary with *Rf4* and *Rf5*): restores fertility to cms-C			181 407
Rf7	?	fertility restorer: partially restores fertility to cms-Y			318
Rg1	3-67	ragged leaves: defective tissue between veins of older leaves, causing holes and tearing	S	P	32
rgd1	6-8	ragged seedling: seedling leaves narrow, thread-like, have difficulty in emerging	S	P	192
Rgd2		(= *Nl2*)			
rgo1	?	reversed germ orientation: embryo faces base of ear; variable frequency, maternal trait			339
rhm1	6-near *rgd1*	resistance to *Helminthosporium maydis*: chlorotic-lesion reaction with race O			371
Ril	4S-27	rind abscission: cob disarticulation; quantitative, one of a family of loci differentiating maize vs. teosinte			119
rMrh		receptor of *Mrh*			327
rMut		receptor of *Mut*			327
Rp1	10S-3	resistance to *Puccinia sorghi*	S	P	223 224
Rp3	3-near *Rg1*	resistance to *Puccinia sorghi*	S		419
Rp4	4S-24	resistance to *Puccinia sorghi*	S		419
Rp5	10S-0	resistance to *Puccinia sorghi*			342
Rp6	10S-near *Rp1*	resistance to *Puccinia sorghi*			419
RPA		Rhone-Poulenc Agrochimie: designator for loci defined by restriction fragment polymorphisms			
Rpp9	10S-near *Rp1*	resistance to *Puccinia polysora* and *P. sorghi*			403
Rs1	?	rough sheath: extreme ligule disorganization			191
rs2	1-near *as1*	rough sheath	S		191
Rs4	7	rough sheath (was *Rs*-1606*): leaf sheaths rough, vascular bundles enlarged			281
rt1	3S-near *cl1*	rootless: secondary roots few or absent	S	P	168
ruq		receptor of *Uq*			115
S		seed color component at *R1*: anthocyanin pigmentation in aleurone; (see also cms-S)			81 387
Sad1	10L-near *bf2*	shikimate dehydrogenase: electrophoretic mobility; plastidial; monomeric			412
sbd1	6L	sunburned (was *wxl*-2292*): sun-exposed leaves greyish-waxy			281
Sdw1	8	semi-dwarf plant (was *Sdw*-1592*): shortened internodes, erect leaves	S		25
se1	4L-near *dp1*	sugary-enhancer: high sugar content with *su1*; light yellow endosperm; freely wrinkled in Ill677a	S		109
sen1	3	soft endosperm (duplicate factor with *sen2*): endosperm soft, opaque			389
sen2	7	soft endosperm (duplicate factor with *sen*)			389
sen3	1	soft endosperm (duplicate factor with *sen1*): like *sen1*			389

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
sen4	?	soft endosperm (duplicate factor with *sen3*)			389
sen5	2	soft endosperm (duplicate factor with *sen6*): like *sen1*			389
sen6	5	soft endosperm (duplicate factor with *sen5*)			389
sft1	?	small flint type: ears on *sft1* plants produce only small flint endosperms; *+/sft* ears are normal			80
Sg1	?	string cob: reduced pedicels		P	117
sh1	9S-29	shrunken: inflated endosperm collapses on drying, forming smoothly indented kernels; sucrose synthase-1 of endosperm (compare *Css1*); homotetramer; *BNL#-SH1* (Pst38 or Pvu55), *NPI15-SH1*()	S	P	163
sh2	3L-149.2	shrunken: inflated, transparent, sweet kernels collapse on drying, becoming angular and brittle; endosperm ADPG pyrophosphorylase subunit (compare *bt2*); *BNL#-SH2* (1050)	S	P	225
sh4	5L	shrunken: collapsed, chalky endosperm	S		401
sh5	5-near *lu1*	shrunken: sides of kernel collapsed	S		383
si1	6L-20	silky (= *ms-si*): multiple silks in ear; sterile tassel with silks			112
sk1	2S-56	silkless ears: pistils abort, no silks	S		176
Sks1	2L-near *v4*	suppressor of sterility: pollen grains developing in presence of *Ms21* are defective and nonfunctional if *sks1*, normal if *Sks1*			205 349
sl1	7L-50	slashed leaves: leaves slit longitudinally by necrotic streaks	S		152
sm1	6L-59	salmon silks: silks salmon color with *P1-RR*, brown in *P1-WW*	S	P	4
Sn1	10L-near *R1*	scutellar node color: anthocyanin in coleoptile, nodes, auricle, leaf blade, etc. (compare *Lc1*)			124
Sod(2-2)	9	superoxide dismutase: *NPI463-SOD*()			422
Sod1	?	superoxide dismutase: electrophoretic mobility; plastidial; Cu-Zn dimeric; intralocus hybrid bands occur			14
Sod2	7L	superoxide dismutase: *NPI419-SOD*()			423
Sod3	?	superoxide dismutase: electrophoretic mobility; mitochondrial; Mn tetrameric; intralocus hybrid bands occur			14
Sod4	1S-near *P1*	superoxide dismutase: electrophoretic mobility; cytosolic; Cu-Zn dimeric; intralocus hybrid bands occur; *NPI412-SOD*()			14 423
Spc1	3L-near *ig1*	speckled (was *Spc*-1376*, *Les*-1376*): brown speckling on leaves and sheath at flowering; supporting tissues weak			284
spc2	1L	speckled (was *spc*-262A*): green seedling with light green speckles			283
spc3	3L	speckled (was *pg*-553C*): green seedling with dark and light green speckles			283
Spm		suppressor-mutator: autonomous transposable element (equivalent to *En*); regulates *dSpm* transposition and function at *a1-m1*, *a1-m2*, *bz1-m13*, etc.			240
spt1	2L	spotted (was *spt*-464*): pale green, weak seedlings with dark green spots	S		283
spt2	4S	spotted (was *pgspt*-1269A*): like *spt1*	S		283
sr1	1S-0	striate leaves: many white striations or stripes on leaves (A.M. Brunson, 1935, unpublished data)	S		101
sr2	10L-98	striate leaves: white stripes on blade and sheath of upper leaves	S	P	174
sr3	10S	striate leaves: virescent and striate to striped	S	P	128
sr4	6L	striate leaves (was *stp*-65A*): seedlings pale luteus, later leaves white-striped			279
Ss2		(see *Css1*)			
Ssu1	4L-near *c2*	ribulose bisphosphate carboxylase small subunit family: *BNL17.05-SSU*(pC1), *NPI1-SSU*(), *RPA9B-SSU*(pZmcRPA:SSU), *NPI331-SSU*()			43 423
Ssu2	2-near *ts1*	ribulose bisphosphate carboxylase small subunit family: *BNL#-SSU*(pC1), *NPI227-SSU*(), *RPA9A-SSU*(pZmcRPA:SSU)			423
st1	4S-62	sticky chromosome: small plant, striate leaves, pitted kernels resulting from sticky chromosomes; *st1-e* heightened by high temperature	S	P	19
stAc	10-near *bf2*	stabilized Activator (P. Chomet, unpublished): RFLP locus			
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 (see Coe et al., 1988)	S	P	66
su2	6L-58	sugary: endosperm glassy, translucent, sometimes wrinkled	S		108
Sup1	?	suppressor: modifies *o2* kernels to semi-transparent			232
Sus1		(= *Css1*)			
sy1	?	yellow scutellum			376
T		reciprocal translocation: general symbol for exchange of parts between two nonhomologous chromosomes; (see also cms-T)	S	P	
Ta1	?	transaminase (possibly = *Got1*): electrophoretic mobility; dimeric; intralocus hybrid bands occur			221
tb1	1L-near *Adh1*	teosinte branched: many tillers; ear branches tassel-like	S		41
td1	5-near *bt1*	thick tassel dwarf: (E.G. Anderson, unpublished)	S		
te1	3L	terminal ear: stalked ear appendages at tip; varying to infolded ears			234
Thc1	?	thiocarbamate sensitive: sensitive to Eradicane			306
tl1	?	tasselless			245
Tlr1	1L	tillered (was *Tlr*-1590*): extreme tillering	S		290
Tp1	7L-46	teopod: many tillers, narrow leaves, many small partially podded ears, tassel simple	S	P	214
Tp2	10L-48	teopod: like *Tp1*		P	304
tpe1	?	thin pericarp: reduced cell number in pericarp (from Coroica)			116
Tpi1	7L-59	triose phosphate isomerase: electrophoretic mobility; plastidial; dimeric; intra/interlocus hybrids occur with *Tpi2*			415
Tpi2	2L-100	triose phosphate isomerase: electrophoretic mobility; plastidial; dimeric; intra/interlocus hybrids occur with *Tpi1*			415
Tpi3	8	triose phosphate isomerase: electrophoretic mobility; cytosolic; dimeric; intra/interlocus hybrids occur with *Tpi4* & *Tpi5*; *NPI344-TPI*()			415
Tpi4	3L-near *cl1*	triose phosphate isomerase: electrophoretic mobility; cytosolic; dimeric; intra/interlocus hybrids occur with *Tpi3* & *Tpi5*; *NPI345-TPI*()			415
Tpi5	8L	triose phosphate isomerase: electrophoretic mobility; cytosolic; dimeric intra/interlocus hybrids occur with *Tpi3* & *Tpi4*			415
Tpi6	5L	triose phosphate isomerase candidate: *NPI346-TPI*()			415 423
tpm1	?	thylakoid peptide modifier: dominant decrease in electrophoretic mobility			259
tr1	2S	two-ranked ear: distichous vs. decussate phyllotaxy in ear axis; quantitative, one of a family of loci differentiating maize vs. teosinte			199
trAc	1S-near *vp5*	transposed Activator sequence			44
tru1	?	tassels replace upper ears: upper ear branches tassel-like, tillers bear ears			361

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
ts1	2S-74	tassel seed: tassel pistillate and pendant; if removed, small ear with irregular kernel placement develops	S		94
ts2	1S-24	tassel seed: like *ts1*, but branches variably pistillate and staminate	S	P	94
ts4	3L-73	tassel seed: tassel compact silky mass, upright, with pistillate and staminate florets; ear silky and proliferated	S	P	308
Ts5	4S-53	tassel seed: tassel upright with scattered, short silks; branches mostly pistillate toward the base	S		99
Ts6	1L-158	tassel seed: tassel pistillate to mixed, compact; ear with irregular kernel placement	S	P	293
ts8		(= *si1*)			
Tu1	4L-101	tunicate: kernels enclosed in long glumes; tassel glumes large, coarse	S	P	60 61
Tub1	1L-near *Adh1*	alpha tubulin family: mRNA expressed primarily in roots; member of tandem repeat (see *Tub2*); *BNL17.04-TUB*(pUC9alpha-1)			43 262
Tub2	1L-near *Adh1*	alpha tubulin family: member of tandem repeat (see *Tub1*)			262
Tub3	?	alpha tubulin family: mRNA expressed in dividing cells			263
ub1	?	unbranched: tassel with one spike	S	P	289
ubi1	4L	ubiquitin candidate: *AGR1002A-UBI*()			266
ubi2	5	ubiquitin candidate: *AGR1002B-UBI*()			266
Ufo1	10S-near *Rp1*	unstable factor for orange: anthers, silks, and most other plant parts orange with *P1-WR* or *P1-RR*; growth retarded			395
UMC		University of Missouri, Columbia: designator for loci defined by restriction fragment polymorphisms			
Uq		ubiquitous: controlling element mediating *a1-ruq*			115
v1	9L-63	virescent: yellowish white seedling, greens rapidly; low temperature accentuates	S	P	71
v2	5L-107	virescent: like *v1*, but greens slowly; low temperature accentuates	S	P	92
v3	5L-45	virescent: light yellow seedling, greens rapidly; low temperature accentuates	S	P	71
v4	2L-83	virescent: like *v2*	S	P	71
v5	7S-24	virescent: like *v1*, but older leaves have white stripes	S	P	71
v8	4L-near *Tu1*	virescent: like *v2*; lethal	S		72
v12	5L-near *ys1*	virescent: like *v3*	S		309
v13	?	virescent: first leaf with green tip; greens slowly			309
v16	8L-14	virescent: like *v2*	S		309
v17	4	virescent: like *v1*, but greening from base to tip	S		309
v18	10	virescent: like *v1*	S		309
v19		(= *g2*)			
v21	8L	virescent (was *v*-25*, *v*-A552*): grainy virescent, greening from tips and margins inward	S		22
v22	1L-near *an1*	virescent (was *v*-8983*): like *v1* (E.G. Anderson, unpublished)	S		
v23	4-near *su1*	virescent (was *v*-8914*): like *v1* (E.G. Anderson, unpublished)			
v24	2L	virescent (was *v*-424*): like *v1*	S		283
v25	1S	virescent (was *v*-17*): greenish white seedling; greens from base upward	S		283
v26	2S	virescent (was *v*-453*): yellowish white seedling with green leaf tip and midrib	S		283
v27	7L	virescent (was *v*-590A*): like *v1*			283
v28	9S	virescent (was *v*-27*): like *v1*	S		283
v29	10L	virescent (was *v*-418*): grainy virescent			283
v30	9L-87	virescent (was *v*-8587*): like *v1*			56
v31	9S-near *yg2*	virescent (was *v*-828*): grainy, light green seedling; small green plant with longitudinal white stripes			148
va1	7L-near *ij1*	variable sterile: variable male and female fertility; cytokinesis fails in anaphase I	S		17
Vg1	1L-85	vestigial glume: glumes very small, cob and anthers exposed	S	P	379
vp1	3L-near *ts4*	viviparous: embryo fails to become dormant, viable if transplanted; some alleles dormant; chlorophyll and carotenoids unaffected; anthocyanins in aleurone suppressed; *BNL-VP1*(pVPM1B)	S		105
vp2	5S-38	viviparous: embryo fails to become dormant; white endosperm, white seedling; anthocyanins unaffected	S	P	105
vp5	1S-1	viviparous: like *vp2*	S	P	328
vp7		(= *ps1*)			
vp8	1L-154	viviparous: embryo fails to become dormant; chlorophyll and carotenoids unaffected; small, pointed-leaf seedlings	S		329
vp9	7S-25	viviparous (also known as *y7*): like *vp2*; *vp9-4889* dormant, pale aleurone, pale green seedling	S	P	329
Vsr1	10L	virescent striped (was *Vsr*-1446*): virescent seedling; greens to white and yellow striped plant	S		290
w1	6L-near *w14*	white: white seedling (yellow with *l1*); plastid transcripts variously aberrant	S		90 91 211
w2	10L-80	white: white seedling (yellow with *l1*); endosperm pitted and spotted (allele *dek21*); plastid DNA content decreased	S		213
w3	2L-111	white: like *vp2*; *w3-8686* pale endosperm, pale green seedling in dim light	S	P	213
w11	9S-54	white: like *w1*	S		72
w14	6L-78	white (was *w*-8657*): like *w1*	S		68
w15	6L-13	white (was *w*-8896*): like *w1*; fails to convert protochlorophyllide to chlorophyllide	S		68
w16	7S-near *vp9*	white: like *w1*			264
w17	7S-near *Hs1*	white: like *w1*			264
w18	1L	white seedling (was *w*-495A*, allelic to *w*-571C*): like *w1*	S		279
Wc1	9L-107	white cap: kernel with pale yellow endosperm (pearly white with *y1*), emphasized in soft-starch crowns	S		195
wd1	9S-near *yg2*	white deficiency: white seedling, deficiency for distal half of first chromomere of short arm (for alleles, see Coe et al., 1988)	S	P	238
wgs1	5L	white green sectors (was *sct*-206B*): white seedling with green sectors			283
whp1	2L	white pollen: duplicate factor with *c2* for pollen color and for anthocyanins; chalcone synthase; *BNL17.03-WHP*(pC2-c46)	S		58
wi1	6L-near *y1*	wilted: chronic wilting, leaves not as cool as normal; delayed differentiation of metaxylem vessels	S		314
Wi2	3	wilted: top leaves wilt under moisture/temperature stress			280
Wi3	?	wilted: like *Wi2*			281
wlu1	3L	white luteus (was *wl*-28*): pale yellow seedling; lethal			283
wlu2	7L	white luteus (was *wl*-543A*): like *wlu1*	S		283
wlu3	8L	white luteus (was *wl*-203A*): like *wlu1*			283
wlu4	9L	white luteus (was *wl*-41A*): like *wlu1*	S		283

SYMBOL	LOCATION	NAME, PHENOTYPE	S	P	REF
wlu5	1L-near *br1*	white luteus (was *wl*-266A*): like *wlu1*	S		279
Wrk1	3S-62	wrinkled kernel (was *Wr*-1020*): kernels small and wrinkled	S		290
Wrp1	?	wrinkled plant: dominant dwarf, leaves and culm longitudinally corrugated; dosage effect			27 280
ws1	?	white sheath: light yellow leaf sheaths; duplicate factor with *ws2*			186
ws2	?	white sheath: see *ws1*			186
ws3	2S-0	white sheath: white leaf sheath, culm, husks	S	P	321
Ws4	1S	white sheath (was Pale green-1589): seedlings and plants lighter green in sheaths			280
wsp		weak streaked plant: maternally inherited reduced plants			35
wt1	2S-60	white tip: tip of first leaf white and blunt	S		384
wt2	4S	white tip (was *cb*-10*): seedling with white leaf tip and crossbands on first 2 leaves	S		283
wx1	9S-56	waxy: amylopectin (stained red by iodine) replaces amylose (blue staining) in endosperm and pollen; starch-granule-bound NDP-starch glucosyl transferase (for alleles, see Coe et al., 1988); *BNL-WX1*(pBF225), *UMC25-WX1*(pBF225), *NPI16-WX1*()	S	P	59
wyg1	7L-near *ra1*	white yellow green seedling			264
y1	6L-17	white: reduced carotenoid pigments in endosperm; some alleles affect chlorophyll in seedlings (e.g. *y1-8549*; see Coe et al., 1988)	S	P	66
y3	2S-near *al1*	pale yellow (compare *al1*)	S		303
y7		(= *vp9-y7*)			
y8	7S-18	pale yellow: pale endosperm	S		169
y9	10S-27	pale yellow: pale endosperm, slightly viviparous; green to pale green seedlings and plants	S		334
y10	3L	pale yellow (was *w*-7748*): pale endosperm; white seedling, lethal	S		330
y11	?	pale yellow: pale endosperm; green seedling	S		382
y12	?	pale yellow: like *y11*	S		382
yd2	3L-near *lg2*	yellow dwarf	S		333
yg1	5L-near *v2*	yellow-green: yellow-green seedling and plant	S		103
yg2	9S-7	yellow-green: like *yg1* (for alleles, see Coe et al., 1988)	S	P	167
YNH		Yale New Haven: designator for loci defined by restriction fragment polymorphisms			
ys1	5L-75	yellow stripe: yellow tissue between leaf veins, reflects iron deficiency symptoms	S	P	15
ys2	1S	yellow stripe: yellow tissue between leaf veins			312
ys3	3L-near *gl6*	yellow stripe: like *ys1*	S		421
Ysk1	4-near *su1*	yellow streaked (was *Ysk*-844*): longitudinal yellow streaks in top 3rd of mature leaves	S		284
z1		(= *vp9-z* = *y7-z*)			
zb1	?	zebra crossbands: yellowish crossbands on older leaves			70
zb2	?	zebra crossbands: crossbands on seedling leaves			393
zb3	5L-near *v2*	zebra crossbands: yellowish crossbands on older leaves (M. Demerec, 1935, unpublished data)	S		101
zb4	1S-19	zebra crossbands: regularly spaced crossbands on earlier leaves; enhanced by cool temperatures	S	P	150
zb6	4-79	zebra crossbands: regularly spaced crossbands on earlier leaves; enhanced by cool temperatures	S		153
zb7	1L-near *Adh1*	zebra crossbands (was *zb*-101*): lighter green crossbands on seedlings; glossy	S		283
Zb8	9-near *l7*	zebra crossbands (was *Atc1*, *Cb*-1443*): yellow-green crossbands on older leaves; strong anthocyanin expression in leaf tip and blade	S		284 290
zein		(see Zp, *ZpB36*, *ZpL*, *Zpr*, *Zps*)			
Zer		Zapalote Chico earworm resistance: designator for earworm resistance factors from Zapalote Chico			256
zn1	10L-29	zebra necrotic: necrotic tissue appears between veins in transverse leaf bands on half-grown or older plants	S	P	161
zn2	?	zebra necrotic: like *zn1*			127
Zp		zein polypeptide: designator for loci determining zein polypeptides			372 373
ZpB36	7-near *ra1*	zein family: *BNL#-B36*(B36)			45
zpg1	?	zebra-stripe pale green			79
ZpL1	4S-near *Ga1*	zein polypeptides: *Zp1La* - *Zp1Lf* complex			420
ZpL2a	4L-near *orp1*	zein polypeptides			420
ZpL2b	7S-near *o2*	zein polypeptides			420
ZpL3a	4L-near *orp1*	zein polypeptides			420
Zpr10/(22)	4-39	zein-protein regulator: elevation of 10kD zein			24
Zps10/(22)	9-near *gl15*	zein-protein structural gene: 10kD zein; RFLP (probe 10kZ-1)			24

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	BNL POS '91		UMC Jan '91		UMC Apr '89		PIO POS '90	AGR	POS '90	NPI	POS '90
A1* (BNL# & AGR# & NPI51-A1)	3	149						3	150	3	139
A* (NPI482-A1)										1	76
A* (NPI468-A1)										2	139
A* (NPI469-A1)										5	137
A* (NPI470-A1)										7	11
A* (UMC189A-A1)	8	110								8	161
Abp1* (BNL#-ABP, = pKlamb+)	3	68									
Aco1	4	59									
Acp1	9	70	9	45						9	53
Acp4	1	208	1	314						1	182
Act1* (BNL# & NPI368-ACT)	8	80								8	
Adh1 (& AGR# & BNL# & NPI21-ADH1)	1	159	1	217				1	236	1	144
Adh2* (& AGR# & BNL# & NPI228-ADH2)	4	46						4	28	4	2
Adp* (NPI#-ADP)										10	17
Adp1* (NPI309-ADP, BNL17.15-BT2)	1	113								1	105
Adp2 (see Agp2)											
Adp3 (see Bt2)											
Ad* (NPI318-ADP)										4	
Adp* (NPI319-ADP)										10	
Adp* (NPI#-ADP, BNL17.16-BT2)	8	74								8	
Agp1* (BNL#-AGP1)	6	115									
Agp2* (BNL#-AGP2 & NPI310-ADP2)	2	106								2	176
AGR#-A1								3	150		
AGR#-ADH1								1	236		
AGR#-ADH2								4	28		
AGR#-BZ1								9	28		
AGR#-CSS1								9	72		
AGR#-P1								1	66		
AGR#-SH1								9	22		
AGR#-WX1								9	47		
AGRc3								6	2		
AGRc4								4	42		
AGRc6								7	120		
AGRc12								6	2		
AGRc20								8	35		
AGRc23								5	23		
AGRc36								7	9		
AGRc39								4	60		
AGRc66								5	7		
AGRc67								6	35		
AGRc94	4	21						4	8		
AGRc203								7	34		
AGRc255A								10	40		
AGRc255B								9	33		
AGRc259A								1	268		
AGRc259B								5	0		
AGRc261								7	0		
AGRc273								9	37		
AGRc300								4	103		
AGRc303								4	95		
AGRc321								2	52		
AGRc329								5	40		
AGRc332								3	58		
AGRc333A								2	110		
AGRc333B								7	50		
AGRc362A								5	36		
AGRc362B								1	204		
AGRc435								3	74		
AGRc445								4	147		
AGRc459								10	74		
AGRc461								3	128		
AGRc467								1	0		
AGRc476A								3	55		
AGRc478								8	48		
AGRc479								2	118		
AGRc489								1	149		
AGRc512								1	128		
AGRc514A								3	56		
AGRc514B								8	35		
AGRc516								1	99		
AGRc525								7	160		
AGRc528								10	36		

	BNL POS '91	UMC Jan '91	UMC Apr '89	PIO POS '90	AGR	POS '90	NPI	POS '90
AGRc539A					2	30		
AGRc542					7	83		
AGRc561					10	13		
AGRc563A					5	125		
AGRc563B					4	78		
AGRc567					4	42		
AGRc568A					3	156		
AGRc568B					8	56		
AGRc569					1	133		
AGRc584					1	106		
AGRc587					1	111		
AGRc593					2	68		
AGRc595					9	84		
AGRc611					6	2		
AGRc634					1	100		
AGRc637					5	65		
AGRc638					3	168		
AGRc669A					5	7		
AGRc669B					1	282		
AGRc690					10	40		
AGRc701					7	82		
AGRc707					1	262		
AGRc714					10	32		
AGRc747					8	42		
AGRc805					2	4		
AGRc814					5	65		
AGRc914					7	42		
AGRc923					3	50		
AGRc926					5	4		
AGRc938					2	4		
AGRc939					2	118		
AGRp40					3	100		
AGRp50					3	52		
AGRp52					5	47		
AGRp53					5	9		
AGRp54					2	65		
AGRp58					2	85		
AGRp62					2	96		
AGRp67					4	44		
AGRp83A					4	49		
AGRp83B					1	155		
AGRp90					5	102		
AGRp91					3	60		
AGRp97					3	58		
AGRp144					6	1		
AGRp166					4	101		
AGRp168A					2	39		
AGRp168B					10	82		
AGRp173					2	75		
AGRr13					2	0		
AGRr18					10	46		
AGRr19					3	58		
AGRr21					8	119		
AGRr22					1	12		
AGRr27					4	85		
AGRr34					1	212		
AGRr37					6	65		
AGRr41					9	10		
AGRr43A					10	0		
AGRr43B					3	163		
AGRr45A					5	141		
AGRr47					6	10		
AGRr49					7	30		
AGRr50A					3	122		
AGRr50B					8	71		
AGRr51					8	85		
AGRr55					7	138		
AGRr57					10	49		
AGRr58					9	35		
AGRr62					4	51		
AGRr64					9	53		
AGRr70					5	55		
AGRr71					1	175		
AGRr73					7	53		
AGRr77					1	7		
AGRr85A					2	74		
AGRr85B					2	82		
AGRr87					6	8		
AGRr88					1	88		
AGRr89					4	42		
AGRr90					9	74		
AGRr92A					1	58		
AGRr92B					9	92		
AGRr101					7	80		
AGRr103A					5	13		
AGRr103B					1	254		
AGRr104					10	49		
AGRr106					5	75		
AGRr109					4	60		
AGRr110					1	182		
AGRr111A					7	50		
AGRr111B					2	110		

	BNL '91	POS '91	UMC Jan '91	UMC Apr '89	PIO	POS '90	AGR	POS '90	NPI	POS '90
AGRr113A							2	51		
AGRr113B							10	74		
AGRr115	4	5					4	0		
AGRr116A							3	30		
AGRr116B							8	27		
AGRr117							1	79		
AGRr118A							6	70		
AGRr118B							9	0		
AGRr125							9	52		
AGRr127							5	71		
AGRr128							7	9		
AGRr131							7	80		
AGRr132							7	87		
AGRr142							5	56		
AGRr144A							3	114		
AGRr144B							8	77		
AGRr147							9	24		
AGRr152							1	22		
AGRr153A							1	80		
AGRr153B							9	65		
AGRr167A							2	51		
AGRr167B							10	67		
AGRr168							7	35		
AGRr169							8	7		
AGRr171							9	67		
AGRr174							7	52		
AGRr175							1	135		
AGRr179							3	60		
AGRr184A							8	94		
AGRr184B							3	72		
AGRr185							1	152		
AGRr186							7	115		
AGRr189							6	12		
AGRr190							4	42		
AGRr193							1	106		
AGRr197							1	91		
AGRr199							5	54		
AGRr200							1	125		
AGRr202							7	125		
AGRr205							9	44		
AGRr206							3	55		
AGRr207							7	82		
AGRr209A							3	2		
AGRr209B							8	38		
AGRr211							5	146		
AGRr213							6	119		
AGRr215							5	77		
AGRr216A							2	75		
AGRr216B							10	52		
AGRr221							6	2		
AGRr222							8	46		
AGRr232							10	47		
AGRr235A							1	201		
AGRr235B							5	47		
AGRr238A							1	206		
AGRr238B							5	36		
AGRr239							2	82		
AGRr241							7	34		
AGRr246							1	207		
AGRr248A							4	130		
AGRr248B							5	65		
AGRr250							1	145		
AGRr252							5	106		
AGRr255A							10	59		
AGRr261							6	85		
AGRr262							8	111		
AGRr265A							7	42		
AGRr265B							2	105		
AGRr267A							7	36		
AGRr267B							2	90		
AGRr269							8	96		
AGRr271							3	69		
AGRr273							4	108		
AGRr274A							3	78		
AGRr274B							8	98		
AGRr278A							1	210		
AGRr278B							5	31		
AGRr286							4	49		
AGRr288							5	122		
AGRr290							1	223		
AGRr291							1	142		
AGRr294A							1	11		
AGRr294B							9	4		
AGRr295							10	64		
AGRr298							5	89		
AGRr299							1	172		
AGRr301							4	42		
AGRr321							4	42		
AGRr322							8	92		
AGRr324							4	98		
AGRx23							1	100		

	BNL '91	POS '91	UMC Jan '91	UMC Apr '89	PIO	POS '90	AGR	POS '90	NPI	POS '90
AGRx43							5	26		
AGRx701							5	95		
AGRx825							2	135		
AGRx975							8	43		
AGR1002A-UBI							4	130		
AGR1002B-UBI							5	63		
AGR1003-ZEINB							6	4		
AGR1004-ZEING							7	30		
AGRx1128							5	68		
AGRx1176							1	170		
Alr1 (NPI348-ALR)	2	128								
Alr2 (NPI349-ALR)	7	40								
Als1* (BNL#-ALS)	4	87								
Als2* (BNL#-ALS)	5	87								
Amp1					1	192				
Amp3	5	76			5	124				
Ars1* (BNL#-ARS1)	2	51								
B1 (& NPI248-B1)	2	49							2	81
B32c3A* (NCR#-B32c3A)	8	78								
B32c3B* (NCR#-B32c3B)	7	83								
B36* (BNL#-ZpB36)	7	32								
B70* (NCR#-B70)	5	69								
Bm2							1	258	1	178
BNL#-A1	3	149								
BNL#-ABP	3	68								
BNL#-ACT	8	80								
BNL#-ADH1	1	159								
BNL#-ADH2	4	46								
BNL#-AGP	6	115								
BNL#-AGP2	2	106								
BNL#-ALS	4	87								
BNL#-ALS	5	87								
BNL#-ARS1	2	51								
BNL#-BT1	5	80								
BNL#-BT2	4	67								
BNL#-BZ1	9	31								
BNL#-BZ2	1	137								
BNL#-C1	9	26								
BNL#-C2	4	117								
BNL#-CSS	9	84								
BNL#-GLN1	10	112								
BNL#-HCF106	2	92								
BNL#-MAH9	5	42								
BNL#-ME2	6	82								
BNL#-MGS1	10	49								
BNL#-MGS2	4	127								
BNL#-NABP1										
BNL#-O2	7	16								
BNL#-ORP1	4	76								
BNL#-ORP2	10	73								
BNL#-PCR1	1	134								
BNL#-PCR2	5	47								
BNL#-PCR3	2	8								
BNL#-PCR4	7	21								
BNL#-PHP1	10	40								
BNL#-PHY1	1	156								
BNL#-PHY2	5	34								
BNL#-PL1	6	55								
BNL#-R1	10	78								
BNL#-RAB17	6	80								
BNL#-RAB30	1	15								
BNL#-SH1	9	29								
BNL#-SH2	3	149								
BNL#-SSU2	2									
BNL#-stAc	10	47								
BNL#-VP1	3	76								
BNL#-WX1	9	56								
BNL#-ZP15	6	18								
BNL#-ZP50	7									
BNL#-ZPB36	7	32								
BNL1.297	3	126								
BNL1.326	1	47								
BNL1.380	5	66							5	36
BNL1.45	8	70			4	83			8	52
BNL1.556	1	83					1	98		
BNL1.80	3	126								
BNL2.369	8	87							8	52
BNL3.02	10	10								
BNL3.03	6	73								
BNL3.04	10	10	10	0 10 0					10	17
BNL3.06	9	53					9	46	9	53
BNL3.18	3	120								
BNL4.24	7	54								

	BNL '91	POS '91	UMC '91	Jan '91	UMC '89	Apr '89	PIO '90	POS '90	AGR '90	POS '90	NPI '90	POS '90
BNL4.36	5	73			5	99						
BNL5.02	5	51			5	67						
BNL5.02B											1	105
BNL5.04	9	74			9	75			9	56		
BNL5.09	9	106	9	105	9	132			9	100		
BNL5.10	9	64									9	53
BNL5.14	3	97										
BNL5.21A	7	65										
BNL5.21B	2	139										
BNL5.24	5	150			5	208	5	189				
BNL5.27	5	56										
BNL5.33	3	123										
BNL5.37	3	84	3	92	3	82	3	118	3	86		
BNL5.40	5	105					5	141	5	77		
BNL5.46	4	54	4	26	4	0	4	54	4	31		
BNL5.47A	6	94			6	84			6	92	6	109
BNL5.47B					8	57						
BNL5.59	1	93	1	120	1	117	1	139	1	109		
BNL5.61A	7	66										
BNL5.61B	2	143										
BNL5.62	1	0	1	0	1	0	1	0	1	28		
BNL5.62B											2	47
BNL5.62C											6	139
BNL5.62D											9	55
BNL5.67	4	141										
BNL5.71	5	91	5	92	5	123	5	127				
BNL6.06	3	69			3	67			3	61		
BNL6.10	5	53			5	74			5	58		
BNL6.16	3	115					3	152	3	105		
BNL6.20	2	151					2	126	2	123		
BNL6.22	5	64							5	58		
BNL6.25	5	13	5	0	5	7	5	32	5	14		
BNL6.27	7	56										
BNL6.29	6	7					6	0				
BNL6.32	1	203	1	300	1	238	1	291	1	271		
BNL7.08	8	76										
BNL7.13	9	75							9	58	9	55
BNL7.20L	4	78										
BNL7.21A	1	73					1	112	1	85		
BNL7.21B							9	88				
BNL7.24A	9	61										
BNL7.24B	5	22										
BNL7.25	1	169			1	198	1	249	1	225	1	157
BNL7.26	3	165										
BNL7.28	6	15										
BNL7.43	5	70										
BNL7.49A	10	97			10	115	10	103	10	92		
BNL7.49B									10	44		
BNL7.50	9	91										
BNL7.56	5	48			5	58	5	84				
BNL7.57	9	110										
BNL7.61	7	77	7	79	7	73			7	80		
BNL7.65									4	76		
BNL7.65	4	106					4	120	4	76		
BNL7.71	5	77	5	83	5	107	5	111	5	63		
BNL8.01	3	95							3	78		
BNL8.04	2	70										
BNL8.05	1	3							1	29		
BNL8.06A	8	72										
BNL8.06B	6	98										
BNL8.08	1	177										
BNL8.10A	1	143			1	169	1	214	1	185		
BNL8.10B									8	35		
BNL8.15	3	10					3	0	3	0	3	6
BNL8.17	9	86					9	65	9	66		
BNL8.21A	7	79							7	80	7	70
BNL8.21B									2	118		
BNL8.23	4	156	4	183	4	171	4	179				
BNL8.26	8	92							8	52		
BNL8.29A	1	183	1	253	1	212	1	261	1	250		
BNL8.29B	5	18										
BNL8.32	7	70					7	81				
BNL8.33	5	0					5	21			5	0
BNL8.35	3	54					3	62	3	45		
BNL8.37	7	78							7	92		
BNL8.39	7	87					7	98				
BNL8.44	7	122					7	126	7	142		
BNL8.45	2	10	2	0	2	1	2	4				
BNL9.07	9	6										
BNL9.08	8	72					8	47				
BNL9.11	8	36	8	30	8	20	8	21	8	14		
BNL9.13	1	30										
BNL9.44L	8	67			8	48						
BNL10.05	4	104							4	87		
BNL10.06	5	68			5	91			5	60		
BNL10.12L	5	96										
BNL10.12U	8	142										
BNL10.13	10	76			10	90	10	90				
BNL10.17	10	0							10	2	10	17
BNL10.17B											2	
BNL10.17C											8	

	BNL '91	POS '91	UMC '91	Jan '91	UMC '89	Apr '89	PIO '90	POS '90	AGR '90	POS '90	NPI '90	POS '90
BNL10.24A	3	89					3	124				
BNL10.24B	8	136					8	114				
BNL10.38	1	45									1	118
BNL10.39	8	63					8	53	8	37		
BNL10.42	2	69							6	36		
BNL10.42A									2	60		
BNL12.06	1	62					1	99	1	76		
BNL12.09	2	82										
BNL12.09											8	70
BNL12.3B											3	136
BNL12.30A	8	103					8	90				
BNL12.30B	3	150										
BNL12.36	2	66										
BNL13.05A	8	21					8	0	8	0		
BNL13.05B	3	63					3	96				
BNL13.05C	8	68										
BNL13.24	7	69										
BNL14.07	7	76	7	79	7	68			7	100		
BNL14.26	9	104										
BNL14.28	9	104	9	107	9	128	9	122				
BNL14.34	7	76										
BNL15.07	4	163							4	120		
BNL15.18	1	154							1	215		
BNL15.20	3	112			3	119			3	102		
BNL15.21	7	46			7	27			7	63		
BNL15.27	4	73										
BNL15.37	6	84					6	83	6	84		
BNL15.40	7	28			7	0			7	32		
BNL15.45	4	73							4	50	4	24
BNL16.06	7	114	7	107	7	118			7	128		
BNL17.01-A1	8	110										
BNL17.02	10	82										
BNL17.03-C2	2	166										
BNL17.04-TUB	1	155										
BNL17.05-SSU	4	115										
BNL17.06	1	130										
BNL17.07	10	77										
BNL17.08-B1	10											
BNL17.09	4	69										
BNL17.10	4											
BNL17.11-PL1	9											
BNL17.12	6	101										
BNL17.13A	7	22										
BNL17.13B	4	35										
BNL17.13C	4	63										
BNL17.14	2	189										
BNL17.15-BT2	1	113										
BNL17.16-BT2	8	74										
BS-1 (see NIU1, NIU2, UMC186A, B)												
Bt1* (BNL#-BT1)	5	80										
Bt2* (BNL#-BT2 & NPI314-ADP)	4	67									4	
Bz1* (AGR# & BNL# & NPI8 & UMC192-BZ1)	9	31	9	17					9	28	3	30
Bz2* (BNL# & UMC181-BZ2)	1	137	1	180								
C1* (BNL#-C1)	9	26										
C2* (BNL#-C2)	4	117										
Cab1* (NPI477 & UMC24-CAB)			3	193	3	124					3	136
Cab2*, Cab3* (NPI478-CAB)											7	53
Cab4* (NPI479 & UMC174-CABM2)					8	50					8	70
Csl*-Sus1* (AGR# & BNL# & NPI121 & PIO# & UMC190-CSS)	9	84	9	62			9	58	9	72	9	63
Dhn1* (UMC170-DHN)			6	99	6	70						
Dia1	2	109										
E1			7	80								
E4			3	54								
E8	3	20	3	10			3	14			3	16
Emu1* (NPI347-EMU)											2	116
Enp1	6	22	6	14					6	20	6	33
G1									10	77	10	62
Gl1											7	28
Glb1* (UMC184A-GLB1)			1	197								
Gln1* (BNL#-GLN)	10	112										
Glu1	10	43	10	62			10	58			10	56
Got2	5	134							5	135		

	BNL '91	POS '91	UMC '91	Jan '91	UMC '89	Apr '89	PIO '90	POS '90	AGR '90	POS '90	NPI	POS '90
Gpa1* (UMC188-GPA)			10	97								
Gpc1* (UMC191-GPC)			4	53								
Hcf106* (BNL#-HCF106)	2	92										
Hex2	6	68									6	85
Hrg1 (UMC145-HRG)					2	87						
Hsp1*											119	70
Idh1	8	118	8	142							8	96
Idh2	6	111	6	155							6	152
J1									8	102		
Lg1									2	15	2	24
M3-4							6	6				
Mah9* (BNL#-MAH9)	5	42										
Mdh1	8	57										
Mdh2	6	113	6	157			6	128			6	152
Mdh3	3	146	3	147							3	139
Mdh4	1	128									1	118
Mdh5	5	55										
Me1* (YNH# & NPI231-ME)	3	40									3	37
Me2* (BNL# & NPI330-ME)	6	82									6	
Mgs1* (BNL#-MGS1)	10	49										
Mgs2* (BNL#-MGS2)	4	127										
Nabp1* (BNL#-NABP1)	7	26										
NCR#-b32c3A	8	78										
NCR#-b32c3B	7	83										
NCR#-b70	5	69										
NCR#-NRA	1	107										
NCR#-NRB	4	74										
NIU1-BS-1 (=UMC186A-BS-1)	8	65										
NIU2-BS-1 (=UMC186B-BS-1)	5	46										
NOR	6	0										
NPI#-ADP0											10	17
NPI#-ADP8											8	
NPI#-B1	2	77										
NPI#-PEP2											5	74
NPI#-PEP3											4	86
NPI1											8	113
NPI2											6	0
NPI3											8	107
NPI4											2	139
NPI6											4	20
NPI7											6	11
NPI8-BZ1											9	30
NPI9											6	148
NPI10											1	121
NPI11											2	90
NPI12											4	94
NPI13											3	80
NPI15-SH1											9	29
NPI16-WX1											9	50
NPI17											4	101
NPI17											9	75
NPI18											4	20
NPI19											1	113
NPI20											1	83
NPI21-ADH1											1	144
NPI22											10	68
NPI23											7	40
NPI24											1	182
NPI27											4	20
NPI28											7	5
NPI29											7	107
NPI30											7	16
NPI32											2	190
NPI33											8	113
NPI34											9	53
NPI35											7	87
NPI36											4	101
NPI37											8	70
NPI38											6	98
NPI39											8	113
NPI40											1	87
NPI41											5	58
NPI42											3	113
NPI43											8	113
NPI44											7	11
NPI45											7	107
NPI46											2	176
NPI47											7	23
NPI48											7	16
NPI49											2	116
NPI50											8	113
NPI51-A1 (& see A1*)											3	139
NPI52											3	90
NPI53											5	74
NPI54											1	124
NPI56											4	106
NPI57											2	51
NPI59											7	87
NPI60											5	95
NPI61											2	180
NPI63											6	118
NPI64											8	63
NPI67											6	109
NPI68											7	49
NPI69											8	124
NPI70											3	80
NPI71											3	0
NPI72											8	117
NPI73											4	20
NPI74											5	137
NPI75											5	18
NPI77											4	20
NPI78											3	99
NPI79											8	17
NPI80											9	57
NPI82											1	166
NPI83											3	70
NPI84											1	87
NPI85											10	56
NPI86											9	19
NPI88											3	125
NPI89											3	65
NPI90											3	84
NPI91											3	125
NPI92											10	62
NPI93											1	23
NPI94											3	99
NPI95											4	20
NPI96											1	83
NPI97	1	18	9	130							9	101
NPI98	6	42									9	118
NPI99											1	151
NPI100											6	5
NPI101											6	11
NPI102											6	109
NPI103											8	113
NPI104											4	113
NPI105	10	44	10	56	10	48					10	52
NPI106											1	34
NPI107											8	170
NPI108											3	80
NPI109	1	27									1	23
NPI110					8	20					8	35
NPI111											7	16
NPI112			7	44	7	12					7	28
NPI113	7	111			7	118					7	87
NPI114	8	0									8	0
NPI115											5	105
NPI116											5	63
NPI118	2	149									2	180
NPI119-HSP70											8	190
NPI120											1	118
NPI121-CSS1											9	63
NPI122											2	132
NPI123					2	152					2	128
NPI201											3	125
NPI202	3	65									3	61
NPI203											4	113
NPI204											8	132
NPI205											1	83
NPI206											8	63
NPI208	4	98									4	68
NPI209A	9	113	9	108	9	121					9	86
NPI209B	1	26										
NPI210					2	194					2	180
NPI211											9	11
NPI212			3	127	3	116					3	113
NPI213											5	58
NPI214					1	100					1	76
NPI215											3	156
NPI216					7	5					7	23
NPI217											7	40
NPI218											8	30
NPI219											3	61
NPI220	8	7	8	0	8	0					8	8
NPI221					2	151					2	151
NPI222											9	53
NPI223	6	60									6	62
NPI224											1	105

	BNL '91	POS '91	UMC '90	Jan '91	UMC '89	Apr '89	PIO '90	POS '90	AGR '90	POS '90	NPI	POS '90
NPI224A	7	24										
NPI224B	8	149										
NPI224C	8											
NPI224D	8											
NPI224E	7											
NPI224F	1											
NPI224G	1	105										
NPI224H	8											
NPI224I	3	32										
NPI225	1	158									1	144
NPI226											1	157
NPI227-SSU											4	86
NPI228-ADH2											4	2
NPI229-PDK											6	95
NPI230-PDK											8	70
NPI231-ME											3	37
NPI232					10	83					10	76
NPI233					5	97					5	70
NPI234					1	57					1	43
NPI235	6	10			6	0					6	18
NPI236	1	115			1	151					1	105
NPI237					5	140					5	117
NPI238	1	178	1	233	1	211					1	178
NPI239	2	5	2	0	8	26					2	0
NPI240											7	49
NPI241					1	215					1	166
NPI242	2	97										
NPI243											1	43
NPI244											2	90
NPI245											6	18
NPI246											1	118
NPI247											3	70
NPI248-B1											2	81
NPI249					3	32					3	47
NPI250											4	46
NPI251-PHY											1	157
NPI252											6	95
NPI253A	9	16									9	0
NPI253B	4	97										
NPI253C	5	122										
NPI253D	6	66										
NPI254	2	20									2	24
NPI255			1	217							1	124
NPI256											5	58
NPI257											3	125
NPI258											1	87
NPI259											4	20
NPI260											8	70
NPI261											6	148
NPI262			1	84							1	63
NPI263	7	68									7	53
NPI264	10	55									10	72
NPI265											6	78
NPI266											9	40
NPI267											4	20
NPI268	8	139									8	124
NPI269A	2	60									2	70
NPI269B	10	66										
NPI270					4	112					4	86
NPI271	2	94									2	95
NPI272											1	83
NPI273	1	29									2	95
NPI274											2	170
NPI275											5	63
NPI276											8	52
NPI277A					7	8					7	16
NPI277B					2	148						
NPI278A					2	191					2	180
NPI278B					7	131						
NPI279											1	83
NPI280					6	128					6	139
NPI281											1	43
NPI282											5	27
NPI283											7	49
NPI283	7	61									7	49
NPI284					4	52					4	20
NPI285	10	23	10	22							10	22
NPI286			1	58							1	48
NPI287					2	60					2	56
NPI288	5	132									5	137
NPI289											4	20
NPI290	10										10	92
NPI291	9	115									9	90
NPI292											4	86
NPI293											9	57
NPI294											2	41
NPI295											5	84
NPI296											3	84
NPI297	2	91									2	90
NPI298	2	147									2	176
NPI299											1	15

	BNL '91	POS '91	UMC '91	Jan '91	UMC '89	Apr '89	PIO '90	POS '90	AGR '90	POS '90	NPI	POS '90
NPI300											9	48
NPI301											8	
NPI302											6	
NPI303											10	56
NPI304											1	76
NPI305											5	22
NPI306	10	90									10	88
NPI307											6	
NPI309-ADP											1	105
NPI310-ADP (see Agp2)											2	176
NPI312											10	
NPI313											5	117
NPI314-ADP (see Bt2)											4	
NPI315											8	190
NPI316											7	11
NPI317											4	
NPI318-ADP											4	
NPI319-ADP											10	
NPI320											2	24
NPI321	10										10	101
NPI322											1	
NPI324											10	
NPI325											8	
NPI327											10	56
NPI328											8	151
NPI329											2	
NPI330-ME											6	
NPI331-SSU											2	116
NPI332											7	
NPI332-PEP											9	53
NPI333											4	113
NPI335											7	53
NPI337											2	151
NPI340											2	70
NPI340A	6	8										
NPI340B	4											
NPI341											2	
NPI343											9	11
NPI344-TPI3											8	52
NPI345-TPI4											3	61
NPI346-TPI5											5	117
NPI347-EMU											2	116
NPI348-ALR											2	128
NPI349-ALR											7	40
NPI350	10	115									10	104
NPI352											2	
NPI353											7	
NPI354											1	43
NPI356											2	116
NPI357											1	157
NPI358											3	70
NPI359											4	
NPI361											5	117
NPI361A	2	202										
NPI361B	7	20										
NPI361C	6	44									6	46
NPI361D	2	169										
NPI361E	7	25										
NPI361F	8	77										
NPI361G	4											
NPI361H	10											
NPI361I	1	198										
NPI361J	4	16										
NPI361K	2	169										
NPI362											5	117
NPI363											5	137
NPI364											3	65
NPI365											2	
NPI366											10	0
NPI367											2	
NPI368-ACT											8	
NPI369-PHY											5	22
NPI370-P1											1	43
NPI371											4	106
NPI372											8	52
NPI373	6	24									6	30
NPI375											7	40
NPI376											8	52
NPI377											6	30
NPI378											10	56
NPI379											3	70
NPI380											7	53
NPI381											1	
NPI382											8	
NPI383											4	20
NPI384											5	63
NPI385											7	53
NPI386											4	7
NPI387											6	30

	BNL POS '91		UMC Jan '91		UMC Apr '89	PIO POS '90	AGR POS '90	NPI	POS '90
NPI388								7	11
NPI390									101
NPI391								7	28
NPI392								7	87
NPI393								6	46
NPI394								7	40
NPI395								4	20
NPI396								4	20
NPI398								8	
NPI399								7	
NPI400	7	12	7	9				7	0
NPI400B	2	196							
NPI401								1	76
NPI402								2	56
NPI403	9	112						9	86
NPI404	1	32						1	23
NPI405								2	139
NPI406								1	15
NPI407								1	151
NPI408								5	74
NPI409	5	8						5	8
NPI410								4	86
NPI411	1	36						1	23
NPI412								5	105
NPI412-SOD								1	43
NPI413								2	170
NPI414	8	152						8	151
NPI415								1	0
NPI416								9	53
NPI417								2	7
NPI418								6	
NPI419								6	148
NPI419-SOD								7	94
NPI420								3	156
NPI421A	2	27						2	31
NPI421B	10	102							
NPI422								10	17
NPI423								1	34
NPI424								5	74
NPI425	3	158						3	160
NPI426								8	107
NPI427	9	96						9	68
NPI428								1	43
NPI429								1	83
NPI430								7	16
NPI431								7	70
NPI432								3	136
NPI433								7	81
NPI434								5	63
NPI435								7	46
NPI436								10	80
NPI437								10	72
NPI438	8	163	8	220				8	160
NPI439								1	43
NPI440								7	40
NPI441								1	118
NPI442								5	105
NPI443	9	101						9	76
NPI444								4	86
NPI445								10	62
NPI445B	10	42							
NPI446	3	54						3	61
NPI446								3	61
NPI447								1	113
NPI448								1	43
NPI449								5	79
NPI450									101
NPI451	4	161						4	128
NPI452								2	170
NPI453								1	63
NPI454								9	53
NPI455								7	49
NPI456	2	111						2	139
NPI457								3	156
NPI458								5	105
NPI459								7	40
NPI461								10	80
NPI463-SOD2*2								9	
NPI465								7	94
NPI467-A1								3	
NPI468-A1								2	139
NPI469-A1								5	137
NPI470-A1								7	11
NPI472-OEC								4	24
NPI473-OEC								2	70
NPI474-OEC								5	36
NPI475-OEC								7	40
NPI476-OEC								8	
NPI471-L3								2	70
NPI477-CAB								3	136
NPI478-CAB								7	53

	BNL POS '91		UMC Jan '91		UMC Apr '89	PIO POS '90	AGR POS '90		NPI	POS '90
NPI479-CAB									8	70
NPI480-O2									7	
NPI481-L2									1	22
NPI482-A1									1	76
NPI500									7	11
NPI553	3	66								
NPI557B	10									
NPI561									6	152
NPI562									5	105
NPI563									10	76
NPI564									7	11
NPI565									3	125
NPI566									1	105
NPI567									4	20
NPI569									5	36
NPI570									4	86
NPI571									5	74
NPI573									1	118
NPI574									4	24
NPI576									2	170
NPI577									2	24
NPI578	10								10	76
NPI579									5	22
NPI580									6	85
NPI580									9	55
NPI581									1	151
NPI582									10	76
NPI583									2	70
NPI584									4	20
NPI585									8	52
NPI587									2	56
NPI589									1	43
NPI590									8	96
NPI591									2	176
NPI593									4	128
NPI594									4	20
NPI595									8	70
NPI596									7	11
NPI597									6	152
NPI598									1	63
NPI599									8	96
NPI601									5	63
NPI602									10	62
NPI605									1	105
NPI606									6	11
NPI607									2	56
NPI608									6	85
NPI610									2	170
NPI611									7	100
NPI613									2	139
NPI614									1	0
NPI615									1	118
NPI616									6	85
NPI617									6	85
O2* (BNL# & NPI480-O2)	7	16							7	
Oec1* (NPI472 & UMC171A-OEC23)				4	6				4	24
Oec2* (NPI473-OEC)									2	70
Oec3* (NPI474-OEC)									5	36
Oec4* (NPI475-OEC)									7	40
Oec5* (NPI476-OEC)									8	
Orp1* (BNL# & UMC193A-ORP)	4	76	4	52						
Orp2* (BNL# & UMC193B-ORP)	10	41	10	73						
P1 (AGR# & BNL# & NPI370 & UMC185-P)	1	57						1	66	1	43
Pcr1* (BNL#-PCR1)	1	134								
Pcr2* (BNL#-PCR2)	5	47								
Pcr3* (BNL#-PCR3)	2	8								
Pcr4* (BNL#-PCR4)	7	21								
Pdk1* (NPI229 & UMC173-PDK)			6	105	6	76			6	95
Pdk2* (NPI230-PDK)									8	70
Pgd1	6	19	6	8				6	19	6	30
Pgd2	3	71								
Pep1* (NPI332-PEP)									9	53

	BNL '91	POS '91	UMC '91	Jan '91	UMC '89	Apr '89	PIO '90	POS '90	AGR '90	POS '90	NPI '90	POS '90
Pep2* (NPI#-PEP2)											5	74
Pep3* (NPI#-PEP3)											4	86
PGE#A4	8	97										
PGE#B5	5	43										
PGE#C2	1	158										
PGE#D1	3											
PGE#E7												
PGE#F8	1											
PGE2	8	59										
PGE3	7	72										
PGE4	9											
PGE14.1	4	112										
PGE132	1	7										
Pgm2	5	38	5	39			5	71			5	27
Phi1	1	171	1	241							1	167
Php1* (BNL#-PHP)	10	40										
Phy1* (BNL# & NPI251-PHY)	1	156									1	157
Phy2* (AGR# & BNL# & NPI369-PHY	5	34							5	26	5	22
PIO#-CSS1							9	58				
PIO#-SH1							9	13				
PIO155					9	63						
PIO1016					6	99						
PIO1025					4	126						
PIO1033					10	76						
PIO60005	10	41	10	64			10	49				
PIO60007	6	29					6	30				
PIO60012							5	95				
PIO100005							9	0				
PIO100005	9	18			9	8	9	0				
PIO100012			2	87	2	90	2	78				
PIO100014							5	122				
PIO100016							6	80				
PIO100017	5	140	5	190			5	207				
PIO100025							4	137				
PIO100033							10	68				
PIO100040							8	14				
PIO100080	3	144					3	188				
PIO150013							10	66				
PIO150018							5	101				
PIO150024							5	98				
PIO150033							3	158				
PIO150037							7	54				
PIO200005			2	138			2	98				
PIO200006							3	56				
PIO200017							2	107				
PIO200020			7	122			7	137				
PIO200026	3	101										
PIO200042			3	38			3	39				
PIO200044							1	167				
PIO200045	6	28					6	21				
PIO200052							9	47				
PIO200071							4	126				
PIO200075A	10	14	10	0			10	0				
PIO200075B	9	60										
PIO200508							3	96				
PIO200509							3	88				
PIO200511							3	84				
PIO200518							1	232				
PIO200521							3	163				
PIO200523	7	109					5	196				
PIO200527							6	8				
PIO200528							6	111				
PIO200531							5	145				
PIO200537							1	17				
PIO200554							9	60				
PIO200557							1	274				
PIO200558							3	87				
PIO200562							4	129				
PIO200563	7	72					4	200				
PIO200566							5	156				
PIO200568A							10	118				
PIO200568B							2	0				
PIO200569							6	133				
PIO200569A	7	50					7	38				
PIO200569B	2	130										
PIO200575							1	151				
PIO200576							3	77				
PIO200581A	7	0					2	159				
PIO200581B	2	194			7	0	7	0				
PIO200589							5	113				
PIO200593			7	103			7	111				
PIO200595							6	129				
PIO200597							4	77				
PIO200599	6	117					6	133				
PIO200603							1	28				

	BNL '91	POS '91	UMC '91	Jan '91	UMC '89	Apr '89	PIO '90	POS '90	AGR '90	POS '90	NPI '90	POS '90
PIO200608			4	155			4	159				
PIO200622							5	92				
PIO200626	10	5					10	2				
PIO200640							1	58				
PIO200644			1	142			1	156				
PIO200646							10	56				
PIO200654							1	137				
PIO200661							1	182				
PIO200668							1	188				
PIO200674							1	148				
PIO200682							1	144				
PIO200684							7	61				
PIO200689							1	32				
PIO200690A	7	107	7	107			7	114				
PIO200690B	7	26										
PIO200708							7	58				
PIO200713	4	31					4	24				
PIO200714							8	66				
PIO200715							5	109				
PIO200719							10	77				
PIO200725	4	30	4	0			4	24				
PIO200726	3	161	3	164			3	208				
PIO200728	7	130					7	145				
PIO200746							7	64				
PIO200793							8	137				
PIO200802							3	92				
PIO200854							6	13				
PIO200855			1	153			1	168				
PIO200870							1	199				
PIO200872							5	86				
PIO200898							5	73				
PIO200904							6	99				
PIO200909							5	193				
PIO1200026							5	161				
pKlamb+ (=Abp1)	3	68										
Pil (& BNL#-PL1)	6	55									6	62
Pr1											5	105
Pro1* (NCR#-b32c3A)	8	78										
R1* (BNL#-R1 & NPI308-R1 & UMC182-R1)	10	78	10	125							10	80
Rab17* (BNL#-RAB17)	6	80										
Rab30* (BNL#-RAB30)	1	15										
RNY1 (see Rab17)												
RPA1					6	66						
RPA2					3	194						
RPA3					4	110						
RPA5A					5	48						
RPA5B					1	179						
RPA6					1	179						
RPA7A			1	239	1	203						
RPA7B			5	7	5	15						
RPA8					9	88						
RPA9A-SSU					2	121						
RPA9B-SSU					4	112						
Rpl									10	20		
Sh1* (AGR# & BNL# & NPI15-SH1 & PIO#)	9	29					9	13	9	22	9	29
Sh2* (BNL#-SH2)	3	149										
Sod2* (NPI419-SOD)											7	94
Sod2*2 (NPI463-SOD)											9	
Sod4* (NPI412-SOD)											1	43
Ssu1* (BNL17.05 & RPA9B & NPI331-SSU)	4				4	112					4	86
Ssu2* (BNL# & RPA9A & NPI227-SSU)	2				2	121					2	116
stAc	10	47										
Su1									4	47	4	20
Sus1* = Css1*												
Tpi4	3	61										
trAc9705	1	28										
Tub1* (BNL17.04-TUB)	1	155										
Ubi1* (AGR1002A-UBI)									4	130		
Ubi2* (AGR1002B-UBI2)									5	63		

	BNL '91	POS '91	UMC Jan '91		UMC Apr '89		PIO '90	POS '90	AGR '90	POS '90	NPI '90	POS '90
UMC1					5	85						
UMC2A			3	175	3	185						
UMC2B			2	101	2	125						
UMC3	3	119			3	117	3	144				
UMC4			2	143	2	182	2	112				
UMC5A	2	122	2	126	2	43						
UMC5B	7	36										
UMC6			2	49	2	152						
UMC7			8	220	8	173	8	144				
UMC8A					1	68						
UMC8B					2	87			2	43		
UMC8C					2	120			2	80		
UMC8E					2	87						
UMC10			3	66	3	48	3	71				
UMC11			1	41	1	58	1	84				
UMC12			8	94	8	79	8	81				
UMC13					1	63	1	94				
UMC14					1	121						
UMC15	4	120	4	124	4	113	4	134				
UMC16			3	134	3	125	3	172				
UMC17					3	125	3	168				
UMC18					3	69						
UMC19	4	137	4	100	4	73	4	103			4	46
UMC20			9	46	9	70					9	53
UMC21			6	89	6	64	6	58				
UMC22					2	185						
UMC23A	1	117			1	148	1	177				
UMC23B							4	82				
UMC24-CAB			3	163	3	174						
UMC25-WX1			9	41	9	55						
UMC26			3	80	3	71	3	107				
UMC27			5	49	5	54						
UMC28	6				6	169			6	128		
UMC29A					1	75						
UMC30			8	136	8	131						
UMC31A	4	40	4	14	4	8	4	44	4	24	4	0
UMC31B	2	157										
UMC32A	3	11	3	0	3	0						
UMC32B	8	63										
UMC33	1	119			1	149						
UMC34			2	68	2	75	2	63				
UMC35	7	137	7	128	5	235						
UMC36A			2	174	2	226	2	153			2	81
UMC36B											2	90
UMC36C											2	116
UMC37	1	134			1	158	1	172				
UMC38			6	118	6	99						
UMC39	3	121	3	125	3	143						
UMC40			5	76	5	97						
UMC42A	4	68	4	58	4	45	4	86				
UMC42B	3	62										
UMC43	5	54	5	60	5	79	5	92				
UMC44A	10	72	10	121	10	93	10	85	10	85		
UMC44B	2	41										
UMC45			7	109	7	114						
UMC46	6		6	101	6	76	6	70				
UMC47	4	70	4	61	4	35	4	78				
UMC47B											3	104
UMC48	8	107			8	134						
UMC49	2	167	2	158	2	212						
UMC50	3	58			3	50						
UMC50B							1	203				
UMC51	5	113	5	123	5	179						
UMC52	4	113	4	134	4	120	4	135				
UMC53	2	23	2	13	2	11	2	11				
UMC54	5	107	5	119	5	171	5	143				
UMC55			2	110	2	136						
UMC56					7	48						
UMC57	10				10	93	10	88				
UMC58	1	99			1	127						
UMC59	6	23			6	17	6	12	6	13		
UMC60	3	73	3	112	3	96			3	99		
UMC61	2	50	2	53	2	57	2	52				
UMC62			6	148	6	155	6	122				
UMC63			3	154	3	169			3	145		
UMC64	10	46			10	68						
UMC65L	6	48	6	56	6	43	6	43	6	58		
UMC66			4	96	4	68	4	99				
UMC66A	5	25										
UMC66B	1	166										
UMC67			1	111	5	93						
UMC68			5	150	5	203	5	174				
UMC68B							5	0				
UMC69					5	22						
UMC70A					9	0						
UMC70B					9	8						
UMC70C					6	56						
UMC71					6	145						
UMC72A	5	36			5	30	5	55				
UMC72B	1	160			1	191						
UMC74					9	63						

	BNL '91	POS '91	UMC Jan '91		UMC Apr '89		PIO '90	POS '90	AGR '90	POS '90	NPI '90	POS '90
UMC75					1	185	1	51				
UMC76			1	35	1	47			1	46		
UMC78					2	33						
UMC80					7	118						
UMC81			9	44	9	63	9	49				
UMC82			3	116	6	147	3	137				
UMC83			1	175	1	160	1	194				
UMC84	1	201	1	283	1	223	1	278				
UMC85	6	7	6	0	6	1			6	0		
UMC86					1	224						
UMC87			4	15	4	4	4	0				
UMC88					2	185						
UMC89	8		8	106	8	76			8	61		
UMC90					5	32	5	47				
UMC91					7	114						
UMC92			3	53	3	46						
UMC93			8	145	8	140						
UMC94					9	49	1	5				
UMC95	9	89	9	67	9	88	9	76				
UMC96			3	168	3	174						
UMC97					3	45						
UMC98					2	154						
UMC102			3	71	3	61						
UMC103					8	32	8	26	8	19		
UMC104			5	181	5	213	5	212				
UMC105			9	28	9	44						
UMC106	1	157			1	196						
UMC107A			1	210	1	187	1	225				
UMC107B							5	62				
UMC108			5	140	5	192	5	167	5	108		
UMC109			9	0	9	1						
UMC110			7	71	7	41	7	70				
UMC111					4	160						
UMC113A							6	51				
UMC113B			9	14	9	21	9	15				
UMC114			9	50	9	77	9	51				
UMC115			1	23	1	34						
UMC116			7	45	7	22	7	43				
UMC117			8	134	8	120						
UMC119	1	97			1	125						
UMC120			8	67	8	51	8	54				
UMC121			3	14	3	18			3	15		
UMC122	2	145	2	146	2	173			2	118		
UMC124			8	53	8	37						
UMC125A			2	144	2	161						
UMC125B			7	78	7	57	7	86				
UMC126			5	117	5	159						
UMC127					9	59						
UMC128	1	133	1	171	1	157	1	190				
UMC129					1	176						
UMC130	10	38	10	53	10	59						
UMC131			2	93	2	106	2	82				
UMC132			6	137	6	137						
UMC133			4	117	4	108	1	131				
UMC134			6	164	6	168						
UMC135	2	108			2	84						
UMC136			7	41	7	16						
UMC137			2	149	2	177						
UMC138					6	103	6	88				
UMC139			2	117	2	154	2	89	2	98		
UMC140			1	201	1	173						
UMC141			5	126	5	179						
UMC142-ATP					5	188						
UMC143					5	0						
UMC144A					1	183						
UMC144B					5	37						
UMC145-HRG					2	87						
UMC146					10	76						
UMC147A	5	28	5	11	5	21						
UMC147B	1	167										
UMC149					7	48						
UMC150A					8	115						
UMC150B					2	197						
UMC150C					6	91						
UMC151					7	118						
UMC152					6	91						
UMC153	9	72			9	70						
UMC154	3	57			3	49						
UMC155	10	40			10	68						
UMC156	4	71			4	52						
UMC157	1	16	1	20	1	29						
UMC158	4	109			4	52						
UMC159	6				10	80						
UMC160					6	1						
UMC161			1	239	1	203						
UMC162					1	50						
UMC163			10	104	10	81						
UMC164			1	4	1	8						
UMC165A					3	87						
UMC165B					8	112						
UMC166			5	55	5	63						

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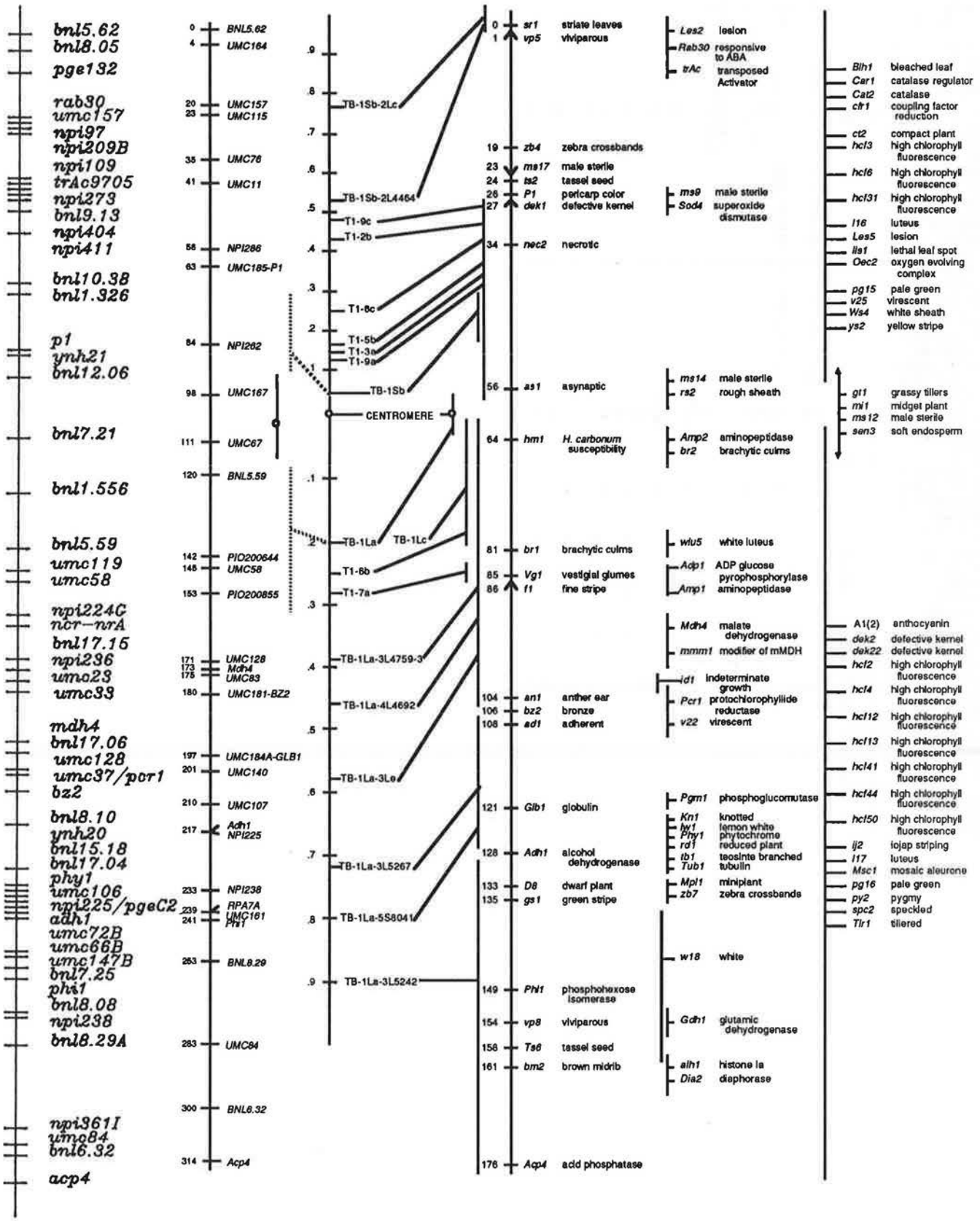
UMC167			1	98	1	100						
UMC168			7	123	7	101						
UMC169			4	171	4	174						
UMC170-DHN			6	99	6	70						
UMC171A-OEC23					4	6						
UMC171B-OEC23					2	206						
UMC172-OEC33			6	16	6	20						
UMC173-PDK			6	105	6	76						
UMC174-CABM2					8	51						
UMC175	3	66			3	48						
UMC176					2	128						
UMC177					6	169						
UMC180-PEP			6	85								
UMC181-BZ2			1	180								
UMC182-R1			10	125								
UMC184A-GLB1			1	197								
UMC184B-GLB			2	89								
UMC185-P			1	63								
UMC186A-BS-1 (= NIU1-BS-1)			8	67								
UMC186B-BS-1 (= NIU2-BS-1)			5	49								
UMC188-GPA1			10	97								
UMC189A-A1			8	161								
UMC190-CSS1			9	62								
UMC191-GPC1			4	53								
UMC192-BZ1			9	17								
UMC193A-ORP1			4	52								
UMC193B-ORP2			10	73								
UMC193C-ORP			7	25								
UMC999A							3	180				
UMC999B							4	75				
Vp1* (BNL#-VP1)	3	76										
Whp1* (BNL17.03-C2)	2	166										
Wx1* (AGR# & BNL# & NPI16 & PIO# & UMC25-WX1)	9	56	9	41	9	55	9	43	9	47	9	50
Y1									6	22	6	33
YNH#-ME1	3	40										
YNH20	1	151										
YNH21	1	58										
Zein15B* (AGR1003-ZEINB)									6	4		
Zein27G* (AGR1004-ZEING)									7	30		
Zp15*	6	18										
Zp50	7											
ZpB36*	7	32										
ZpL1a	4	29										
ZpL1b	4	31										
ZpL1c	4	31										
ZpL1d	4	34										
ZpL1e	4	30										
ZpL1f	4	33										
ZpL2a	4											
ZpL2b	7	21										
ZpL3a	4											

CHROMOSOME 1



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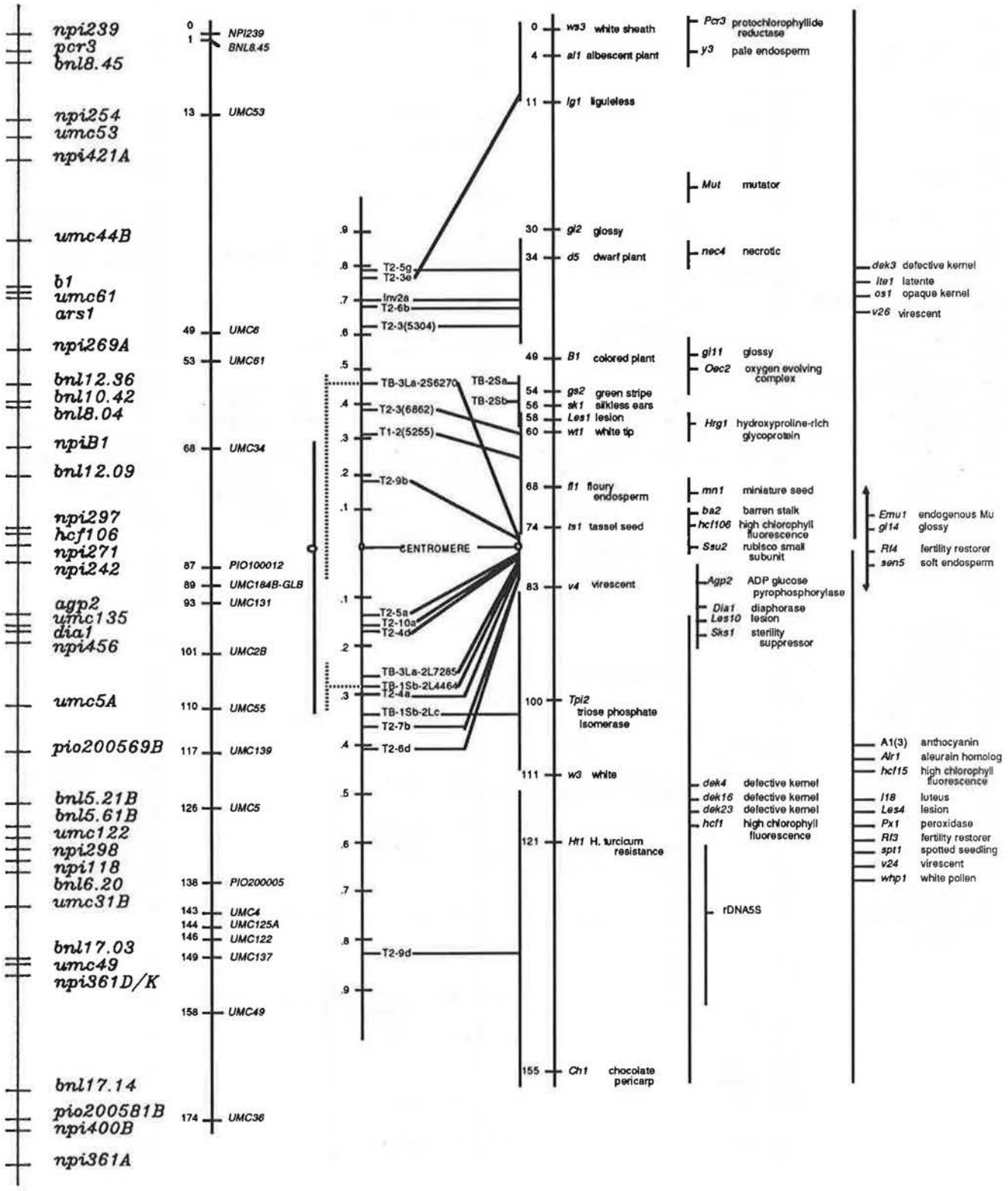

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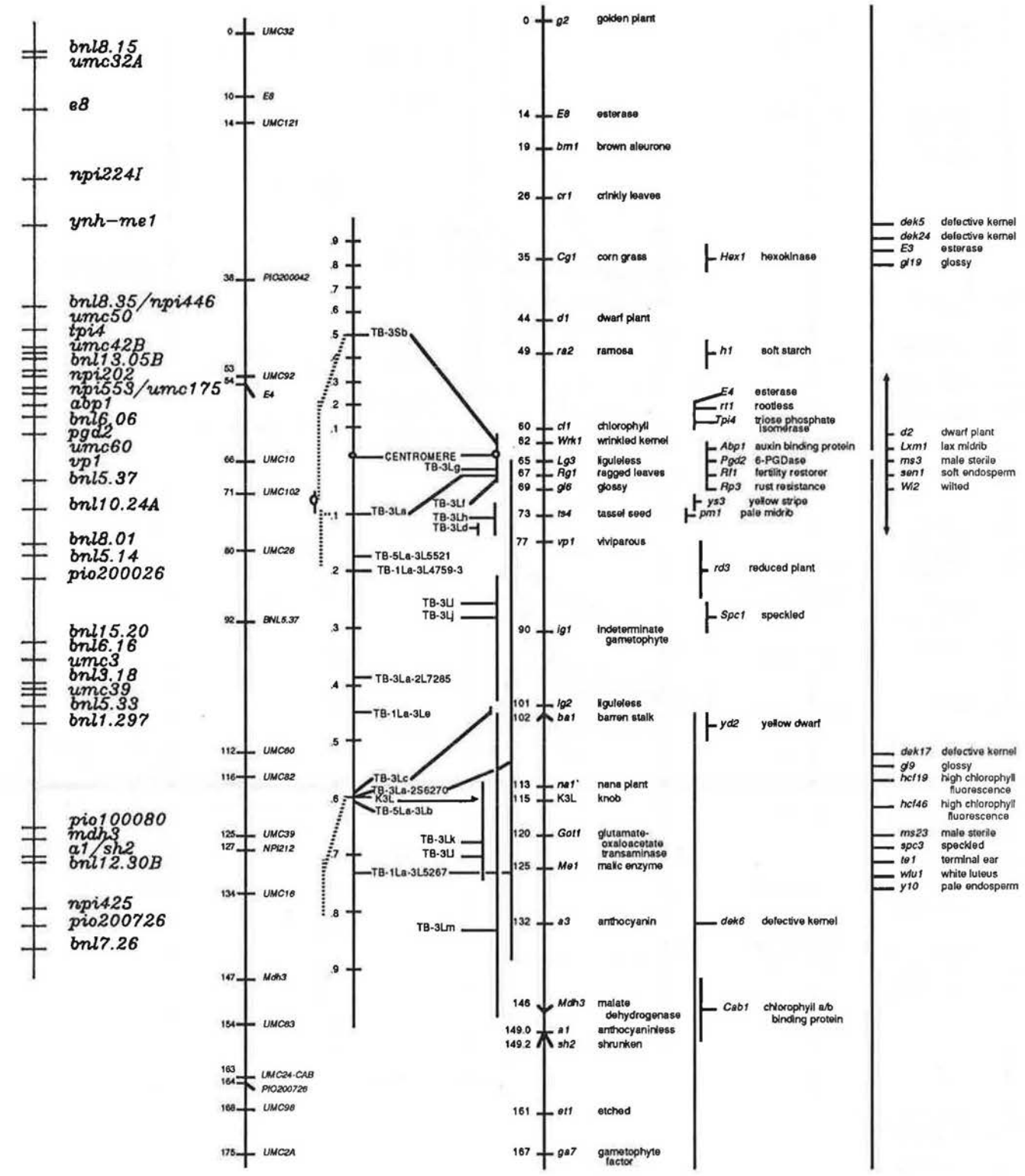

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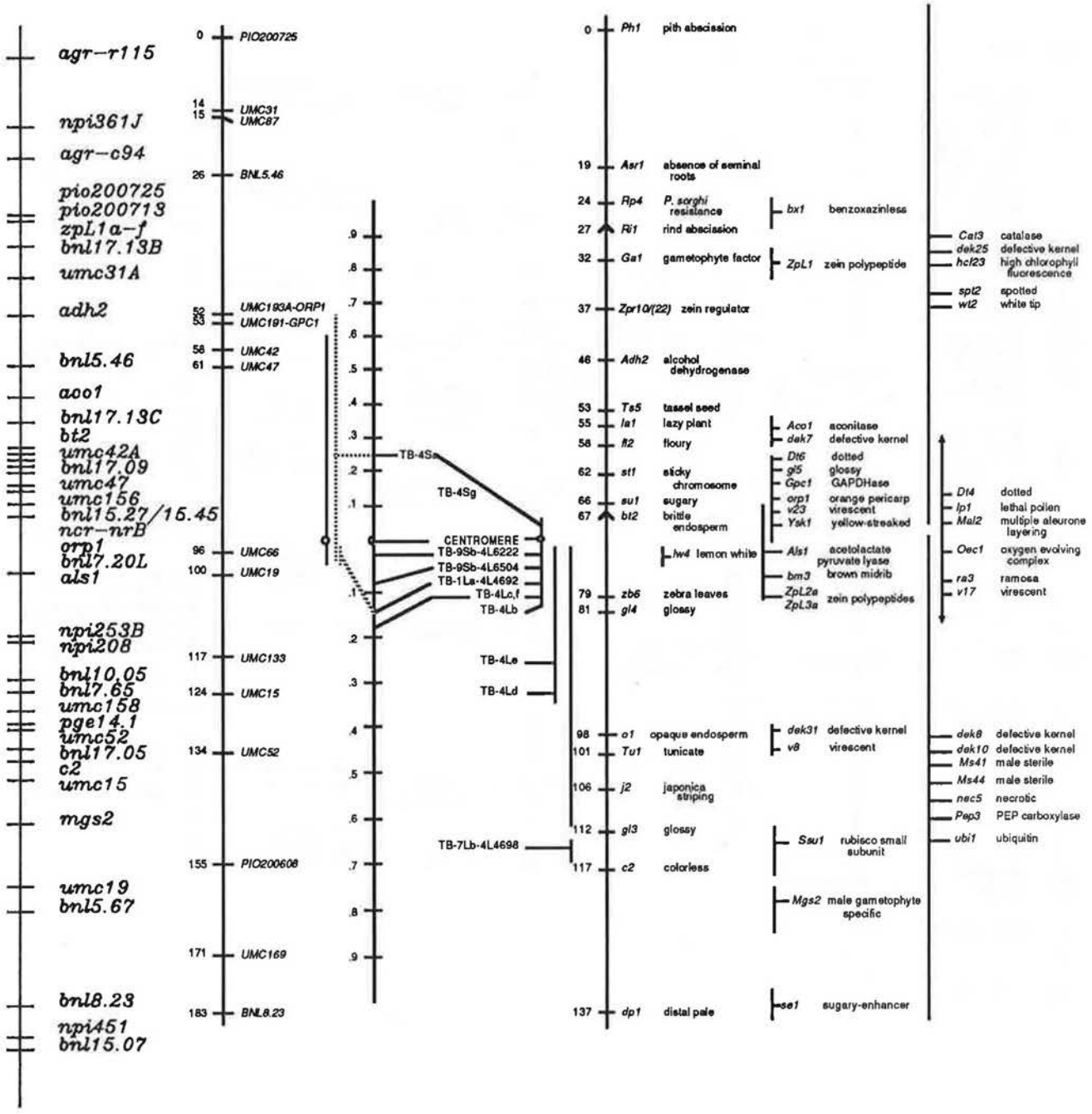

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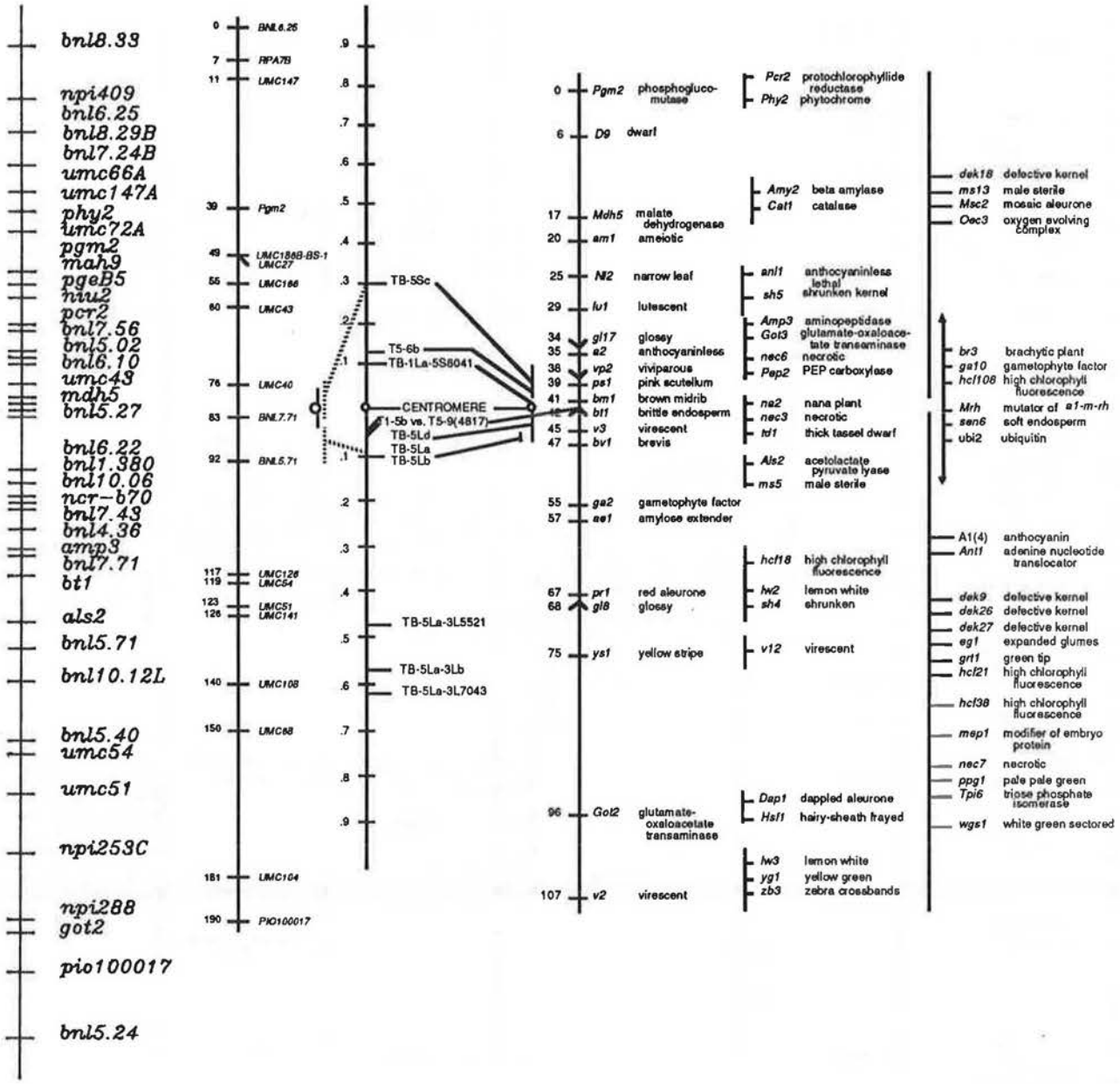

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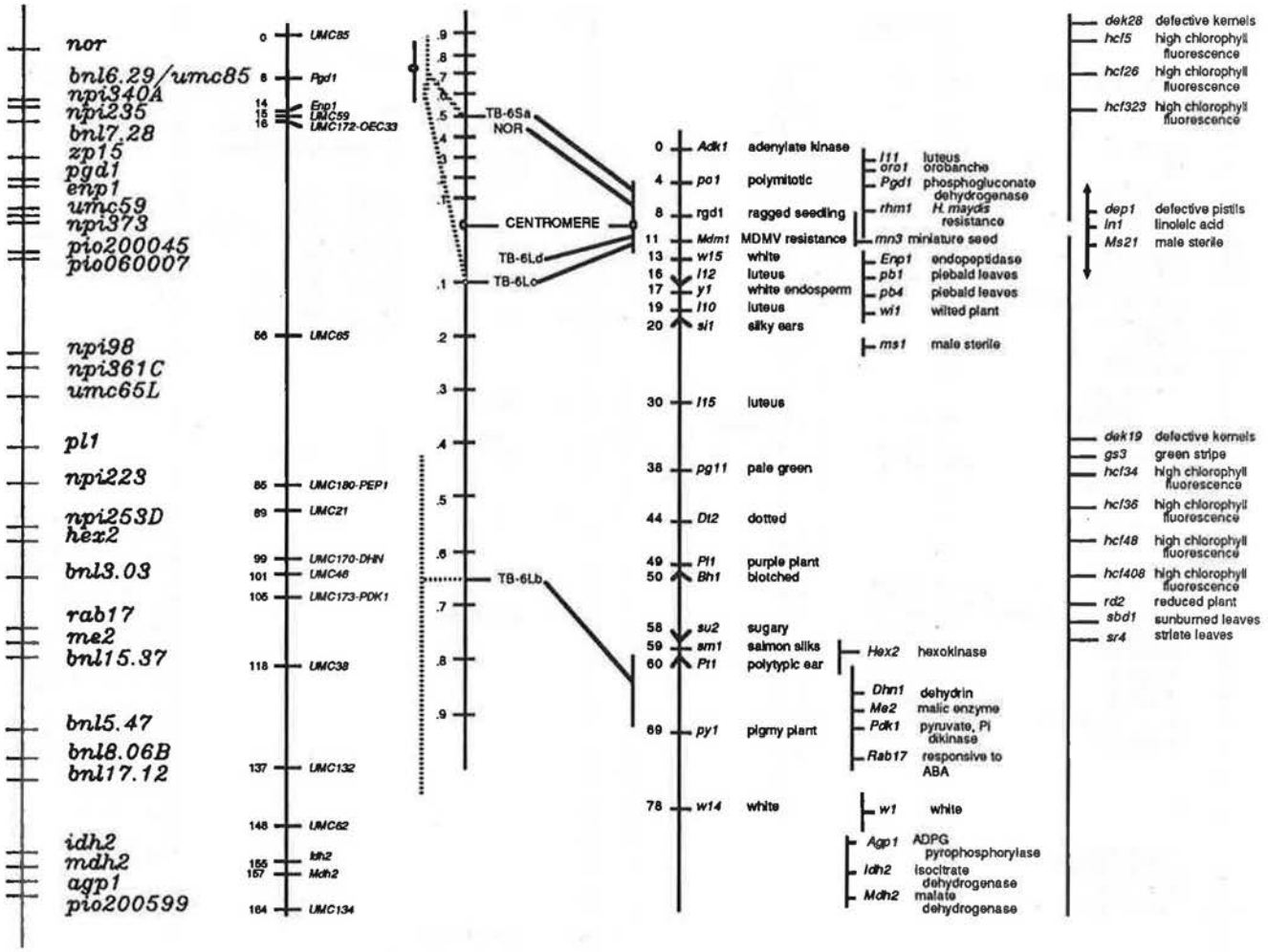

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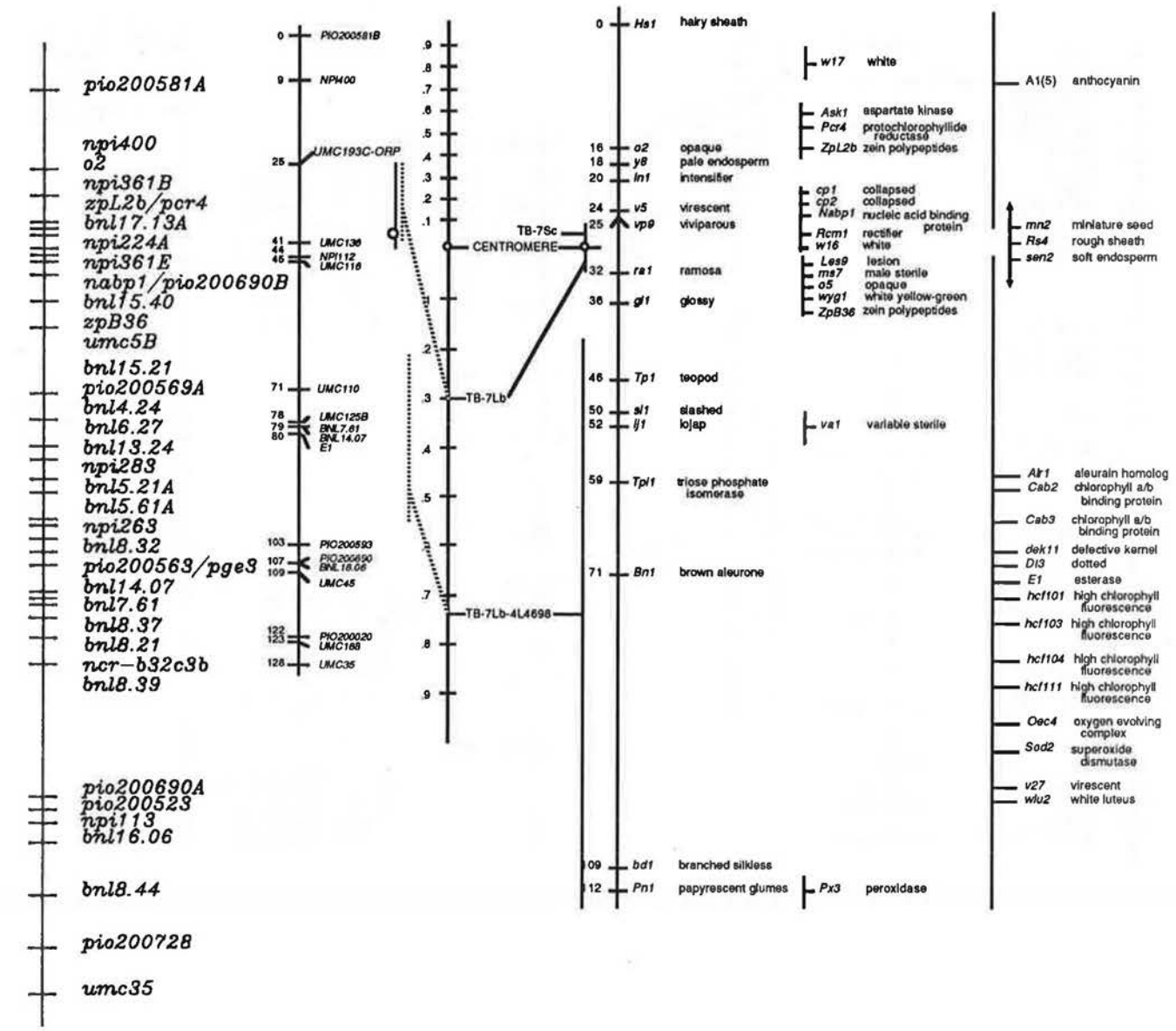

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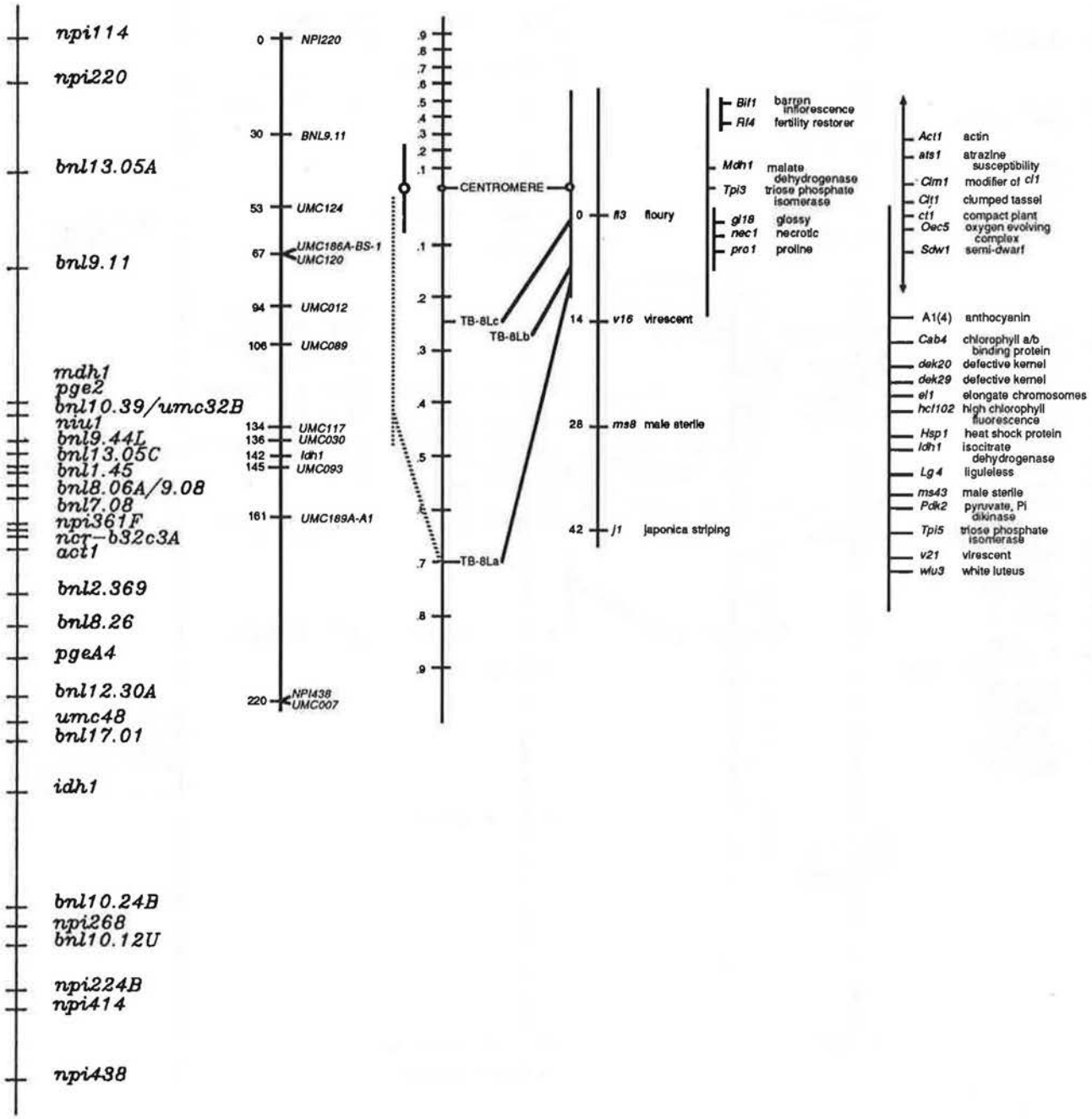
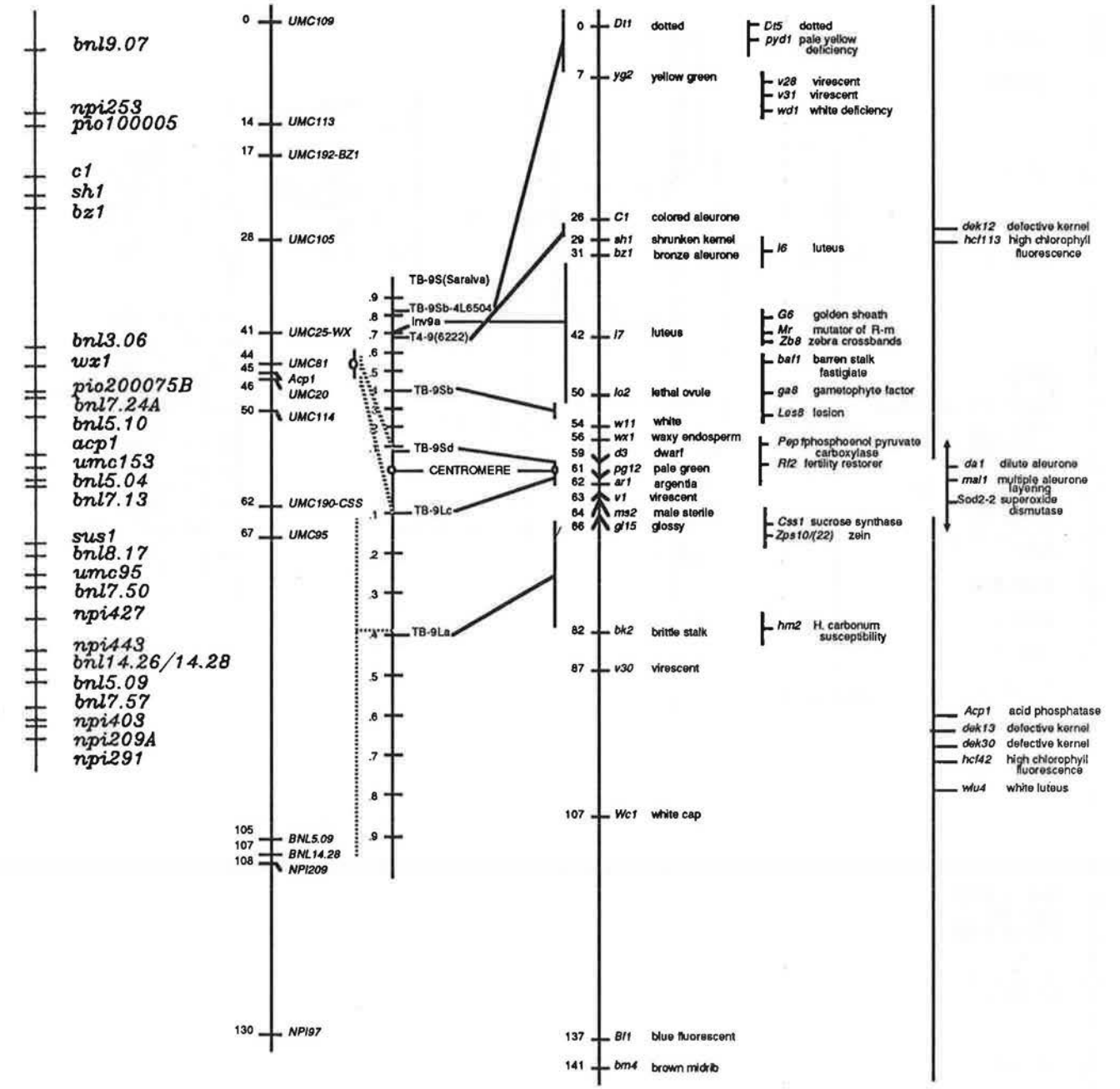


CHROMOSOME 9



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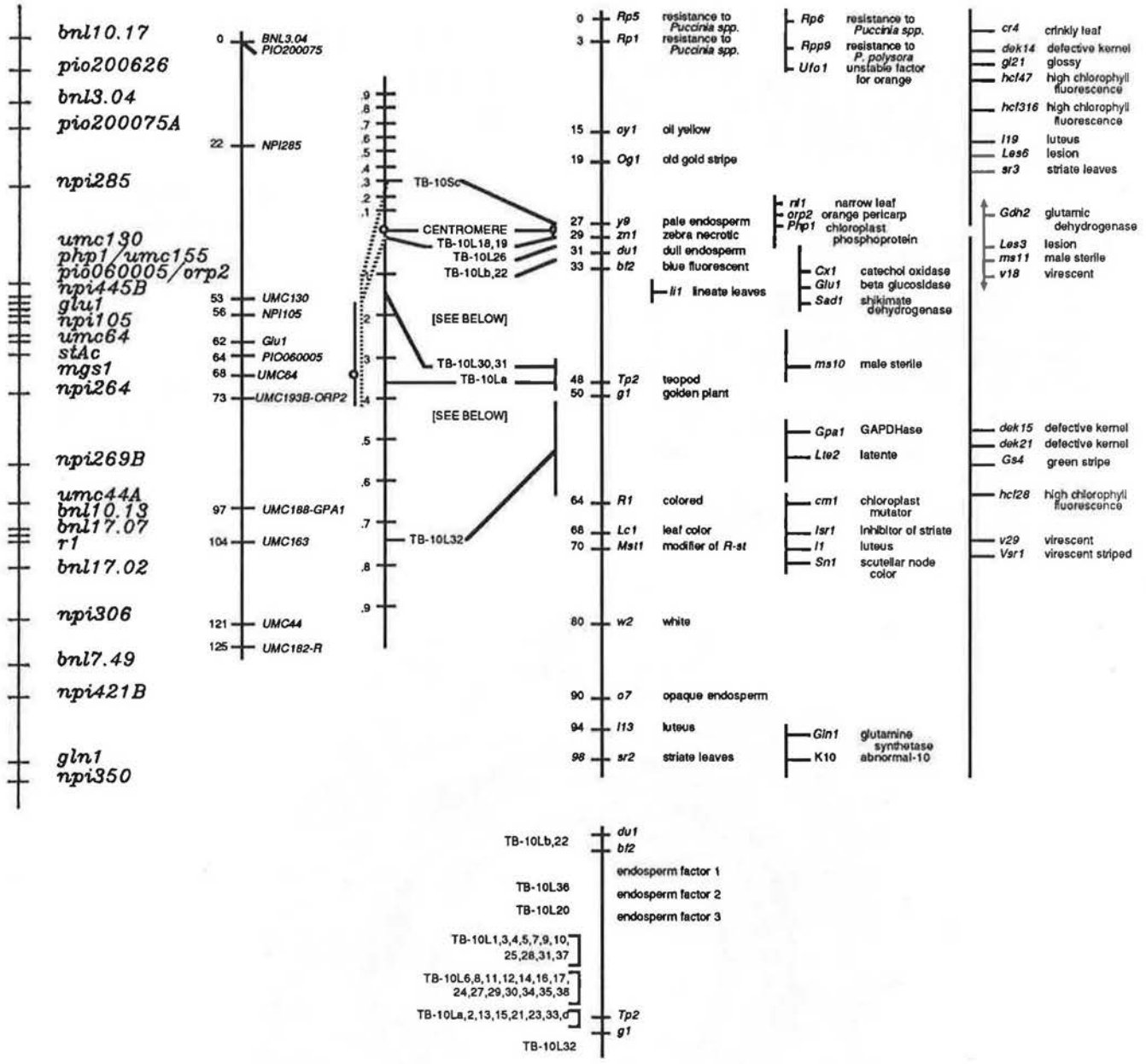

GENETIC MAP OF THE *ZEA MAYS* PLASTID CHROMOSOME

Steven Rodermel, Department of Botany, Iowa State University, Ames, IA 50010

The complete sequences of seven maize plastid genes have been reported in the past year. Their locations are shown on the map below, and their gene products are briefly described in the following table. The sequence of the plastid chromosome is @75% complete.

See the 1987-1989 News Letters for descriptions of other sequenced genes: **MNL 62**:148; **MNL 63**:155; **MNL 64** :164.

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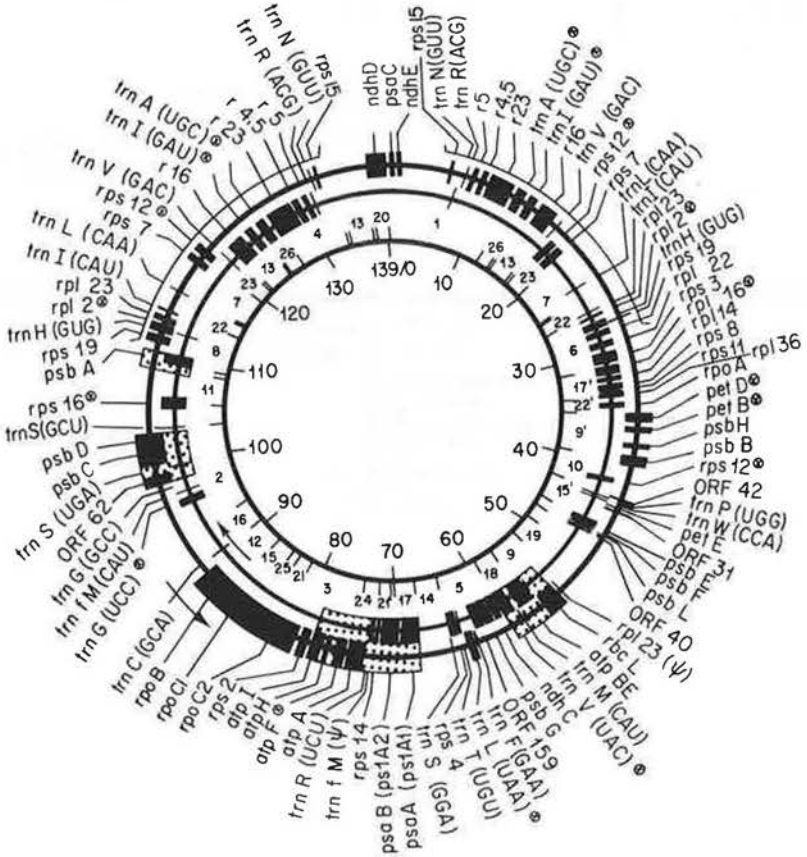
Stahl, D., S.R. Rodermel, A.R. Subramanian and L. Bogorad. 1990. Nucleotide sequence of a 3.46 kb region of maize chloroplast DNA containing the gene cluster *rpo*C2- *rps*2- *atp*I- *atp*H. **Nucleic Acids Res. 18**: 3073.

MAIZE PLASTID GENE SEQUENCES REPORTED IN 1990

GENE PRODUCT	GENE	REFERENCE
ATP Synthetase:		
Subunit IV of CF_0	*atp*I	Stahl et al., 1990
70S Ribosomal Proteins:		
S2	*rps*2	Stahl et al., 1990; Igloi et al., 1990
S15	*rps*15	Fitzky and Subramanian, 1990
L2	*rpl*2	Kavousi et al., 1990
Plastid RNA Polymerase:		
B subunit	*rpo*B	Igloi et al., 1990
B' subunit	*rpo*C_1	Igloi et al., 1990
B" subunit	*rpo*C_2	Igloi et al., 1990; Stahl et al., 1990



VI. MAILING LIST

Abbott, Albert G., 338 Long Hall, Clemson University, Clemson SC 29634-1903
Abler, Brenda S., Dept of Genetics, North Carolina State Univ, Box 7614, Raleigh NC 27695-7614
Agrawal, B. D., G. B. Pant Univ Agr Tech, Pantnagar 263145, INDIA
Agrema, Hesham A., 42 Mumphis Street, Camp Caesar, Alexandria 21321 5970525, EGYPT
Aguiar-Perecin, Margarida L. R., Depto Genetica - ESALQ/USP, CP 83, Piracicaba SP 13.400, BRAZIL, 0194-33-0011, ext 125
Ainsworth, Thomas C., Ainsworth Seed Company, Mason City IL 62664, 217-482-3281
Albertsen, M. C., Pioneer Hi-Bred Internatl, P. O. Box 258, 7250 NW 62nd Ave, Johnston IA 50131, 515-270-3648
Alexander, Larry, Sigco Research, Box 289, Breckenridge MN 56520, 218-643-2155
Alfenito, Mark R., 16 Divinity Ave., Harvard Biol. Labs., Cambridge MA 02138
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
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, Joint FAO/IAEA Division, IAEA, Wagramerstrasse 5 , P.O. Box 200, A-1400 Vienna, AUSTRIA, FAX: (43)-1-234564
Anderson, Joe, Institute Biol. Chem., Washington State University, Pullman WA 99164-6340, (509)335-1958
Andrew, R. H., Department of Agronomy, University of Wisconsin, Madison WI 53706, 608-262-2587
Anghel, I., Biol-Lab Genet Molec, Univ Bucuresti, Aleea Portocalelor nr.1-3, Bucuresti 77206, ROMANIA
Antonelli, N. Michele, Dept. of Genetics, Iowa State University, Ames IA 50011
Armstrong, Katherine, Dow Chemical Company, Building 1701, Midland MI 48640, 517-636-9270
Armstrong, Charles L., Monsanto, GG4H, 700 Chesterfield Village Pkwy, St. Louis MO 63198, 314-537-7229, FAX: 314-537-6567
Arnold, Jerry M., Asgrow Seed Co., 9866 Highway 66, Newburgh IN 47630
Aromose, O. S. A., Biological Sciences Dept, University of Benin, Benin City, NIGERIA
Arruda, P., Univ. Estadual de Campinas, Cidade Univ. Zeferino Vaz, Distrito de Barao Geraldo, Campinas - SP, BRAZIL
Arthur, Gary D., Holden's Foundation Seeds Inc, RR 1, Box 149, Franklin IN 46131, 317-535-8357
Ashman, R. B., Dept of Botany, Purdue University, W. Lafayette IN 47907, 317-494-4634
Atherly, Alan G., 8 Curtiss Hall, Genetics Dept, Iowa State Univ, Ames IA 50011
Athma, Prasanna, Cold Spring Harbor Lab, P.O. Box 100, Cold Spring Harbor NY 11724
Atkinson, Burr, Univ. of Western Ontario, Dept. of Plant Sciences, Biological & Geological Bldg, London, CANADA
Attewell, John, McCurdy Seed Co., 522 East Main St., Fremont IA 52561, 515-933-4292
Auger, Donald L., Biology Department, Box 8238, University Station, University of North Dakota, Grand Forks ND 58202, 701-777-3674
Ayala-Osuna, Juan, Depto Agrar Vet Fac Cienc, Universidade Estad Paulista, Rodovia Carlos Tonnani, Km 5, 14870 Jaboticabal SP, BRAZIL
Aycock, Harold S., 8 Meadow Lane, Athens OH 45701
Ayers, J. E., Dept Plant Pathology, 211 Buckhout Lab, Pennsylvania State Univ, University Park PA 16802, 814-865-7776, FAX: 814-863-7217
Badenhorst, Coerie, Saffola, Box 36, Boskop 2528, SOUTH AFRICA
Baier, John W., 1255 Fifield Hall, University of Florida, Gainesville FL 32611
Bailey-Serres, Julia, Botany & Plant Sciences, Batchelor Hall, Univ. Calif. Riverside, Riverside CA 92521, (714)787-3738
Baker, Raymond, Pioneer Hybrid Seed Co, Johnston IA 50131, 515-278-2108
Baker, Barbara, USDA, Plant Gene Expression Center, 800 Buchanan Street, Albany CA 94710, 415-559-5912
Banks, Jody, 1205 Docteur Penfield Avenue, Dept of Biology, McGill University, Montreal, PQ H3A1B1, CANADA, 301-467-1414
Banuett, F., Dept Biochem Biophys, Univ of California, San Francisco CA 94143-0448, 415-476-4985, FAX: 415-476-0943
Bar-Tsur, Avri, Dept Maize Breed/Genet, Newe Yaar Exper Sta 31999, PO Haifa, ISRAEL, 04-933-186
Barkan, Alice, Institute of Molecular Biology , University of Oregon, Eugene OR 97403
Barry, Edward G., Dept of Biology, CB# 3280, Univ of North Carolina, Chapel Hill NC 27599, 919-962-6933
Barry, B. Dean, 243 Ag. Engr., University of Missouri, Columbia MO 65211, 882-1116
Bass, Hank W., Botany Dept, Box 7612, North Carolina State Univ, Raleigh NC 27695-7612, 919-737-3570
Baszczynski, C. L., Allelix Inc, Agric Division, 6850 Goreway Drive, Mississauga, Ont L4V 1P1, CANADA, 416-677-0831
Bates, Lynn S., Alteca Ltd., 731 McCall Road, Manhattan KS 66502, 913-537-9773
Baulcombe, David, Cytogenetics, Plant Breeding Inst., Cambridge CB2 2LR, UNITED KINGDOM
Baxter, Donald Ray, Curtis Hall, University of Missouri, Columbia MO 65211, 314-882-3856
Bayliss, Michael W., Imperial Chem Ind PLC, Internatl Seeds Business, Jealott's Hill Research Stn, Bracknell Berks RG12 6EY, UNITED KINGDOM, 0344-424701
Beach, Larry R., Pioneer Hi-Bred Internatl, Department of Biotechnology, Box 38, 7300 NW 62nd Ave, Johnston IA 50131, 515-270-3633
Bear, Robert P., Noble Bear, Inc, PO Box 950, Decatur IL 62525, 217-422-5621
Beavis, W. D., Pioneer Hi-Bred Int., Box 85, Johnston IA 50131
Beckendorf, Joy, Dept Soil & Crop Science, Texas A & M University, College Station TX 77843
Beckert, M., INRA Station D'Amelioration des Plantes, 63039 Clermont Ferrand, Cedex, FRANCE
Beckett, Jack, Curtis Hall, University of Missouri, Columbia MO 65211, 314-882-8214
Beckman, Diana, Northrup King Co., Route 19, Stanton MN 55081, 507-645-5621
Becraft, Philip, Genetics Department, Univ of California-Berkely, Berkeley CA 94702, 415-642-7948
Bedinger, Patricia, Dept of Biol, 010A Coker Hall, U of North Carolina, Chapel Hill NC 27599-3280
Behr, Fritz, 222 W. Pearl St., Geneseo IL 61254, (309)944-6500
Belanger, Faith, 1102 S Goodwin Street, Urbana IL 61801
Bendbow, Evelyn, Curtis Hall, Univ of Missouri, Columbia MO 65211
Bennett, M. D., Jodrell Laboratory, Royal Botanic Gardens, Kew, Richmond, Surrey, TW9 3AB, UNITED KINGDOM, 81-940 1171, FAX: 81-948 1197
Bennetzen, Jeff, Dept of Biological Sciences, Purdue University, W. Lafayette IN 47907, 317-494-4763, Maize@edu.purdue.bio.bilbo
Benson, Dirk L., E. J. Funk & Sons, Inc., 601 Funk Pkwy., Kentland IN 47951
Benton, W. D., Genbank c/o Intelligenetics, 700 E. El Camino Real, Mountain View CA 94040, 415-962-7360

Benzion, G., 303 W. Lanvale St., Baltimore MD 21217, 301-529-3386
Bergquist, Richard R., Plant Pathologist, 401 East Sixth Street, El Paso IL 61738, 309-527-6000
Bergstrom, Gary C., 316 Plant Sci Bldg, Cornell University, Ithaca NY 14853, 607-255-7849
Berlyn, Mary, Dept. of Biology, Yale University, New Haven CT 06520, 203-432-3536, 203-432-5145
Berman, Judith, Dept. of Plant Biology, 220 Bio Science Center, Univ. of Minnesota, St. Paul MN 55108, 612-625-1971
Bernardo, Rex, Lifaco Seed Co., Box 6055, Champaign IL 61821, (217)398-8968
Bertaux, Philippe, Lifaco Seed Corp, PO Box 278, Kirkland IL 60146, 815-522-3246
Berthaud, Julien, CIMMYT, Lisboa 27 , Aptdo. Postal 6-641, 06600 Mexico, D. F., MEXICO
Bertrand, Ralph L., Univ. of CA - Berkeley, Dept. of Plant Biology, 111 Genetics Plant Bio. Bldg., Berkeley CA 94720, 415-642-7948
Berville, A., INRA, Station d'Amelior des Plantes, Lab de Genet et Mutag, BV 1540, 21034 Dijon Cedex, FRANCE
Bhave, Mrinal R., Univ. of Florida, Dept. of Plant Path., % 934 S.W. 16th Avenue, Gainesville FL 32601, 904-392-7237
Bianchi, Angelo, Ist Sper Cerealicoltura, Via Cassia, 176, 00191 Rome, ITALY, 06-3285705
Bigbee, Kevin, Lifaco, RR1 Box 29A, S. Amana IA 52334
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, 919-787-8452
Bjarnason, Magni, CIMMYT, Apdo. Postal 6-641, 06600 Mexico, D.F., MEXICO
Blakey, Cynthia Ann, Curtis Hall, University of Missouri, Columbia MO 65211, 314-882-2033
Bocanski, Jan, Faculty of Agriculture, University of Novi Sad, 21000 Novi Sad, YUGOSLAVIA
Bodeau, John, Dept Biol Science, Stanford University , Stanford CA 94305-5020, (415)723-2609
Bogorad, L., Biol Labs, Harvard Univ, 16 Divinity Ave, Cambridge MA 02138, 617-495-4292
Bohning, Kermit, Cargill Research, 311 River Ave North, P.O. Box 307, Belmond IA 50421
Bokde, S., 3400 Dana Drive, Burnsville MN 55337
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, 519-679-2111 ext. 6467, FAX: (519)661-3935
Bongard-Pierce, Deverie K., Univ. of CA - Berkeley, Dept. of Genetics, 345 Mulford Hall, Berkeley CA 94720, 415-642-8058
Boothe, Joe G., Plant Biotechnology Institute, Saskatoon, S7N 0W9, CANADA
Borman, Dan, Box 254, Deer Harbor WA 98243
Borner, T., Sekt Biol, Humboldt Univ, Invalidenstr 43 DDR-1040, Berlin, FEDERAL REPUBLIC OF GERMANY
Boston, Rebecca S., Box 7612, Dept Botany, North Carolina State Univ, Raleigh NC 27695-7612, (919)737-2727, FAX: (919)737-3436
Bouchard, Robert A., 2006 Blair Blvd., Wooster OH 44691, 216-263-2026
Bouck, Dan, Box 4741, Agway Inc Crop Serv, Syracuse NY 13221
Bowen, Benjamin, Pioneer Hi-Bred Internatl, 7300 NW 62nd Avenue, Johnston IA 50131, (515)270-3647, FAX (515)270-3444
Boyer, C., Dept Hort Penn State Univ, 103 Tyson Building, University Park PA 16802, 814-863-3371
Brady, Thomas, Room 321, 1800 G St. NW, Washington D.C. 20550
Brakke, M., Dept of Plant Path, Univ of Nebraska-Lincoln, Lincoln NE 68583, 402-472-3166
Brar, G. S., Agracetus Corp, 8520 University Green, Middleton WI 53562, 608-836-7300
Brawn, Robert I., 15 Douglas Drive, Ayr, Ontario NOB 1E0, CANADA, 519-632-7954
Bredenkamp, C., Northrup King Co., Route 19, Stanton MN 55081
Brettell, R., CSIRO Div Plant Industry, P.O. Box 1600, Canberra City ACT 2601, AUSTRALIA, 062-46-5581
Bretting, Peter K., Indiana Crop Impr Assoc Inc, 3510 U.S. 52 South, Lafayette IN 47905, 317-474-3494
Brewbaker, James, Horticulture, Univ of Hawaii, 3190 Maile Way, Honolulu HI 96822, 808-948-7985
Briggs, Steven, Pioneer Hi-Bred Internatl, P.O. Box 38, 7250 NW 62nd Ave., Johnston IA 50131
Briggs, Robert W., Funk Seeds International, 1300 W Washington Street, P.O. Box 2911, Bloomington IL 61702-2911, 309-829-9461
Brinkman, Mark, 1203 Agronomy Hall, Iowa State University, Ames IA 50011-1010
Britt, Anne Bagg, Plant Growth Lab., U of CA, Davis CA 95616
Brown, R. P., Ohio Foundation Seeds, Croton OH 43013, 614-893-2501
Brown, Steven, Northrup King Research Center, Stanton MN 55081, (507)663-7633
Brown, D., Dept of Biology, Bishops University, Lennoxville J1M1Z7 Quebec, CANADA, 819-822-9632
Browne, C., Curtis Hall, University of Missouri, Columbia MO 65211, 314-882-2674
Bruder, Karin, Inst Getreidef Bernburg-Hadmer, Mitschurinstr. 22, Bernburg, FEDERAL REPUBLIC OF GERMANY
Brunklaus-Jung, E., Ludwig-Maximillians Univ., Botanisches Institut, Mezingerstr.67, 8000 Munchen 19, FEDERAL REPUBLIC OF GERMANY, 89-1792200, fax: 89-338297
Bubeck, David, Crop Sci. Dept., P.O. Box 7620, NC State Univ., Raleigh NC 27695, 919-737-2704
Buckner, Brent, Div. Science, Science Bldg, NE Missouri State Univ., Kirksville MO 63501, 816-785-4181
Buescher, Pat, Custom Farm Seed, Box 160, Momence IL 60954, 815-472-2433
Bullock, W. Paul, 424 Oliver Avenue, Ames IA 50010
Burgess, Diane, DNA Plant Technology, 6701 San Pablo Avenue, Oakland CA 94608, 415-547-2395
Burmood, T., Jacques Seed Company, 720 St. Croix Street, Prescott WI 54021, 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, burr@bnlux1.bnl.gov
Butnaru, Gallia, Institutul Agronomic Timisoara, Disciplina de Genetic, C. Post 136; O.P. 1, Timisoara 1900, ROMANIA
Byrne, Patrick, CIMMYT, Lisboa 27, Apdo. Postal 6-641, 06600 Mexico, D.F., MEXICO
Callaway, M. Brett, 604 Bradfield Hall, Dept of Plant Breeding, Cornell Univ, Ithaca NY 14853
Callis, Judy, Dept of Biochem & Biophysics, Univ of CA, Davis CA 951616, 916-752-1015, FAX: (916)752-3085
Camara-Hernandez, J., Altolaguirre 1295, Buenos Aires 1427, ARGENTINA, 51-6464
Cambolive, M., GIE Pioneer-France, Epuiseau, 41290 Oucques, FRANCE
Campbell, Wilbur H., Dept. of Biological Sciences, Michigan Technological Univ., Houghton MI 49931, 906-487-2214, FAX:906-487-2245
Campbell, Gerald, S.L.C. Communications, 11815 Spruce Orchard Drive, St. Louis MO 63146
Cande, Zac, Dept of Molec & Cell Biology, Box 341 LSA, Univ of California, Berkley CA 94720, (415) 642-1669, FAX: 415-643-6791
Capelaro, Ademir, CIBA GEIGY QUIMICA S/A, R. Eduardo Marquez, 56, Caixa Postal, 585, Uberlandia-MG 38.400, BRAZIL, (034) 236-6520
Carlson, Peter S., Crop Genetics International, 7170 Standard Dr., Hanover MD 21076

Carlson, Wayne, Botany Department, University of Iowa, Iowa City IA 52242, (319)335-1316
Carlson, Lawrence A., 7 N. Winthrop Street, St. Paul MN 55119, 612-738-8812
Carlson, John E., Biotechnology Lab, 6174 Univ Boulevard, Univ of British Columbia, Vancouver, BC V6T1W5, CANADA, 604-228-4733
Carolina Bio Supply Co, Rainey Bldg. Library, 1308 Rainey St., Burlington NC 27215
Carson, Chris, Univ FL, Vegetable Crops Dept, 1255 Fifield Hall, Gainesville FL 32611
Caruso, John L., Dept Biol Sci, University of Cincinnati, Cincinnati OH 45221
Cavanaugh, Kevin, Agronomy, Lilly Hall, Purdue Univ., West Layfayette IN 47907
Chairman, Agronomy Dept, 210 Waters Hall, University of Missouri, Columbia MO 65211, 314-882-2801
Chandlee, Joel M., Dept Plant Science, Univ Rhode Island, Kingston RI 02881, 401-792-2529
Chandler, Vicki, Department of Biology, Institute of Molecular Biology, University of Oregon, Eugene OR 97403, 503-346-5136
Chang, Ruying, Agronomy Dept, Iowa State Univ, Ames IA 50011, 515-294-7883
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, Shiaoman, Dept. of Agronomy, Curtis Hall, University of Missouri, Columbia MO 65211, 314-882-3698
Chapa, J. A., Escuela de Agron. y Zootecnia, Universidad de Guanajuato, Apdo. Postal 311, Irapuato GTO, MEXICO
Chase, Sherret S., Chase Road, P.O. Box 193, Shokan NY 12481, 914-657-2392
Chase, Christine D., Vegetable Crops Dept, 0514 IFAS, Univ of Florida, Gainesville FL 32611, 904-392-7920
Chaudhary, V. K., Dept Genetics, Rajendra Agr Univ, Bihar, Pusa (Samastipur)-848125, INDIA
Chaudhary, D. N., Pioneer Overseas Corporation, G/F, Renaissance Bldg., 215 Salcedo St., Legaspi Vlg., Makati, Metro Manila, PHILIPPINES
Chaudhuri, Sumita, Rutgers State Univ, Waksman Institute, P.O. Box 759, Piscataway NJ 08855
Chen, Che-Hong, Sogetal, Inc., 3876 Bay Center Place, Hayward CA 94545, 415-785-1881
Chen, Weicheng, Dept. of Agronomy, Henan Agric. College, ZhengZhou, CHINA
Chen, Jychian, Institute of Molec Biol, Taipei, TAIWAN, 02-789-9208, FAX 02-782-6085
Cheng, Ping-chin, Dept Elect & Comp Engineering, 317 Farber Hall, State University of New York, Buffalo NY 14214, (716)636-3868
Chilton, Scott, Dept of Botany,, Box 7612, N. C. State University, Raleigh NC 27695-7612
Chismark, Stacie, Dept Biol Sciences, Illinois State Univ, Normal IL 61761
Choe, Bong-Ho, Agronomy Dept, College of Agriculture, Chungnam National University, Dae-Jon 300-31, KOREA
Choi, Keun-Jin, Dept. of Corn Breeding, Upland Crop Div., Crop Experiment Station, Suwon, KOREA
Chojecki, Jan, Imperial Chem Industries PLC, Jealott's Hill Research Sta, Bracknell Berks RG12 6EY, UNITED KINGDOM, 344-414814, FAX: 344-414818
Chomet, Paul, 345 Mulford Hall, Genetics Dept, Univ California, Berkeley CA 94720
Chou, Tau-San, Geo. J. Ball, Inc., P.O. Box 335, West Chicago IL 60185, 708-231-3600
Chourey, Prem, USDA-ARS, Plant Pathology Department, University of Florida, Gainesville FL 32611, 904-392-7237
Christensen, Dean W., Ciba-Geigy Seed Div., P.O. Box 18300, Greensboro NC 27419, 919-547-1017
Chughtai, Sajjad R., Maize Program, NARC, P.O. NIH, Islamabad, PAKISTAN, 82005226
Civardi, Laura, Dept. Genetics, Iowa State Univ., Ames IA 50011, (515)294-0337
Clark, Jan, Purdue University, Dept of Biological Sciences, W. Lafayette IN 47906, 317-494-4919
Clark, Raymond L., USDA Plant Introduction Sta, Iowa State University, Ames IA 50010, 515-294-3255
Close, Timothy J., Dept. Bot. & Plant Sci., Univ. of California Riverside, Riverside CA 92521, 714-787-3318, FAX: 714-787-4437
Clutter, Mary, BBS, National Science Foundation, 1800 G Street, N. W., Washington D. C. 20550
Cobb, B. Greg, Texas A & M University, Dept. of Hort. Science, College Station TX 77843, 409-845-8615
Cocciolone, Suzy, Tucker Hall, University of Missouri, Columbia MO 65211, (314)882-1168
Coe, E. H., 206 Heather Lane, Columbia MO 65203, , Agrocoe@UMCVMB
Coe, E. H., Curtis Hall, University of Missouri, Columbia MO 65211, 314-875-5349, FAX: 314-875-5359, Bitnet: Agrocoe@UMCVMB
Colasanti, Joseph J., Cold Spring Harbor Lab, P.O. Box 100, Cold Spring Harbor NY 11724, 516-367-8469, FAX: 516-367-8369, colasant@cshl.org
Colbert, Terry, Funk Seeds International, P.O. Box 921, Union City TN 38261, 901-885-7538
Colless, J. M., Agric Research Sta, Grafton, NSW 2460, AUSTRALIA, 066-447210
Cone, Karen C., Biological Sciences, University of Missouri, Columbia MO 65211, 314-882-2118
Conrad, Peter L, Dept of Biol Sciences, State Univ of New York, Plattsburgh NY 12901, 518-564-5271
Cook, Bill, 308 Tucker, University of Missouri, Columbia MO 65211, 314-882-3481
Cooper, Pamela S., Univ. of Missouri , Div. of Biological Science, Columbia MO 65211, 314-882-8033
Coors, James G., Department of Agronomy, University of Wisconsin, 1575 Linden Drive, Madison WI 53706, 608-262-7959
Cormack, Jean, Garst Seed Co., Highway 210, P.O. Box 500, Slater IA 50244, 515-685-3574
Cornu, A., Sta d'Amelior des Plantes, B V 1540, 21034 Dijon, FRANCE, 80-237221
Corriveau, Joseph, Dept. of Agronomy, Univ. of Maryland, College Park MD 20742
Coruzzi, Gloria M., Lab Plant Mol Biology, Rockefeller Univ, 230 York Ave., New York NY 10021
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 Ctr, USDA-ARS, 1815 N. University Street, Peoria IL 61604, FTS 360-4011, 309-685-4011 EXT 371
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 Haas 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., Highway 210, P.O. Box 500, Slater IA 50244, 515-685-3574
Cross, John, Pfizer Central Research, Dept. of Plant Genetics, Groton CT 06340
Cross, Harold, Dept Crop & Weed Sci, North Dakota State Univ, Fargo ND 58102, 701-237-8138
Cruz-Alvarez, Marilyn, Rutgers State Univ., Waksman Institute, P.O. Box 759, Piscataway NJ 08855
Cuany, Robin L., Department of Agronomy, Colorado State University, Fort Collins CO 80523, 303-491-6832
Cummings, D. P., Dekalb-Pfizer Genetics, Box 367, Windfall IN 46076, 317-945-7121
Curtis, Christine A., Dept of Botany & Plant Science, Univ of California, Riverside CA 92521, (714)787-3946

Cutter, Gary L., Cargill, Inc, 324 Troy Street, Covington OH 45318, 513-473-5265
D'Halluin, Kathleen, Plant Genetic Systems, Lab Gent, Jozef Plateaustraat 22-B 9000, Gent, BELGIUM, (32) (97) 24255, (32) (97) 255473
D.M.L. Associates, Inc., 420 Lexington Avenue, Room 406, New York NY 10170
Da Silva, W. J., Genet Evol, Univ Campinas, Cidad Univ Barao Geraldo, Campinas SP, BRAZIL
Dales, David, RR #1, Petrolia, Ont. N0N 1R0, CANADA, 519-882-2847
Dallmier, Donald J., Sturdy Grow Hybrids Inc, Box 94, Arcola IL 61910, 217-268-3838
Damerval, Catherine, Station de Genetique Vegetale, Ferme du Moulon, 91190 Gif Sur Yvette, FRANCE, (1)69 41 67 24, FAX: 33(1)69 41 27 90
Dankov, Toma, Ivan Assen Str.-93, Sofia 1124, BULGARIA, 43-82-73
Darrah, L., 110 Curtis Hall, University of Missouri, Columbia MO 65211, 314-882-7707
Das, Prem, Rutgers, The State Univ., Waksman Institute, P. O. Box 759, Piscataway NJ 08855
Dash, Sudhansu, G-412 Agronomy, Ames IA 50011, 515-294-7883
Davis, David W., Horticultural Science Dept, 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, Peter R., Center for Ag Molec Biology, Cook College, Rutgers Univ, Env. & Nat'l. Sci Bldg. P.O. 231, New Brunswick NJ 08903-0231, 908-932-8165, FAX: 908-932-6535
Day, Janet, Dept Plant Biol, 289 Morrill, University of Illinois, 505 S. Goodwin, Urbana IL 61801, 217-333-2919
De Francisci, B., Dept. of Genetics, Univ. of California, Berkeley CA 94720
De Jong, A. W., Zelder B. V., Postbus 26, 6590 AA GENNEP, NEDERLAND
De Leon, Carlos, CIMMYT, P.O. Box 9-188, Bangkok 10900, THAILAND
De Mars, Sandra, Agrigenetics Advanced Sci. Co., 5649 E. Buckeye Road, Madison WI 53716, 608-221-5000
Dellaporta, Steve, Department of Biology, P.O. Box 6666, Yale University, New Haven CT 06511
DeLong, Alison, Yale University, Biology Dept OML 350, P.O. Box 6666, New Haven CT 06511, 203-432-3894
Demopulos, J. T., U. of Alabama-Birmingham, UAB/Immunogenetics Program, Birmingham AL 35294, 205-934-2516
Dempsey, Ellen, Biology Dept, Indiana University, Bloomington IN 47401, 812-855-4061
Dennis, Elizabeth, CSIRO, Division of Plant Industry, P.O. Box 1600, Canberra, ACT 2601, AUSTRALIA
Deutsch, James A., CIMMYT, Lisboa 27, Apdo. Post. 6-641 , 06600 Mexico D. F., MEXICO
DeWald, Steve, Sandoz Crop Protection, Dept Plant Biotechnology, 975 California Ave , Palo Alto CA 94304-1104
Dhillon, B. S., Dept. of Plant Breeding, Punjab Agricultural University, Ludhiana 141 004, INDIA
Di Fonzo, Natale, Ist Sper Cereal, Via Stezzano 24, 24100 Bergamo, ITALY, 035-313132
Dilworth, Machi, USDA ARS OGPS, Room 112, W. Auditors Bldg., 15th & Independence Ave., SW, Washington DC 20251
Dodd, J. L., Professional Seed Research,Inc, 7 South 437 Dugan Road, Sugar Grove IL 60554, 708-466-1060, Fax 708-466-1068
Doebley, John F., Plant Biology Department, University of Minnesota, St. Paul MN 55108, Fax:612-625-3702, jdoebe@uminn1
Doehlert, Douglas C., USDA/ARS N. Regional Res. Ctr., 1815 N University Street, Peoria IL 61604
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
Dombrink-Kurtzman, Mary Ann, Northern Regional Res. Center, USDA, ARS, 1815 N. University St., Peoria IL 61604, (309)685-4011
Dooner, Hugo K., DNA Plant Technology Corp., 6701 San Pablo Avenue, Oakland CA 94608, 415-547-0291
Doring, H.-P., Inst Genetik, Univ Koln, D-5000 Koln 41, Weyertal 121, FEDERAL REPUBLIC OF GERMANY, 0221-4702465
Dotson, Stanton, Dept of Agronomy & Pl Genetics, Borlaug Hall, 1991 Buford Circle, St. Paul MN 55108
Dowe, Mike E. Jr., Dept Biochemistry, Spaulding Life Sci Bldg, Univ New Hampshire, Durham NH 03824
Dowling, Edward J., C/O Jacques Seed Co, RR #2, Lincoln IL 62656, 217-735-2770
Dowty, Jean L., Dept. Plant Sciences, Forbes Bldg #36, Univ. of Arizona, Tucson AZ 85721, (602)621-1977, JDOWTY@ARIZRVAX
Doyle, G. G., Curtis Hall, University of Missouri, Columbia MO 65211, 314-882-8215
Dudley, Mark, Plant Science Institute, Dept of Biology, University of Pennsylvania, Philadelphia PA 19104-6018, (215)898-8915
Dudley, John W., 1102 S Goodwin Ave, University of Illinois, Urbana IL 61801, 217-333-9640
Duncan, Dave, Dept Agron, S-332 Turner Hall, University of Illinois, 1102 S. Goodwin, Urbana IL 61801, 217-333-9465
Duvick, Jonathan P., Pioneer Hi-Bred Internatl, Dept of Biotechnology Res, 7300 N.W. 62nd Ave, Box 38, Johnston IA 50131, 515-270-3176
Duvick, Donald N., P.O. Box 446, Johnston IA 50131, 515-276-2923
Earle, Elizabeth D., Dept Plant Breed & Biom, Cornell University, 516 Bradfield, Ithaca NY 14853-1902, 607-255-3102
Earp, David , Plant Gene Expression Center, 800 Buchanan St., Albany CA 94710, (415)559-5919
Eberhart, Steve, USDA ARS Natl Seed Storage Lab, Colorado State Univ, Fort Collins CO 80523, 303-484-0402
Echt, Craig, Dept. of Plant & Soil Sciences, Montana State Univ., Bozeman MT 59717, 602-621-3381
Edwards, Marlin, Green Giant, 1201 N. 4th Street, Le Sueur MN 56058, 612-665-3515
Eggleston, Bill, Laboratory of Genetics, University of Wisconsin, Madison WI 53706, 608-262-3286
Eichholtz, David A., Monsanto, AA3E, 700 Chesterfield Village Pkwy, St. Louis MO 63198, 314-537-6227
Ellingboe, A. H., Dept. Plant Pathol., Univ. Wisconsin, Madison WI 53706
Elliott, Beth, Cold Spring Harbor Lab, P. O. Box 100 , Cold Spring Harbor NY 11724, 516-367-8330
Emons , A. M. C., Dept. Plant Cytology, Wageningen Agricultural Univ., Arboretumlaan 4, 6703 B D Wageningen, NETHERLANDS
Empig, L. T., c/o MIAC, 230 Gentry, University of Missouri, Columbia MO 65211
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
Epp, Melvin D, Genetics & Tissue Culture, 6560 Trinity CT, Arco Plant Res, Dublin CA 94568
Erber, Mike, National Corn Growers Assoc., 1000 Executive Parkway, Suite 105, St. Louis MO 63141, 314-275-9915, FAX: 314-275-7061
Esen, Asim, Dept Biology, Va Polytech Inst & State Univ, Blacksburg VA 24061, 703-961-5894
Eubanks, Mary W., 4110 Hulon Drive, Durham NC 27705, (919)490-5380
Everett, Herbert L., Cornell University, 520 Bradfield Hall, Ithaca NY 14853, 607-255-1667
Evola, S. V., CIBA-GEIGY Corporation, P.O. Box 12257, Research Triangle Park NC 27709-2257, 919-549-8164
Fahey, Jed, Crop Genetics International, 7170 Standard Drive, Hanover MD 21076, 301-379-0030
Falbel, Tanya, Box 347, MCD Biology, University of Colorado, Boulder CO 80309, 303-492-8893

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, 801-581-5192
Fedoroff, Nina, Carnegie Inst Wash, 115 West Univ Parkway, Baltimore MD 21210, 301-467-1414
Feiler, Heidi, 413 W. Main #3, Urbana IL 61801, 217-333-1518
Feix, G., Institut Fur Biologie III, Universitat Freiburg, Freiburg, FEDERAL REPUBLIC OF GERMANY, 0761-2032724
Fergason, V., Custom Farm Seed, 2773 N Main, Decatur IL 62526, 217-875-2826
Ferl, R. J., Dept Vegetable Crops, Univ Florida, Gainesville FL 32611, 904-392-2370
Filion, W. G., Erindale Campus Biology, University of Toronto, 3359 Mississauga Road, Mississ., Ont. L5L1C6 LL5L1C6, CANADA , 416-828-5326
Filner, P., 2050 Concourse Drive, San Jose CA 95131-1818
Fincher, Robert R., Pioneer Hi-Bred Internatl, Dept of Biotechnology Research, P.O. Box 38, Johnston IA 50131, 515-270-3650
Fink, Gerald R., Whitehead Inst. Biomed. Res., Cambridge Center, Cambridge MA 02142
Flick, Christopher E., EniMont America Inc., 2000 Cornwall Rd., Monmouth Junction NJ 08852, , FAX:908-422-0084
Flores, Catalina, Ceres Int. A.P. 484, Los Mochis, Sinaloa, Mexico 81200, MEXICO, 681-2-1502
Foley, Robert, RPA-C Associates, 505 W. Saginaw, P.O. Box 54, Breckenridge MI 48615
Foley, Terry, Holden's Foundation Seeds Inc, P.O. Box 839, Williamsburg IA 52361, 319-668-1100
Fowler, Tom, Ohio State Biotechnology Ctr, 244 Rightmire Hall, 1060 Carmack Road, Columbus OH 43210, 614-292-3349
Fowler, John, Freeling Lab, 111 GPBB, Dept. Plant Biology, Berkeley CA 94720, (415)642-8058
Fowler, John, Department of Genetics, 345 Mulford Hall, University of California, Berkeley CA 94720, 415-642-8058
Fraley, Robb, Monsanto, BB3B, 700 Chesterfield Village Pkwy, St. Louis MO 63198, 314-537-6204
Francis, T. R., Northrup King Co., R. R. 1, Arva, N0M 1C0, CANADA, 519-659-5130
Frankel, Rafael, Volcani Center, POB 6, Bet Dasan 50 250, ISRAEL
Freeling, Michael, Dept of Genetics, Univ of California, Berkeley CA 94720, 415-642-7085
Frei, Mark, Northrup King Seed, Via Comparina 2, Madignano 26020, ITALY, 065-381797
Freymark, Peter J., 146A University Village, Ames IA 50010, 515/296-7500
Friedman, Robert B., American Maize-Products Co , 1100 Indianapolis Blvd, Hammond IN 46320-1094
Friedrich, James W., Maize Genetic Resources, Inc, 421 Angier Highway, Benson NC 27504, 919-934-9908
Frisch, David, Department of Agronomy, University of Minnesota, St. Paul MN 55108, 612-625-5215
Fromm, Michael, Monsanto Co. AA2G, 700 Chesterfield Village Pkwy, St. Louis MO 63198, 314-537-6048, FAX: 314-537-6480
Fuerstenberg, Susan, Botany Dept - U of M, 220 Biological Sciences Building, 1445 Gortner Avenue, St. Paul MN 55108
Fujiyama, Kazuhito, ICBiotech, Faculty of Eng., Osaka University, Yamada-oka 2-1, Suita-shi, Osaka 565, JAPAN, 06-877-5111 Ext.4392, Fax: 81-6-876-9036
Gabay-Laughnan, S., Plant Biology/289 Morrill Hall, University of Illinois, 505 S. Goodwin Avenue, Urbana IL 61801, 217-333-2919
Gain, Jeffrey E., National Corn Growers Assoc., 1000 Executive Parkway, Suite 105, St. Louis MO 63141
Galinat, Walton C., Eastern Agric. Center, U. Mass., 240 Beaver Street, Waltham MA 02154-8096, 617-891-0650, FAX 617-899-6054
Gallais, A., G.I.S. du Moulon, Ferme du Moulon, 91190 Gif sur Yvette, FRANCE
Garcia, Ramon, EniChem Americas Inc., 2000 Princeton Park Corp Ctr, Monmouth Jct. NJ 08852
Garcia-Olmedo, F., Dept Biochemistry, E T S Ingenieros Agronomos, 28040-Madrid, SPAIN, 1-24444807
Gardiner, Jack, Curtis Hall - Agronomy, University of Missouri, Columbia MO 65211, 314-882-2033
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
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, 41-61-697-4457, Fax: 41-61-697-6194
Geadelmann, J. L., Holden's Foundation Seeds, Inc, Rt. 1, Box 112, Stanton MN 55018, 507-263-3476
Gebauer, Juan E., Corn Breeding, Casilla 190, Buin, CHILE
Geiger, Hartwig H., Univ Hohenheim, Inst Pflanzenzucht, Postfach 700562(350), D-7000 Stuttgart 70, FEDERAL REPUBLIC OF GERMANY
Genetics Library, Curtis Hall, University of Missouri, Columbia MO 65211
Gengenbach, B.G., Agron & Plant Genetics, Univ of Minnesota, St Paul MN 55108, 612-625-6282
Georgiev, Trifon, Technical University, "A. Kanchev", Rousse 7004, BULGARIA, 082-23469
Gerdes, James, 405C Eagle Hts, Madison WI 53705, 608-233-6730
Getschman, R. J., Dekalb Plant Genetics, Box 408, Dayton IA 50530, 515-547-2550
Gierl, Alfons, Max-Planck Inst., Egelspfad, D-5000 Koln-30 Vogelsang, FEDERAL REPUBLIC OF GERMANY, 49-221-5062-242
Gillies, Christopher, Botany Bldg A12, Univ of Sydney, Sydney NSW 2006, AUSTRALIA, 02-692-2688
Gilna, Paul, Genbank T10 Mailstop K710, Los Alamos Natl. Lab., Los Alamos NM 87544
Giroux, Michael, Veg Crops Dept, Univ of Florida, IFAS, Gainesville FL 32611
Glover, David V., Dept of Agronomy, Purdue University, W. Lafayette IN 47907, 317-494-8067
Godshalk, E. Brent, Dekalb-Pfizer Genetics, 3100 Sycamore Road, Dekalb IL 60115, 815-758-9372
Goff , Steve, USDA-ARS-PGEC, 800 Buchanan Street, Albany CA 94710
Goldberg, Robert, UCLA Biology Dept, 405 Hilgard Ave, Los Angeles CA 90024
Goldman, Stephen, Dept of Biology, University of Toledo, Toledo OH 43606, 419-537-4197
Golubovskaya, I. N., Dept of Genetics, N I Vavilov , All-Union Res Inst Plant Ind, Hertzen Street, 42, 190 000 Leningrad, USSR
Gonzalez de Leon, Diego, Av de Val de Mont Ferrand, B.P. 5035, 34032 Montpellier, FRANCE
Goodman, Howard M, Mass. Gen. Hosp., Molecular Biol., Blossom St., Boston MA 02114
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
Gouache, Jean C., Box29A, Route 1, South Amana IA 52334, (319)668-1814, FAX: (319)668-2401
Gould, Alan R., United Agriseeds, Inc., P. O. Box 4011, Champaign IL 61820, 217-373-5300
Gracen, V. E., Cargill Hi-Bred Seed, Box 9300, Minneapolis MN 55440, 612-475-7244
Grant, David, Pioneer Hi-Bred Internatl, Box 38, 7300 N.W. 62nd Avenue, Johnston IA 50131, 515-270-3503
Green, M. M., Dept of Genetics, University of California, Davis CA 95616, 916-752-1189 EXT 6295
Greenblatt, I. M., U-131, Molecular & Cell Bio, Univ of Connecticut, 354 Mansfield Rd, Storrs CT 06268, 203-486-2915
Greene, Ben A., USDA PGEC, 800 Buchanan St., Albany CA 94710, (415)559-5922

Greiser, P., Institut fur Getreideforschung, Bernburg-Hadmersleben, Bernburgh 4351, FEDERAL REPUBLIC OF GERMANY
Grier, Stephen L., Rogers NK Seed Co., NK Research Center, Hwy 19, Stanton, MN 55081, 507-663-7662
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
Grotewold, Erich, Cold Spring Harbor Lab, P.O. Box 100, Cold Spring Harbor NY 11724
Grula, John, Phytogen, 101 Waverly Drive, Pasadena CA 91105, 818-792-1802
Guffy, Richard D, Dept of Genetics, North Carolina State Univ, Box 7614, Raleigh NC 27695-7614, 919-737-2280
Gupta, Manju, 2050 Concourse Drive, San Jose CA 95131-1818, (408) 433-9800
Haag, Wayne L., Global 2000, Inc., Kotoka International Airport, Private Mailbag , Accra, GHANA
Hagemann, Kim M., G412 Agronomy, Ames IA 50011, (515) 294-7883
Hagemann, R., Dept Genetics, Martin Luther University, Domplatz 1, 4020 Halle/S., FEDERAL REPUBLIC OF GERMANY
Hake, S., USDA-ARS-PGEC, 800 Buchanan Street, Albany CA 94710, 415-559-5907
Hakiza, J., Agronomy Dept., Room 426, 2021 Coffey Road, Ohio State University, Columbus OH 43210-1086, (614)292-2001, FAX: (614)292-7162
Hallauer, Arnel R., Agronomy Building, Iowa State University, Ames IA 50010, 515-294-3052
Hallberg, Boone, Cent de Grad, Inst Tec Oaxaca, Calzs . Technologico Y, Gral. Wilfrido Massieu S/N., Oaxaca, Oax. C.P. 68000, MEXICO, 6-44-13, 6-17-22 Oaxaca
Hamilton, R. I., Agriculture Canada, Plant Research Centre, Forage Bldg 12, Ottawa, Ontario K1A 0C6, CANADA
Han, Changdeok, Cold Spring Harbor Laboratories, P.O. Box 100, Cold Spring Harbor NY 11724, (516)367-8467
Hancock, Denis C., 210 Curtis Hall, University of Missouri, Columbia MO 65211, 314-882-2674, Agrohanc@UMCVMB
Hannah, L. C., Veg Crops Dept, Univ of Florida, IFAS, Gainesville FL 32611, 904-392-7919
Hansel, W. C., Hansel Cons & Mgmt, Box 283, Carrollton MO 64633, 816-542-1616
Hansen, Leon A. , Rogers Brothers Seed Co., P.O. Box 4727, Boise ID 83711-0727, 208-322-7272
Hansen, Dale J., NPI, 417 Wakara Way, Salt Lake City UT 84108
Hanson, Maureen R., Section Genetics/Develop., Cornell Univ., Bradfield Hall, Ithaca NY 14853, 607-256-2100
Harberd, Nicholas, AFRC/IGER Inst Plant Sci Res, Cambridge Lab, Maris Lane, Trumpington, Cambridge CB2 2JB, UNITED KINGDOM, 011-44-233-840932
Hardeman, Kris, Department of Biology, Institute of Molecular Biology, University of Oregon, Eugene OR 97403
Harper, Lisa, Dept. Plant Biology, Genet. & Plant Biol. Bldg., University of CA, Berkeley CA 94720, (415)841-1539, (415)642-7948
Harris, John W., Dept of Biology, Tennessee Tech Univ, Cookeville TN 38505, 615-372-3143
Harris, Steven, Dept of Biology, University of Michigan, 830 North University, Ann Arbor MI 48109, (313)763-5285
Harris, Linda, Institute of Molecular Biol, Univ of Oregon, Eugene OR 97403
Harris-Cramer, Jane, Agrigenetics, 5649 East Buckeye Road, Madison WI 53716, 608-221-5000 ext 353
Harrison, V., Dept of Science, 470 E Lockwood Av, Webster University, St. Louis MO 63119-3194, (314)968-7079
Hartley, Tony, Sungene Technologies Corp., 2050 Concourse Drive, San Jose CA 95131-1818
Hartman, Carl J., 155 South St. Rd. 2, Valparaiso IN 46383
Hauptli, Holly, Calgene Inc, 1920 Fifth Street, Davis CA 95616, 916-753-6313
Hawk, James A., Dept Plant Science, University of Delaware, Newark DE 19711, 302-738-2531
Headrick, John, Monsanto Co., 700 Chesterfield Village Pkw, St. Louis MO 63198
Hedges, Bradley, Dept of Genetics, Curtiss Hall, Iowa State University, Ames IA 50011
Hegge, David J., Rt 1, Box 99, Climax MN 56523, (218) 281-3818
Heinlein, Manfred, Inst. of Genetics , Univ. of Cologne, Weyertal 121, D-5000 Cologne 41, FEDERAL REPUBLIC OF GERMANY
Helentjaris, Tim, Dept. of Plant Sciences, Univ. of Arizona, Tucson AZ 85721, 602-621-8746, HELNJARS@ARIZRVAX
Heller, Stephen R., Bldg 005, BARC-W, Beltsville MD 20705-2350, 301-344-2909, FAX: 344-5467, SRHELLER@UMDARS
Helm, James, Agronomy Department, North Dakota State University, Fargo ND 58105
Henderson, Clarion B., Illinois Foundation Seeds, PO Box 722, Champaign IL 61824, 217-485-6260
Hendrickx, Margaretha, Dept Biol Sci, Purdue University, W. Lafayette IN 47907
Heredia-Diaz, Oscar, 302 Curtis Hall, University of Missouri, Columbia MO 65211, 882-2033
Herrmann, R., Botany Department, Ludwig-Maximilans Univ Munich, Manzenger Str 67, 8 Munchen 19, FEDERAL REPUBLIC OF GERMANY
Heslop-Harrison, J. S., Karyobiology Group, Jl Centre for Plant Science Research, Colney Lane, Norwich NR4 7UH, ENGLAND
Hile, Glenn C., Northrup King Co, 1276 Lititz Rd., Mannheim PA 17545
History file, Curtis Hall, University of Missouri, Columbia MO 65211
Ho, David, Department of Biology, Washington University, St. Louis MO 63130, 314-889-4632, FAX: 314-726-4432, HO@BIOLGY.WUSTL.EDU
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
Hofstra, H., CIVO-TNO, P.O. Box 360, 3700 AJ Zeist, NETHERLANDS, 31-3404-52244, fax: 31-3404-57224
Hoisington, David, CIMMYT, Lisboa 27, Aptdo. Postal 6-641, Col. Juarez, MEXICO, 011-52-595-41069, fax: 011-52-595-41069
Holden, A., Corn States Hybrid Serv, 2505 McKinley Ave., Des Moines IA 50321, 515-285-3091
Hole, David , Plant Soils & Biometeorology Dept., Utah State Univ., Logan UT 84322-4820, (801)750-2235, fahole @ usu
Holley, Randall N., P.O. Box 638, Bell Arthur NC 27811
Hoo, B., 2050 Concourse Drive, San Jose CA 95131-1818
Hooker, A. L., Plant Molecular Biology Ctr, Northern Illinois Univ, DeKalb IL 60115, 815-753-7841
Horejsi, Thomas, G412 Agronomy, Ames IA 50011, (515)294-7883
Howell, Casey, Curtis Hall, University of Missouri, Columbia MO 65211
Huang, Yi Xiang, Crop Breed & Cult Res Inst, Sichuan Academy Agr Sci, Chengdu, Sichuan Province, CHINA
Hubbard, Jon, Asgrow Seed Company, PO Box L, San Juan Bautista CA 95131
Hulbert, Scott, Kansas State University, Dept. of Plant Pathology, Manhattan KS 66506, 913-532-6176
Hunsperger, John P., HaploGenetics, 6700B Princevalle St., P.O. Box 2217, Gilroy CA 95021-2217, (408)848-1161
Hunt, Marjorie, Div Biol Sci, Tucker Hall, Univ Missouri, Columbia MO 65211
Inoue, Yasuaki, National Grassland Res Inst, 768 Nishinasuno, Tochigi 329-27, JAPAN
Irish, Erin, Department of Botany, 312 Chemistry-Botany Bldg., Iowa City IA 52242
Isaac, Peter, The Nickerson Seed Co Ltd, Cambridge Science Park, Milton Road, Cambridge CB4 4GZ, UNITED KINGDOM, 0223-

862933
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 , Kannondai Tsukuba, Ibaraki 305, JAPAN
Jackson, Paul J., Life Sci Div, LS3 MS M886, Los Alamos Nat Lab, Los Alamos NM 87545, 505-667-2775, FAX: 505-665-3024, JACKSON@flovax.lanl.gov
Jaesan, Kim, Dept. of Agron. & Plant Genet., St. Paul MN 55108
Jakob, Karl M., Department of Plant Genetics, Weizmann Inst Sci, Rehovot, ISRAEL, 054-83301
James, Martha G., G403 Agronomy Hall, Iowa State University, Ames IA 50010, 515-294-8202
Jardine, Douglas J., Throckmorton Hall, Kansas State University, Manhattan KS 66506, 913-532-5810
Jauhar, Prem P., USDA ARS, Forage & Range Res Lab, Utah State Univ, Logan UT 84322-6300, 801-750-3074
Jewell, David C., CIMMYT - Maize Program, Lisboa 27, Apdo Postal 6-641, Mexico D.F. 06600, MEXICO, 525-761-3311 EXT 1110
Jimenez, J., Biology Dept, Purdue Univ, Fort Wayne IN 46805, 219-481-6100
Johal, G., Cold Spring Harbor Lab., P. O. Box 100, Cold Spring Harbor, NY NY 11724
Johns, Mitrick A., Dept Biological Sciences, Northern Illinois University, DeKalb IL 60115, 815-753-7836
Johnson, M. W., Dept of Agronomy, Pennsylvania State Univ, University Park PA 16802, 814-865-0324
Johnson, W., USDA-ARS, Plant Pathology Dept, Univ of Florida, Gainesville FL 32611
Johnson, Scott, Agrigenetics Co, Slater Research Sta. P.O. Box 508, 321 Main St., Slater IA 50244, 515-684-3080, FAX: 515-684-2291
Johnson, E. C., Escagen Corp., 830 Bransten Road, San Carlos CA 94070, 415-595-5335
Johnson, Elizabeth, CIBA-GEIGY Corporation, P.O. Box 1830, Kaunakakai HI 96748, 808-567-6146
Johri, M. M., Molecular Biology Unit, Tata Inst Fundamental Res, Homi Bhabha Road, Bombay 400 005, INDIA, 91-22-495-2971 ext 255, FAX 91-22-495-2110
Jondle, Doug, Cargill Hybrid Seeds, Box 328, St. Peter MN 56082
Jones, Todd, E. I. DuPont de Nemours & Co, Experimental Station, P.O. Box 80402, Wilmington DE 19880-0402, 302-695-4372
Jones, John Edward, 8429 Meadow Green Way, Gaithersburg MD 20877
Jones, R. K., Dept Agron & Plant Genetics, Univ of Minnesota, St. Paul MN 55108
Jones, J. K., Dept Agric Bot, University of Reading, Whiteknights, Reading RG6 2AS, UNITED KINGDOM, 0734-875123 EXT 7950
Jones, Jonathan, Sainsbury Laboratory, John Innes Inst., Colney Lane, Norwich NR4 7UH, UNITED KINGDOM, (44)603-52571, 56844 (FAX)
Jordan, Berry, Dept Foods & Nutrition, Dawson Hall, Univ Georgia, Athens GA 30602
Julstrom, P., Dekalb Plant Genetics, 3100 Sycamore Road, Dekalb IL 60115
Juvik, J. A., Dept of Horticulture, University of Illinois, 1103 West Dorner Drive, Urbana IL 61801, 217-333-1966
Kahler, A. L., Biogenetic Services, Inc, 2308 6th Street E., P.O. Box 710, Brookings SD 57006, 605-697-8500
Kalia, V., Regional Research Station, Dhaulakuan-173001, Distt Sirmur (H. P.), INDIA
Kamimura, Shoji, 421-19 Furuichi-machi, Maebashi-shi, Gunma-ken 371, JAPAN
Kang, M. S., Department of Agronomy, Louisiana State University, Baton Rouge LA 70803, 504-388-2110
Kangasjarvi, Jakko, Agron & Plant Genetics, Univ of Minnesota, St. Paul MN 55108
Kannenberg, L. W., Dept of Crop Science, University of Guelph, Guelph, Ontario N1G 2W1, CANADA, 519-824-4120 EXT 2506
Karp, Robert, Eukaryotic Genetics Program, National Science Foundation, Washington DC 20550
Karpoff, A. J., Dept of Biology, Univ of Louisville, Louisville KY 40292, 502-588-6771
Kasha, K. J., Dept of Crop Science, Univ of Guelph, Guelph, Ontario N1G 2W1, CANADA, 519-824-4120 EXT 2507
Kaszas, Etienne, Lab G412, Agronomy Hall, Iowa State University, Ames IA 50010, 515-294-7883
Kato, Akio, National Grassland Res Inst, 768 Nishinasuno, Tochigi 329-27, JAPAN
Kato Y., T. A., Centro de Genetica, Colegio de Postgraduados, Chapingo 56230, MEXICO, (5)5-85-07-28
Katsuta, Masumi, Laboratory of Ecological Genetics, Dept Genetic Resources, Natl Inst Agrobiol Resources, Tsukuba, Ibaraki 305, JAPAN, 02975-6-7458
Katt, Maria, Pioneer Hi-Bred Intl., Inc., 7250 N.W. 62nd Avenue, Johnston IA 50131, 515-270-3651
Kazco, Dept #KAA-FM-036U, ATTN: Mr. King, P.O. Box 1898, Hoboken NJ 07030
Keeling, Peter, Garst Seed Co., Highway 210, P.O. Box 500, Slater IA 50244
Keim, Kent R., Sunseeds Genetics, Inc., 1200 Anderson Corner Road, Parma ID 83660, 208-674-2208
Kelley, Phil, University of Nebraska, School of Biology, Lincoln NE 68588, 402-472-1683, PKELLEY@UNLVAX1
Kellogg, Elizabeth A., 166 Vassal Lane, Cambridge MA 02138
Kendra, David F., Enimont America Inc., 2000 Cornwall Rd., Monmouth Junction NJ 08852, (201) 442-3674, FAX: (201) 442-0084
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, 12773 Castlebar Dr, St. Louis MO 63146
Khavkin, Emil E., All-Union Res Institute of, Agricultural Biotechnology , 12 Pskovskaya str., bld. 4, I27253 Moscow, USSR
Kiefer, Mike, Northrup King Co., Route 19, Stanton MN 55081
Kim, Soon Kwon, Internat Inst Tropical Ag, (IITA), AMEG, 11511 Katy Freeway, Suite 300, Houston TX 77079
Kindiger, Bryan, Wilson Hybrids, Inc, P.O. Box 391, Harlan IA 51537, 712-755-3841
Kinugawa, Kenjiro, 4-23-3, Takakuradai, Sakai, Osaka 590-01, JAPAN, 0722-91-5497
Kirihara, Julie, Plant Science Research, Inc., 10320 Bren Road East, Minnetonka MN 55343, 612-936-6101
Kirschman, Jane A., Agrigenetics Advanced Sci. Co., 5649 E. Buckeye Road, Madison WI 53716, 608-221-5000
Kiss, Charles, Northrup King Semences, 18 Avenue Gollieni, 49130 Les Ponts de Ce, FRANCE, 41-44-86-45
Kleese, Roger, Plant Science Research Inc, 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, 603-862-2455, Fax: 603-862-4013, A Klei@UNHH.unh.edu
Klein, Ted, USDA-ARS-PGEC, 800 Buchanan Street, Albany CA 94710
Kloeckener, Barbara, Univ. of CA - Berkeley, Dept. of Genetics, 345 Mulford Hall, Berkeley CA 94720, 415-642-8058
Koeppler, Shawn, Dept. Agron. & Plant Genet., 1991 Buford Circle , St. Paul MN. 55108
Koester, Ruth, Dept of Genetics , Box7614, North Carolina State University, Raleigh NC 27695-7614
Kohashi, Josue, Dept of Botany, Colegio de Postgrad, Chapingo, Edo de Mex, MEXICO
Koinuma, Keiichi, Kyusyu Natl. Agric. Exp. Stn., Yokoichi, Miyakonojo, Miyazaki, 885, JAPAN
Konstantinov, Yu., Siberian Inst Plant Phys Bioch, USSR Academy of Sciences, P.O. Box 1243, Irkutsk-33, 664033, USSR
Konstantinov, Kosana, Maize Research Institute, P.O. Box 89, 11081 Zemun, YUGOSLAVIA

Kowalewski, Shirley, Curtis Hall, University of Missouri, Columbia MO 65211, 314-882-3856
Kowles, R. V., Biology Department, St Marys College, Winona MN 55987, 507-457-1554, Fax: 507-457-1633
Krivov, N. V., Inst Ecological Genet, st. Lesnaya 20, Moldavian SSR, Kishinev-277018, USSR
Kriz, A. L., Dept Agronomy, Turner Hall, Univ Illinois, Urbana IL 61801
Kudirka, Dalia, Univ. of Western Ontario, Dept. of Plant Sciences, Biol. & Geol. Bldg., London, CANADA
Laible, Charles A., Research Department, Funk Seeds International, Bloomington IL 61701, 309-829-9461
Lakra, Alejandro Blanco, CINVESTAV, Apdo Postal 629, Irapunto, GTO, MEXICO
Lambert, A., Semences Nickerson, 49160 Longue, Jumelles, FRANCE
Lamkey, Kendall, Agronomy Building, Iowa State University, Ames IA 50010, 515-294-7826, FAX: 515-294-9359
Lampoh, E., Crops Res Inst, PO Box 20, KPEVE, Volta Reg, GHANA, 2 ext 5
Lanahan, Mike, Dept of Biology, Campus Box 1137, Washington Univ., St. Louis MO 63130
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
Lang, Lee, Molec & Cell Biol Dept, University of Arizona, Tucson AZ 85721
Langdale, Jane, Department of Plant Sciences, University of Oxford, South Parks Road, Oxford OX1 3RB, ENGLAND, 011-44-865-275024, Fax: 011-44-865-275144
Langston-Unkefer, Pat J., INC-4, Mail Stop C345, Los Alamos National Lab, Los Alamos NM 87545, 505-665-2556
Laohawanich, Chirayus, Natl. Corn & Sorghum Res. Ctr., Kasetsart University, Pakchong, Nakorn Rajasima 30130, THAILAND
Larkin, John C., Dept Biochemistry, Univ Minnesota, 1479 Gortner Ave, St Paul MN 55108
Larkins, B., Dept Plant Sciences, University of Arizona, Building #36, Tucson AZ 85721, (602)621-1977, FAX: (602)621-7186
Larrinua, I M, Lilly Research Laboratories, 307 E McCarty St, Indianapolis IN 46208
Larson, Steven R., Dept of Quantitative Genetics, Northrup-King Co., Stanton MN 55081
Last, Robert, Boyce Thompson Inst Plant Res, Inc., Tower Road, Ithaca NY 14850
Laughnan, John R., Plant Biology/289 Morrill Hall, U of Illinois/505 S Goodwin Av, Urbana IL 61801, 217-333-2919
Laurie, David, JI Centre for Plant Science Res., Colney Lane, Norwich, Norfolk NR4 7UJ, UNITED KINGDOM, (0603) 52571, Fax: (0603)502270
Lavapaurya, Tavat, Dept Horticulture, Kasetsart University, Bangkok 10900, THAILAND
Law, Colin N., Cambridge Laboratory, JI Centre for Plant Science Research, Colney Lane, Norwich, Norfolk NR4 7UJ, UNITED KINGDOM, 0603-52571, FAX: 0603-502270
Lazarowitz, Sondra G., Dept. of Microbiology, U. of Ill./131 Burrill Hall, 407 S. Goodwin Av., Urbana IL 61801
Leaver, C. J., Dept Plant Sciences, University of Oxford, South Parks Road, Oxford Ox1 3RB, UNITED KINGDOM, 031-667-1081 EXT 3304
LeClerc, M. Christian, PROCOSEM SA, Domaine du Chaumoy, 18570 - LE SUBDRAY, FRANCE, Telex: 760 127, FAX: 48 64 76 45
Lee, Sharon K., Department of Botany, University of Rhode Island, Kingston RI 02881, 401-792-2625
Lee, Norman K, Department of Mathematics, Ball State University, Delaware Dr & Trl, Muncie IN 473032
Lee, Michael, Department of Agronomy, Iowa State University, Ames IA 50011, 515-294-3052
Lee, Philip K, Health Physics, 8 Res Park Development Bldg, University of Missouri, Columbia MO 65211
Lee, Elizabeth, Dept. of Agronomy, Curtis Hall, University of Missouri, Columbia MO 65211, 314-882-7709
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
Leroy, Philippe, Biocem Laboratoire, Campus Universitaire des Cezeaux, 24 Av des Landais, 63170 Aubiere, FRANCE, (33) 73-42-79-70, fax: 33-73-27-57-36
Leto, K. J., Agric Prod Dept, DuPont Co, Stine Laboratory S215, Elkton Road, PO Box 30, Newark DE 19714, 302-366-5170
Levings, C. S., Department of Genetics, North Carolina State Univ, Box 7614, Raleigh NC 27695-7614, 919-737-7115
Levites, E. V., Inst Cytol Genetics, Novosibirsk, USSR
Levy, Avraham, Dept Biol Science, Stanford University, Stanford CA 94305
Li, Jigeng, Institute of Genetics, Academia Sinica, Beijing, CHINA
Li, Jingxiong, Chinese Acad Agric Sci, Institute Crop Breeding & Cult, 30 Bai Shi Qiao Road, Beijing, CHINA
Lillis, Marcella, Sogetal, Inc., 3876 Bay Center Pl., Hayward CA 94545
Lim, Yong Pyo, Division of Genet. & Physiol., Korea Ginseng & Tobac Res Inst, #302 Shinseong-Dong, Yusong-Gu, Taejon, KOREA, (042)822-5070
Lin, Bor-yaw, Genetics Institute, 87 Cambridge Park Drive, Cambridge MA 02140, 617-876-1170
Livini, C., Ist Sperimentale de Cereali., Via Stezzano 24, Bergamo, ITALY, 39-35-313132/341422, fax: 39-35-316054
Loeffel, F. A., NAPB Res. Center, Rural Route #2, Brookston IN 47923, 317-563-3111
Long, Sharon, Biological Science, Stanford University, Stanford CA 94305
Lonsdale, D. M., Cambridge Laboratory, IPSR, Colney Lane, Norwich, UNITED KINGDOM, FAX: 603 502270, LONSDALE@uk.ac.afrc.jii
Lorenzoni, C., Istituto di Genetica Vegetale, Universita Cattolica, 29100 Piacenza, ITALY
Lorz, Horst, Institut Allgemeine Botanik, Universitat Hamburg, Ohnhorststrasse 18, 2000 Hamburg 52, FEDERAL REPUBLIC OF GERMANY
Lowe, Brenda, PGEC - USDA, 800 Buchanan St, Albany CA 94710
Lower, W., 235 Environ Trace Ctr, Univ of Missouri, Columbia MO 65211, 314-445-8595
Ludwig, Steven, Department of Botany, University of Georgia, Athens GA 30602
Luehrsen, Ken, Dept of Biological Sciences, Stanford Univ, Stanford CA 94305, 415-723-2609
Lundquist, Ron, Plant Science Research, Inc., 10320 Bren Road East, Minnetonka MN 55343
Lysikov, V. N., Inst Ecological Genet, Lesnaya str 20, Moldavian SSR, Kishinev-277018, USSR
Ma, Hong, Cold Spring Harbor Laboratories, P.O. Box 100, Cold Spring Harbor NY 11724
Ma, Yu, Agrigenetics, 5649 East Buckeye Road, Madison WI 53716, 608-221-5000
MacDonald, M. V., Botany Department, University of Cambridge, Downing Street, Cambridge CB2 3EA, UNITED KINGDOM, 0223-333900
MacKenzie, David R., CSRS-USDA, Room 330L Aerospace Building, 901 D Street, S. W., Washington, DC
Mackenzie, David, 1905 Chula Vista, Belmont CA 94002, 415-591-1507
Mackey, C. J., DeKalb-Pfizer Genetics, Pfizer Central Research, Eastern Point Road, Groton CT 06340
MacRae, Amy F., Tucker Hall, Division of Biological Sciences, University of Missouri, Columbia MO 65211

Magill, Clint, Texas A & M University, Dept. of Plant Pathology, College Station TX 77843, 409-845-8250, FAX: 409-845-6483
Magoja, J. L., Enrique Py 580, 1846-Adrogue (Prov. Buenos Aires), Buenos Aires, 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
Maisadour CCL AP, Rte de St Sever BP 27, Haut Mauco, 40001 Mont de Marsan , Cedex, FRANCE
Malone, Carl, 205 McKinley Avenue, Milford IL 60953, 815-889-4079
Mans, Rusty J., Dept of Biochem/J245 JHMHC, Univ of Florida, Gainesville FL 32610, 904-392-2332
Manzocchi, Lucia A., Consiglio Nazionale Ricerche, Istituto Biosintesi Vegetali, Via Bassini 15, 20133 Milano, ITALY, (2)70600170, (2)2362940
Mapping file, Curtis Hall, University of Missouri, Columbia MO 65211
Marchand, J. L., F Chemin de l'IRAT, Ligne Paradis, 97410 St. Pierre, REUNION
Marquez-Sanchez, F., Campo Agricola Experimental, Valle de Mexico, Coordinacion de Maiz, Zoua Sur, Chapingo, Mex. 56230, MEXICO, 585-0728
Marshall, Lori, Holden's Foundation Seeds, Inc., P.O. Box 839, Williamsburg IA 52361, 319-668-1100, FAX: 319-668-2453
Martienssen, R. A., Cold Spring Harbor Labs, P.O. Box 100, Cold Spring Harbor NY 11724, 516-367-8322
Martin, Michael J., Garst Seed Co., P.O. Box 500, Slater IA 50244
Martin Sanchez, J. A., UPC-IRTA Centro R S D, Alcalde Rovira Roure 177, 25006 Lerida, SPAIN, 73-247240, Fax: 73-238301
Marton, Csaba Lajos, 2462 Martonvasar, P.O.B. 19, 26-45774
Mascarenhas, Joseph P., Department of Biol Sci, State University of New York, Albany NY 12222, 518/442-4388, FAX: 518/442-4767
Mascia, P., Cargill Hi-Bred Seed Division, RR 3, Box 751, Aurora IL 60506, 708-892-4331
Mashnenkov, A. S., Krasnodar Res Inst Agric, Krasnodar, USSR
Masson, Patrick, Carnegie Inst Wash, 115 West Univ Parkway, Baltimore MD 21210, 301-467-1414
Mather, Diane E., Plant Sci Dept - Macdonald Col, McGill Univ - 21111 Lakeshore, Ste-Anne-de-Bellevue, Quebec H9X 1C0, CANADA, (514) 398-7854
Mathern, J., USDA-ARS-PGEC, 800 Buchanan Street, Albany CA 94710
Mathews, Dennis, Agron & Plant Genetics, Univ of Minnesota, St. Paul MN 55108
Mathur, D. S., Div. of Genetics, Indian Agr Res Inst, New Delhi, INDIA
Matz, Eileen C., P.O. Box 1300, Cutchogue NY 11935
Matz, Eileen C., Copy 2, P.O. Box 1300, Cutchogue NY 11935
Mazoti, Luis B., C. Croce 145, 1832 Lomas de Zamora, ARGENTINA
McBride, A. C., Phyto Dynamics, Inc., 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, 904-392-9087
McClain, Eugene F., Dept Agronomy & Soils, Clemson University, Clemson SC 29634-0359, 803-656-3102
McClintock, Barbara, Cold Spring Harbor Laboratory, Cold Spring Harbor NY 11724, 516-367-8385
McConnell, R. L., Dept of Corn Breeding, Pioneer Hi-Bred International, Box 85, Johnston IA 50131
McConnell, D. J., Genetics, Trinity College, University of Dublin, Dublin 2, IRELAND
McCormick, Sheila, USDA-ARS-PGEC, 800 Buchanan Street, Albany CA 94710, 415-559-5906
McCullough, Andrew, Plant Biology, Rm 289 Morrill Hall, Univ Illinois, Urbana IL 61801, (217)333-8785
McCurdy, Leroy, P.O. Box 77, McCurdy Seed Co, Fremont IA 52561, 515-933-4291
McDaniel, R. G., Dept of Plant Sciences, University of Arizona, Tucson AZ 85721, 602-261-3357
McFerson, John, Asgrow Seed Co., 1060 E. Highway 14, RR #1, Janesville WI 53546, (608)755-1777
McMullen, Michael, USDA ARS, Department of Agronomy, OARDC, Wooster OH 44691, 216-263-3894
McWhirter, K. S., 127 Victoria Road, West Pennant Hills, N.S.W. 2125, AUSTRALIA, 02-484-7417
Mead, Doug, Northrup King Co., Route 19, Stanton MN 55081
Melchinger, Albrecht E., Universitat Hohenheim, Institut fur Pflanzenzuchtung, Fruwirthstr. 21, 7 Stuttgart 70, FEDERAL REPUBLIC OF GERMANY, , FAX: 49 711 459 3295, whao@ds 0rus 1
Melia-Hancock, Susan, Curtis Hall - Agronomy, University of Missouri, Columbia MO 65211
Mello-Sampayo, Tristao, Estacao Nacional Melhoramento Plantas, Inst Gulbenkian de Ciancia, 7351 Elvas, PORTUGAL
Messing, Jo, Rutgers, The State Univ, Waksman Institute, P.O. Box 759, Piscataway NJ 08855, 201-932-4256
Mets, Laurens J, Mol Genetics & Cell Bio, 1103 E 57th St, Chicago IL 60621
Meyer, Annette, 240 PABL, 1201 W. Gregory, University of Illinois, Urbana IL 61801-3838
Meyerowitz, Elliot, Biology Dept 156-29, California Inst Tech, Pasadena CA 91125
Michel, Kenneth, Department of Biology, Slippery Rock University, Slippery Rock PA 16057, 412-794-7326
Michelini, Luiz Antonio, 44B Schilletter Village, Ames IA 50010
Michelmore, Richard W, Vegetable Crops, UC Davis, Davis CA 95616
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, 309-685-4011 EXT 372
Mies, David, Northrup King, Box Z, St. Joseph IL 61873
Mihm, John A., CIMMYT, Apdo Postal 6-641, Deleg Cuauhtemoc, 06600 Mexico, DF, MEXICO, (5)761-3311,(595)421-00, FAX: (595)410-69
Miksche, J. P., USDA NPS, Bldg 005, Rm 239, BARC-W, Beltsville MD 20705, , jnet%"jmiksche@umdars"
Mikula, Bernard C., Defiance College, Defiance OH 43512, 419-784-4010 EXT 426
Miles, Donald, Tucker Hall, University of Missouri, Columbia MO 65211, 314-882-7933
Miranda, L. T. De, Instituto Agronomico, Caixa Postal, 28, 13.100 Campinas SP, BRAZIL, 0192-420235
Mitchell II, Roger E., Dept Genet & Cell Biol, University of Minnesota, St. Paul MN 55108, (612)624-3015
Mock, James J., Northrup King Company, 7500 Olson Memorial Highway, P.O. Box 959, Minneapolis MN 55440
Modena, Stephen, Dept. of Crop Science, North Carolina State Univ., Box 7620, Raleigh NC 27695, 919-737-2246
Moehlenkamp, Cynthia, Biological Sciences, University of Missouri, Columbia MO 65211
Mogford, D. J., Asgrow S. Africa, P.O. Box 912-653, 0127 Silverton, SOUTH AFRICA
Molina, M. C., Inst Fitotecnico de Sta. Catalina, C.C. 4, 1836, Llavallol, Buenos Aires, 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, Plant Biotech, Plant Res Ctr, Ottawa, Ontario K1A 0C6, CANADA, 613-995-3700 EXT 7560
Moore, Paul H., USDA ARS PWA, Experiment Station HSPA, P.O. Box 1057, Aiea HI 96701, 808-487-5561, FAX: 808-486-5020

Moore, Kathy, Molec & Cell Biol Dept, University of Arizona, Tucson AZ 85721
Mordvinova, V., Genet. Academy of Sciences, Moldavian SSS, Lesnaya str. 20, 277018 Kishinev, USSR
Moreno, Maria Angela, Yale Univ - Dept Biology, P.O. Box 6666, New Haven CT 06511, 203-432-3894
Morris, Roy O., 322 Chemistry, Biochemistry Dept, Univ of Missouri, Columbia MO 65211
Morse, Richard, Cargill Hybrid Seed, 3864 Preemption Rd, Geneva NY 14456, 315-789-8626
Mottinger, John, Department of Botany, University of Rhode Island, Kingston RI 02881, 401-792-2625
Mousel, Alan, CIBA-GEIGY - Seed Division, PO Box 417, Bluffton IN 47614
Muenchrath, Deborah A., Dept Agronomy , Iowa State Univ., Ames IA 50011, 515/294-0359, FAX: 515/294-3163
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
Mulligan, T. P., P. O. Box 523, Cold Spring Harbor NY 11724, 516/367-8829, H: 516/692-4926
Murphy, C. F., USDA NPS, Bldg 005, Rm. 239, BARC-W, Beltsville MD 20705, 301-344-1560
Murphy, T. C., The Upjohn Company, 9612-50-1, Kalamazoo MI 49001, 616-384-2651
Murray, Michael G., Agrigenetics, 5649 E Buckeye Rd, Madison WI 53716, 608-221-5000
Murrell, K. D., North Central Region, USDA-ARS MWA, 1815 N. University, Peoria IL 61604
Muszynski, Michael G., Cytogenetics, G412 Agronomy, 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
Nadel, Daniel, IBN Givirol 3, Jerusalem, ISRAEL
Nagle, B. J., Dekalb-Pfizer Genetics, Box 33, Mason City IL 62664, 217-482-3213
Naranjo, Carlos, Inst Fitotecnico de Santa Catalina (UNLP), C.C.4 (1836) Llavallol, Buenos Aires, ARGENTINA
Nash, J., Dept Biol Science, Stanford Univ, Stanford CA 94305
Nasser, DeLill S., Eukaryotic Genetics Program, Division of Cell Biology, National Science Foundation, Washington DC 20550, 202-357-0112
Natli, Tom, Harris-Moran Seed Co., P.O. Box 980, Nampa ID 83653, 208-467-6341
Natti, Thomas A., Harris Moran Seed Co., 1832 Garrity Blvd., P.O. Box 980, Nampa ID 83653, 208-467-6341, Fax: 208-466-8188
Nebiolo, Christine M., Dept of Biology, Allegheny College, Carnegie Hall, Meadville PA 16335, 814-724-5360
Nel, P. M., Dept of Genetics, Univ of The Witwatersrand, P.O. WITS, Transvaal 2050, SOUTH AFRICA, 011-716-2154
Nelsen, Terry, USDA-ARS, 1815 N. University, Peoria IL 61604
Nelson, Timothy, Department of Biology, PO Box 6666, Yale University, New Haven CT 06511-8112, 203-432-3860
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
Newton, K., Dept Biol Sci, University of Missouri, Columbia MO 65211, 314-882-4049
Nguyen, Henry T., Dept of Agronomy, 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
Noble, Jr., Stephen W., Dept of Corn Breeding, Pioneer Hi-Bred Internat Inc, P.O. Box 409, Johnston IA 50131-0409, 515-270-3318
Notani, N. K., Bhabha Atomic Res Centre, Biology Division, Mod Lab, Trombay, Bombay 400085, INDIA, 5510323
Nubel, Douglas, Pfister Hybrid Corn Co, El Paso IL 61738, 309-527-6008
Nykaza, Scott, Callahan Seeds, 1122 East 169th Street, Westfield IN 46074, 317-896-5551
O'Brien, David, Dept of Botany, University of Rhode Island, Kingston RI 02881
Ober, Eric, 1-40 Agriculture Bldg., University of Missouri, Columbia MO 65211 65211, (314)882-3971, FAX: 314-882-1467, AGROERIC@UMCVMB
Oberthur Caldwell, Elizabeth E., G412, Agronomy Building, Agronomy Department, Iowa State University, Ames IA 50010, 515-294-7883
Ogle, Charles W., P.O. Box 484, Sugar Grove IL 60554, 312-466-4742
Ohta, Y., Inst Agr & Forestry, Univ of Tsukuba, Tsukuba City, Ibaraki-Ken 305, JAPAN, 0298-53-6691
Oishi, Karen, Molec & Cell Biol Dept, University of Arizona, Tucson AZ 85721, 602-621-3381
Okagaki, Ron, Lab of Genetics, 445 Henry Mall, Univ of Wisconsin, Madison WI 53706, 608-262-3287
Okuno, K., Field Crops Div., Hokuriku National Agr Exp Sta, 1-2-1 Inada, Joetsu, Niigata 943-01, JAPAN
Oliver, Carmen, Plant Biol/289 Morrill Hall, Univ Illinois, 505 S. Goodwin Avenue, Urbana IL 61801, 217-333-2919
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., Dpt. of Genetics and 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
Pages, Montserrat, Jordi Girona Salgado, 18-26, 08034 Barcelona, SPAIN
Paiva, R., Dept. of Agronomy-Turner Hall, 1102 S Goodwin Av, Univ. Illinois, Urbana IL 61801
Pan, Yong-Bao, 714 Pammel Court, Ames IA 50010, 515-296-7946
Pan, David, Laboratory of Genetics, B14 Genetics Building, University of Wisconson, Madison WI 53706, 608-262-3287
Pardee, William D., 252 Emerson Hall, Cornell University, Ithaca NY 14853
Pareddy, Dayakar R., United AgriSeeds, Inc., P.O. Box 4011, Champaign IL 61820
Parlov, Dragomir, Inst Breeding & Prod Field Crops, Marulicev trg 5, 41000 Zagreb, YUGOSLAVIA
Parrish, Fred, 1100 Robert E Lee Blvd, New Orleans LA 70124
Paterniani, Ernesto, Univ Sao Paulo Esc Sup Agr, Luiz de Queiroz, P.O. Box 83, 13400 Piracicaba, BRAZIL
Patterson, Earl, Agron Dept, S-116 Turner Hall, University of Illinois, 1102 S. Goodwin Avenue, Urbana IL 61801, 217-333-9641, FAX: 217-333-9817
Patterson, Garth, Department of Biology, Institute of Molecular Biology, University of Oregon, Eugene OR 97403
Paul, Anna-Lisa, Dept Veg Crops, 1255 Fifield Hall , Univ Florida, Gainesville FL 32611, 904-392-2370
Paulis, Jerrold, USDA/ARS, 1815 N. University St., Peoria IL 61604, 309-685-4011 ext.359
Pauly, Michael H., Rogers Brothers Seed Co., P.O. Box 1069, Nampa ID 83653, 208-466-0319
Pawar, S. E., Biol/Agric Div, Bhabha Atomic Research Center, Trombay Bombay 40085, INDIA, 5514910 ext 2340

Payne, Peter I., Plant Breeding International, Cambridge, Maris Lane, Trumpington, Cambridge CB2 2LQ, UNITED KINGDOM
Peacock, W. J., Division of Plant Industry, CSIRO, Canberra ACT 2601, AUSTRALIA, 062-465250
Pepe, John, Cargill Seed Research, P.O. Box 774, Grinnell IA 50112
Perani, Laura, Calgene, 1910 5th St, Davis CA 95616
Periodical Department 660/90, The Book Home, Inc., 220 East Monument Street, Colorado Springs CO 80903
Peschke, Virginia M., Dept Biology, Campus Box 1137, Washington Univ, St. Louis MO 63130, (314)889-4728
Peters, D. W., Asgrow Seed Company, R.R. 2, Box 291, Queenstown MD 21658
Petersen, William L., Agrigenetics Advanced Sci. Co., 5649 E. Buckeye Road, Madison WI 53716, 608-221-5000
Peterson, Thomas A., Cold Spg. Harbor Lab, P.O. Box 100, Cold Spg. Harbor NY 11724, 516-367-8825
Peterson, Peter A., Dept of Agronomy, Iowa State University, Ames IA 50011, 515-294-9652
Phillips, Ronald, Agronomy & Pl Genetics, University of Minnesota, St. Paul MN 55108, 612-625-1213, 612-625-1208
Pienaar, R. de V., Dept Genet & Plant Breeding, University of Stellenbosch, 7600 Stellenbosch, SOUTH AFRICA
Pierce, Dorothy, 2000 Princetonpark Corp Center, Monmouth Junction NJ 08852, FAX: 201-422-0084
Pinter, Lajos, Crop Science Dept, Univ Keszthely, P.O. Box 71, H-8361 Keszthely, HUNGARY
Pitas, Jan M.W., Agrigenetics Advanced Sci. Co., 5649 E. Buckeye Road, Madison WI 53716, 608-221-5000
Plesset, Judith, Division of Cellular Biosci., National Science Foundation, Washington, D. C., 202-357-7989
Podolskaya, Anna P., N. I. Vavilov All Union Inst, of Plant Industry, 44 Herzen Street, 190000, Leningrad, USSR
Poethig, R. Scott, Biology Department, Leidy Labs, Univ Penn, 415 University Ave, Philadelphia PA 19104-6018, 215-898-8915
Poggio, Lidia, Inst Fitotecnico de Santa Catalina (UNLP), C.C.4 (1836) Llavallol, Buenos Aires, ARGENTINA
Poggio, E. A., Inst Fitotecnico de Sta. Catalina, C.C. 4, 1836, Llavallol, Buenos Aires, ARGENTINA
Pogna, N. E., Ist Sper Cerealicoltura, Via Mulino, 3, 20079 S. Angelo Lodigiano, Milan, ITALY
Polacco, Mary , Dept 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, 73-62-43-01
Pollak, Linda, Dept of Agronomy, Iowa State Univ, Ames IA 50011, 515-294-3052
Pollmer, W. G., Universitat Hohenheim, Postf 700562-304, Garbenstrasse 9, 7000 Stuttgart 70, FEDERAL REPUBLIC OF GERMANY
Polson, David E, Canners Seed Corporation, PO Box 18, Lewisville ID 83431, 208-754-8666
Poneleit, Charles G., Agronomy, University of Kentucky, Lexington KY 40546-0091, 606-257-4934
Porter, Hedera L., R.R. 2, Box 268, Blackville SC 29817
Pratt, Richard C., Agronomy Department, OSU/OARDC, 1680 Madison Avenue, Wooster OH 44691, 216-263-3972
Price, Carl, Waksman Inst, Piscataway NJ 08855-0759, 908-932-2920, Fax: 908-932-5735, Price@Biovax
Price, S. C., Office of Biotechnology, 1301 Agronomy, Iowa State University, Ames IA 50011-1010
Pring, D. R., Dept of Plant Pathology, University of Florida, Gainesville FL 32611, 904-392-3638
Prioli, Laudenir , Dept. of Genetics, North Carolina State Univ., P.O. Box 7614, Raleigh NC 27695, (919)737-7116, FAX: (919)737-7115
Pryor, A. J., Div Plant Ind CSIRO, PO Box 1600, Canberra Act 2601, AUSTRALIA, 062-46-5896
Puckett, Jane L., Dept Agronomy, Turner Hall, Univ Illinois, Urbana IL 61801
Purugganan, Michael, Dept of Botany, Univ of Georgia, Athens GA 30602
Qin, Minmin, Dept Plant Pathology, Univ Wisconsin, Madison WI 53706
Qin, Tai-chen, Department of Agronomy, Jiangsu Agriculture College, Jiangsu, Yangchou, CHINA
Quail, Peter H., USDA-ARS-PGEC, 800 Buchanan Street, Albany CA 94710, 415-559-5900
Qualset, Calvin O., Genetic Resources Cons Program, Univ of California, Davis CA 95616, 916-757-8920
Quayle, Tom, Institut Biologie III, Universitat Freiburg, Schanzlestrasse 1, 78 Freiburg I. Br., FEDERAL REPUBLIC GERMANY
Quebedeaux, Bruno, Horticulture Dept., Univ of MD, College Park MD 20742
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
Radicella, Pablo, Institute of Molecular Biol, University of Oregon, Eugene OR 97403
Radtke, Jim, Agrigenetics, 321 Main St., P.O. Box 508, Slater IA 50244, 515-685-3080
Ragot, Michel, Dept. of Genetics, NC State University, Box 7614, Raleigh NC 27695-7614, (919)737-2289
Rakha, Farouk A., Dep Genetics Fac Agric, Alexandria University, Alexandria, EGYPT, 966386
Ralston, Ed, DNA Plant Technology Corp, 6701 San Pablo Avenue, Oakland CA 94608, 415-547-2395
Ramsey, Ann, RR2, Box 15, Rochester IL 62563, 217-498-9374
Rao, P. N., Dept of Botany, Andhra University, Visakhapatnam 530003, INDIA, 54871 ext 390
Rapela, Miguel, Northrup King Semillas, Hipolito Yrigoyen 1628 - Piso 13, 1344 Burnos Aires, ARGENTINA, Tel. 54 1 498621/26, FAX 54 1 9534453
Rashid, Abdul, Agronomy Dept, Iowa State University, Ames IA 50011
Rayapati, P. John, Department of Agronomy, Iowa State University, Ames IA 50011
Rayburn, A. L., Department of Agronomy, Turner Hall, 1102 South Goodwin Avenue, Urbana IL 61801
Reddy, A. R., School of Life Sci, Univ of Hyderabad, Hyderabad-500001, INDIA, 253901, extn:226
Reddy, G. M., Department of Genetics, Osmania University, Hyderabad-500007, INDIA
Redei, G. P., Curtis Hall, University of Missouri, Columbia MO 65211, 314-882-6434
Rees, Hubert, Agricultural Botany Dept, Univ College of Wales, Aberystwyth, Wales, UNITED KINGDOM, 0790-3111
Reid, W. R., Asgrow Seed Company, P.O. Box 460, Parkersburg IA 50665
Reksodihardjo, S., Pioneer Agricultura Ltda, Caixa Postal 131, 86100 Londrina PR, BRAZIL, 0432 271512
Reyes, Luz M., North Carolina State Univ., Hort. Sci. Dept., Box 7609, Raleigh NC 27695-7609, (919)737-3167
Rhoades, M. M., Biology Dept, Indiana University, Bloomington IN 47401, 812-855-9319
Rhodes, C. A., Sandoz Crop Protection Corp., 975 California Ave., Palo Alto CA 94304-1104, 415-354-3527
Rice, Thomas B., Dekalb-Pfizer Genetics, 3100 Sycamore Road, Dekalb IL 60115
Ries, Matthew N., Cargill Hybrid Seeds, Box 405, Seward NE 68434, 402-643-2549
Rinehart, Karl, Box 187, Marshalltown IA 50158, 515-752-2170
Ritchings, Bruce, Gurley Lab, Microbiol & Cell Sci/IFAS, 3103 McCarty Hall, University of Florida, Gainesville FL 32611
Rivin, Carol, Dept. of Botany, Oregon State University, Corvallis OR 97331, 503-754-4433
Robbins, Jr., W. A., P.O. Box 158, Ag. Alumni Seed Imp. Assn., Romney IN 47981, 317-538-3145
Roberts, Rich, Cold Spring Harbor Laboratories, P.O. Box 100, Cold Spring Harbor NY 11724

Roberts, Jean L., Lilly Research Labs, Greenfield IN 46140, 317-467-4788
Robertson, Donald S., Department of Genetics, Iowa State University, Ames IA 50010, 515-294-3908
Robison, Glenn, Dekalb Plant Genetics, Sycamore Road, Dekalb IL 60115, 815-758-9531
Rocheford, Torbert R., Agron. Dept. S-116 Turner Hall, University of Illinois, 1102 S. Goodwin Av., Urbana IL 61801
Rodermel, S., Department of Botany, Bessey Hall, Iowa State University, Ames IA 50011, 515-294-7172
Rodrigues, Delmo Diogo, Sem Cargill Ltda, Sitio Sao Jao Barao Geraldo, Caixa Postal, 6553, Campinas SP 13.100, BRAZIL, 0192-39-13-81
Roebbelen, G., Inst f. Pflanzenbau, Von Sieboldstr. 8, D-3400 Goettingen, FEDERAL REPUBLIC OF GERMANY
Rogers, Sharon, Lakeside Biotechnology, Inc., 2201 W. Campbell Park Drive, Chicago IL 60612
Romero-Severson, Jeanne, Agrigenetics, 5649 E. Buckeye Road, Madison WI 53716, 608-221-5000
Romig, R. W., Northrup King Co., P.O. Box 959, Minneapolis MN 55440
Roseman, Robin R., Department of Botany, University of Iowa, Iowa City IA 52240, 319-335-1331
Rosenkrans, Leonard, Schweitzer Hall, University of Missouri, Columbia MO 65211
Rosielle, A., Asgrow Seed Co, 634 E. Lincoln Way, Ames IA 50010, 515-232-7170
Roth, Brad, Pioneer Hi-Bred Internat, Inc., 7300 NW 62nd Ave., Johnston IA 50131
Roura, Lluis Bosch, Departament d'Agronomia, Escola Superior d'Agricultura, Urgell 187, 08036 Barcelona, SPAIN, 93-2304207
Rowland, Lisa Jeannine, Univ. of Florida, USDA-ARS, % 2922 N.E. 16th Terrace, Gainesville FL 32609, 904-392-7237
Rubenstein, Irwin, 220 Bioscience Center, Dept Plant Biology, University of Minnesota, St Paul MN 55108, 612-624-2716
Russell, W. A., Dept of Agronomy, Iowa State University, Ames IA 50010
Russell, W. Ken, Garst Seed Co, PO Box 500, Slater IA 50244
Russell, Doug, Washington University, Dept. of Biology, Campus Box 1137, St. Louis MO 63130, 314-889-4728
Ryan, G. S., E.E.A. INTA, 5988 Manfredi, (Cordoba), ARGENTINA
Sachan, J. K. S., Division of Genetics, I.A.R.I., New Delhi-110012, INDIA
Sachs, M. M., Department of Biology, Campus Box 1137, Washington University, St. Louis MO 63130, 314-889-4631, SACHS@BIOLGY.Wastl.edu
Saedler, H., Max-Planck Inst Zuchtungsf, Egelspfad, D 5000 Koln 30 Vogelsang, FEDERAL REPUBLIC OF GERMANY, 221-5062200
Saghai, Mohammad, CSES Department, VPI & SU, Blacksburg VA 24061
Saha, B. C., Tirhut College of Agriculture, P.O. Dholi, Dist. Muzaffarpur, Bihar-813210, INDIA
Sahi, Shivendra V., Botany Dept., Box 7612, North Carolina State Univ., Raleigh NC 27695-7612
Salamini, Francesco, Max Planck Inst Zuchtungsf, Abt Pflanzenz Ertragsphysiol, D-5000 Koln 30, FEDERAL REPUBLIC OF GERMANY
Santos, M. A., Dep Biol Molec Agro, Centro Invest Desarrollo, Gorge Girona 18-24, 08034 Barcelona, SPAIN
Sanwo, Monica M., Dept. of Botany & Plant Sci., U of CA, Riverside CA 92521, (714)787-3580
Saraiva, L. S., Genetica Dept Biol Geral, Univ Fed Vicosa, 36570 Minas Gerais, BRAZIL
Sarkar, K. R., Division of Genetics, Indian Agric Res Inst, New Delhi 110012, INDIA
Saura, Fulgencio, Div Agron Basica, Dpto Tecnologia , Univ Nacional Lujan, CC 221-6700 Lujan(Bs As), ARGENTINA
Sauvaire, D., ORSEM, Le Rezeau, 49800 Andard, FRANCE, 41 54 9721
Savidan, Yves H., ORSTOM/CIMMYT, Aptdo. Postal 6-641, 06600 Mexico D.F., MEXICO, (52)595-42100, Fax: (52)595-41069
Sayed, Sayed Galal, PO Box 953, Cairo, EGYPT, 3491619
Scanlon, Mike, G403 Agronomy Hall, Iowa State University, Ames IA 50010
Schertz, K. F., USDA-ARS, Soil & Crop Sciences, Texas A&M University, College Station TX 77843, FTS 527-1252, Comm 409/260-9252
Schiefelbein, John W., 4085 Natural Science Bldg., University of Michigan, Ann Arbor MI 48109, 313-764-3579, FAX: 313-747-0884
Schillinger, John, Asgrow Seed Company, 9672-190-16, 7000 Portage Rd., Kalamazoo MI 49001
Schmidt, Daria, 1571 Agronomy, Iowa State University, Ames IA 50011
Schmidt, Robert J., Univ of California-San Diego, Dept of Biology, C-016, San Diego CA 92093, 619-534-1636
Schnable, Patrick, Dept of Genetics, Curtiss Hall , Iowa State Univ, Ames IA 50011
Schneerman, Martha C, Dept Biol Sciences, Illinois State Univ, Normal IL 61761
Schulman, Alan H., Helsinki University, Institute of Biotechnology, Karvaamokuja 3, 00380 Helsinki, FINLAND, 358-0-434-6034, 358-0-434-6046, ASCHULMAN@CC.HELSINKI.F1
Schwall, Michael, Im Rheinfeld No. 5, 7550 Rastatt, FED REP GERMANY
Schwartz, Drew, Biology Dept, Indiana University, Bloomington IN 47401, 812-855-6060
Schwer, Joseph F., Agrigenetics Corporation, Centre Plaza North, 35575 Curtis Blvd, Suite 300, Eastlake OH 44094, 216-942-2210
Scott, Gene E., Agronomy Dept, Box 5248, Mississippi State University, Mississippi State MS 39762, 601-325-2311
Selzer, Gerald, Room 312, National Science Foundation, Washington DC 20550
Sendo, Shigeyuki, Hokkaido Plant Genet Res S Ctr, 363-2 Minami Takinokawa, Takikawa City 073, Hokkaido, JAPAN
Serials Dept, Ellis Library, Univ of Missouri, Columbia MO 65201-5149
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 and Gwen Krill-, 928 N. 123rd St., Wauwatosa WI 53226
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, N. D., Division of Biochemistry, Indian Agric Res Inst, New Delhi 110012, INDIA
Shattuck-Eidens, Donna M., NPI, 417 Wakara Way, Salt Lake City UT 84108
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
Shepherd, Nancy, Du Pont, E328/148B, Wilmington DE 19898, 302-695-1519, FAX: 302-695-7556, SHEPHERD % ESVAX@DUPONT.COM.
Sheridan, W. F., Biology Department, Univ of North Dakota, PO Box 8238 Univ Station, Grand Forks ND 58202, 701-777-4479 or 777-4206
Shilo, Judith, Dept of Biological Chemistry, The Hebrew University, Jerusalem 91904, ISRAEL
Short, Kent E., CIMMYT, Mid Altitude Maize Res Stn, P.O. Box MP 163, Mt Pleasant, Harare , ZIMBABWE, 35534, Telex 2462 CIMMYT ZW
Siddiqui, Khushnood A., Plant Genetics Division, A.E.A.R.C., Tandojam Sind, PAKISTAN

Simcox, K., OARDC-OSU, Wooster OH 44691, 216-263-3894, FAX: 216-263-3658, Simcox%00%OARDC@gate. vs.Ohio-State.Edu
Simonds, S. Caprice, 7560 Harmony Road, Sheridan OR 97378, (503)843-3021
Sims, Lynne E., Pioneer Hi-Bred Internatl, Dept Biotechnology, P.O. Box 38, 7300 NW 62nd Ave, Johnston IA 50131
Singh, N. N., Cereal Research Lab, Indian Agricultural Res Inst, New Delhi 110012, INDIA
Sinha, Neelima, Dept of Genetics, Univ of California, Berkeley CA 94720
Sinibaldi, Ralph, Sandoz Crop Protection Corp., 975 California Ave, Palo Alto CA 94304-1104, 414-354-3435, FAX: 415-858-0116
Sisco, P. H., USDA-ARS, Dept. Crop Science, North Carolina State Univ, P.O. Box 7620, Raleigh NC 27695-7620, , FAX: (919)737-7959
Smith, Margaret E., Cornell Univ, 252 Emerson Hall, Dept of Plant Breeding, Ithaca NY 14853, 607-255-1654, FAX: 607-255-6683
Smith, Oscar S., Pioneer Hi-Bred Internatl, Plant Breeding Division, P.O. Box 85, Johnston IA 50131, 515-270-3300
Smith, Steve, Pioneer Hi-Bred Internatl, Plant Breeding Div, PO Box 38, 7300 N.W. 62nd Avenue, Johnston IA 50131, 515-270-3353
Smith, Alan G., Dept Horticultural Science, 356 Alderman Hall, Univ. of MN, 1970 Folwell Av., St Paul MN 55108, 612-624-9290
Smith, James D., PO Box 2132, Department Soil & Crop Sci, Texas A & M University, College Station TX 77843, 409-845-8276
Snape, John W., Inst Plant Sci Res, Cambridge Laboratory, Colney Lane, Norwich, UNITED KINGDOM, 0603-52571
Sollinger, John, Dept. of Botany, Oregon State University , Corvallis OR 97331
Somers, David A., Dept Agron & Plant Genet, University of Minnesota, St. Paul MN 55108, 612-625-5769
Somerville, Chris, MSU/DOE Plant Research, Michigan State U, East Lansing MI 48824
Sorenson, John C., Experimental Ag Sci, The Upjohn Co, Kalamazoo MI 49001, 616-385-4582
Sorrentino (Brown), Jennifer, Cold Spring Harbor Lab, P.O. Box 100, Cold Spring Harbor NY 11724, 516-367-9808
Sotomayor-Rios, A., Tropical Agric Res Stn, P.O. Box 70, Mayaguez PR 00709
Sozinov, Alexey A., All-Union Academy of Agric. Sci., Suvorov Str., 9, Kiev-10, USSR, 290-10-85, Fax: (044)2905134
Sperling, David W., Robson Seed Farms Corp, PO Box 270, Hall NY 14463, 716-526-5879
Spielmann, A., ORSAN, 16 av. de la Baltique, 91953 Les Ulis Cedex, FRANCE, 33-1-69073919, fax: 33-1-69077312
Sprague, G. F., Dept Agron, S-120 Turner Hall, University of Illinois, 1102 S. Goodwin Avenue, Urbana IL 61801, 217-333-4254
Spray, Clive R., Dept. of Biology, 405 Hilgard Av, UCLA, Los Angeles CA 90024-1606, (213)825-3177
Springer, Patricia, Dept Biol Sci, Purdue University, W. Lafayette IN 47907, (317) 494-4919
Springer, Warren, Northrup King Company, P.O. Box 959, Minneapolis MN 55440, 612-593-7285
Sriwatanapongse, Sutat, CIMMYT, P.O. Box 39 Emek, Ankara, TURKEY
Stack, Stephen, Department of Biology, Colorado State University, Fort Collins CO 80523
Stadler, Joan, 8 Curtiss Hall, Genetics Dept, Iowa State University, Ames IA 50011, 515-294-0337
Staiger, Chris, Dept. of Cell Biology, John Innes Institute, Colney Lane, Norwich NR4 7UH, UNITED KINGDOM
Stapleton, Ann, Dept of Biol, 010A Coker Hall, U of North Carolina, Chapel Hill NC 27599-3280, 919-962-6943
Starlinger, P., Inst Genetik, Univ Koln, Weyertal 121, D-5000 Koln 41, FEDERAL REPUBLIC OF GERMANY, 221-470-2465
Staub, Rick W., Dept of Biology, Carleton College, Northfield MN 55057, 507-663-4385
Steffensen, D. M., 506 Morrill Hall, Cell Biol, Univ of Illinois, Urbana IL 61801, 217-333-3087
Stelly, David M., Dept Soil & Crop Science, Texas A & M Univ, College Station TX 77843-2474
Steyaert, Mary Ann, Curtis Hall, University of Missouri, Columbia MO 65211, 882-2033
Stiles, J. I., Dept. of Pl & Molec Physiology, Univ. Hawaii at Manoa, Honolulu HI 96822, 808-948-7354
Stinard, Philip, Dept Genetics, Iowa State Univ, Ames IA 50011
Strelow, Lisa, 759 Leland #3N, St. Louis MO 63130
Strissel, Jerry, Wilson Hybrids, Inc, P.O. Box 391, Harlan IA 51537, 712-755-3841
Strommer, Judith, Dept of Hort. Sci., Univ of Guelph, Guelph, Ont. NIG 2WI, CANADA, 519-824-4120
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
Styles, E. D., Biology, Univ of Victoria, Victoria, British Columbia V8W 2Y2, CANADA, 604-721-7101
Subramanian, R. A., Max-Planck Molec. Genet., Ihnestrasse 73, 33 (Dahlem), D-1000 Berlin, FEDERAL REPUBLIC GERMANY, FAX: (030)830-7380
Suga, Tetsuya, Japan Tobacco Inc, Plant Brdg Genetics Res Lab, 700 Higashibara Toyoda-cho, Iwata-gun Shizuoka-ken438, JAPAN
Sullivan, Sue, Garst Seed Co., Highway 210, P.O. Box 500, Slater IA 50244
Sullivan, Tom, Laboratory of Genetics, 445 Henry Mall, University of Wisconsin, Madison WI 53706, 608-262-3287
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, 912 Research Building, Beijing 100094, 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, 203-432-3858, 203-432-3844
Swaminathan, M. S., B-4/142, Safdarjang Enclave, New Delhi 110029, INDIA, 67-9069
Sylvester, Anne W., Dept of Molecular & Cell Biol., LSA 341, Univ. of California, Berkeley CA 94720, 415-643-8279, FAX: 415-643-6791
Tadic, Bojana, Maize Research Institute, P.O. Box 89, 11081 Zemun, YUGOSLAVIA, 38-11-617-434, FAX: 38-11-197-890
Talbert, Luther, Dept of Plant & Soil Science, Montana State University, Bozeman MT 59717
Talo , Anthony A., Dept of Biology , UCLA, Los Angeles CA 90024-1606, (213)825-3177
Tang, Ching-Yan, Maize Program, CIMMYT, Lisboa 27, Apdo Postal 6-641, Col. Juarez , 06600 Mexico D.F., MEXICO
Tanksley, Steven D., Dept. Plant Breeding, Cornell University, 252 Emerson Hall, Ithaca NY 14853
Tavares, J., Board on Agriculture, National Research Council, 2101 Constitution Ave, Washington DC 20418
Taylor, Brian H., Dept of Biology, Texas A&M Univ, College Station TX 77843-3258, 409-845-7754
Taylor, Loverine P., Hort. and Landscape Architecture, Pullman WA 99164-6414, 509-335-9502
Taylor, W. C., CSIRO, Division of Plant Industry, G.P.O. Box 1600, Canberra ACT 2601, AUSTRALIA, (61-6)246-5223
Thatiparthi, V. R., 412 Agronomy, Iowa State University, Ames IA 50010
The Research Scientist (Maize), National Agri. Res. Project, Maize Research Station, GAU, Godhra - 389 001, INDIA
Thomann, Estela, Dept. of Botany, Oregon State University, Corvallis OR 97331
Thome, Cathy, CIMMYT, Apdo Postal 6-641, Deleg Cuauhtemoc, 06600 Mexico, DF, MEXICO
Thompson, Jack C., FFR Coop, P.O. Box 10, Providence Forge VA 23140, 804-966-2888
Thompson, Steven A., United Agriseeds, P.O. Box 4011, Champaign IL 61824-4011, 217-373-5300
Thompson, Don L., BioTECHNICA Agriculture, Inc., 7300 W. 110th St. Suite 540, Overland Park KS 66210, (913) 661-0611

Thornbury, David W., S-305 Ag Science Center N, Dept Plant Path, University of Kentucky, Lexington KY 40546, 606-257-2681
Thorstenson, Yvonne, Dept of Plant Biology, Univ of California, Berkeley CA 94720, 415-642-8058
Tierney, Mary L., Biotechnology Center-OSU, Rightmire Hall, 1060 Carmack Road, Columbus OH 43210-1002, 614-422-3348
Timmermans, M., Rutgers State University, Waksman Institute, P.O. Box 759, Piscataway NJ 08855
Timothy, David H., Dept of Crop Science, North Carolina State Univ, Box 7620, Raleigh NC 27695-7620, 919-737-2718
Ting, Yu-Chen, Biology, Boston College, Chestnut Hill 67, Boston MA 02167, 617-552-3551
Ting, Chu-Po, Shandong Agricultural College, Taian, Shandong, CHINA
Tingey, Scott V., Du Pont Company, Experimental Station, P.O. Box 80402, Wilmington DE 19880-0402, , TINGEYSV%ESVAX%DUPONT.COM@RELAY.CS.NET
Tjhen, K., P.O. Box 2132, Dept. Soil & Crop Science, Texas A & M University, College Station TX 77843
Tomes, Dwight T, Pioneer HI-BRED Intl, 7300 NW 62nd Ave, Johnston IA 50131
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 Plant Genetics, 3100 Sycamore Road, Dekalb IL 60115, 815-756-3671
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
Tuttle, Annmarie, Ciba-Geigy Corporation, P.O. Box 12257, Research Triangle Park NC 27709-2257
Ueda , Tak, Rutgers State University, Waksman Institute, P.O. Box 759, Piscataway NJ 08855
Uhr, David V., Crop Science Department, Box 7620, N.C.S.U., Raleigh NC 27610, 919-737-2704
Ujiie, Katsuhiro, HOKUREN Naganuma Res. Stn., Minami-2, Higashi-9, Naganuma-Cho, Yuubari-Gun, Hokkaido 069-13, JAPAN
Ulrich, V., Div Plant & Soil Sci, West Virginia Univ, Morgantown WV 26506, 304-293-6258
Underwood, Douglas, Dept. of Botany, Oregon State University, Corvallis OR 97331
Usami, Satoru, Plant Brdg Genetics Res Lab, Japan Tobacco Inc, 700 Higashibara Toyoda-cho, Iwata-gun Shizuoka- 438ken438, JAPAN
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, SOUTH AFRICA
van Lammeren, Andre A. M., Dept. Plant Cytology & Morph., Arboretumlaan 4, Wageningen Agricultural Univ., 6703 B D Wageningen, NETHERLANDS
Van Montagu, M., Lab Genetics, K L Ledeganchstr 35, 9000 Gent T-2406, BELGIUM, 091 227 827
Van Schaik, N., Dept Genetics/U Witwatersrand, PO Wits 2050, SOUTH AFRICA, (011) 716-2125
Vanderslice, Olin L., Agway Inc, 2 Parkwood Circle, Cortland NY 13045
Vantoai, Tara T., USDA Agronomy, 590 Woody Hayes Dr, Columbus OH 43210, 614-292-9806
Varagona, Rita, Michigan State Univ, MSU-DOE Plant Research Lab, Plant Biology Bldg, E. Lansing MI 48824-1312, 517-353-3519
Vasal, S. K., Lisboa-27, Apartado Postal 6-641, Mexico 6, D.F., MEXICO, 5-85-43-55 EXT 115
Vasil, Indra K., Dept Vegetable Crops, University of Florida, Gainesville FL 32611, 904-392-1193
Veit, Bruce, USDA ARS PGEC, 800 Buchanan Street, Albany CA 94710, 415-559-5907
Veitenheimer, Erich E., Dekalb-Pfizer Genetics Inc, 603 W. Main St., Palmyra PA 17078
Veldboom, Lance, Dept. of Agronomy, 1529 Agronomy Hall, Iowa State Univ., Ames IA 50011
Verbeke, Pat, D M L Associates, 420 Lexington Ave., Suite 406, New York NY 10170
Veretnik, Stella, Dept Genet & Cell Biol, University of Minnesota, St. Paul MN 55108
Verma, S. C., Botany Department, Panjab University, Chandigarh-160014, INDIA
Vierling, Richard, Agronomy Dept., MS2122, Lubbock TX 79409-4169, 806-742-2831
Vilbrandt, Lisa, Agrigenetics Advanced Sci. Co., 5649 E. Buckeye Road, Madison WI 53716, 608-221-5000
Villemur, Richard, Dept of Genetics/Cell Biology, University of Minnesota, 250 BioSciences Ctr, 1445 Gortner Ave, St. Paul MN 55108
Violic, Alejandro D., CIMMYT, Cristobal Colon 3773 Dep. 23, Santiago (Las Condes), CHILE, (562)228-6685
Viotti, Angelo, Ist Biosintesi Vegetali, Via Bassini 15, 20133 Milano, ITALY
Viveros, Isolina Aguilar, CINVESTAV, Apdo Postal 629, Irapunto, GTO, MEXICO
Vodkin, Lila Ott, Dept Agron, S-110 Turner Hall, Univ Illinois, 1102 S. Goodwin, Urbana IL 61801, 217-244-6147
Vogel, Julie, Department of Plant Biology, 111 Genet. & Plant Biol. Bldg., University of California, Berkeley CA 94720, 415-642-8058
Vollbrecht, Erik, USDA, % PGEC, 800 Buchanan St., Albany CA 94710, 415-559-5907
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
Vulchinkov, Stefan, Maize Research Institute, Kneja, BULGARIA
Waines, J. Giles, Dept of Botany/Plant Sciences, University of California, Riverside CA 92521, 714-787-3706
Waiss, Tony, Western Regional Research Ctr., USDA/ARS, Albany CA 94710
Walbot, V., Dept Biol Sci, Stanford Univ, Stanford CA 94305, 415-723-2227, fa.vir@stanford
Walden, D. B., Dept of Plant Sciences, Univ of Western Ontario, London N6A 5B7, CANADA, 519-679-6757
Walker, John, Biol Sci, 110 Tucker Hall, Univ of Missouri, Columbia MO 65211, 314-882-3583, FAX: 314-882-0123, Walkbio@UMCVMB
Walko , R., USDA-ARS-PGEC, 800 Buchanan Street, Albany CA 94710
Wallace, Nancy, Dept Agronomy, Turner Hall, Univ Illinois, Urbana IL 61801
Walton, Jonathan, DOE Plant Research Lab, Michigan State University, East Lansing MI 48824, 517-353-4885
Wan, Yuechun, Dept of Agronomy, Turner Hall, 1102 S. Goodwin Ave, Urbana IL 61801, (217)333-9465
Wang, Yunxia, Department of Agronomy, University of Illinois, Urbana IL 61801
Wang, Andrew S., Northrup King Co., Research Center, Hwy. 19, Stanton MN 55081, (507) 663-7658
Ward, Kathy, Rutgers State University, Waksman Institute, P.O. Box 759, Piscataway NJ 08855
Warmbrodt, Robert, National Agricultural Library, Room 1402, 10301 Baltimore Blvd., Beltsville, MD 20705, (301)344-3875
Warnock, S. J., Campbell Institute for, Research & Technology, Route 1, Box 1314, Davis CA 95616-9610, 916-753-2116
Wassom, Clyde E., Agronomy Dept, Kansas State Univ, Manhattan KS 66506, 913-532-7253
Weaver, Sam H., The Quaker Oats Company, P.O. Box 9001, Chicago IL 60604-9001

Weber, David, Dept of Biological Sci, Illinois State Univ, Normal IL 61761, 309-438-2685
Weber, A., Institut Alle. Bot., Ohnhorststrasse 18, D-2000 Hamburg 52, FEDERAL REPUBLIC GERMANY
Weck, Edward A., Northrup King Co., Route 19, Stanton MN 55081
Weeks, Donald, Zoecon Corp, 975 California Ave, Palo Alto CA 94303
Weerakoon, Kaushalya, Dept Biol Sciences, Illinois State Univ, Normal IL 61761
Wehr, Wolfgang, Institut Fur Genetik, Universitat Koln, Weyertal 21 5 Koln 41 , FEDERAL REPUBLIC OF GERMANY
Weil, Cliff, Botany Department, Miller Plant Science Bldg., University of Georgia, Athens GA 30602, 404/542-1857
Wendel, J. F., Department of Botany, Bessey Hall, Iowa State University, Ames IA 50011, 515-294-7172
Werner, Georgina, Rhone-Poulenc Ag Co., P.O. Box 12014, Research Triangle Park NC 27709
Wessells, Catherine P. B., 28015 Stonehenge Lane, Eugene OR 97402
Wessler, Sue, Dept of Botany, Univ of Georgia, Athens GA 30602, 404-542-3732
West, Dennis R., Dept Plant and Soil Sci, Univ of Tennessee, Knoxville TN 37901-1071, 615-974-8826
West, D. P., Research Dept, Jacques Seed Company, Prescott WI 54021, 715-262-3162
Westhoff, Peter, Ins. Ent. Mol. Bio. Pflanzen, Heinrich-Heine-Univ Dusseldorf, D-4000 Dusseldorf 1, FEDERAL REPUBLIC OF GERMANY, (0277)377-2338
Whalen, R. H., Dept of Biology, South Dakota State Univ, Brookings SD 57007, 605-688-4553
Whitwood, W., Robson Seed Farms, 1 Seneca Circle, Hall NY 14463, 716-526-5879
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 , FEDERAL REPUBLIC OF GERMANY
Wilkes, H. Garrison, Biology-College II, Univ of Mass/Boston, Boston MA 02125-3393, 617-929-8448
Williams, Terrill E., Pioneer Hi-Bred Internatl, P.O. Box 128, New Holland PA 17557
Williams, T., Int. Fund for Ag Research, 1611 N. 10th St., Rosslyn Plaza, Suite 600, Arlington VA 22209
Williams, W. P., Crop Sci Res Lab, PO Box 5248, Miss. State MS 39762, 601-325-2311
Williams, Robert E., PO Box 294, Pittsfield IL 62363, 217-285-2530
Willman, Mark R., Beatrice/Hunt-Wesson Inc, 463 U.S. Hwy. 30 East, Valparaiso IN 46383, 219/477-2233
Wilson, Richard, Plant Intro Station, Agron. Bldg. Rm. G212, Iowa State University, Ames IA 50011
Wilson, Curtis M., USDA ARS NCAUR, 1815 North University Street, Peoria IL 61604, 309-685-4011, FTS 360-4011
Winkler, Rodney G., Univ. of CA - Berkeley, c/o Freeling Lab - Genet. Dept, 111 Plant Sci/Genetics, Berkeley CA 94720, 415-642-8058
Wise, Roger, USDA-ARS, Dept. Plant Pathology, Iowa State Univ., Ames IA 50011-1020
Witherspoon, W. David, Garst Seed Co., Rt 2 Box 16, Marshall MO 65340, 816-886-9877, fax: 816-886-9877
Wong, James R., Sogetal, Inc., 3876 Bay Center Place, Hayward CA 94545, 415-785-1881
Woodman, James C., Molec Genetics Inc, 10320 Bren Road East, Minnetonka MN 55343, 612-935-7335
Woodruff, Dorde, 6366 Cobblerock Lane, Salt Lake City UT 84121-2304, 801-277-5526
Wright, James, Pioneer Hi-Bred Internat Inc, PO Box 278, Old Troy Rd, Union City TN 38261
Wright, Allen, Curtis Hall, University of Missouri, Columbia MO 65211
Wright, S., Linkage Genetics, 417 Wabara Way, Salt Lake City UT 84108, (801) 582-0144
Wu, Madeline, Dept Biol Science, Univ of Maryland, Baltimore Co., Catonsville MD 21228
Wurtzel, Eleanore, Dept Biol Sci, Davis Hall, Lehman College, City Univ New York, Bronx NY 10468, 212-960-8483
Xie, You-Ju, College of Biology, Beijing Agricultural Univ, Beijing, CHINA
Xu, Wenwei, Waters Hall, University of Missouri, Columbia MO 65211
Yamada, Minoru, Dept Plant Breeding, Nat Grassland Res Inst , 786 Nishi-nasuno, Tochigi, 329-27, JAPAN, 0287-36-0111 ext. 285
Yano, Masahiro, Hokuriku Natl Agr Exper Sta, 1-2-1, Inada, Joetsu, Niigata 943-01, JAPAN
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
Yerk , Georgia, Dept. Agronomy & Plant Genet., Univ. of Minnesota, 1991 Buford Cir., St. Paul MN 55108, (612)625-1208
Youngquist, Wayne C., Agronomy Dept, Univ of Nebraska, Lincoln NE 68583-0915, 402-472-1531
Zabala, Gracia, Plant Biol/289 Morrill Hall, Univ Illinois, 505 S. Goodwin Avenue, Urbana IL 61801, 217-333-2919, FAX: 217-244-3499
Zabulionis, Ray, Department of Plant Sci, B & G Building, University of Western Ontario, London, Ontario N6A 5B7, CANADA
Zaitlin, David, Agrigenetics, 5649 E. Buckeye Road, Madison WI 53716
Zarowitz, Michael, Escagenetics, 830 Bransten Road, San Carlos CA 94070, 415-595-5335
Zeng, Mei, Washington University, Dept. of Biology, Campus Box 1137, St. Louis MO 63130, 314-889-4728
Zeng, M. Q., 100012 Institute of Genetics, Academia Sinica, Beijing, CHINA, 44,6551-556
Zhao, Zuo-Yu, Biotechnology Research, Pioneer Hi-Bred Int'l , 7300 NW 62nd Ave. P.O. Box 38, Johnston IA 50131, 515-270-3644, FAX: 515-270-3444
Zhao, Ji-ping, Department of Agronomy, Shandong Agric University, Taian, Shandong Province, CHINA
Zidwick, Mary Jo, Cargill Research Department, Box 9300, Minneapolis MN 55440
Zimmer, Elizabeth, Lab of Molecular Systematics, Support Ctr. Nat'l Museum Nat. History, Smithsonian Inst, Washington DC 20560, 301-238-3025, FAX: 301-238-3667, ZIMMERE@SIMSC

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IX. AUTHOR AND NAME INDEX

("r" refers to numbered references in the Recent Maize Publications section)
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March, 1991

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This is an informal newsletter by which working research information on the genetics and cytogenetics of maize is shared. The information and data are shared by Cooperators with the understanding that they will not be used in publications without their specific consent.

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