MAIZE GENETICS COOPERATION

NEWSLETTER

67

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The encounter that changed the world - Three ships, three crops, the global merging of humankind.

Drawing by Walton C. Galinat, 1992

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The working research information here is shared with the understanding that each item is unpublished and is not to be cited in publications without specific consent of the authors.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 suggested guidelines may ensure against misunderstanding of our Newsletter:

1) In publications, whenever permitted, refer to MNL notes in the text, rather than 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 Specify "unpublished data", or "personal communication" (i.e., with the colleague's consent).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) When preparing your MNL notes, emphasize 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 supply notes, tables and figures in electronic form, and this greatly facilitates the editing and compiling.

Gifts to the Endowment Fund for support of the Newsletter now total over $80,000. Please see the listing, in the front of this issue, of donors whose generosity has made this total grow. We are all grateful for the support of our colleagues and of organizations with which we have common interests. The continuity and support necessary for collecting genetic and molecular information, evaluating it, and preparing gene lists, maps, and similar syntheses, however, is made possible only by sustained and ongoing encouragement of this work within the Agricultural Research Service, USDA, specifically as part of my regular research project and most recently as part of the Plant Genome Initiative project for development of a database prototype for maize.

A warm acknowledgment for help, advice and ideas during the past year is given to my colleagues, Gerry Neuffer, Mary Polacco, Georgia Davis, Pat Byrne, Shiaoman Chao, Larry Darrah, Jim Birchler, Karen Cone, Kathy Newton, Jack Beckett, Guri Johal, Mike McMullen, Don Miles, and John Walker. Their advice and encouragement, not to mention tolerance, is greatly appreciated.

Shirley Kowalewski skillfully edited and nurtured the contents from rough into fine form, surprised the word-processor by getting it to do what was needed, structured the year's literature, and gave creative advice at critical moments. Mary Ann Steyaert booked addresses and subscriptions through the year, carried out literature searching and verifications with efficiency and accuracy, and artfully prepared the mockup. Denis Hancock steadily and enthusiastically enhanced our computer efficiency to a higher art. Kudos for vigilance and communications are given to Lou Butler and Evelyn Bendbow, for their contributions of accuracy to the gene list and other places. At University Printing Services, Yvonne Ball and the printshop staff again efficiently ensured the job was done promptly and well.

For submission of notes for the next issue (Number 68, 1994), 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.

The next Maize Genetics Conference is scheduled at Pheasant Run, March 24-27, 1994. Details about the Conference will be mailed to former attendees in November, 1993; others may request the mailing by providing their address to Coe. The program and abstracts are provided by Bill Sheridan.

The Steering Committee for the Maize Genetics Conference is:

Kathy Newton, chair
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Karen Cone
Alfons Gierl
Tim Helentjaris
Tom Peterson
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ALBANY, CALIFORNIA
Plant Gene Expression Center

Gene conversion-like rearrangement at the *Kn1-0* tandem duplication

--Julie Mathern and Sarah Hake

The homeobox-containing *knotted* (*kn1*) locus of maize is characterized by a series of dominant alleles affecting leaf development. All but one of the *Kn1* mutations are caused by insertions of transposable elements into introns. The exceptional *Kn1-0* allele consists of a tandem array of two 17kb repeat units (Veit et al., Genetics 125:623, 1990), each containing the entire *Kn1* transcription unit. Analysis of *Kn1-0* derivatives indicates that the duplication itself conditions the mutant phenotype due to the novel context of the proximal transcription unit. Insertion mutagenesis of the *Kn1-0* allele produced four derivatives which altered the mutant phenotype to nearly normal. Three of the insertions are Mutator elements, and have inserted within a 316bp region 5' to the proximal transcription unit. One of the derivatives, *Kn1-174*, contains a *Mu1* element. We have shown that the mutant phenotype of *Kn1-174* is regulated by methylation of the *Mu1* element (Lowe et al., Genetics 132: 813, 1992). When the *Mu1* element is hypomethylated, or active, the phenotype is near normal, with mild ligule displacement on early leaves. Conversely, when the *Mu1* element is methylated, or inactive, the phenotype is mutant, resembling the *Kn1-0* allele.

Further genetic analysis of the *Kn1-174* derivative yielded progeny which were completely wild type in phenotype. Unlike the progenitor, this new derivative, *Kn1-174a*, is stably normal; knotted progeny have not been found in a population of 500 heterozygous *Kn1-174a*/+ individuals. Sequence data show that *Kn1-174a* has lost the *Mu1* element and instead has sustained a rearrangement. The rearrangement places sequences outside the tandem duplication, 5' to the proximal transcription unit (Fig. 1). Thus both transcription units now have the same 5' sequences for approximately 2.4kb. According to our model for the *Kn1-0* mutant phenotype, both transcription units are now in a "wildtype" context.

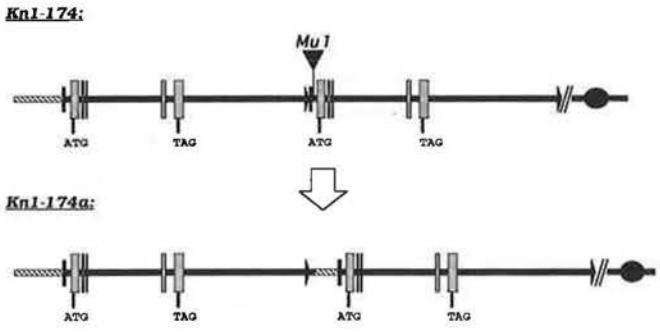

Figure 1.

We propose that gene conversion is responsible for the *Kn1-174a* rearrangement. We previously documented a high frequency of loss of one of the tandem repeats in *Kn1-174* and the other *Mu* insertion alleles at *Kn1-0*. The high frequency of repeat loss required active *Mu* elements (Lowe et al., Genetics 132: 813, 1992). When the *Mu* element excises, the broken chromatid ends are highly recombinogenic and pair with homologous sequences. Since the *Kn1-0* sequences are duplicated, pairing could occur at two different places, at the proximal repeat that contained the *Mu* element, or at the distal repeat. We propose that the pairing in *Kn1-174a* was misaligned thus bringing sequences normally 5' to the distal repeat in a position 5' to the proximal repeat.

A new mutation affecting tassel and ear morphology

--Laurie G. Smith and Sarah Hake

A recessive inflorescence mutation has been recovered that has some phenotypic similarity to previously described *barren stalk* and *barren inflorescence* mutations, but is generally more severe. Though the phenotypic expression of this mutation is somewhat variable, mutant individuals recovered after outcrossing to various inbreds and selfing consistently show a severe reduction in tassel branching and in the production of spikelets in both the tassel and ear. In the most severely affected mutants, the apical inflorescence consists of an elongated, unbranched, bare rachis lacking any floral structures whatsoever, which becomes coiled up to produce a corkscrew-like form (Fig. 1). The effects of the mutation on ear morphology have not been examined in detail, but are similar. The vegetative development of these mutants appears normal, except that severe mutants are dwarfed as adults, perhaps as an indirect consequence of the failure to initiate normal floral development.

Figure 1.

Plants heterozygous for this inflorescence mutation were crossed to dominant *barren inflorescence* (*Bif*) mutants; *Bif* F1 progeny were selfed to generate F2 families segregating both mutant phenotypes. Among F2 families segregating both mutations, three quarters of the plants showed the *Bif* phenotype (heterozygotes and homozygotes) and one quarter showed the severe bare rachis phenotype described here; no normal individuals were found among a total of 60 F2 individuals scored. This suggests that the new inflorescence mutation is linked, or possibly al-

lelic, to *Bif*, which has been mapped to 8S. Allelism tests with the recessive *barren inflorescence* mutations (*ba1*, *ba2*, and *ba3*) have not been done. Two independently derived alleles of this new inflorescence mutation have been recovered from the progeny of self-pollinated individuals with active Mutator transposons; these mutations may therefore be caused by *Mu* element insertions. Segregating families for these mutations can be made available upon request.

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_Ac-st_ and automutagenesis of _Kn1-2F11_

--Erik Vollbrecht and Sarah Hake

Knotted1 (Kn1) encodes a homeodomain-containing protein that is expressed in vegetative and floral meristems. Dominant mutations (*Kn1*) that alter leaf development apparently still provide *Kn1* function: *Kn1* mutants produce wildtype gene product (KN1) and express KN1 in meristems in a wildtype pattern. Additional, ectopic expression of KN1 in leaves causes the knotted phenotype in the mutants. A small, x-ray generated deletion of chromosome 1 that includes *Kn1* (*kn1-del*) is lethal to the pollen but transmits through the female, and is an embryonic lethal when uncovered by TB-1La. Although *kn1-del* does not delete RFLPs closely linked to *Kn1*, the extent of the deletion is unknown and it may include additional loci. Thus while analysis of the wildtype and dominant mutant alleles has revealed much about the role of *Kn1* in development, no loss of function mutation at the locus has yet been recovered.

The *Kn1-2F11* mutation was used in a mutagenesis experiment designed to generate germinal derivatives with loss of *Kn1* function. *Kn1-2F11* is caused by a *Ds2* insertion in an intron 150bp from an intron-exon junction. Transposon induced mutant alleles serve as excellent starting material for mutagenesis experiments. The resident transposon can act as a mutagen to create DNA alterations through events such as imprecise excisions, intragenic transpositions or rearrangements. One can screen for changes from the mutant phenotype, such as reversion to wildtype or alteration to a novel mutant phenotype. Critical to this approach is a mutant phenotype that is easily scored and reliably manifests when the mutation is present. *Kn1-2F11* confers a mild knotted phenotype that is evident principally in the first one to three seedling leaves. The *Kn1-2F11* mutation is also *Ac* dependent: penetrance and expressivity of the mutation increase markedly when an active *Ac* element is present in the genome (Vollbrecht and Hake, MNL64:4). Interactions between transposase and *Ds2*, and not transposition-associated DNA changes, appear to underlie the *Ac* dependence of *Kn1-2F11* expressivity. In our *bz2-m* background, penetrance of *Kn1-2F11* is 0-0.1 when no *Ac* is present (0% to 10% of those plants containing *Kn1-2F11* manifest the knotted phenotype). When a standard *Ac* is in trans, penetrance increases to between 0.4 and 0.8. Most standard *Ac* elements are similar in their effect on *Kn1-2F11* when compared in similar genetic backgrounds. We recently discovered that McClintock's *stabilized Ac* (*Ac-st*) is exceptional in its effect on *Kn1-2F11*. When *Ac-st* is present, penetrance of *Kn1-2F11* is nearly 100%, and expressivity increases dramatically such that both seedling and adult leaves display a severe knotted phenotype. When *Ac-st* is present, *Kn1-2F11* satisfies the criteria for an automutagenesis of the type described above. The data reported here are results of a pilot mutagenesis experiment, carried out in the field in which the *Ac-st* effect was first observed.

Given that *kn1-del* does not transmit through the male, this experiment used the *Kn1-2F11* parent primarily as the female, although *Kn1-2F11* was also used as the male in a few crosses. The basic cross was:

F0 (*Ac11*/-; *Kn1-2F11*; *bz2-m*; *R*) X (*kn1*; *bz2-m*; *Ac-st*/-; *r-m3*/*R*)

F1 All (*Kn1-2F11*/*kn1*; *bz2-m*); ±*Ac-st* and ±*Ac11*.

Ac11 is a standard *Ac* element present in the *bz2-m* tester stock, and causes early transposition in the *bz2-m* background (Dawe and Freeling, MNL65:33). A few *Kn1-2F11* lines containing other standard *Ac* elements were in the field and were also used. Standard *Ac* is included in the *Kn1-2F11* parent to induce transposition of the resident *Ds2* and thus potentially generate change of state derivatives, and *Ac-st* is in the outcross parent to enhance the knotted phenotype when *Kn1-2F11* transmits unchanged to the F1. F1 kernels showing aleurone mutability (bronze to purple) were sorted into two classes: (1) *Ac-st* kernels: those exhibiting a spotting pattern typical of *Ac-st* (± a superposed *Ac* spotting pattern; *Ac-st* and *Ac* show little or no dosage interaction and can be scored simultaneously) and (2) Generic mutable kernels: those exhibiting a spotting pattern that was either typical of standard *Ac* dosage or unclassifiable. Kernels were planted in the greenhouse and seedlings were screened for alterations from the *Kn1-2F11* phenotype, i.e., for wildtype or novel individuals. In most families the *Ac-st* class showed sufficiently high *Kn1-2F11* penetrance to suggest non-knotted individuals had truly undergone changes at the *kn1* locus (Table 1). Penetrance in the "generic mu-

Table 1. F1 seedling phenotypes, kernels with *Ac-st* spot pattern.

Ac element^	family #	# kernels	# wt sdlgs	% wt
	Kn1-2F11 transmitted as female.			
Ac11	E29	62	5	8
	E30	75	9	12
	E31	50	3	6
	E32	60	3	5
	E33	90	8	9
	E34	40	4	10
	E35	35	2	6
	E36	18	3	17
	E59	30	4	13
	Total *Ac11*	460	41	8.9
wx-m9	E45	40	3	7.5
	E46	24	5	21*
	E47	80	14	18*
wx-m7	E48	14	5	36*
	E51	76	6	7.9
r-nj:m	E53	54	many	high*
	E54	58	many	high*
	E55	55	many	high*
	E56	67	14	21*
	E57	38	2	5
	E58	27	0	0
	Total *nj:m*	65	2	3.1
bz1-m2	E49	80	1	1.3
Total, as female**		721	53	7.4
	Kn1-2F11 transmitted as male.			
Ac11	E37	64	4	6
	E38	24	2	8
	E39	40	2	5
	E40	34	1	3
	E41	39	1	3
	E42	23	1	4
	E43	40	7	18*
	E44	24	0	0
Total, as male**		248	11	4.4

Table 2. F1 seedling phenotypes, generic mutable kernels.***

Ac element^	family #	# kernels	# wt sdlgs	% wt
	Kn1-2F11 transmitted as female.			
Ac11	E32	120	11	9
	E59	90	6	7
wx-m9	E46	90	7	8
r-nj:m	E57	42	3	7
Total, as female**		342	27	7.9
	Kn1-2F11 transmitted as male.			
Ac11	E37	185	4	2
	E38	135	8	6
Total, as male**		320	12	3.8

Table 3. Combined F1 phenotype totals.**

Kn1-2F11 as female	1063	80	7.5%
Kn1-2F11 as male	568	23	4%
Total, female + male	1631	103	6.3%

Table 4. Southern analysis of F1 plants (putative revertants).**

Summary of Southern data for 103 seedlings.

3	no DNA
2	self contaminants
23	Contained Kn1-2F11 (low expressivity)
75	revertants
103	total

Net reversion frequencies of *Ds2* from *Kn1-2F11* for different *Ac*'s.

Ac element^		# kernels	# wt	% wt^^
Ac11	as female	670	47	7
	as male	568	19	3.3
	total *Ac11*	1238	66	5.3
wx-m9	as female	130	5	3.8
wx-m7	as female	76	1	1.3
r-nj:m	as female	107	2	1.9
bz1-m2	as female	80	1	1.3
Totals	as female	1063	56	5.3
	as male	568	19	3.3
Combined molecular total		1631	75	4.6

NOTES on tables:
^Refers to the *Ac* element effecting transposition in the F0.
^^Indicates reversion frequency per chromosome.
*When occurrence of WT seedlings was > 15%, we assumed poor penetrance (<85%) rather than high reversion, and these families were not investigated further.
**Families of assumed poor penetrance (see above) are not included.
***Generic mutable refers to spotted kernels in which unambiguous classification of mutability pattern (*Ac-st* or *Ac*) was not possible.

table" class was lower and only a few of these families were useful (Table 2). In reciprocal crosses involving the *Ac11* lines, the recovery of wildtype was lower through the male (~4.1%, Table 3) than through the female (7.5%, Tables 1 and 2).

Wildtype seedlings were transplanted, tissue samples were taken for Southern analysis of genomic DNA and plants were eventually selfed. Hybridization of genomic DNA blots with a series of *Kn1* fragments scanned 11kb of DNA. This region includes most of the *Kn1* transcription unit, and 1.5kb of 5' sequences. Southern analysis identified 2 self contaminants (showed no outcross parent RFLPs), 23 non-excision individuals that still contained the *Kn1-2F11* mutation, and 75 excision individuals that no longer contained *Ds2* (Table 4). The 75 excision plants showed no novel RFLPs within the 11kb scanned by the genomic Southerns. No intragenic transpositions, deletions or rearrangements were detected. Southerns would have detected even short-range transpositions, as the restriction enzyme used (*Bam*HI) cuts within *Ds2*. To date, all 75 excision events appear to be reversions to *kn1* that transmit normally and confer a wildtype phenotype. Molecularly corrected reversion frequencies per chromosome still proved higher through the female than through the male (5.3% vs. 3.3%, Table 4). The higher frequency of reversions conditioned by *Ac11* (5.3%) compared to other *Ac* elements (2.3 % combined) could reflect *Ac11*-induced somatic ear (or tassel) sectors due to early transposition events, or could reflect an increase in *Ac11*-induced transpositions. Our data do not favor one interpretation over the other.

No germinal derivatives with imprecise excisions, rearrangements or intragenic transpositions were detected in this experiment. We assume that some DNA rearrangements or alterations would cause loss of *Kn1* function and would be recessive to wildtype *kn1*. Lack of recovery of a recessive was not likely due to inability of *kn1* mutations to transmit, since *kn1-del* transmits normally through the female. While our Southern analysis may not have detected imprecise excisions changing ≤50bp (the excision *Bam*HI fragment is 5kb), if they do exist in the excision population we can only infer that either such alterations (within an intron) do not affect *Kn1* gene expression or their effects are unimportant for screened aspects of the phenotype. We similarly assume that some intragenic *Ds2* transpositions would result in *kn1* (or *kn1-m*) alleles. Of the many (>12) transposon-induced *Kn1* dominants that have been recovered, all but one have insertions into the same intron, near the site of the *Ds2* insertion in *Kn1-2F11*. Thus we infer the intron region is unique, perhaps containing sequences relevant for *Kn1* regulation, and insertions elsewhere within the gene would likely not cause the knotted phenotype and perhaps cause loss of *Kn1* function. We may have missed intragenic transpositions in our screen if they conferred a knotted phenotype. It was reported for *Wx-m5* that intragenic *Ds* transpositions (unidirectional, within a ~4kb region) occurred at a frequency of 5.5×10^{-4} (Weil et al., Genetics 130:175-185, 1992). Although our experiment would have detected bidirectional *Ds2* transpositions within an 11kb interval, our population size may have been too small to generate such derivatives in this pilot experiment. The results of this experiment, however, suggest the *Kn1-2F11* + *Ac-st* system can be utilized to generate and recover germinal change of state derivatives, including the elusive *kn1* mutation. We have begun a much larger scale reversion experiment, with *Kn1-2F11* plants containing one dose of *Ac11* crossed by homozygous *Ac-st* lines. 15,000 F1 plants were screened in the field and roughly 500 putative wildtypes identified. Wildtype plants were selfed and F2 progeny screening is in progress.

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Mutables at *in*

--Peter A. Peterson

in-m918732-13. This is an early sectoring *in*-mutable. It arose in an *En/Spm* isolation plot. In the cross *c2/c2 in-o/in-o* x *c2/c2 in-m/in-o* the following segregation ratio was observed.

pale	pale cl-areas	cl	Total
86	199	175	460

Whereas the pale class is expected to be 50%, it is seriously deficient. Such a ratio arising from male transmission has the earmarks of a gamete factor, though none have been reported in chromosome 7. This aspect will be pursued.

in-m918732-14. This *in*-mutable arose in a plot containing *En/Spm*. The mutability pattern shows heavy (early) colorless sectors on a pale colored background from the cross *c2 c2 in-m/-* x *c2 c2 in in*. The following segregation was observed. The *in-m*

parent would be homozygous or heterozygous.

	pale	pale cl-sectors	cl	Total
92 0236-3/0336	133	71	15	219
92 0236-5/0301	58	126	53	237

Plant -3 could be heterozygous. Plant -5 could be homozygous with a high incidence of changes to pale (non-mutable) and colorless via excisions. The sectors for this mutable allele are early-occurring.

in-m918732-4. This mutable *in-m* arose in an *En/Spm* plot. In a confirming cross *c2/c2 in-m/in-o* x *c2/c2 in-o/in-o*, the following segregation was seen.

	pale	pale-cl areas	cl	Total
92 1201y-21/0528	88	59	35	171

The high frequency of colorless (cl) indicates a high rate of revertants, which agrees with the pattern of mutability showing early colorless areas.

in-m918732-19. This *in*-mutable arose in an *En/Spm* isolation plot. From a self of *c2/c2 in-m/in-o*, kernels with colorless sectors on a pale background were selected. In the cross *c2/c2 in-o/in-o* x *c2/c2 in-m/in-o* the following segregation was observed.

	pale	pale-cl areas	cl	Total
92 0303/0245-9t	1	122	90	213

The segregation indicates that plant -9t was homozygous *in-m/in-m* and would appear to be autonomously mutable (lack of pale segregants). The high frequency of colorless (cl) is likely due to revertants but must be tested.

There are approximately 35 other *in*-mutables that are currently being confirmed. All came from an *En/Spm* containing plot. Though all contain and show an *En*, none has been confirmed as to a system in a strict co-segregation test. There has been a crucial lack of a confirmed usable reporter allele for *En* but this is now corrected.

C-bk897174

--Peter A. Peterson

In an isolation plot that was *C C* and included the *Dt* element, crossed by *c sh wx*, a kernel was isolated that showed colorless sectors on a colored background. This appeared to be a loss of *C*, exposing *c*. In crosses with the *wx-m1* reporter, *Ac* activity was not evident. From a self of a plant that was *C-Bk/c*, the following segregation was observed.

	Colored	Sectored	Colorless	Total
92-3212-12@	126	62	202	390

Because 1/2 of the progeny of this self are colorless, it would appear that the sectored type is not male transmissible. The colored class could come from crossovers of the *breaker* locus with the *C* allele. This hypothesis will be tested further. There are a number of *breaker* phenotypes, unrelated to *Ac-Ds* that are being explored.

En2 (wx86 0246x) - potency of S and M functions

--Peter A. Peterson

En2 is a derivative of *En1 (wx844)* that is deficient in ORF2 and part of ORF1 of the *En* element. The remainder of the element (11 exons) is intact. When tested for the two genetic functions of *En*, the *S* function is expressed. Not, however, completely, because gown coloration is evident in *En2 a-m15719* (*a-m15719* is colored in the absence of *En*). The low frequency of spots with this same combination indicates that the M function is minimally expressed. These conclusions are limited to the observation of *En2* with *a-m15719*. Do these observations define the functionality of the element? When another responder is used, a totally different picture is seen. With *a-m(Au)pale m(r)871618W-1* the response is relatively quite high mutability. It is this writer's conclusion that the *a-m15719* allele does not adequately gauge the potency of *En2*, implying therefore that the limits of the full functionality of ORF1 have not been determined. A further observation of this functionality of the TNPA (S) function is seen in the progeny of a self of *Wx/wx860246x*. When heavy gown color is selected, they are all found to be *Wx*. If this proves to be consistent, it would mean that the dosage (*Wx/Wx/wx860246x* vs. *wx860464/wx860246x*) of the TNPA protein is significant in expressing the potency of the S function.

c2-m857272 - A geneticist interprets molecular events

--Peter A. Peterson

This allele is a low mutating type with very few excision events (2b), however, sectors that are motley appear. The motley kernels, though they look like *R r r*, are proven to be *R R*. When the 2b is crossed to an *En* reporter allele, *En* activity cosegregates with 2b. Thus, the 2b allele is a low mutating *En* at the *c2* locus. When motley germinals are backcrossed (x *c2 c2*), the low mutating type appears. The segregating pattern indicates that a separate independent factor converts the 2b pattern to a motley pattern. It seems that the 2b is autonomously mutable (*En*) at *c2* and it is methylated, but from a near quiescent (2b) state shows a motley appearance. This is proven by testing with *wx-m8* which shows sectoring when the motley (active) appears, but no sectoring when it is quiescent.

The volatility of the maize genome

--Peter A. Peterson

When appropriate searches are made for new instabilities, one is confronted with many surprises. Though a specific transposon is relegated to the search and the target, other transposons appear in the final analyses. This is the case for *Ac-Ds*. This has occurred in several instances and some of these are related here.

The first instance is that of the activation of quiescent *Uq*s, some of which have turned out to be *Ac*. This became apparent with the use of reporter alleles. In this case the reporter allele was *c-ruq*. Thus, in ordinary breeding lines, these events may occur with no cognizance of the activation of these elements because appropriate reporter alleles are not present. Thus, these particular transposons in this particular instance were quiescent due possibly to methylation, and subsequently, and in periods of normal growth and development, are periodically activated. There has been no stress induced activation such as a bridge-breakage-fusion cycle that has been particularly targeted for these events, but during normal processes of development. Note again that these would not appear except for the presence of the appropriate reporter allele.

Other cases are when specific genes are targeted. In a number of instances they appear as chromosomal breakers. Chromosomal breakers, because a number of markers are lost simultaneously. This is the case with *C-I-b836024*. In this case, it turns out to be a fully active *Ac*. It is not related to any two-element arrangement

but certainly all the evidence suggests that it is a fully functional *Ac*. Its activity includes changes of state and transposition of the element to nearby sites.

Other elements are also known and these are elements that induce the same kind of event that has also been identified. This includes *C-m897140* (1992 Newsletter). This also arose as an independent event in an isolation plot where the *C* locus was targeted. And, in this instance, a number of elements were present, however, only the *Ac* was identified with the breaker event.

It would appear that the corn genome is highly volatile. That is, these transposons are now known to be quiescent in the genome. They appear also to be spontaneously activated. This is evident in almost any line one looks at with appropriate reporter alleles as indicated in the paper by Pan and Peterson (1988), where sectors were pervasive in these lines. It would appear, therefore, that the corn genome has a built-in system of activation and redeployment of genome segments and in a final result, leads to a great deal of variability.

Quantitative genetic variation near *an1* and *bx1*

--M. Lee and L. R. Veldbloom

We have been investigating the relation between quantitative trait loci (QTL) detected by RFLP mapping and loci defined by alleles with qualitative effects (mutants). As proposed by Robertson (J. Theor. Biol. 117:1-10, 1985) qualitative and quantitative genetic variation may trace to the same loci. At a locus, it may be possible to recover alleles conferring a nearly continuous range of effects on the phenotype of interest. Alleles with extreme effects (possibly defective products or nulls) are easily recognized by their mutant phenotypes (dwarf plants, defective kernels, pigmentation, etc.). In contrast, alleles with more subtle effects (quantitative) would be more difficult to assess and characterize. The proposed relation between quantitative and qualitative variation may seem intuitively obvious to some; however, direct tests of the hypothesis have been difficult to design and conduct. We have attempted to conduct such tests by using clones of functionally defined genes as RFLP probes in mapping experiments of quantitative genetic variation. As the various maize maps (RFLP, mutant, and cytological) are merged and more clones of defined function become available, rigorous review of the presumed relationship should be possible. If the relationship is generally true, information from quantitative and qualitative approaches to mapping and genetic analysis should be complementary on the basis of their relative strengths for identifying interesting regions (via QTL mapping) and defining candidate loci (via mutants). In this note, we briefly report two cases supporting Robertson's hypothesis.

In the first case we used a genomic probe at or tightly linked to *an1* as a marker in the analysis of genetic variation for plant height. The probe was provided by S. Briggs, Pioneer Hi-Bred Intl. The *an1* locus is defined by a recessive, GA-responsive dwarf phenotype. The probe was mapped in a F2 population of 150 plants created by crossing inbreds Mo17 (ca. 167cm tall) and H99 (ca. 103cm). The same plants and their selfed progeny were evaluated in replicated experiments for several quantitative traits including plant height. The *an1* probe was placed to the long arm of chromosome one and was closely linked to a QTL for plant height. The probe defined an interval on the genetic map which accounted for 40% of the phenotypic variation for plant height with additive effects estimated at 15cm (i.e. on average, plant height is predicted to increase by 15cm with each substitution of the "allele" from Mo17 for the "allele" from H99). We have observed similar effects on plant height by this region in other populations. Of course, there are other loci in the vicinity of the *an1* QTL (e.g. *D8*) or other plant height QTL in other populations (Beavis et al., Theor. Appl. Genet. 83:141-145, 1991) that could contribute to variation in plant height. Determining which locus, if any, is the primary source of genetic variation in the population represents a major obstacle for direct tests of the hypothesis for most traits.

In the second case, we used the same materials and evaluated them for resistance to leaf blade feeding by the European corn borer. H99 and Mo17 are highly resistant and susceptible, respectively. The QTL with the largest contribution to resistance accounted for 17% of the variation in this population and was located (genetically) to an interval at the end of the short arm of chromosome four. This is also the vicinity of the *bx1* locus. This locus is defined by a recessive phenotype lacking cyclic hydroxamates which inhibit corn borer larvae (Mo17 and H99 are +/+ at *bx1*). Currently, it is the only known maize locus with such effects. Therefore, alleles with quantitative effects at the *bx1* locus seem to be plausible sources of genetic variation in this population.

A second putative QTL for resistance to leaf blade feeding was located to the long arm of chromosome one. The interval defining the QTL accounted for nearly 16% of the variation. To our knowledge, loci affecting resistance to European corn borer have not been described in this region. Certainly, mutant alleles with qualitative effects have not defined such loci (or molecules) in this region. In a crude way, QTL mapping studies may identify the functional significance of genetic regions in a very efficient manner. Once more efficient techniques have been developed for map-based cloning and for more thorough, directed evaluation of genomic regions, information from QTL mapping studies should provide important clues about starting positions and targets of the search.

Integrating maize and sorghum RFLP maps

--C. Spike and M. Lee

We are using a recently completed sorghum RFLP map and established maize maps to investigate the extent and nature of genetic linkage between these organisms and to integrate our sorghum and maize maps. Sorghum and maize are diploid members of the tribe Andropogoneae with 2n=20. Previous reports have documented a high degree of homology and conserved linkage between maize and sorghum as determined by low-copy maize probes (Hulbert et al., PNAS 87:4251-4255, 1990; Whitkus et al., Genetics 132:1119-1130, 1992).

The information in this note focuses on linkage group F of our sorghum map. This map was created from a population of 78 F2 plants from a cross between CK60 and PI229828. The map consists of 201 loci placed into 10 linkage groups. Most (134) of the loci were defined by previously unmapped maize cDNA clones. The maize mapping population consisted of 150 F2 individuals from a cross between inbreds Mo17 and H99. The sorghum and maize maps were created from restriction fragment length polymorphisms detected by probes of several sources, Brookhaven National Laboratory (bnl), University of Missouri-Columbia (umc), Pioneer Hi-Bred Intl., Inc. (pio), and Native Plants, Inc. (npi). Other probes for the maize map were provided by C. Hannah, Univ. of Florida (*agp2*) and M. Scanlon and M. James, ISU (*dek*-1047*, *dek*-326*). Additionally a library of maize cDNA clones (jc) was

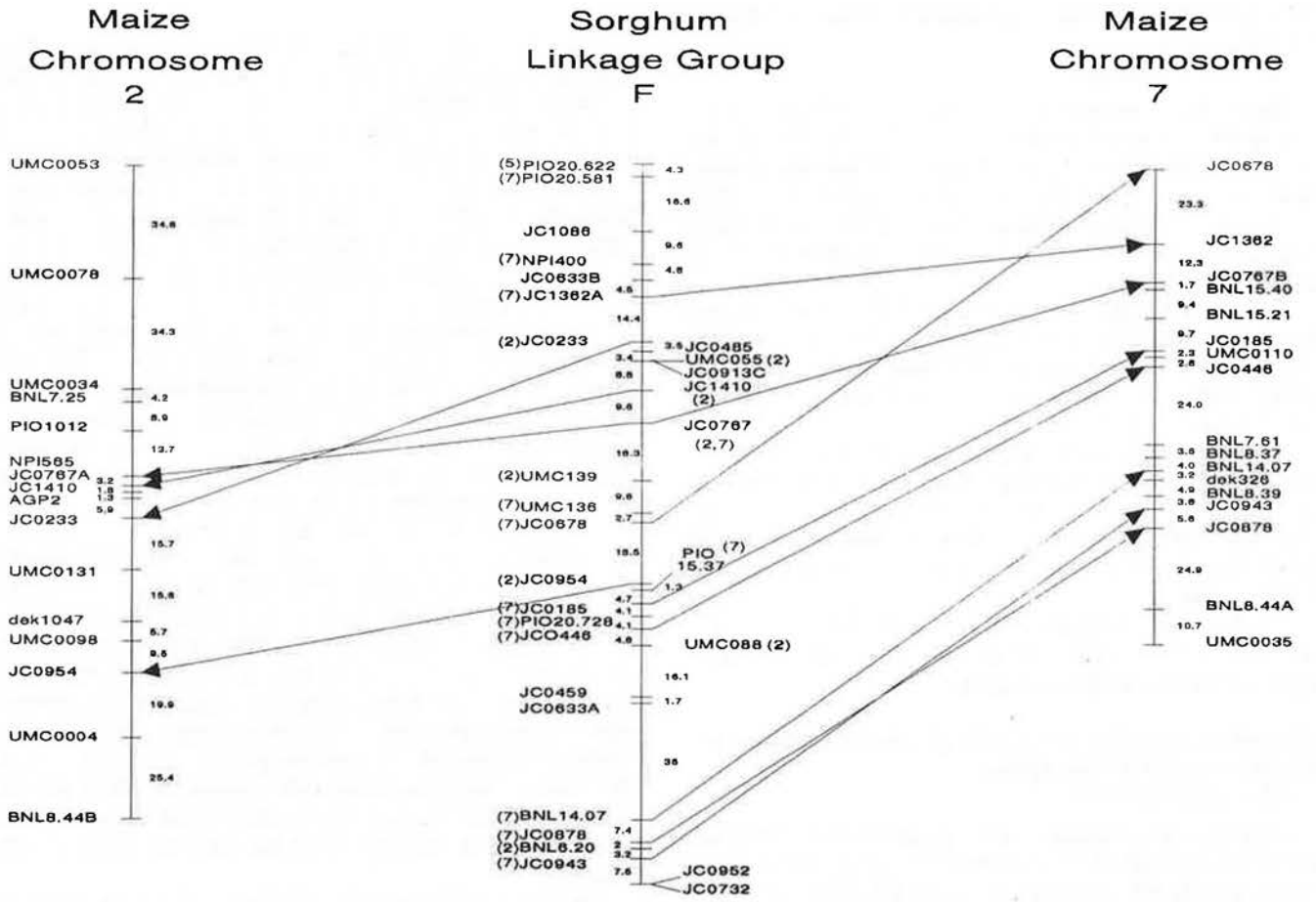

Figure 1. A comparison of RFLP linkage maps between sorghum linkage group F and maize chromosomes 2 and 7. Numbers directly adjacent to the vertical lines identify genetic distances in cM. Numbers in parentheses next to loci on the sorghum linkage group indicate the maize chromosome containing that locus. Capital letters at the end of locus designators indicate duplicate loci.

developed at Iowa State University and used as a source of probes for the sorghum map. The jc clones are the primary focus of this investigation because they identify most of the loci on the sorghum map and their map positions were unknown in maize.

jc clones which detect polymorphisms for the parents used to create the maize map were chosen, and those located on sorghum linkage group F were mapped in the maize population. The RFLP segregation data were analyzed and integrated into the established maize map by MAPMAKER (Figure 1). For assigning linkage, a LOD threshold of 3.0 and recombination frequency of 0.4 were used.

The map clearly indicates that jc clones from sorghum linkage group F mapped to maize chromosomes 2 and/or 7. This is consistent with previous observations that sorghum linkage groups often contain loci which map to two maize chromosomes. The order of the loci is substantially conserved, with only three obvious rearrangements. Two putative inversions are present, one spanning three loci on maize chromosome 2 (*jc0767a, jc1410, jc0233*), and the other involving two closely linked loci on maize chromosome 7 (*jc0943, jc0878*). Another rearrangement detected by *jc0678* is also evident on the short arm of maize chromosome 7. Estimates of genetic linkage distances (Haldane) were compared for regions characterized by high colinearity. Excluding the putative inversions, map distances were very similar with the exception of the interval defined by *pio20.728* and *bnl14.07*.

Among low copy sequences, there appear to be fewer duplicate loci in sorghum. Most probes detected one locus in this sorghum population although several probes (e.g. *jc1362, jc0943* and *jc0767*) also hybridized to two or more monomorphic bands which could represent additional loci. When the same probes are hybridized to DNA of maize inbreds, additional monomorphic bands are detected; usually one or two more than detected in hybridizations with sorghum DNA. Two loci which map to sorghum linkage group F have duplicate loci in either sorghum or maize. *jc0676* maps to maize chromosomes 2 and 7, but only to linkage group F in sorghum. *jc1362* maps to maize chromosome 7, but detects a duplicate locus in sorghum on linkage group I. This locus on linkage group I is flanked by loci which map to chromosomes 3 and 8 in maize.

Explanations for these types of rearrangements (Whitkus et al., Genetics 132:1119-1130, 1992) often include either (1) differential duplication of chromosome segments in maize or (2) polyploidy in the ancestry of maize and sorghum. Segmental duplication predicts that duplicated segments should be found only in maize. This is consistent with our sorghum map which shows no significant regions of duplication. However, polyploidy could account for the observed relationships if there has been a differential loss of duplicated segments in sorghum, and/or if duplicated regions in sorghum exist but have not been discovered. Our observations currently do not strongly favor either theory.

The future of the Mutator stocks produced by Robertson at Iowa State University

--Donald S. Robertson

Many in the maize genetic community know that I have been in poor health for the last couple of years. Six months after the completion of chemotherapy, a CAT scan last December revealed no evidence of Hodgkin's disease. Side effects of the chemotherapy, however, have made it obvious to me that I will not be able to continue field work. Thus, I have no more need for any of my research material.

I plan to go through all of my stocks in 1993 and make a list of any of them that I think would be useful to other maize workers. This list will be included in the 1994 News Letter. Also any stocks I think are appropriate, will be forwarded to the Maize Genetic Cooperation Stock Center.

Stocks on this list will be made available to anyone requesting them. Any worker presently aware of stocks I have that would be useful in their research program should feel free to request them at any time. Also, anyone who would like to come to Ames to look for stocks of interest to them would be welcome at a mutually agreeable time.

My plans (God willing) are to have all stocks that might be useful to others distributed by the end of 1994. After that date, any remaining stocks will be discarded.

Deletions with the *wd* phenotype in the short arm of chromosome nine induced by the Mutator system

--Donald S. Robertson

Robertson and Stinard (Genetics 45:353-361, 1986) described the induction of deletions involving the *yg2* locus, which had no or reduced transmission through the male. To further characterize these deletions, crosses were made with heterozygous TB-9Sb and *wd1* stocks. In these tests, six of these deletions produced albino seedlings, which indicated that they were viable in the hemizygous state and were "allelic" to *wd1*. ("Allelic" will be used in this report when crosses between two different deletion stocks yield a mutant phenotype. Because deletions do not represent a single locus, the term allele is not technically accurate.) McClintock (Genetics 29:478-502, 1944) gave the symbol *wd* to terminal deletions of the short arm of chromosome 9, which included the terminal knob, the adjacent chromatin thread, and extended through approximately half of the first chromomere. Plants heterozygous for these deletions and the recessive *yg2* allele were yellow green. When plants heterozygous for these deletions were self-pollinated, the progeny segregated for albino seedlings.

Because some of our deletions produced seedlings with the albino phenotype when hemizygous or when crossed to the standard *wd1*, it was obvious that the Mutator system was producing *wd*-like deletions. We have available over one hundred putative deletion stocks involving the *yg2* locus. To determine if some of these could be short deletions that would give the *wd* phenotype when homozygous, ten kernels from the male outcrosses of the original isolates (genotype *yg2/*"deletion"*)* to standard lines (genotype *Yg2 Yg2*) were sown, and the resulting plants self pollinated. Approximately 50 kernels from each selfed ear were seedling tested and scored for the segregation of albino seedlings, which were expected in half of the outcross plants, if a short deletion had been induced similar to those responsible for the *wd* phenotype.

Included in these tests were the five deletion stocks that had segregated for seedlings with the *wd* phenotype in the TB-9Sb and *wd* crosses, which previously had been described in the 1985 paper in Genetics. As expected, some of the outcross plants of each of these five deletion lines segregated for the *wd* phenotype.

Seven new heterozygous deletion stocks were found, which segregated for albino seedlings in the progeny of the self pollinated plants. Six of these (given the symbols *wd-Mu1* through *wd-Mu6*) were tested to determine if they were "allelic" to each other. Kernels from the selfed ears of each of the six *wd-Mu* deletion stocks were sown and the resulting plants of each deletion line were intercrossed in all possible combinations. Sufficient crosses were made to insure that a positive result would be obtained if "allelism" was present. Positive results were obtained in all combinations. Thus, all the deletions had in common the region responsible for the *wd* phenotype. Crosses of these six deletions with plants heterozygous for the standard *wd1* plants deletion proved that they were all "allelic" to this deletion.

Crosses also were made to determine if the six new *wd-Mu* deletions involved the region of the short arm missing in the *pyd* deletions. The *pyd* stocks were generously supplied by Dr. Barbara McClintock. She found these deletions to be shorter than the *wd* deletions. They included the terminal knob of chromosome 9 and the adjacent chromatin, but did not include the first chromomere. Heterozygous *yg2/pyd* plants are green. Thus, the *pyd* deletion does not extend through the *yg2* locus (Genetics 29:478-502, 1944). Crosses of plants heterozygous for all six *wd-Mu* deletions with heterozygous *pyd* plants resulted in the segregation of pale yellow seedlings, which are expected of *pyd/wd* heterozygotes.

The five *wd-Mu* deletions reported in the 1986 paper in Genetics were also tested for "allelism" with *pyd* and proved to be "allelic". These deletions have been given new symbols as follows: 108-8 - *wd-Mu7*, 110-9 - *wd-Mu8*, 110-8 - *wd-Mu9*, 114-1 - *wd-Mu10*, 117-8 - *wd-Mu11*.

It is not known how much of the deleted regions of the *pyd* and *wd1* deletions are responsible for their respective phenotypes. It is known that the *pyd* deletion is shorter than the *wd1* deletion and that the latter deletion uncovers the *yg2* locus and includes approximately half of the first chromomere of the short arm of chromosome 9. The genetic evidence indicates that all 11 *wd-Mu* deletions uncover the regions of chromosome 9 responsible for the *wd1* and the *pyd* phenotypes.

The *wd-Mu* deletions involve the region of the short arm of chromosome 9 responsible for the *wd1* and *pyd* phenotype. This, however, does not necessarily mean that the *wd-Mu* deletions are terminal deletions, as are the *pyd* and *wd1* deletions. Cytological analyses are needed to determine the extent of the *wd-Mu* deletions. Cytological studies of the *wd-Mu* deletions are being undertaken at this time, but because of the genetic background of these stocks, it is difficult to obtain good cytological preparations. The determination of whether or not some or all of the *wd-Mu* deletions are terminal awaits further cytological studies.

Four-point linkage data for *ae pr lw2 gl8* on 5L

--Philip S. Stinard and Patrick Schnable

We report the results of a four-point linkage test for *ae pr lw2 gl8* (Table 1). The linkage test was set up as a modified backcross as indicated in Table 1. Kernels from the backcross ear

Table 1. Four-point linkage data for *ae pr lw2 gl8*.

Testcross: *Ae Ae Pr Pr Lw2 Lw2 Gl8 Gl8* x (*ae pr Lw2 gl8*/*Ae Pr lw2 Gl8*)

Reg.	Phenotype	No.	Totals
0	*ae pr + gl8*	144	
	+ + lw2 +	142	286
1	*ae + lw2 +*	11	
	+ pr + gl8	12	23
2	*ae pr lw2 +*	2	
	+ + + gl8	0	2
3	*ae pr + +*	1	
	+ + lw2 gl8	0	1

no multiple crossovers observed
% recombination *ae--pr*=7.4±1.5
% recombination *pr--lw2*=0.64±0.45
% recombination *lw2--gl8*=0.32±0.32

were planted in the field, the resulting plants selfed, and the selfed ears scored for *ae*, *pr*, and *lw2*. Approximately 50 kernels from each selfed ear were grown to seedling stage in a sandbench, and the seedlings were scored for *lw2* and *gl8*. The following linkage relationships were established (distances presented in centimorgans): *ae*-7.4-*pr*-0.64-*lw2*-0.32-*gl8*. These data are consistent with the reported linkage relationships between *ae*, *pr*, and *gl8* (*ae*-10-*pr*-1-*gl8*), and would place *lw2* between *pr* and *gl8*. However, it should be noted that the standard errors for the map distances in the *pr-lw2-gl8* interval are large compared to the distances themselves. Also, no double crossovers were observed. Therefore, the placement of *lw2* between *pr* and *gl8* must be viewed as tentative until more data are collected. For now, the suggested map revision is:

```
                                    lw2
--ae----------------------pr--gl8--
 57                        67  68
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etched2 (_et2_), a new etched endosperm/albino seedling mutant, is located on 2S

--Philip S. Stinard, Michael Scanlon and Patrick Schnable

A new recessive etched endosperm kernel mutant was found segregating on the selfed ear of a Mutator outcross plant (87-2352-23) grown in Donald S. Robertson's nursery. The mutant was given the designation *et*-2352*. When seedling tested, mutant kernels gave rise to off-white albino seedlings, with occasional greening of leaf tips. Some mutant seedlings had fine, green revertant sectors typical of *Mu*-induced seedling mutants. Upon further propagation, stable lines were obtained. Mutant seedlings die after the kernel nutrient reserves are exhausted. Outcrosses of heterozygous mutant plants were placed in Patrick Schnable 's TB block, and crossed by a series of B-A translocations involving 19 of the 20 chromosome arms. Only crosses by TB-2Sb uncovered the mutant endosperm phenotype. Nonmutant kernels from the positive TB test ears were sown in the sand bench, and gave rise to seedlings segregating for off-white albinos (putative hypoploids). Mutant kernels from the same ears gave rise to green seedlings (putative hyperploids) as expected. We conclude that *et*-2352* is located on the short arm of chromosome 2, distal to the TB-2Sa breakpoint. Since there are no other mutants with this phenotype located on the short arm of chromosome 2, we have given this mutant the designation *etched2* (*et2*).

A unique white endosperm viviparous allele of _opaque5_

--Philip S. Stinard, Patrick Schnable and Donald S. Robertson

A new recessive pale yellow/sugary endosperm, embryo lethal mutant was found segregating on the selfed ear of a plant (83-3038-42) from an active Mutator population. Upon subsequent propagation, the mutant phenotype was found to vary from pale yellow to white endosperm, from sugary endosperm to empty pericarp defective, and from viviparous to embryo lethal. Outcrosses to standard of heterozygous mutant plants were crossed by a series of B-A translocations involving 19 of the 20 chromosome arms of maize. Only crosses involving TB-7Lb uncovered the mutant endosperm phenotype. Seedling tests of mutant endosperm kernels from the TB-7Lb cross produced both yellow-green and wildtype seedlings. Since our TB-7Lb source also carries the *opaque5* reference allele, which has a yellow-green seedling phenotype, we suspected that the wildtype seedlings arising from mutant endosperm kernels of the TB-7Lb cross were the expected hyperploids, and the yellow-green seedlings were indicative of allelism of our mutant with *o5*. Subsequent allele tests conducted in our 1991-92 winter nursery proved this to be the case. We now designate our mutant as *o5-3038*.

We have not yet been able to rescue the viviparous embryos of homozygous *o5-3038* kernels in order to determine a seedling phenotype for our mutant. However, when heterozygous with the *o5* reference allele, mutant seedlings are yellow-green. A small minority of mutant seedlings are mutable, with the fine green revertant sectors indicative of late reversion events and commonly found in *Mu*-induced seedling mutants. While it is likely that the reversion events are occurring in the *o5-3038* allele, we have not yet ruled out the possibility that the reversion is occurring in the (normally stable) *o5* reference allele in the presence of an active Mutator system.

Kernels that are heterozygous for both *o5-3038* and the *o5* reference allele are distinctly pale yellow, and range from sugary to shrunken-opaque. This is a phenotype intermediate between that of the two homozygous alleles (homozygotes of the *o5* reference allele have yellow, shrunken-opaque endosperms in our genetic backgrounds). Furthermore, the intermediate pale yellow endosperm phenotype of the mutant heterozygotes indicates that the white endosperm aspect of the *o5-3038* phenotype is due to mutation at the *opaque5* locus, and is not due to a coincident mutation or deletion at some other closely linked (and currently unknown) white endosperm locus.

The white endosperm, viviparous embryo phenotype of *o5-3038* suggests that the gene product of the *O5* gene may be involved in the carotenoid biosynthetic pathway. Why the other known *o5* mutants do not have white or pale endosperms as well remains a mystery. Perhaps the other *o5* mutant alleles are leaky and produce enough wildtype gene product to give normal appearing endosperm carotenoid levels, or perhaps *opaque5* is a complex locus with separate components affecting endosperm carotenoids, seedling carotenoids (some *o5* mutants produce albino seedlings, some produce yellow-green or luteus seedlings), and/or plastid structure and development. (See Stinard, MNL65:18-19, for a discussion on how nuclear genes affecting both endosperm and seedling phenotypes could be affecting plastid structure and development. It is conceivable that a mutation that disrupts plastid structure or development could create deleterious lesions in the membranes of the plastids in which carotenoid biosynthesis occurs, resulting in a reduced carotenoid content in both endosperm and seedling. Lesions in the endosperm amyoplasts could also affect starch and protein biosynthesis, leading to the sugary/opaque endosperm phenotype of mutant *o5* kernels.)

_gl23_ and _gl18_ are allelic

--Philip S. Stinard and Patrick Schnable

The mutant *glossy23* (*gl23*), first reported by Sprague (MNL64:110), conditions seedlings with shiny leaf surfaces to which water adheres in droplets. In the course of our studies of the biology of glossy mutations, we obtained a *gl23* stock from Dr. Sprague. Since *gl23* had not yet been located to chromosome arm, we conducted experiments to determine its chromosomal location. In our 1991-92 winter nursery, we crossed plants homozygous for *gl23* by a series of B-A translocations covering 19 of the 20 chromosome arms. We planted kernel samples from each TB cross in the sandbench, and found that only the seedlings grown from crosses involving TB-8Lc segregated for glossy.

In an unrelated experiment elsewhere in our winter nursery, we had produced an F1 between *gl23* and *gl18*, also known to be located on 8L (our *gl18* stock was obtained from Donald Robertson). We planted kernels from this cross in the sandbench, and found that they gave rise to glossy seedlings, indicating allelism. We repeated the allele test in our 1992 summer nursery and obtained the same results. We conclude that *gl23* and *gl18* are allelic. We also note that the expression of *gl23* is similar to that of *gl18* (weak expression).

opaque12 (_o12_) is located on 4S, and conditions aleurone mosaicism

--Philip S. Stinard and Patrick Schnable

In the course of our studies of putative Mutator-induced mutants with sugary/etched endosperm phenotypes, we collected mutants from other sources with similar phenotypes for the purpose of allele tests. One of these mutants, *o12*, first described by Oliver Nelson (MNL55:68), was obtained from the Maize Stock Center. Kernels homozygous for *o12* were originally described as having opaque, etched/scarred endosperms of variable size, giving rise to chlorophyll-deficient seedlings. When we propagated the colorless aleurone *o12* stock, we noted its similarity in phenotype to the mutants *dek5*, *dek7*, and *cp2*, which in addition to the above phenotypes, produce aleurone mosaicism in a colored aleurone background. In order to study the relationships between these mutants, we outcrossed the colorless *o12* line to an M14/W22 colored aleurone line and selfed the F1. The F2 segregated for aleurone color, and the colored *o12* mutant kernels did indeed show aleurone mosaicism as evidenced by colorless, depressed areas of endosperm cells interspersed with areas of normal appearing colored aleurone cells.

Since *o12* had not yet been placed to chromosome, we crossed plants heterozygous for *o12* by a series of B-A translocations covering 19 of the 20 chromosome arms. Only crosses by TB-4Sa uncovered the mutant endosperm phenotype. Seedling tests of nonmutant kernels from the positive TB test ears showed segregation for pale green seedlings (putative hypoploids). Mutant kernels from the same ears gave rise to green seedlings (putative hyperploids) as expected. We thus conclude that *o12* is located on the short arm of chromosome 4 distal to the TB-4Sa breakpoint. Allele tests of *o12* with *dek7*, also located on 4S, are in progress.

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Two independent mutants designated as _shrunken6_ (_sh6_) are allelic

--Philip S. Stinard, Patrick Schnable and M. G. Neuffer

In two reports in the 1992 MNL, one from Ames (MNL66:4) and one from Columbia (MNL66:39), we independently reported new recessive mutants on 7S, both with shrunken-opaque kernel phenotype and both giving rise to pale green seedlings. We exchanged stocks of our mutants, and conducted allelism tests in our summer nurseries. (The Ames tests were performed in the nursery of Patrick Schnable.)

In Ames, kernels from the outcross to standard of heterozygous *sh6-8601* (the ISU allele) plants were sown in the field and grown to maturity. The second ears of these plants were self-pollinated, and the first ears were pollinated by plants grown from wildtype kernels of selfed segregating ears of *sh6-1295* (the UMC allele). The latter plants were also self-pollinated. In each instance in which the self-pollinated ears on both plants segregated for mutant kernels, the ears from the allele test cross also segregated for mutant kernels, confirming the allelism of these two mutants. Seedling tests of mutant kernels from the allele test crosses also gave back the mutant pale green seedling phenotype. Allele tests conducted in Columbia gave similar results.

Slight phenotypic differences between the two alleles were noted in the Ames grown materials. Mutant kernels of *sh6-1295* were uniformly opaque, more extremely shrunken than mutant kernels of *sh6-8601*, and were frequently embryo lethal. Mutant kernels of *sh6-8601* varied from glassy to opaque, were less shrunken than *sh6-1295* (and closer to *shrunken1* in appearance), and usually produced viable embryos. Kernels heterozygous for both mutant alleles were similar in appearance to kernels homozygous for the *sh6-8601* allele. Whether these differences are due to differences in genetic background, or whether *sh6-8601* is a less severe, leaky allele, we do not know at this time.

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The _Spm_ transposable element has an unusual, enhancer-insensitive promoter

--Ramesh Raina and Nina Fedoroff

The transpositional activity of the maize Suppressor-mutator (*Spm*) element is regulated by both positive and negative mechanisms that include alternative splicing of the element's primary transcript, inactivation by an epigenetic mechanism, reactivation mediated by the element-encoded TnpA, and transcriptional inhibition by the same gene product (Fedoroff et al., MNL66:14-15, 1992). TnpA is a DNA-binding protein and there are multiple copies of its 12bp binding site located at the element's 5' end, just upstream from the transcription start site, as well as at the element's 3' end, adjacent to its 13bp terminal inverted repeat sequence. To gain further insight into molecular mechanisms regulating *Spm* transposition at the transcriptional level, we have identified and analysed the element's promoter sequence using

transcriptional fusions to a firefly luciferase gene. Plasmids containing the transcriptional fusions, together with a standard CaMV 35S-chloramphenicol acetyl transferase gene (used to normalize the values obtained with different constructs), were introduced into cultured tobacco cells by microprojectile bombardment (Cook and Fedoroff, MNL66:11-12, 1992). We report here that the *Spm* promoter has unusual properties that contribute not only to minimizing expression, but which render it resistant to the influence of a nearby enhancer.

Identical levels of promoter activity were obtained with 5' terminal *Spm* fragments comprising the first 220 or the first 578bp of the element's sequence. These fragments were selected to contain the sequence upstream of the transcription start site at nucleotide 209 (a region we previously designated the 'upstream control region' or UCR), or the upstream sequence together with most of the untranslated GC-rich leader sequence of the first exon (a region we previously designated the 'downstream control region' or DCR) (Banks et al., Genes Dev. 2:1364-1380, 1988). This observation implies that the element's promoter sequence is contained entirely within the sequence upstream of the transcription start site. Progressive deletions through the UCR resulted in the gradual loss of promoter activity. The complete *Spm* promoter is a weak one, supporting luciferase expression at 1-2% of that observed with the CaMV 35S promoter.

The most surprising property of the *Spm* element's promoter is that it is insensitive to the influence of a nearby strong enhancer. When the 35S promoter's enhancer was placed upstream of either the *Spm* promoter consisting of just the UCR or of both UCR and DCR, luciferase activity increased only by a factor of 3-4, in sharp contrast to the 100-200-fold enhancement observed with the 35S minimal promoter. The *Spm* promoter was converted to a much more highly enhancer-responsive form by introducing an optimal TATA box sequence and by removing the DCR. Although each change had an effect individually, the effects of the two together were more than additive. The sequence that was converted to a TATA box was the TATGAAT sequence at -24 to -30 upstream of the transcription start site identified by others as the element's putative TATA box (Pereira et al., EMBO J. 5:835-841, 1986). This is not a sequence essential for *Spm* promoter activity, since changes by site directed mutagenesis that either decreased or increased its resemblance to a conventional TATA box sequence had no effect on promoter activity. Nonetheless, even a single base change of the G at -27 to an A residue increased the responsiveness of the *Spm* promoter to the enhancer, and conversion of the sequence to TATATAA, the 35S promoter's TATA sequence, made the UCR highly responsive to the enhancer. The GC-rich DCR makes a major contribution to the enhancer-insensitivity of the *Spm* promoter, despite the absence of any perceptible effect of this sequence on the basal activity of its own promoter in the transient assay system.

Thus the inherent properties of the *Spm* element's unusual promoter region contribute to minimizing transposition frequency and making the element's activity position-independent. That is, *Spm*'s promoter is both a weak TATA-less promoter and it is quite insensitive to enhancement. Taken together with our recent observations that the UCR and DCR are the sites of de novo methylation in transgenic tobacco (see Schläppi and Fedoroff, this issue) and are associated with transcriptional inactivation of the element, these observations suggest that the element's control sequences are indeed primarily or entirely within the 5' terminal 0.6kb UCR + DCR region.

TnpA trans-activates de novo methylated Suppressor-mutator transposable elements in transgenic tobacco

--Michael Schläppi and Nina Fedoroff

We report that the maize Suppressor-mutator (*Spm*) transposable element is subject to epigenetic inactivation in transgenic tobacco, as it is in maize (Schläppi et al., Genetics, 1993, in press). *Spm* inactivation in tobacco is correlated with increased methylation of sequences near the element's transcription start site. To determine whether element-encoded gene products can promote the reactivation of an inactive element, as has been reported in maize, we investigated the effects of introducing individual cDNAs for *tnpA*, *tnpB*, *tnpC*, and *tnpD*, the element's four known protein-coding sequences. Each cDNA was expressed from the strong 35S CaMV promoter and introduced into plants containing one or more copies of the *Spm* element and an excision assay plasmid with an internally deleted *dSpm*-disrupted β-glucuronidase (GUS) gene. Introduction of the CaMV 35S-*tnpA* cDNA into the transgenic tobacco plants promoted the reactivation of the inactive resident *Spm* element, as judged by the appearance of regenerants with very early excision events and transposed elements. By contrast, none of the other CaMV 35S controlled cDNAs affected the activity of the resident *Spm* element. Similar results were obtained when the element-encoded cDNAs were introduced either by *Agrobacterium*-mediated retransformation or by a genetic cross. Reactivation of an inactive *Spm* by CaMV 35S-*tnpA* is accompanied by reduced methylation of several methylation-sensitive restriction sites near the element's transcription start site, but not elsewhere in the sequence. Maintenance of the reactivated *Spm* element in an active state requires the continued presence of the CaMV 35S-*tnpA* cDNA. Elimination of the CaMV 35S-*tnpA* cDNA locus by genetic segregation generally results in decreased element activity, as judged by its ability to promote excision of the *dSpm* element from the excision assay plasmid, and is accompanied by increased methylation of the element's 5' end. Exceptions resembling the phenomenon of "presetting" are also observed in which progeny plants that did not receive the CaMV 35S-*tnpA* cDNA locus maintain high excision activity and exhibit low methylation levels. Together with the finding in maize that a *weak Spm* element can transcriptionally activate an inactive *Spm* (Kolosha and Fedoroff, MNL66:9, 1992), we hypothesize from the tobacco reconstitution experiments that *tnpA* activates transcription from the *Spm* 5'-end. The following observations, summarized below, support the hypothesis.

The transcription start site of *Spm* has been located at nucleotide 209 from the element's 5' end (Pereira et al., EMBO J. 5:835, 1986). TnpA binds in vitro to a 12bp motive repeated in direct and inverted orientation several times within 200bp of the element's 5' end and within 600bp of its 3' end (Gierl et al., EMBO J. 7:4045, 1988). In a transient expression assay recently established in the laboratory, it has been shown that the luciferase reporter gene fused to different *Spm* 5' segments is active after introduction into NT1 tobacco suspension cells by microprojectile bombardment (Cook and Fedoroff, MNL66:11, 1992). A 600bp *Spm* 5' end segment containing sequences both upstream and downstream from the transcription start site, as well as a 220bp DNA segment without the G+C-rich downstream sequences of the untranslated leader, are active in the transient expression system.

In addition, minimal promoter activity can be detected using deletions to within -41 from the transcription start site, but deletions including the transcription start site abolished promoter activity. All of these constructs are also active in transgenic tobacco (M. Schläppi, unpublished), but become inactivated as rapidly as full length *Spm* elements. It will be determined whether this inactivation correlates with methylation of the *Spm* 5' sequences as it does in the full-length element.

Constitutive expression is observed when the 5' *Spm*-luciferase construct is introduced into tobacco suspension culture cells by microprojectile bombardment (Cook and Fedoroff, MNL66:11, 1992; Raina and Fedoroff, this issue). The same is true when the *Spm* 3' end is added to the 3' end of the luciferase construct (R. Raina, unpublished). A similar 5' *Spm*-luciferase-*Spm* 3' construct was linked to the GUS excision assay plasmid and introduced with the CaMV 35S*tnpD* cDNA into tobacco by *Agrobacterium* mediated co-transformation. Small plantlets removed from selected calli that were analyzed for luciferase activity at a very young stage give low to moderate levels of luciferase gene activity (two- to five-fold above background). In older leaves, the luciferase activity is on average only two-fold above background. Such leaves were retransformed with the *tnpA* cDNA controlled by promoters that differ in strength. Since the CaMV 35S-*tnpD* cDNA had previously been introduced into the retransformed plants, the simultaneous effect of *tnpA* on *dSpm* excision from the excision assay locus and on transcription activation from the *Spm* 5' end (fused to luciferase) can be monitored in this experiment, because *tnpA* and *tnpD* are necessary and sufficient for *Spm* excision (Frey et al., EMBO J. 9:4037, 1990; Masson et al., Plant Cell 3:73, 1991). The results show a positive correlation between excision of the linked *dSpm* and trans-activation of the luciferase gene controlled by the *Spm* 5' end. That is, whenever a high frequency of *dSpm* excision is observed in the presence of the *tnpA* cDNA, a correspondingly high luciferase activity, five- to sixty-fold above background, is observed. In some cases, little or no *dSpm* excision correlates with a high luciferase activity. But we have never observed excision in plants that exhibit no luciferase activity. Hence, the results indicate that *tnpA* introduced by transformation can trans-activate 5' *Spm*-luciferase constructs that are stably integrated into the tobacco genome. In contrast, when a *tnpA* cDNA is co-bombarded into cultured cells with an 5' *Spm*-luciferase construct in the transient assay, it inhibits luciferase expression (Cook and Fedoroff, MNL66:11, 1992). It is therefore possible that *Spm* 5' sequences are targets for DNA methylation and that TnpA activates the *Spm* promoter in vivo indirectly by interfering with methylation. Experiments are underway to determine the TnpA concentrations required for promoter activation or repression and to directly analyze the TnpA interaction with cis-acting sequences in vivo.

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Dosage of 10kDa zein influenced by the modifier-genes on the background of *opaque2*

--Shun-hong Dai and Meng-qian Zeng

Zein is one of the most important components of maize storage proteins. Taking advantage of the triploidy of the endosperm, we found the 10kDa zein had a dosage modified by the modifier-genes

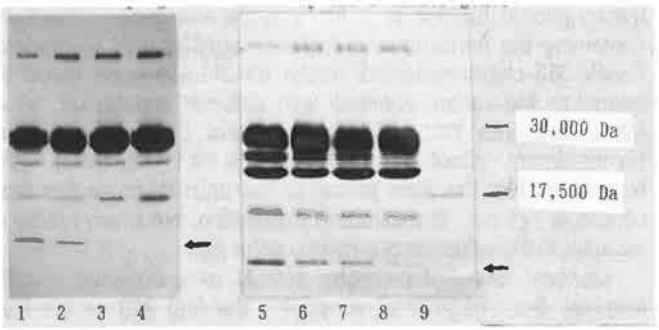

Figure 1. SDS-PAGE of total zeins. Mo17 *o2* (lane 1), Mo17 *o2*/Zhongxi042QPM (lane 2), Zhongxi042QPM/Mo17 *o2* (lane 3), Zhongxi092QPM (lane 4), Mo17 *o2* (lane 5), Mo17 *o2*/Zhongxi092QPM (lane 6), Zhongxi092QPM/Mo17 *o2* (lane 7), Zhongxi092QPM (lane 8), MW marker (lane 9).

on the background of *o2*. Mo17 *o2*, Zhongxi092QPM, Zhongxi042QPM and the reciprocal crosses between Mo17 *o2* and the two QPMs were used in our research. We harvested the 20 DAP endosperms, extracted zeins with 70% ethanol and 2% mercaptoethanol at 60 C and analysed zeins with SDS-PAGE. In Figure 1, it can be found that Mo17 *o2* contains more 10kDa zein than the two QPMs and there is a dosage among the parents and their progenies caused by the modifier-genes.

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Separation of corn albumins and globulins by improved lactate-PAGE procedure

--T. M. Song, D. H. Zheng, Q. S. Yang and G. H. Song

An acidic lactate-PAGE procedure was developed from the basic method of wheat cultivar electrophoretic identification (Draper, 1987), but modified in extraction solution, polyacrylamide concentration, the constitution of the stock solutions and pH of buffer solution (Table 1). This procedure was proved to be suitable for separating albumin and globulin fractions of corn seed proteins and had strong resolving power, so that it could be used in corn cultivar identification, purity testing and other research purposes.

Table 1. Recipes for stock, extraction, and buffer solutions.

	Gel mixed volume	
Stock solutions	separate	concentrate
1. Acr 95g Bis 3.8g H_2O 500ml	14ml	
2. Sodium lactate 2.8ml+lactic acid to pH 3.2 H_2O 100ml	2ml	
3. Ascorbic acid 0.48g ferrous sulphate ($7H_2O$) 8ml H_2O 100ml	2ml	1ml
4. Sodium lactate 3ml+lactic acid to pH 5.6 H_2O 100ml		1ml
5. Acr 26g Bis 5.2g H_2O 200ml		2ml
6. Ap 11.41g H_2O 100ml	80µl each +2ml H_2O	80µl

Extraction: 0.5M NaCl 100ml sucrose 15g methyl green 3mg
Buffer: Glycine 4g+lactic acid to pH 3.4 H_2O 2000ml

Figures 1 and 2 show the electrophoregrams of 11 corn inbreds and 11 hybrids. It is very clear that every inbred or hybrid has its own specific band pattern distinguishable from the others. These patterns are also very stable and repeatable, so could be considered as a kind of 'fingerprint'.

Based on the mobility, the electrophoregrams were classified

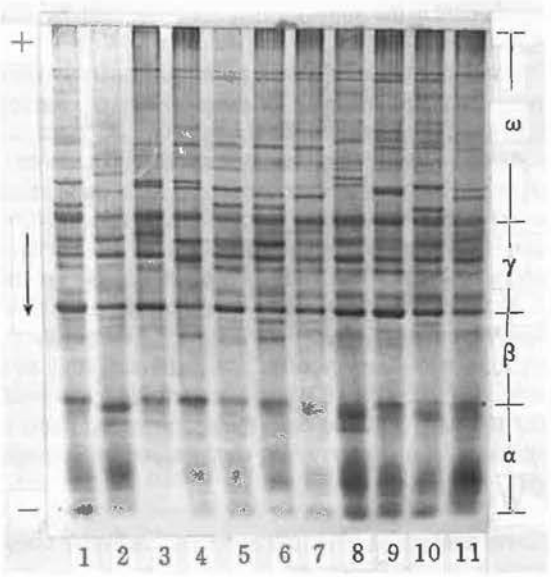

Figure 1. Lactate-PAGE of albumins and globulins from 11 inbred seeds. 1) Gy237, 2) 478, 3) Ye4, 4) 8112, 5) Gy798, 6) Mo17, 7) Gy246, 8) Zong31, 9) Gy220, 10) 5003, 11) 1127.

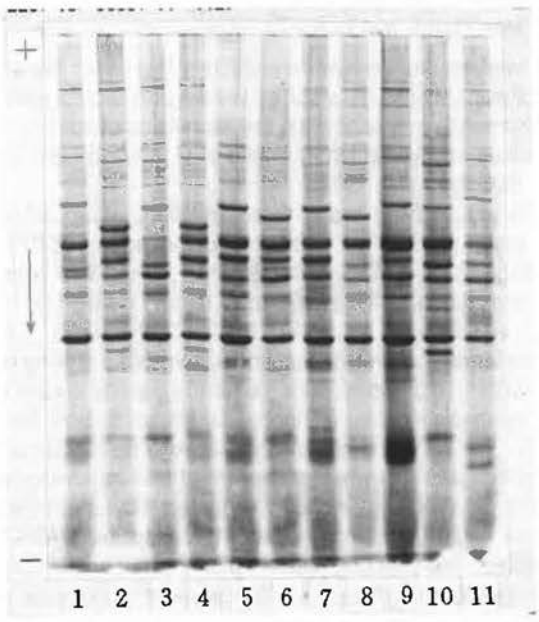

Figure 2. Lactate-PAGE of albumins and globulins from 11 hybrid seeds. 1) Gy237x478, 2) F135x1127, 3) Bs3xBs5, 4) T23xW3-9, 5) Ye4x8112, 6) Dan340x478, 7) Zong31x5003, 8) Gy220x1127, 9) Ye4xGy798, 10) Gy237x8112, 11) Zong31xGy246.

into four groups designated as α, β, γ and ω respectively with three common bands as their boundary markers (Fig. 1). α was the fast moving, ω the slow moving, and β and γ the intermediate groups. γ and ω were the main groups in cultivar identification. They included 25-32 bands, most of which were very clear and distinctive. It probably indicated the genotypic characteristics.

The lactate-PAGE band patterns of corn whole kernel, embryo, and endosperm

--D. H. Zheng, T. M. Song, Q. S. Yang and G. H. Song

Figure 1 is the comparison of the electrophoregrams of whole kernel, embryo and endosperm of inbred Gy237 and 8112. It shows that the band patterns of the whole kernel and the embryo

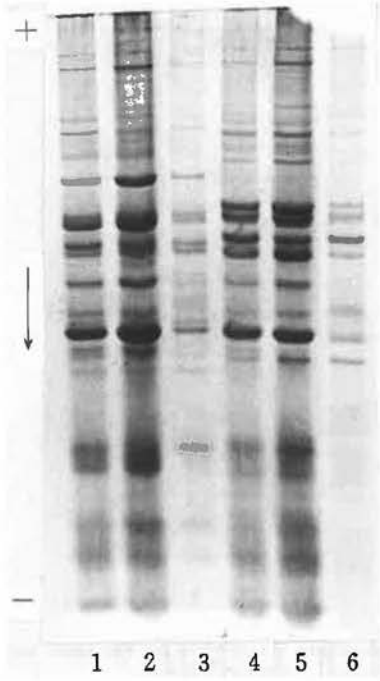

Figure 1. Lactate-PAGE of albumins and globulins from whole kernel, embryo and endosperm of inbred Gy237 and 8112. 1) Gy237 whole kernel, 2) Gy237 embryo, 3) Gy237 endosperm, 4) 8112 whole kernel, 5) 8112 embryo, 6) 8112 endosperm.

are basically the same no matter the band number, mobility, width, intensity or stain patterns. Electrophoregram of endosperm not only has fewer band numbers and weaker staining than the whole kernel and embryo, but they almost are completely overlapping with the embryo bands. This indicated that both electrophoregrams of whole kernel or embryo could be used as the 'fingerprint' of corn inbreds or hybrids, but because the embryo dissecting is time consuming and rather difficult, our suggestion is to use the electrophoregrams of whole kernel in most cases.

F1 hybrid electrophoregrams predicted and demonstrated by their parent inbreds

--T. M. Song, D. H. Zheng, Q. S. Yang and G. H. Song

Figure 1 (next page) shows the electrophoregrams of inbred Gy237, 478, hybrid Gy237x478, 478xGy237 and the electrophoregram from mixed extraction solution of inbred Gy237 and 478. From this plate, it could be found that at least 8 bands were obviously different between inbred Gy237 and 478 in γ and ω groups. The band number of the F1 is just equal to that of the common bands plus the specific bands of the two parents. The band patterns of reciprocal crosses were exactly the same. It indicated that the number of electrophoretic bands was a hereditary trait controlled by nuclear genes. Preliminary conclusions drawn from the data are: 1) presence of bands is dominant over absence; 2) strong band is dominant over weak band. Another interesting phenomenon we have found is that electrophoregrams of mechanically mixed extraction solution of two inbreds were equal to that of their F1 hybrid. From these results, we could easily predict the F1 electrophoregram from that of their two parents, and also could easily demonstrate the F1 electrophoregram from that of the mixed extraction solution of their parents.

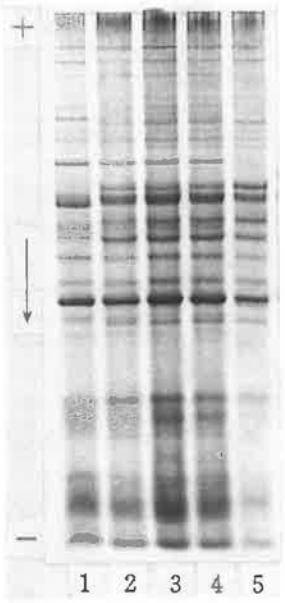

Figure 1. Electrophoregrams of corn hybrids and their parent inbreds. 1) Gy237, 2) Gy237x478, 3) 478xGy237, 4) Gy237+478, 5) 478.

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Molecular analysis of unstable alleles at the *o2* locus

--Detlef Michel, Francesco Salamini, Mario Motto and Hans-Peter Döring

Transposon tagging, i.e., insertional mutagenesis using transposons, has proved to be a powerful technique for the isolation of maize genes. We have recently used the transposable element *Ac* to tag and clone the *opaque2* (*o2*) gene, a positive trans-acting transcriptional activator of zein gene expression. The source of the *Ac* system of controlling element was a strain bearing the *wx-m7* mutation. Homozygous plants from variegated *wx-m7* kernels were crossed to a stable *o2* tester line according to the following mating scheme: *wx-m7 Ac O2* x *Wx o2 R*.

Among a population of about 1,000,000 kernels, variegated F1 kernels, i.e. kernels showing vitreous and opaque sectors, were selected from mature ears. Plants originating from these kernels were selfed to confirm the presence of variegated phenotypes in the F2 generation. In eight cases the variegated phenotype observed in the F1 kernels was heritable, giving rise to the mutable alleles termed *o2-m5* to *o2-m15*. The frequency of induction of mutable alleles at the *o2* locus was approximately 1.2×10^{-5}, a value consistent with that reported for the induction of mutable alleles by maize transposable elements (Döring, Maydica 34:73-88, 1989). One of these mutations, the *o2-m5* allele, was caused by the insertion of an *Ac* element into the *o2* locus (Motto et al., Mol. Gen. Genet. 212:488-494, 1988). To establish if the remaining 7 unstable mutable *o2* alleles (*o2-m7, 8, 9, 10, 11, 12,* and *13*) were also generated by *Ac* elements, plants derived from *o2* mutable kernels were crossed to a *C-I wx Ds* tester strain to evaluate the presence of endosperm variegation due to chromosome breakage (BFB cycle) and crossed to an *o2* tester line. The presence of variegated *o2* kernels did not induce *Ds* breakage due to an *Ac* element. These results excluded the presence of a functionally active *Ac* element in the autonomously controlled mutable alleles *o2-m7, 8, 9, 10, 11, 12, 13*.

The seven unstable *o2* alleles were subsequently examined by Southern restriction mapping for the presence of insertions. No *Ac* or *Ac*-like elements were found. However, we found in six mutable alleles an insert which has a restriction map identical to the known map of the *Spm/En* element. In one allele we found an insertion which is very similar though not identical to an autonomous *Bg* element. The insertion points of the elements are distributed over the whole *o2* locus. This finding indicates that the *wx-m7* strain which was used for tagging mutagenesis carried not only an active *Ac* element, but also active *Spm/En* and *Bg* elements. A similar observation was also reported by Patterson and coworkers (Genetics 127:205-220, 1991) who used maize strains with active Mutator elements for transposon mutagenesis, but found two mutable alleles of the *B* locus which were caused by *Spm*-related insertions.

Functional expression of the transcriptional activator *opaque2* of *Zea mays* in transformed yeast

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Investigations on genetic mutations that affect the accumulation of zein proteins have suggested the presence of several regulatory mechanisms controlling the expression of specific members of the zein multigene family (Motto et al., Oxford Surv. Plant Mol. Cell. Biol. 6:87-114, 1989).

We are studying one of these mutations, *opaque2* (*o2*). The *O2* gene product is a basic leucine-zipper (bZIP) protein (Hartings et al., EMBO J. 8:2795-2801, 1989) that is necessary for transcription of the 22kD family of zein genes and for the *b-32* gene (Lohmer et al., EMBO J. 10:617-624, 1991). A computer search for related protein sequences revealed a striking homology with GCN4, which is responsible for the general control of amino acid synthesis in yeast (Hinnebusch, Microbiol. Rev. 52:248-273, 1988). The structural homologies between O2 protein and GCN4 prompted us to test whether they might be functionally related.

The *O2* cDNA was cloned in the yeast expression vector pEMBLyex4 under the control of the hybrid inducible UASGAL/CYC1 promoter. The resulting plasmid pIOP2 was used to transform to Ura^+ the X4004-3A strain. Transformed yeast cells produced specific *O2* mRNA and a specific immunoreactive polypeptide of about 65kD during growth in galactose. The expression of O2 protein does not produce appreciable alterations during batch growth at 30 C. The heterologous protein is properly translocated into the yeast nuclei, as demonstrated by immunofluorescence, indicating that the nuclear targeting sequences of maize are recognized by yeast cells. To test if O2 protein can substitute for GCN4 functions we transformed a *gcn4* mutant (L1502 strain) with pIOP2 plasmid. The *gcn4* mutants fail to grow in the presence of aminotriazole (AT), and the expression of O2 proteins restores a significant resistance to AT. Since the AT resistance is related to the expression of the *HIS3* gene, which is controlled by GCN4, this confirms the hypothesis that O2 protein activates transcription of GCN4 regulated genes.

The interaction at the molecular level between O2 protein and *HIS4* promoter was tested by band-shift and DNAse I footprint assays. O2 protein was obtained by expressing the *O2* gene as an

IPTG-inducible glutathione-S-transferase (GST) fusion protein, GST-02-55 in BL21 (psB161) (Lohmer et al., EMBO J. 10:617-624, 1991).

To test the potential capacity of O2 protein to bind the *HIS4* promoter gel retardation assays were performed using purified GST02-55 protein and a fragment of *HIS4* promoter containing one of the three GCN4 binding sequences. Incubation of purified O2 protein with labelled *HIS4* promoter fragment gave two protein-DNA complexes of reduced electrophoretic mobility compared to free DNA. The formation of the complexes was specific because i) purified GST protein alone was not able to bind the labelled *HIS4* promoter region and ii) a 1000 fold molar excess of an unspecific competitor DNA does not prevent binding. The binding could be abolished, however, by a 100-fold molar excess of unlabelled *HIS4* promoter fragment. The O2 binding site was mapped by DNAse I footprinting, in which the same fusion protein and end-labelled promoter fragment were used as in the bandshift assay. The sequence protected by O2 corresponds to the sequence TGACTC, which is a GCN4 binding site. These results support the idea that basic mechanisms of transcription control have been highly conserved in eukaryotes.

Identification and mapping of RAPD markers in maize

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The development of nuclear restriction length polymorphism (RFLP) technology has been useful for the genetic analysis of various species and is of potential value in crop improvement as an aid to plant breeders in the selection of desired genotypes. However, RFLP technology is expensive and time consuming and technically difficult to use in some species with large and complex genomes.

The recently developed "Random Amplified Polymorphic DNA" (RAPD) marker system of Williams et al. (Nucl. Acid. Res. 18:6531-6535, 1990), that uses the polymerase chain reaction techniques to generate random amplified DNA markers, holds great promise for quickly placing markers on linkage groups. Accordingly, we have examined in our laboratory the reliability and reproducibility of the RAPD marker system with maize DNA and with a sample of random 10-mers.

The inbreds A7 and B73 were chosen from the maize germplasm collection present at the Experimental Station for Cereal Crops in Bergamo, Italy. The F1 generation and 45 F2 individuals segregating from the cross B73 x A7, which were already characterized by RFLP analysis, were used for the genetic mapping of RAPD markers. Genomic DNA was isolated from about 20 shoots of 10 to 15 day old seedlings derived from each progeny. DNA was purified according to the CTAB method as performed by Saghai-Maroof et al. (Proc. Natl. Acad. Sci. USA 81:8014-8018, 1984). Forty-seven random 10-mers (Operon Technologies Inc., Alameda, CA, USA) were used. The primers had 50-70% G+C content and no internal inverted repeats.

In order to decrease the background and to arrive at reproducible amplification patterns we have optimized the conditions described by Williams et al. (Nucl. Acid. Res. 18:6531-6535, 1990). To determine the effect of primer concentration, we tested three primers (OPA03, OPA04 and OPA05) at 0.1μM, 0.2μM, and 1.0μM final concentration, on genomic DNA extracted from B73, A7, and their F1 hybrid (B73 x A7). In addition, we evaluated three Mg final concentrations (1.5, 2.5, and 3.5mM) combined with OPA03 and OPA04 on the three genotypes. The effect of template concentration was assayed by amplifying in a final volume of 25μl, 2, 4, 10, and 50ng of genomic DNA from the inbred line A7 with the primers OPA02, OPA03 and OPA04. We have also verified hot (Taq-polymerase added as the last reagent to samples already at 94 C) versus cold start and the effect of UV light in reducing artifacts due to contamination with exogen DNA. Final volumes of 25μl and 50μl, with annealing temperatures of 36 C and 37 C, were also assayed. The results obtained suggest that 1) using 1.0μM primers a higher background was obtained, coupled with a lower number of amplification products compared to 0.2μM. Using 0.1μM primer concentration we obtained the same pattern observed with 0.2μM, but fainter bands. 2) Compared to lower concentrations, the use of 3.5mM Mg resulted in a more reproducible amplification of a larger number of distinct bands. 3) The best patterns were obtained using 4-10ng of template DNA in a reaction final volume of 25μl. Larger amounts of DNA gave rise to a non-specific smear. 4) The hot versus the cold start, annealing temperatures of 37 C versus 36 C, and the differences in the final volume had no evident effect. 5) The exposure of reagents to 280nm UV light for 60' significantly decreased the amplification of artifacts observed in the control reactions in our first attempts of producing RAPDs.

The optimal amplification conditions were obtained in a 25μl volume amplifying 4ng of DNA with 0.4U of Taq polymerase (Promega) at a final concentration of 10mM TrisHCl, pH 8.3, 50mM KCl, 3.5mM $MgCl_2$, 200μM dNTPs, 02.μM primer, 0.01% BSA, 0.1% Tryton X-100. To verify the reproducibility of the results all experiments were replicated three to five times. Amplification was conducted with an MJ Programmable Thermal Controller (MJ Research Inc.) for 45 cycles of 1' at 94 C, 1' at 36 C and 2' at 72 C. After the last cycle samples were incubated for 10' at 72 C, and then conserved at 4 C. Samples were loaded on a 1.4% agarose gel, electrophoresed, stained with ethidium bromide, photographed and the distribution of markers among progenies recorded. The 1kb ladder (Bethesda Research Laboratories) was used as molecular weight standard. RAPD markers were named according to the origin (OP) followed by primer identification code and size of the amplification product in base pairs.

Out of 47 primers tested, 35 yielded, on agarose gel, distinct amplification products: the remaining 12 produced a smear. The number of fragments amplified averaged 2.57±1.12, and ranged from 1 to 5. Twelve of the 35 primers revealed polymorphic fragments. These primers produced a total of 17 clearly scorable products which were polymorphic between the two inbred lines B73 and A7 and additive in the F1 genome (Table 1). All non-clear, faint and non-additive bands were excluded from further analysis. In the genotypes evaluated here, the level of polymorphism found with RAPDs was comparable to the level of polymorphism found with RFLP. This suggests that RAPD markers can be used for the construction of genetic maps in maize and other polymorphic crops. Using a 10bp primer, the expected average number of amplified products from the amplification of the maize genome is 9.1 (Williams et al., Methods in Enzymology, Orlando, FL, USA, Academic Press, 1991). In soybean, the authors previously cited observed a higher than expected average number of products and proposed that a 95% homology between primers and annealing sites was sufficient for the PCR amplification. In our experiments, however, the average number of fragments scored was lower than expected. This may indicate that a 100% matching of

Table 1. Primers used in RAPD analysis, quality of amplified products, maximum number of bands amplified in either parent and number of bands polymorphic between A7 and B73 inbreds.

Name	Quality	Max n. of bands	N. of polymorphic bands
OPA01	+	3	
OPA02	+	4	
OPA03	+	2	2
OPA04	+	2	
OPA05	+	3	
OPA06	+	1	
OPA07	+	5	3
OPA08	+	3	
OPA09	+	3	1
OPA10	+	4	
OPA11	+	3	
OPA12	-		
OPA13	-		
OPA14	-		
OPA15	+	2	
OPA16	+	1	
OPA17	+	2	
OPA18	-		
OPA19	+	2	
OPA20	-		
OPB01	+	2	
OPB02	+	3	
OPB03	+	1	
OPB04	+	1	
OPF06	+	2	
OPF14	+	3	2
OPM01	-		
OPM02	+	5	
OPM03	-		
OPM04	+	3	1
OPM05	+	2	1
OPM06	+	3	
OPM07	-		
OPM08	+	1	1
OPM09	+	4	1
OPM10	-		
OPM11	+	3	
OPM13	+	3	
OPM14	+	5	
OPM15	+	2	
OPM17	+	2	2
OPM18	+	2	
OPM19	+	1	
OPM20	+	2	1
OPO03	-		
OPO08	-		
OPO14	-		
Total	47	90	17
Average		2.57	
Standard error		1.12	

+ = distinct amplification products of good quality
- = smear or faint products of bad quality
Average and standard error refer to the average number of amplified products detected per primer giving good amplification products

the primer with the template DNA was necessary to obtain amplification in our laboratory conditions.

The segregation of the 17 RAPD markers was assessed in 45 individuals from the F2 progeny derived from the F1 hybrid B73 x A7, previously analyzed with RFLPs using MAPMAKER computer software (Lander et al., Genomics 1:174-181, 1987). The statistical analysis confirmed that RAPDs behaved as dominant Mendelian factors. Sixteen RAPDs have been located on a 1800cM maize genetic map already covered by 70 evenly distributed RFLP markers. They mapped on chromosomes 1, 2, 3, 4, 5, 6, 7 and 10; interestingly, *opm082000*, located on the short arm of chromosome 7, mapped 28.0cM distal to *pio200581*, the most distal mapped probe. This observation, together with the fact that RAPD probes are often not polymorphic and they may identify repetitive DNA sequences, suggests that RAPDs map in regions where polymorphic RFLP markers are rare.

The transposable element *Bg* from maize transposes in tobacco

--H. Hartings, E. Lupotto, N. Lazzaroni, V. Rossi, M. Motto and F. Salamini

The *Bg-rbg* system of transposable elements consists of autonomous *Bg* elements which encode all the functions needed for its transposition, and non-autonomous members (*rbg*) which can only transpose if an autonomous element is present in the same cell. Both elements have been isolated and characterized at the molecular level. The *Bg* element, isolated at the waxy locus, is 4,869bp in length, carries terminal inverted repeats of 5bp and generates 8bp long target site duplications upon transposition. With respect to the autonomous element, the receptor element has undergone numerous deletions but it has retained the terminal inverted repeats of the *Bg* element as well as a 76bp long subterminal sequence. This subterminal region is supposed to be of importance for transposition, since it contains a number of 6bp long motifs, both in direct and inverted orientation. A similar motif has been found in the subterminal region of the *Ac* transposon of maize, and it has been demonstrated that the putative transposase of *Ac* binds to these sequences.

In order to study the mechanism of transposition of the *Bg* element, we have introduced the autonomous element in tobacco plants. For this purpose we have constructed two types of binary vectors both based on pBIN 19. The first vector contains the complete *Bg* element flanked by small fragments of the *waxy* gene. This construct was denominated pBINBg. The second binary vector contains a CaMV 35S promoter and a *rolC* gene from *Agrobacterium rhizogenes*. The transcription of the *rolC* gene in this vector has been interrupted by placing the complete *Bg* element plus flanking *waxy* sequence between the CaMV promoter and *rolC*. *Bg* was inserted in both orientations between promoter and *rolC* giving rise to the constructs pBINBg53, carrying the CaMV and *Bg* promoter in the same orientation, and pBINBg35 with the two promoters in opposite directions. Tobacco plants were transformed by the leaf-disk method and 84 transgenic plants were obtained. The excision of the *Bg* element from its original position was monitored by PCR analysis with two oligos complementary to the flanking *waxy* sequences. Only if *Bg* excises will the PCR analysis give rise to an amplification product since the oligos span a distance of almost 5kb in the presence of *Bg*. All plants tested showed amplification of specific products, indicating the excision of the *Bg* element. The PCR products were subcloned and sequenced. All fragments contained the expected *waxy* sequence with an empty donor site for the *Bg* element. Moreover, typical footprints were observed. A second series of experiments to demonstrate excision of *Bg* in tobacco was carried out with the use of the pBINBg53 and pBINBg35 plasmids. Plants transformed with these constructs are expected to express *rolC* upon excision of the *Bg* element. This would originate plants with pale green to yellow sectors. Sixteen plants transformed with pBINBg53 and 26 plants with pBINBg35 were analyzed. All showed characteristic sectors on the leaves. No differences were observed between the two types of constructs.

In order to investigate whether the excision of *Bg* was followed by reintegration of the element, Southern analyses were performed on plants transformed with the pBINBg53 and pBINBg35 constructs. Upon digestion with *Eco*RI or *Hin*dIII restriction enzymes and hybridization with a *Bg* probe, expected fragments were seen in most of the plants analyzed. A number of plants showed additional bands, indicating a reintegration of the *Bg* ele-

ment. Some plants showed no hybridization with the *Bg* probe, indicating the loss of the element upon excision. This phenomenon was observed in plants transformed with both the pBINBg53 construct and the pBINBg35 construct. Striping of the blots and rehybridization with a *rolC* probe produced bands for all plants, confirming that all plants tested were actually transformed. The majority of plants integrated a single copy of the construct.

The available data indicate that the *Bg* element is capable of excising from and reintegrating into the tobacco genome. Furthermore, the addition of a strong promoter does not influence the rate of transposition of the *Bg* element in tobacco. Studies aimed at quantifying the amount of *Bg* transcript in plants containing the different constructs are in progress.

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The order of liguleless gene function

--Lisa Harper and Michael Freeling

Phenotype of *lg1* and *lg2* homozygotes. Recessive mutants in two unlinked genes remove the ligule and auricle of the maize leaf: *lg1* and *lg2*. In a Mo17 background, they have different phenotypes which are distinguishable in seedlings as well as in adult plants. In *lg1* mutant plants, no auricle is found on any leaf, and ligule only on the last few adult leaves before the tassel. In *lg2* plants, only the first and sometimes second leaves are completely liguleless and auricleless. On juvenile leaves, the ligule and auricle appear as small wedges at the margin, making the adaxial side appear to have a broad midrib. Each subsequently formed leaf has more ligule and auricle than the previous. The two auricle wedges on either side of the broad midrib area can also be displaced relative to each other. Ligule is never formed at the midrib.

Mosaic analysis results. Earlier results of mosaic analysis on *lg1* (Becraft et al., Dev. Bio. 141:220-232, 1990) indicate that $Lg1^+$ product acts in a cell autonomous way. Borders of *lg1* sectors in a wildtype leaf correspond exactly to the cell autonomous marker used. In addition, the progression of ligule maturation from midrib to margin is aborted by the presence of a *lg1* sector. Ligule and auricle reinitiate on the marginal side of the mutant sector, as if they were starting new from the midrib. This suggests that $Lg1^+$ is involved in the progression or propagation of a "make ligule-make auricle" signal.

Preliminary results of mosaic analysis of *lg2* suggest a different role. Inv3a/*lg2 y10* plants were X-rayed to induce segmental monosomic sectors on a wildtype leaf. The presence of a *lg2* sector on an otherwise wildtype leaf sometimes causes the disruption or lack of ligule. However, the presence or absence of ligule in a mutant sector shows no correlation with the presence or absence of *Lg2+* in any particular layer or layers of the leaf at the blade-sheath boundary in 39 sectors studied. No correlation was found between sector width, or distance from the midrib, with the presence or absence of ligule. Since no obvious correlative factor was found, other factors, such as a signaling pathway acting in the longitudinal dimension, cannot be ruled out. The data suggest that $Lg2^+$ function does not confine itself to the cells in which $Lg2^+$ product is made.

In addition, the progression of ligule and auricle on the marginal side of the mutant sector is not disrupted by the presence of a *lg2* sector. Thus, the presence of a *lg2* mutant sector does not impede the "make ligule-make auricle" signal.

Aneuploid analysis. TB-2Sb was crossed as a male to *lg1-0/lg1-0* (the Coop reference allele), and to a dwarf tester. Liguleless and dwarf progeny were observed, respectively, in non-Mendelian ratios. The *lg1* plants appeared no different than their mothers with respect to ligule and auricle. Likewise, TB-3Lg, *lg2/+* was crossed as a male to *lg2-0/lg2-0* (the Coop reference allele). The cross was set up this way in order to compare *lg2/lg2* sibs to *lg2/-*. The progeny were >50% *lg2*. Hypoploids of TB-3Lg are runts, so *lg2/lg2* could be distinguished from *lg2/-*. There was no apparent difference in the ligule and auricles of the 2 classes of *lg2* plants. Thus, both *lg1-0* and *lg2-0* mutants are acting genetically as nulls.

Double mutant analysis. Double mutants of *lg1-0* and *lg2-0* were constructed by the following crosses: ((*lg1 gl2/lg1 gl2* X *lg2/lg2*) X *lg1 gl2/lg1 gl2*) self, and testcross to *lg2/lg2*, selecting for liguleless and glossy phenotype after the second cross. Of the families that segregate 1:1 for *lg2* in the testcross, all progeny of the self showed the *lg1* phenotype; i.e., they appeared totally liguleless and auricleless when scored at the three leaf stage. For comparison, sibs of the *lg2/lg2* test plants show auricle and ligule on the margins of the second leaf, at the approximate place where the ligule is expected. This is in agreement with segregation of the (*lg1 gl2/lg1 gl2* X *lg2/lg2*) self, where 104 adult plants were scored, and segregated 67 wildtype to 24 *lg1*-like phenotype to 13 *lg2*-like phenotype. Statistical analysis suggests this is more likely a 9:4:3 segregation than a 9:3:3:1 (chi squared equals 3.57 versus 10.94 respectively). No novel phenotypes were observed, and there was no significant loss of germination in either planting of double mutants. In a strict sense, *Lg1* is epistatic to *Lg2*, however, the nature of the two phenotypes does not allow us to conclude that $Lg1^+$ and $Lg2^+$ are acting in the same biochemical pathway. Since both behave as nulls, we can conclude that they are both involved in the same biological developmental program.

Conclusion. Mutants of *lg1* have a more severe phenotype than mutants of *lg2*. One might expect the double mutant to display the more severe phenotype, and this is indeed the case. In terms of developmentally ordering gene function, however, $Lg1^+$ cannot act earlier in development than $Lg2^+$. Several observations point to this conclusion. $Lg1^+$ acts in a cell autonomous manner. This precludes having a non-cell-autonomously acting gene downstream. Mosaic analysis has shown that $Lg1^+$ is responsible for reception and/or propagation of a "make ligule-make auricle" signal, but is not the signal itself. Scanning electron microscopy and sectioning have shown that the *lg1* mutation blocks the epidermal periclinal and longitudinal anticlinal divisions in the pre-ligule, pre-auricle area (Sylvester et al., Development 110:985-1000, 1990; Becraft et al., Dev. Bio. 141:220-232, 1990). This is relatively late in development.

$Lg2^+$ gene function acts in a non-cell autonomous way, suggesting that the $Lg2^+$ gene product is itself mobile, or causes another product to be mobile. This mobile product is not likely to be the "make ligule-make auricle" signal itself, because *lg2* mutants, even in embryonic leaves, make ligule and auricle. The lesion in *lg2* mutants affects the placement of ligule and auricle on the leaf, and the coordination of the auricle wedges on either side of the midrib. This suggests that $Lg2^+$ is involved in making the region competent to receive the "make ligule-make auricle" signal.

Mapping of *y10* on chromosome 3

--Lisa Harper and Michael Freeling

I am using *y10* as a cell autonomous marker for mosaic analysis of *lg2*, and need to know the distance between *y10* and *lg2*. Both mutations are recessive, and *y10* dies as a white seedling. Thus, a typical mapping testcross to a double recessive homozygote is not possible. Robertson mapped *y10* approximately 11 map units from *lg2* (Robertson, Genetics 46:649-662, 1961). Beckett showed by TB translocations that *y10* is distal to *lg2* (J. Hered. 69:27-36, 1978). I obtained data from selfs of 5 double heterozygotes of *lg2* and *y10* in coupling.

		Phenotypes		
	Parentals		Recombinants	
Family	W.T.	*lg y10*	*y10*	*lg*
510.1 self	42	5	5	4
510.3 self	32	11	0	4
510.4 self	26	5	2	2
510.5 self	83	31	11	11
510.7 self	75	21	3	6
TOTAL	258	73	21	27

In order to calculate the recombination frequency, I used two methods, and made the assumption that recombination frequency between these 2 genes is the same in the male and female. Using the product method (Immer, Genetics 15:81-98, 1930), the recombination frequency comes out to 0.134, with a probable error of 1.3%. Using maximum likelihood (Mather, The Measurement of Linkage in Heredity, Wiley and Sons, 2nd ed., 1951), the recombination frequency comes out to 0.135, with a standard error of 1.9%.

Thus *y10* is 13.4 map units distal to *lg2* on chromosome 3.

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β-glucosidase occurs as large aggregates in "null" genotypes of maize

--Asim Esen

In maize, the catalytically active form of β-glucosidase is a dimer. Genotypes whose zymograms are devoid of detectable enzyme bands and thus are thought to be homozygous for a null allele at the *Glu1* locus are known. This is puzzling in view of the data implicating the enzyme in important functions that are critical to plant growth and development. We have shown that both null and normal genotypes have similar amounts of the enzyme protein and activity, but the enzyme occurs mostly as insoluble or poorly soluble aggregates in nulls and does not enter the gel; thus, it is not detected by zymogram techniques (Esen and Cokmus, 1990).

Further studies were carried out on β-glucosidase isolated from "null" genotypes in order to determine the nature of the interactions responsible for aggregation as well as the sizes of enzyme aggregates. Coleoptile extracts from inbreds K55 (normal) and H95 (null) were made with 50mM sodium acetate buffer, pH 5, and subjected to size fractionation through a column of Sephacryl HR 300 (exclusion limit, 1.5 million Daltons) using appropriate calibration standards. Column fractions were assayed for enzyme activity spectrophotometrically and for the enzyme monomer by SDS-PAGE. The gel filtration data show that nearly 80% of β-glucosidase activity in extracts of H95 appeared in the flow-through fraction suggesting that it had a molecular mass equal to or greater than 1.5 million Daltons while only about 20% of the activity eluted as a dimer (120,000 Daltons) (Table 1). In contrast, nearly all of the activity in extracts of K55 eluted from the column as a dimer. These data clearly indicate that the enzyme in extracts from "null" genotypes occurs as large aggregates.

Table 1. Distribution of β-glucosidase activity in normal and "null" phenotypes.

Phenotype	Est. Mol. Wt. (Daltons)	
	120,000	≥1.5 million
Normal (K55)	98%	2%
Null (H95)	22	78%

Estimated by gel filtration (Sephacryl HR 300)

In order to determine the nature of the interaction responsible for aggregation, the enzyme was extracted from null inbreds with buffers containing SDS, or SDS was added to the supernatant fluid after extraction without SDS in buffer. These samples were electrophoresed through native, IEF and SDS-gels. All of the 8 different "null" genotypes tested yielded zymograms with active bands in all of these gel systems after extracts were added or made with SDS-containing buffers. Figure 1 provides evidence for the fact that the enzyme from a typical "null" (H95) does not enter the gel until SDS is either added to a final concentration of 0.5% to the extract or extraction is made with a buffer containing at or above 0.5% SDS. These results suggest that the basis of

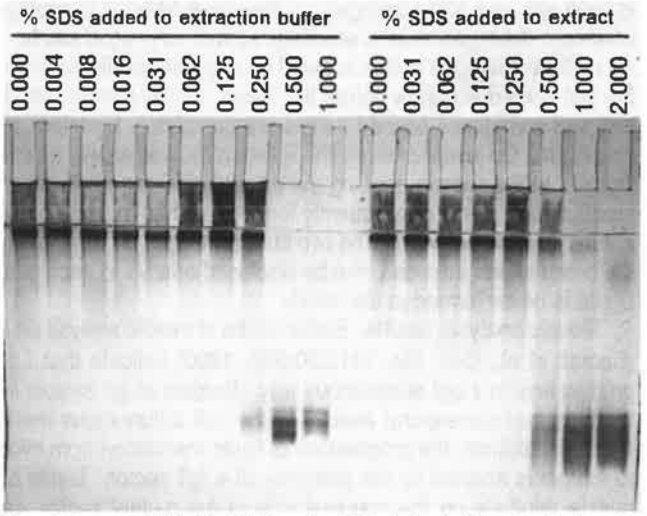

Figure 1. Zymogram (6% native PAGE; anode at bottom) showing the entry of β-glucosidase from a null genotype (H95) into the gel after SDS was added to the extract or the extraction buffer at a final concentration of 0.5% or higher. Note that enzyme aggregates remain in the stacking gel and form a band at the top of the resolving gel.

interaction leading to aggregation is very likely to be hydrophobic because it could be disrupted by SDS, releasing catalytically active dimeric enzyme. However, the question of whether or not the aggregates are formed between enzyme molecules after interaction with one another or between enzyme molecules and other protein or nonprotein components is not known and under investigation. Likewise whether the aggregates are formed as an artifact during extraction or they occur also in vivo is worthy of further investigation.

On the basis of the data above it can be concluded that none of the maize genotypes classified as "null" is truly null, and apparent null phenotype is due to formation of large quaternary associations of the enzyme which can be dissociated with SDS. Thus the observed monogenic inheritance for "null" phenotype appears to be for an allele of another locus whose product either interacts with the enzyme or causes it to aggregate in vivo or during isolation.

Tissue-specific expression of β-glucosidase

--Asim Esen

Soluble and total protein were extracted from a variety of plant organs and parts in order to quantitatively determine enzyme activity and the amount of antigen reacting with anti-β-glucosidase sera in extracts of each of these organs and parts. The plant organs and parts used were: pollen, male spikelet, rachis, silk, husk, ear axis, ovule, mature leaf, germ and endosperm dissected from seeds on day 0, 3, and 5 after germination (DAG), and coleoptile. Figure 1 shows that the highest enzyme activity was measured in the coleoptile, primary root, husk, and ovules. Low levels of enzyme activity were measured in the silk, spikelet, mature leaves, and germ (3 DAG and 5 DAG), while no activity was detected in extracts of whole mature kernels, pollen, rachis, ear axis, and endosperm. The immunoblotting results (Figure 2) confirm those from spectrophotometric assays in that only the organs and parts that have detectable enzyme activity have a 60kD (arrow) immunoreactive polypeptide (β-glucosidase monomer).

The overall results from studies on organ-specificity of β-glucosidase expression show that the enzyme is exclusively found in actively growing plant parts (e.g., primordial leaves, shoot apex, mesocotyl, primary roots) of young seedlings or female reproductive organs (e.g., ovule, silk, and husk). Plant parts that are terminally differentiated and matured (e.g., endosperm, ear axis, and rachis) are either devoid of enzyme activity or the enzyme is not extractable with aqueous buffers even after SDS has been added to a 0.5% final concentration. Moreover, the presence of SDS in the extraction buffer did not affect the amount of activity extracted, except in the case of roots, where SDS helped extract additional activity. These results suggest that β-glucosidase is a key enzyme with critical role(s) in normal plant growth and development. Our laboratory could not find any evidence for cyanogenesis in maize coleoptile extracts using either maize or sorghum β-glucosidase as the enzyme source. Interestingly, the coleoptile (coleoptile proper, mesocotyl, primordial leaves, and shoot apex) is the richest source of the maize enzyme, but it is not a cyanogenic plant part, suggesting that the reactions catalyzed by the enzyme in young shoots do not involve cyanogenic glucosides as substrates.

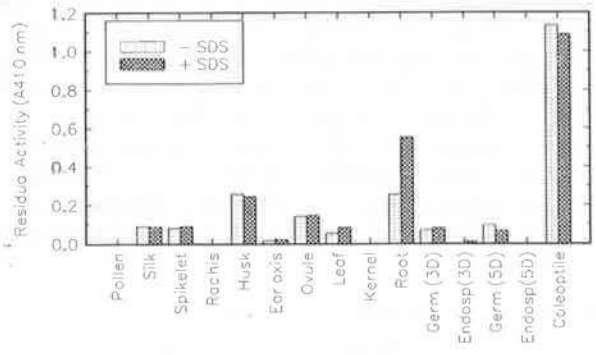

Figure 1. Tissue-specificity of maize β-glucosidase expression. The enzyme was extracted from freeze-dried powder of each plant part with 0.1M Tris-HCl, pH 8.0, containing minus or plus 1% SDS, and the extracts were assayed for activity after 100X dilution with the standard enzyme assay buffer. Note that the highest total activity was measured in the coleoptile extract followed by root, husk, and ovule extracts, respectively.

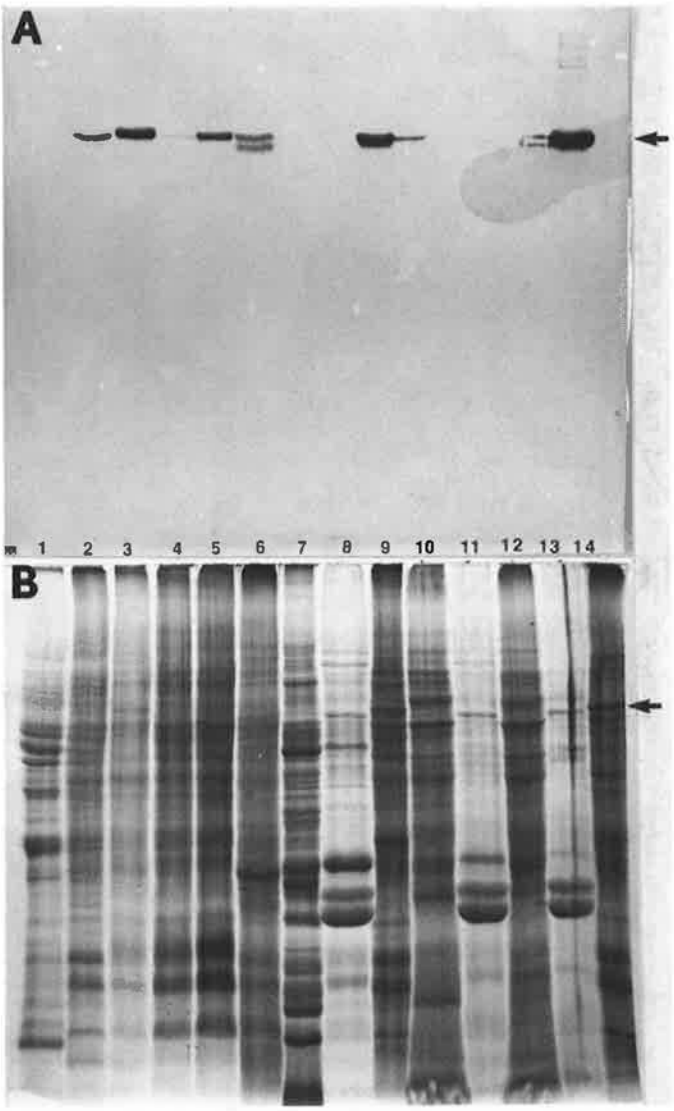

Figure 2. Tissue-specificity of maize β-glucosidase expression. The extracts used for activity assays (refer to Figure 1) were subjected to SDS-PAGE, blotted onto nitrocellulose, and probed with anti-β-glucosidase serum. Lane 1, pollen; 2, silk; 3, husk; 4, ear axis; 5, ovule; 6, leaf; 7, germ at day 0; 8, endosperm at day 0; 9, primary root; 10, germ at day 3; 11, endosperm at day 3; 12, germ at day 5; 13, endosperm at day 5; and 14, coleoptile.

Subcellular localization of maize β-glucosidase

--Asim Esen and David A. Stetler

The seedling parts (primary and secondary roots and coleoptile node, which contains the shoot apex and primordial leaves) that are known to be rich in enzyme activity were fixed, sectioned, and incubated with anti-β-glucosidase sera and protein A-conjugated to 20nm gold particles, respectively. Preimmune and nonimmune sera served as controls. The examination of the distribution and intensity of gold labelling revealed that the label was found exclusively in plastids in root cells and proplastids and immature chloroplasts in shoot cells (Figure 1, B). There was no gold labelling in sections incubated with pre-immune serum (Figure 1, A) while there was fairly intense labelling in all sections incubated with immune serum indicating that labeling was specific for the enzyme. At this point it appears that the enzyme is localized to the stroma portion of the plastid; but this conclusion needs to be con-

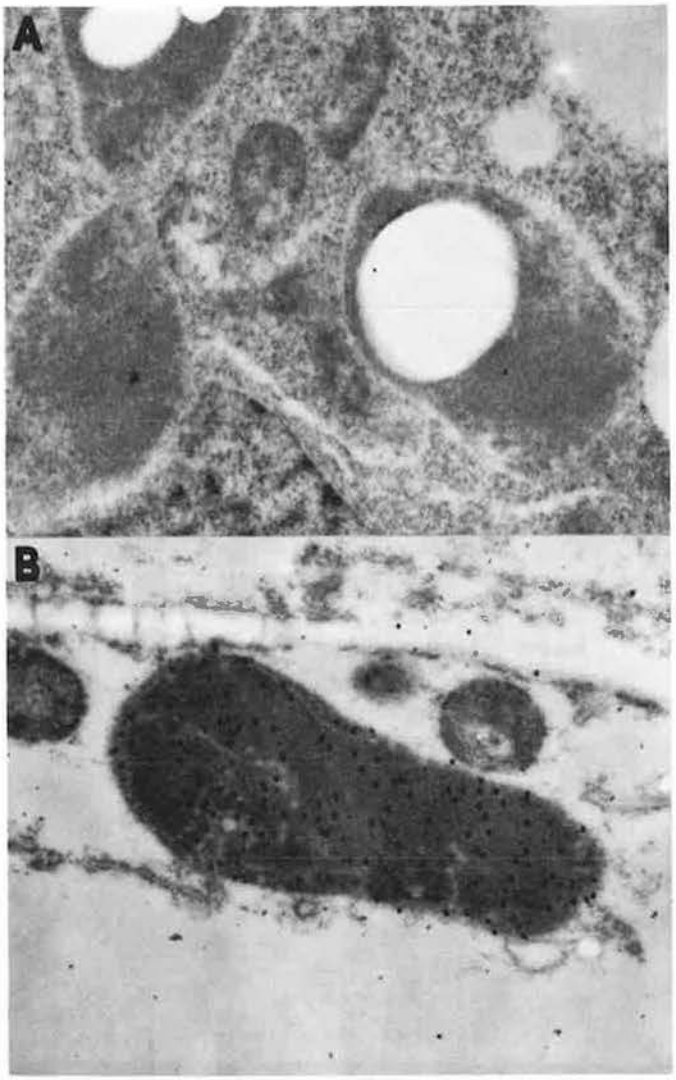

Figure 1. Immunocytochemical localizations of maize β-glucosidase to proplastids in shoots. A. A cell with several plastid sections showing no labelling after immunostaining with the preimmune serum. B. Section of proplastids in cells from the shoot apex region showing intense gold labelling (20nm) after incubation with anti-β-glucosidase antiserum and gold conjugation protein A.

firmed by further studies. The cell wall, which is the site of β-glucosidase localization in legumes and other dicots, does not seem to have any β-glucosidase in maize. Sorghum (Thayer and Conn, 1981), oats (Nisius, 1988) and rice (Muslim and Esen, unpublished) β-glucosidases have been also localized to the plastid, suggesting that this organelle is the site of the enzyme in all grasses, if not in monocots.

The localization of maize β-glucosidase to the plastid by immunocytochemistry was also confirmed by two related approaches. First, etioplasts and chloroplast were isolated from young seedling parts (shoots including primordial leaves, and primary roots) and stained histochemically by infusing the substrate 6-bromo-2-naphthyl-β-D-glucoside plus a coupling dye or X-glu. Only the plastids showed staining (β-glucosidase activity). The same results were obtained when isolated plastids were incubated with substrates. Second, young seedling parts were free-hand sectioned and incubated with the same substrate and coupling dye combination as that used for plastids. Microscopic examinations showed clearly that the enzyme activity (i.e., staining) was associated with discrete organelles, identified as plastids. Histochemical staining was judged to be specific for β-glucosidase because it was not observed in the presence of such known inhibitors of β-glucosidase activity as δ-gluconolactone, silver or mercury ions. Moreover, in the case of primary roots, histochemical staining was highest in the root cap and in the zone of elongation including root hairs. In fact, root hairs had discrete packages of β-glucosidase-positive organelles (plastids) occurring at intervals along their entire length as well as their tips. The implications of these findings are profound because they suggest that (1) the precursor enzyme monomer will include a transit sequence to target it to the plastid, and (2) the plastid is the organelle in which the enzyme performs its catalytic functions and also a likely site for the enzyme's physiological substrates. Alternatively, the plastid might be the site for sequestering and storing the enzyme.

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Nature of dominant inhibitor of aleurone color, *C1-I*

--S. B. Allagikar-Nadiger and N. K. Notani

I is considered allelic (i.e., *C1-I*) to *C1* (colored aleurone) and the recessive allele *c1* (colorless aleurone). *I*, an inhibitor of aleurone color, is dominant over *C* which in turn is dominant over *c*. Based on a superficial similarity to a super-repressed mutation in the regulatory gene of β-galactosidase, which is dominant over the wildtype allele, it was suggested by us that *I* could be a regulatory gene of *C* (Chandra Mouli and Notani, MNL42:6-10). However, all tests of allelism are consistent with the interpretation that *I* is allelic to *C*. We may then ask, in what way is *I* different from *C* and what is its origin? In a spontaneous mutation experiment, we showed that out of 35,000 kernels scored in the progeny of *I/I* crossed by *C/C*, we got only one mutation and that was towards colorless (*i* or *c*) (Chandra Mouli and Notani, MNL41:11-12, 1967). We have reported a mutable *I* manifesting coloured dots on colorless background. We interpreted this mutability in terms of insertion of a transposable element in the DNA sequence corresponding to a DNA-binding (Operator) region of the protein product (Allagikar et al., J. Genet. 70:33-41, 1991). Saedler's group (Paz-Ares et al., EMBO J. 9:315-321, 1990) showed by heteroduplex mapping that at the DNA level, *I* (source Ed Coe) differs from *C* in at least 3 regions, while there is considerable homology in the rest of the DNA stretch. We have recently examined recombination in the *I Sh Bz* interval in a stock isolated locally which has not only an *I* allele but also has an enigmatic *Sh1 Bombay* allele (see accompanying note). Because of the presence of the *Sh1 Bombay* allele, crossover data could be obtained only for the *I-Bz* interval.

Table. Recombination from the cross *I sh1-B Bz/C sh1 bz* x *C sh1 bz*

	Parental		Crossover	
	Colorless	Bronze	Purple	Crossover %
1	226	220	21 x 2	9
2	110	120	7 x 2	6
3	62	50	4 x 2	7
4	40	38	4 x 2	10
5	97	95	6 x 2	6

There is no conspicuous reduction in the crossover values; if anything there is a slight increase--5% (standard value) vs. 7.6%.

Our *I* allele may or may not be similar to *C1-I:Coe*, however, there is little effect on the recombination frequency in the *I-Bz* interval. The present recombination data do not allow strong deductions about rearrangements, if any, between *I* and *C* alleles.

Breeding behaviour of *sh1-B* allele–further analysis

--S. B. Allagikar-Nadiger and N. K. Notani

A new allele, designated *sh1-B* (*shrunken1-Bombay*) had been isolated locally which has a somewhat unorthodox breeding behaviour. Upon self-pollination, *sh1-B* yielded kernels with variable phenotypes. Upon testcrossing to the American tester *sh1-A sh1-A* all progeny kernels are completely shrunken (Allagikar et al., J. Genet. 70:33, 1991). *sh1-A wx/sh1-B Wx* heterozygotes were self-pollinated and the progeny kernels were classified for shrunken phenotype (Table 1).

Table 1. Classification of progeny kernels following self-pollination of plants *sh1-A wx/sh1-B Wx*.

Progeny no.	Kernels (shrunken)	Kernels (phenotypically non-shrunken)	Total	Remarks
11:39-1	251	3	254	--
11:40-1	221	3	224	1 kernel half sectorial for shrunkenness; 1 kernel waxy mutable
11:40-2	154	2	156	1 kernel waxy mutable
11:40-3	258	2	260	--
11:40-4	223	2	225	--

It may be inferred: i) some instability prevails at the *sh1* locus and ii) two kernels showed typical transposable element-induced instability at the *waxy* locus.

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Teosinte in Guerrero near the Oaxacan border, Mexico

--Garrison Wilkes and Jesus Sanchez

Teosinte has been located in a region of Guerrero where there are no previous reports of its occurrence. The significance of this new locality is that it forms a bridge with the large population in the drainage of the Rio Balsas and the recently relocated (MNL60:29) but isolated populations in Oaxaca of annual teosinte. Travelling East from Iguala, Guerrero on the road to Huitzuco de los Figueroa and South to Atenango del Rio, teosinte was found on ejido land at Paseo Morelos (Long. 99° 1' - Lat. 18° 14'). The exact locality is called Plan de Timbres (1225m), an irrigated valley floor planted to maize. We were told of other localities in the surrounding region and did indeed verify one of the sites. The region is seasonally dry to arid and definitely over-grazed, so teosinte could exist only in fenced protected zones, usually maize fields. This region has recently had road access, and further exploration toward Copalillo, Olinalá and the Rio Tlapaneco region might well turn up new collection sites. Because of the significance of this zone to the origin and subsequent evolution of maize further exploration seems appropriate. The local name for annual teosinte is Maiz de Pajaro (bird corn is the same as that of other parts of Guerrero). The plant is definitely a small seeded form of the race Balsas. Seed for experimentation can be obtained from Dr. Jesus Sanchez, Departamento de Recursos Genéticos en el Campo Agrícola Auxiliar Valle de Zapopan INIFAP, km 10 Carretera Tlanomulco a San Miguel Cuyutlan, Guadalajara, Jalisco, Mexico.

Teosinte in the Valley of Toluca, Mexico

--Garrison Wilkes and Sukitoshi Taba

Teosinte is widespread on the South side of Toluca from Metepec to Rayóu (Long. 99° 37', Lat. 19° 10'-15'), a distance of 11km, on the road Toluca-Ixtapan de la Sal in the State of Mexico. How could this population exist for so long without being discovered? For starters there are only cultivated fields and teosinte exists as a corn mimic in a fashion comparable to Chalco in the Valley of Mexico. There are no hillsides with wild populations. Secondly, teosinte is of short stature comparable to the maize in the fields, Conico. In all other maize-mimetic coevolutions teosinte is a tall plant and may exceed the height of the maize. Lastly teosinte around Toluca dries a gray brown, the same color as Conico. All other teosinte populations dry a bright tan, which is several shades lighter than maize and easily spotted. This color difference led to the discovery of the Ciudad Hidalgo population by me thirty years ago.

How I could have missed the Toluca population for so many years is beyond me. A week before the discovery I had driven alone the exact same road and not seen it. A week later Sukitoshi Taba was driving and I was watching the fields which were near harvest condition and I said "that's teosinte" and we both agreed it couldn't be because it had never been reported in the valley, and then there were more plants and we continued to see plants for 11 more kms, from 2600m to 2800m, which is higher than teosinte populations above Amecameca in the Valley of Mexico. The Valley of Toluca teosinte populations on the side of the Nevado de Toluca are the highest in elevation of all teosinte populations in Mexico and unique because they are such excellent mimics of the maize race Conico. The seed is very large and comparable to the race Chalco. At the present time we do not wish to make a racial determination for the population. Professor T. Kato is currently studying the chromosome knob positions, which should help resolve the racial classification question.

The significance of this discovery is that so many of us have driven by but not seen the population. This begs the question, are there more regions in Mexico yet to be discovered that we have repeatedly driven by and not seen? Seed of this population can be supplied by Dr. Sukitoshi Taba, Head CIMMYT Maize Germplasm Bank, Lisboa 27 A.P. 6-641, 06600 Mexico D.F. Mexico.

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From teosinte to maize: knobs as a means to maintain wild x domestic characters in blocks

--Luiz Torres de Miranda, Luiz Eugenio Coelho de Miranda, Omar Vieira Villela, Hermano Vaz de Arruda, Joaquim A. Machado and Vera Lucia Monelli

The birth cradle of a species in domestication shows sympatric populations in disruptive selection. There is selection for polymorphism, with one population being selected for wild characters important to reproduction and the other being selected by man to facilitate the harvest. Whenever polymorphism and/or selection

for epistatic effects exist we can see the occurrence of supergenes, blocks of co-adapted genes. In many cases, for a successful process, there is the necessity of building genetic mechanisms of isolation between the two populations. Small grain cereals (wheat, barley and oat) with complete flowers are open-pollinated in the wild state. Under domestication they became self-pollinated. Rye could be the exception that confirms the rule. Rye developed as a weed among the other cereals maintaining cross-pollination. In the case of maize, with female flowers in the ear and male flowers in the tassel, more elaborate devices were necessary.

Four main characteristics distinguish teosinte from maize: (1) two rowed ears (ts) versus decussated ears (tr); (2) simple spikelets (pd) versus bifloral spikelets (Pd); (3) horny glumes covering the grain (such as *Tu*), versus soft glumes and naked grains; and (4) rind abscission (ri) and pith abscission versus a non-shattering rachis and shelling ear.

What we want to show is that for maize the evolutional solution was to join in strings these characteristics, the knobs being the mechanical device to maintain them together. The study of this matter is extensively presented by Ford (Ecological Genetics, London, Chapman and Hall, p. 442, 1975). He concluded that always when there is polymorphism, there is the formation of supergenes, or blocks of co-adapted genes. In his work he presents many cases with animals but discusses only one case of a wild plant (*Primula sinensis*), the Chinese primrose. In this plant with complete flowers there is the formation of a block of genes to force cross-pollination by several ways: anther position in relation to the stigma, gametophytic effects, etc. The most complete botanical study of ecological genetics in maize is that from Mangelsdorf (Corn, its Origin, Evolution and Improvement, Harvard Univ. Press, MA, p. 262, 1974). His proposal talks about six lineages from different origins, indicating the polyphyletic origin of maize races. Beckett et al. (MNL52, 1978) presented the comparative position between cytogenetic and genetic markers in maize, including four knobs. With K representing the knob, L the long arm, S the short arm, the decimal digit being the physical cytological position and the whole number the position in the map, the correspondence between the two maps is roughly K5L.72=64; K6L.63=66; K6L.74=68; K7L.63=64. Comparing these data with Figure 1, a reproduction of Kato's map in McClintock (Maize Breeding and Genetics, ed. D. B. Walden, Wiley & Sons, NY, p. 794, 1978), we see that only the first (K5L) falls outside the distance of a supergene, when compared with our estimates on krn and pd position (typically within 15cM).

It is seen that the knob distribution is biased. In chromosomes 6, 8 and 10 there is a string in the long arm formed by 3 knobs in chromosome 6, 2 knobs in chromosome 8 and 4 knobs in chromosome 10. In the remaining chromosomes there are two knobs, one in each arm, all in distal positions and far away from one another. Over all chromosomes, taking the strings as units, there are 17 knob sites. In the Maize Genetics Coop. Newsletter working maps, we see the knob K3L on the position 115, mapped by Dempsey (MNL45, 1975). Without taking account the knobs on zero terminal position in chromosomes 4, 7 and 9. With these data, it is possible to map K6L near the position 41, corresponding to that of Miranda et al. (MNL61, 1987), for five translocations with Portugues Fasciado. The main conclusion is that pd (paired) corresponds to fas (fasciation) in maize, and tr (two-ranked) to krn (kernel row number). The original data were analyzed again by Miranda et al. (next article) by a model describing the action of two

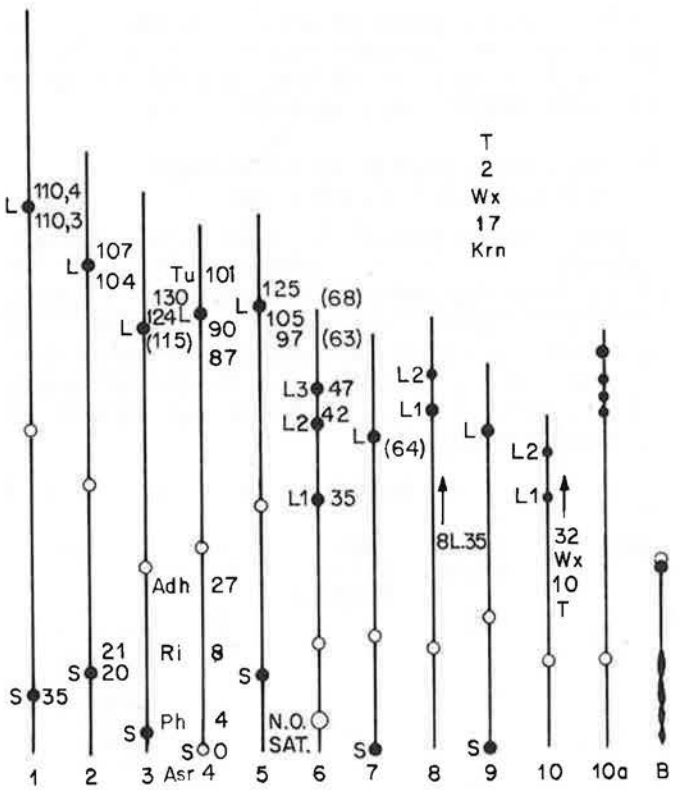

Figure 1.

pairs of alleles in the same chromosome, affecting the same characters, kernel row number and ear fasciation by genes tr (krn) and Pd (fas). Based on the position K7S.0 from Kato, the authors obtained Krn17 and Fas17 at 0, T at 57, Fas7 at 64 and Krn7 at 66. The position of K7L from Beckett corresponds to position 64, a perfect fit. A typical super-gene usually spans a 15cM interval. Miranda et al. (MNL61, 1987) analyzed the recombination values in F2 progenies of tr x pd crosses with chromosome 1 to 6 genetic markers from Langham's data. The biometrical model used was the classic one, for a segregation in F2 of 9:3:3:1 classes. The authors obtained the results presented in Table 1, using the new model developed in this work. The distances Pd Tr vary from 4 to 20cM, with a mean around 16cM.

Table 1.

chr1	chr2	chr3	chr4	chr5	chr6
f1 86	*lg1* 28	*lg2* 93	pd4 3	*pr* 67	*py1* 65
pd 117	*gl2* 30	pd3 113	tr4 7	tr5 125	tr6 99
tr 132	tr2 30	tr3 118	ts5 53	pd5 166	pd6 119
bm2 161	pd2 45	*a* 141			

We could suggest a rough correspondence of pd tr with knobs.

Miranda et al. (MNL64, 1990) proposed for chromosome 2 the sequence ltp at 20, krn at 22, lte at 30, lsc at 35, *b1* at 49, *fl1* at 68, krn12 and p2 at 103. Miranda et al. (MNL65, 1991) mapped ltp in position 17, with n=1013 in F2 progenies, confirming the ltp position.

Kato (Mass. Agric. Exp. Stn. Bull. 635, 1976), concluded that the basic teosinte chromosome morphology has no knobs. He also observed that in the presence of inversions like in 4S and 9S involving knob sites, the knobs were absent. Kato and Lopes (Maydica 35:125-141, 1990) observed that in five sympatric populations of maize and teosinte, each pair had the same knob

complex. McClintock (1978) presents an exhaustive discussion on knobs, together with a knob map from Kato, which we reproduce here. Figure 1 shows the distinctive components of the maize chromosome complement that were recorded in this study. From left to right the chromosomes are numbered according to the relative size, 1 to 10; 10a refers to Abnormal-10; B refers to the B-type chromosome. On the cytological map are superimposed the knob positions explained in the text of this report and also the new positions published in the MNL, 1992.

Classical biometrical models have been insufficient to solve the question. With the same Langham data, we developed a biometrical model for two pairs of alleles in the same chromosome affecting the same characters (tr and pd) in relation to a standard marker. This problem was already solved for a backcross case by Miranda et al. (MNL64, 1990). We propose now a model for F2 progenies.

With F2 progenies in repulsion, the gamete frequencies in relation to the marker gene are: pq, p(1-q), q(1-p) and (1-p)(1-q). Putting them in a 4x4 entry table, we obtain the genotypic expectations: p^2q^2, $p^2q(1-q)$ etc., until $(1-p)^2(1-q)$, $(1-p)^2(1-q)^2$ as in Table 2, where we see the genotypic expectations of classes, when two pairs of alleles affecting the same character (tr or pd) are calculated in relation to a marker within their span (repulsion phase):

Table 2.

Gametes	pq	p(1-q)	q(1-p)	(1-p)(1-q)
pq	p^2q^2	$p^2q(1-q)$	$pq^2(1-p)$	$pq(1-p)(1-q)$
p(1-q)	$p^2q(1-p)$	$p^2(1-q)^2$	$pq(1-q)(1-p)$	$p(1-p)(1-q)^2$
q(1-p)	$pq^2(1-p)$	$pq(1-p)(1-q)$	$q^2(1-p)^2$	$q(1-p)^2(1-q)$
(1-p)(1-q)	$pq(1-p)(1-q)$	$p(1-p)(1-q)^2$	$q(1-p)^2(1-q)$	$(1-p)^2(1-q)^2$

The 16 genotypic classes or only 4 phenotypic classes can be identified as a, b, c and d, as usual. To class a corresponds the intersection of the 1st and 2nd lines with the 1st and 2nd columns; to b, the intersection of the 1st and 2nd lines with the 3rd and 4th columns and so on.

To class a the expectations are:

$$p^2q^2 + p^2q(1-q) + p^2q(1-q) + p^2(1-q)^2$$

Factoring for p and adding we get

$$p^2[q^2 + 2q(1-q) + (1-q)^2]$$
$$= p^2(q^2 + 2q - 2q^2 + 1 - 2q + q^2)$$
$$= p^2(1)$$

p^2 is the estimate for the a class. Effecting the same steps for b, c and d: p(1-p) is the b/n and c/n estimates and $(1-p)^2$ is the d/n estimate.

For the logarithmic likelihood expression, l being the normal log, and a, b, c and d the phenotypic classes observable we have:

$$L = 2\,a\ln p + b\ln(1-p) + c\ln(1-p) + 2d\ln(1-p)$$

Derivating in relation to p and adding we get

$$\frac{\partial L}{\partial p} = \frac{2a}{p} + \frac{b(1-2p)}{p(1-p)} + \frac{c(1-2p)}{p(1-p)} - \frac{2d}{(1-p)}$$

Putting the minimum common multiplier, factoring and equating to zero:

$$\frac{\partial L}{\partial p} = \frac{2a(1-p) + b(1-2p) + c(1-2p) - 2dp}{p(1-p)} = 0 \quad (I)$$

As we usually do, solving the quadratic equation we get the p value:

$$p = \frac{2a + b + c}{2(a + b + c + d)}$$

Making $\frac{\partial^2 L}{\partial p^2}$ to obtain the variance error we get:

$$V = \frac{p(1-p)}{2n} \quad (II)$$

Since $V = -1/\frac{\partial^2 L}{\partial p^2}$ the negative result offers a logical sense, following a Cramer-Rao inequality. This is the 11th theorem.

We can get an alternative simpler solution. If we first derivate the 16 classes and only after we pool them in classes a, b, c and d, we get:

$$p = \frac{2a + b + c}{2(a + b + c + d)} \quad (III) = (I)$$

The variance is obtained as usual:

$$p = 1/2\,\frac{(2a + b + c)}{(a + b + c + d)}$$
$$Vp = 1/4\,V\,\frac{(2a + b + c)}{a + b + c + d}$$
$$Vp = 1/4\,V\frac{(2a + b + c)}{n}$$
$$Vp = 1/4n^2\,V\,(2a + b + c)$$
$$Vp = 1/4n^2\,(4Va + Vb + Vc)$$

being $Vb = \frac{b(n-b)}{n}$

The same is true for Vc and Vd. This is the 12th theorem.

For coupling, the formulae are:

$$2p(a + b + c + d) - (b + c + 2d) = 0 \quad (V)$$

or

$$p = \frac{b + c + 2d}{2(a + b + c + d)} \quad (VI) = (V)$$

Table 3 shows the analysis results of original data from Langham (Genetics 25:88-107, 1940). From the first column, chromosome number; genetic composition; values in cM of p calculated for a 9:3:3:1 inheritance, including the allometric effects alpha and beta; values in cM resulting from the analysis by the new model proposed in this report, for duplicated pairs of alleles in the same

Table 3.

Chromosome	Genotype	cM (conventional)	cM (new model)	s	Genotype	cM (Σ differences)	s
1	*fl* pd	52	20.6	2.1	*fl*	86	0
	fl tr	85.2	22.8	2.1	tr11	110.3	1.5
	bm2 pd	128	23	2.1	pd11	110.4	1.5
	bm2 tr	49	25.7	2.4	*bm2*	161	0
2	*lg1* pd	70	22.4	1.9	*lg1*	11	0
	lg1 tr	59.9	26.7	1.9	pd2	20	1.4
	gl2 pd	81.9	24.4	1.9	tr2	21	1.4
	gl2 tr	66.4	29.1	1.9	*gl2*	30	0
	b pd				*v4*	83	0
	b tr	71.1			pd12	104	1.9
					tr12	107	2.7
	v4 pd	86.3	20.7	1.9			
	v4 tr	83	23.9	1.9			
3	*lg2* pd	36.5	21	2.5	*lg2*	101	0
	lg2 tr	59.3	25.1	2.5	tr3	124	1.7
	al pd	59.9	13.7	2.5	pd3	130	1.7
	al tr	58.2	27.9	2.5	*al*	149	0
4	*ts5* pd	56.1	32.6	2.7	*ts5*	53	0
	ts5 tr	45.6	50.4	3.3	*su1*	63	0
	su1 pd		27.5	3.4	pd14	87	3.4
	su1 tr		16	3.4	tr14	90	4.8
5	*pr* pd	99.2	30.1	3.1	*pr*	67	0
	pr tr	58.2	38	3.1	pd15	97	3.1
					tr15	105	4.4
6	*y1* pd		24.1	1.9	*y1*	17	0
	y1 tr	70.6	28.5	1.9	pd6	42	1.4
	py pd	54.9	26.6	1.9	tr6	47	1.2
	py tr	34.3	21.7	1.8	*py*	69	0

chromosome, as tr2 pd2 and tr12 pd12, affecting the same character; the standard errors; chromosome map distances (including markers used), obtained by the sum or differences in cM, intrapolating for chromosome 1 (pd2 and tr2) and for chromosomes 3 and 6. By difference, extrapolating for pd12 tr12 in chromosome 2 and in chromosomes 4 and 5. Note the overall strong linkage between pd's and tr's. The values in the 7th column were calculated with data from the 3rd column.

To check it, with Langham's data, *lg* pd, a = 160, b = 38, c = 50 and d = 9. Applying the models (I) and (III) we get the same result p = 0.2217899. As there is no available reference, we demonstrated the solutions in two ways, to cross-check it.

The results presented in Table 3 were calculated as follows:

1. The distances to each marker were calculated for pd and tr.
2. The p values were then converted to cM using Haldane's formula.
3. The distances between pd and tr were obtained by differences of the cM values to the common marker.

For chromosome 1 the values obtained were interpolated within the *fl bm2* distance as being pd11 at 103.4 and tr11 at 110.3, since probably there is a krn1 in position 35 as reported by Miranda et al. (MNL61, 1987).

For chromosome 2, interpolating within *lg2 gl2* we get pd2 at 20 and tr2 at 22 since we had already an estimation of krn2 in position 22 (Miranda et al., MNL64, 1990). The values with *v4* were used to get the sequence pd12 at 104 and tr12 at 107.

For chromosome 3, interpolating we get tr3 at 124 and pd3 at 130; this agrees with Miranda et al. (MNL61, 1987) and with K3L at 115 from Dempsey (1975).

For chromosome 4, extrapolating we get pd14 at 87 and tr14 at 90 because probably there is already a tr4 as suggested by Miranda et al. (MNL59, 1985). Miranda et al. (MNL63, 1989) proposed the sequence pd4 at 3, krn4 at 7 and *ts5* at 53. The same authors, using the original Galinat's data (MNL49, 1975), measured ri *su* = 29, e and ph ri = 12. With *su* now in position 47 as a basis, this would lead to ph at 6 and ri at 15.

In chromosome 5 we get pd5 at 97 and tr5 at 105, probably the same as proposed by Miranda et al. (MNL61, 1987) as krn5 at 125.

In chromosome 6, interpolating we get tr6 at 42 and pd6 at 47, probably identical or one of a string reported by Miranda et al. (MNL61, 1987) as being in position 35. See the accompanying article putting K6L, with Dempsey's data, at position 41.

We can see that now the distance pd tr varies from 0.1 to 8.0 with a mean of 5, which demonstrates a much more biologically minded model.

In chromosome 7 krn7 at 64 is postulated by Miranda et al. (MNL61, 1987).

In chromosome 8, Miranda et al. (MNL58, 1984) reported results with translocations at 8L.09; 9S.16; 9S.31, studying flint characteristics in crosses with the Cateto line C2. Dent characteristic linkage was detected in individual grains as a dimple in the crown, as those exerted in baby cheeks in the act of smiling. As shown in the same report, as krn, fas and flt are linked together, another pair, tr and pd, is demonstrated in a distal position to the latter marker.

In chromosome 9, the same authors found the sequence T - 2 - wx - 4 - flt9 - 13 - krn9 with the inversion at 9S.7; 9L.9. Nothing more can be inferred since there are knob sites in both arms of the chromosome.

In chromosome 10 the order and direction of T - 10 - *wx* - 32 - flt10 is given for a T at 9S.13; 10S.40 (Miranda et al., MNL61, 1987). The position is confirmed by the presence of knob sites only in the end of the long arm.

Of particular interest to this work is that from Kato (Evolut. Biol. 17:219-253, 1984). On knobs with restricted distribution he states:

a) Mean Pacific Migration Path. This migration path is illustrated by the distribution pattern shown by the medium and large knobs in the short arm of chromosome 4. This knob appears to be concentrated in collections of the races Zapalote Chico and Zapalote Grande in Oaxaca-Chiapas.

b) Northern Migration Path from Central Mexico. This migration path is exemplified by the distribution of the medium knob at the 6L1 position. The center of distribution of this knob is located in the Central Highlands of Mexico (Mesa Central) and is found in races such as Conico, Palomero Toluqueno, Arrocillo Amarillo and Chalqueno.

c) Pepitilla Migration Path. This path is illustrated by the distribution of the large knob at the 6L3 position. It is characteristic of Pepitilla and Maiz Ancho.

d) Mexican East Coast Migration Path. The specific knob that characterizes this migration path is the distribution of the large knob at the 9L2 position. It is characteristic of Tuxpeno.

e) Migration Path from Highland Guatemala. The highland maize, typically represented by the races San Marceno, Serrano, Quicheno, Negro and Salpor, is characterized by the predominance of a combination of small knobbed and knobless positions. There is one specific small knob at the 10L1 position.

f) Maize from South America is characterized by a typical knob distribution in chromosomes 6 and 7.

The author cites Galinat, who suggested that knobs may be linked to gene complexes controlling the essential taxonomic traits differentiating maize from teosinte. Beadle (Maize Breeding and Genetics, ed. D. B. Walden, 1978) states that teosinte pops, and we remember that most primitive maize, including archaeological maizes, are popcorns.

In conclusion, the contention of Galinat (Adv. Agron. 47:203-231, 1992) that there is only one tr in chromosome 2 and only one pd in chromosome 3 seems untenable. Pego (Genetic Potential of Portuguese Maize with Abnormal Ear Shape, Ph.D. Thesis, Iowa State Univ., 1982) cites a report about P40, a fasciated popcorn inbred with 40 kernel rows in the ear. It seems reasonable that, since a tr pd pair accounts for 4 rows, there is a necessity of at least 10 pairs to achieve that row number.

Part of these conclusions are corroborated by Doebley (Trends Genet. 8:302-307, 1992). See also that in the recent chromosome 4 working map, published in the Maize Genetics Cooperation Newsletter (1992), the genetic cartography *asr1* at 0; *rp4* at 5; ri1 at 8; *ga1* at 13; *adh2* at 27 and *su1* at 47 seems stimulating since it resembles the teosinte x maize affair. *ga1* is just another way to flare the limits and *asr1* and *adh2* could find excellent biological reasons to be in the same neighbourhood.

From teosinte to maize II

--Luiz Eugênio Coelho de Miranda, Luiz Torres de Miranda and Omar Vieira Villela

In the preceding report a biometric model mapping the case in which two pairs of alleles affect the same characters was applied to Langham's F2 data from crosses of teosinte to maize markers.

Tr and pd are duplicated in the same chromosome. In MNL61:29-31 data were presented for crosses of Portugues Fasciado with the standard translocations in IAC Maya, backcrossed to IAC Maya *wx*. With data of Table 1 in that note, we reanalyzed using the new model for the backcross case. The formulae for repulsion were applied to all data with krn and fas, see Miranda et al. (MNL64:35-36, 1990), p=(a+b)/n, q=(a+c)/n, and for coupling p=(c+d)/n, and q=(b+d)/n. The only case in which consistent results were obtained was for the translocation at 7L.63, for which the results in repulsion transformed into centimorgans are presented.

km17	53.7±5.9	T	9.6±2.8	km7
fas17	53.7±5.9	T	6.0±2.2	fas7
km17	60.2±6.4	*wx*	6.0±2.8	km7
fas17	60.2±6.4	*wx*	6.0±2.8	fas7

The T *wx* recombination of p=39.5 was excessively large and ignored in what follows. In MNL62:38-39, 1988, p was calculated as 1.39±0.49 with data of Anderson et al. (MNL39:106-109, 1965). Also the K7S is terminal in the position in Kato's knob map.

K7L was mapped in Miranda and Miranda (MNL61:34-35) in position 64 with Cateto Palha Roxa.

In MNL52:142, 1978, J. B. Beckett et al. present a cytogenetic map which shows one of the zones of interest, krn7 paralleling and showing position 64 corresponding to 7L.63 in the cytogenetic map. So we have plenty of points of reference to anchor our analysis results.

The data presented here are independent from those and lead to the steps indicated below.

The krn17 fas17 distances were gotten as a mean from T and *wx* markers. T to *wx* distance was gotten from the cited report. fas7 to krn7 was obtained as a single comparison, by difference.

fas7 to krn7 was gotten as the difference *wx* krn7 - *wx* fas7, giving the distances krn17 fas17 - 57±3.1 - T - 1.5±0.5 - *wx* - 6.0±1.6 - fas7 - 1.8±4.7 - krn7.

Anchoring it in the distal knob in position 7S.0 in Kato's cytogenetic knob map we get in a working map:

km17 fas17	T	*wx*	fas7 krn7
0	57	58.5	64.5 66.3

Only chromosomes 6, 8 and 10 have a string of knobs. The others have only one knob site in each arm. Each knob of a string must have come from a different domestication, and their distribution study will lead to the original cradles.

From teosinte to maize. III. Allometric genetics 12th theorem - a more involved three point test

--L. E. C. de Miranda and L. T. de Miranda

E. Dempsey (MNL45:58-59) presented the sequence, centromere - *y* - *pg11*. If p is the *y-pg11* distance and q the *pg11*-K distance we complete these data with the expectations:

Coded	Pedigree	Class frequency	Total	Expectation of regions
+ - - (0)	*Y pg11* k	12		
- + + (0)	*y Pg11* K	6	18	p(1-q) a
+ + + (1)	*Y Pg11* K	10		
- - - (1)	*y pg11* k	6	16	(1-p)(1-q) b
+ - + (2)	*Y pg11* K	3		
- + - (2)	*y Pg11* k	1	4	pq c
+ + - (3)	*Y Pg11* k	0	not found	
- - + (3)	*y pg11* K	0	not found	(1-p)q d

The plants with crossover phenotypes were purposely selected and even so the last two classes did not appear. So the linkages calculated with these data are stronger than if calculated with a non-skewed sample.

By the additive or Emerson method, which is the same as the maximum likelihood of Fisher, we get p=(b+c):n

p for *y-pg11*=57.9±8.0 and q for *pg*-K=10.5±5.0

By the product moment method, whose theory for backcrosses is demonstrated in MNL61:32, we get

p for *y-pg11*=57.0±8.0 and q for *pg11*-K=9.3±4.7

With the expectations shown above we can mount a new maximum likelihood model as:

$$L = 18\,[\ln p + \ln(1-q)] + 16[\ln(1-p) + \ln(1-q)] + 4(\ln p + \ln q)$$

Derivating in relation to q to calculate *pg11* to K distance and following through we have

$$\frac{\delta L}{\delta q} = -q(a + b + c + d) + c + d = 0$$

$$\text{and } q = (4 + 0) : 38 = 0.105 = 10.5\%$$

Making the same for p we get p = 57.9, the same as by the additive method.

The distance calculated here in the three models as about 57 or 58 is known to be 19. The value obtained is triple the real one. Thus dividing by 3 the results obtained by the product moment method we have 3.0. This would put K in position 41 within our 35-42 positions proposed for tr (krn) in our prior report. The work of Kato (Mass. Agric. Exp. Stn. Bull. 635, 1976) is most illuminating in understanding the process of conversion of wild teosinte to cultivated teosinte, maize.

In five different sympatric races of teosinte and corn, there were the same chromosome knob complexes, and the knobless genome is the "basic chromosome morphology". This is a result of domestication, knobs.

Even today men in the field talking around a bonfire keep tinkering with it. Beadle (Maize Breeding and Genetics, 1978) states that teosinte pops. This explains the polyphyletic origin of maize, in the beginning, all popcorns.

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A chromosome fragment that rescues lethal oil yellow genotypes

–Dick Brock and Tony Pryor

The *oil yellow* gene on the short arm of chromosome 10 of maize has both dominant (*Oy*) and recessive (*oy*) mutant alleles in addition to the normal green of the wildtype (+), and different genotypes give the following phenotypes:

+/+ ;	*+/oy*	-- green
Oy/+;	*oy/oy*	-- viable oil yellow
Oy/Oy ;	*Oy/oy*	-- lethal yellow

A mutant oil yellow variegated seedling was recovered after ^{60}Co γ-irradiation of wildtype pollen used to cross a plant heterozygous for the dominant *Oy* gene (*Oy/+*). The variegated phenotype was recovered in progeny crosses and analysed by test crossing to various oil yellow mutant stocks.

Cytogenetic studies have demonstrated that the variegated phenotype is invariably associated with the presence of a small or 'Mini' chromosome and two classes of variegated plants can be identified:

	Genotype	Phenotype
Type I	[*Oy/+*; Mini] [*oy/oy*; Mini]	oil yellow plus green stripes
Type II	[*Oy/Oy*; Mini] [*Oy/oy*; Mini]	yellow plus green and oil yellow stripes

The Mini divides regularly at PMC meiosis (cf. Maguire, Genome 29:744-777, 1987) but with somewhat more delayed separation relative to normal chromosome segregation. The Mini is transmitted to 5% of progeny via the egg and to 10% via pollen. Plants containing two Mini's have not been recovered. Plants with tassel sectors lacking the Mini (determined from tapetal mitoses and PMC meiosis) are observed, but this has not been shown to coincide with the green/yellow plant sectors that can occur in the mature tassel. The reason for plant sectoring is unknown but is possibly due to late division and/or delayed separation of the Mini chromosome at mitosis resulting in cell lineages with and without the Mini chromosome. It is assumed that the Mini represents part of the short arm of chromosome 10 and carries the wildtype (+) oil yellow gene and thus rescues the otherwise lethal *Oy/Oy* and *Oy/oy* genotypes. Plants carrying the Mini and homozygous *Oy/Oy* produce *Oy* pollen and when crossed onto females homozygous for the recessive *oy/oy* all the F1 plants are yellow lethal (*Oy/oy*) except for the 10% which inherit the Mini via the pollen. This genetic system could be used to select for maternal inheritance events.

CHESTNUT HILL, MASSACHUSETTS
Boston College

Mutagenic effect of anther culture per se

--Y. C. Ting

In the last few years, several hundred microspore-plants were obtained by anther culture in vitro. Among these plants, variations in gross morphology as well as in chromosome number and behavior were frequently found. For gross morphology, changes such as leaf chlorophyll content, plant height, leaf shape and flowering time were manifested. Leaf chlorophyll content had a tendency to change from dark green to different kinds of chlorophyll deficiency such as striate (*sr*), zebra (*zb*), yellow-stripe (*ys*), virescent (*v*), and japonica (*j*). However, in contrast to the frequent appearance of albino plants from anther culture of rice and wheat, albino plants of maize rarely occurred. Plant height tended to change from the normal parental height to short stature. Chromosome number varied from haploids, to diploids, polyploids and aneuploids. Since these abnormalities did not appear among the plants of the continuously selfed progenies of the parental plants, they must have originated de novo. More studies of the changes, presumably mutations, follow.

A mutation affecting meiotic chromosome behavior was found and it was designated *cfu* (chromosome fusion at meiosis). This phenomenon was observed in the microspore-plants of KH-13. In the last summer, the inheritance of this mutant was studied again. Transmission was confirmed by examining meiosis of selfed progeny plants. Another mutant, curling leaf (*cur*), is subject to further tests. It was demonstrated again that this mutant was controlled by a single recessive gene.

It was also found in the summer of 1991 that three inbred lines derived from anther culture of Dan-San 91 were resistant to lodging. In the last summer, these inbreds were grown along with their parental plants for testing. After a rain storm together with strong wind (40 miles per hour) many of the parental plants fell to the ground. All of the inbreds stood up without any damage. This suggests that by anther culture per se, valuable mutations such as resistance to lodging can be brought about spontaneously.

Effect of silver nitrate on the differentiation of callus lines

--Y. C. Ting

The recent literature has repeatedly reported that silver nitrate ($AgNO_3$) might increase the frequency of the differentiation of friable calli of maize and the other cereals. As a result of this, it led to the increase of somatic embryogenesis. The amount of silver nitrate employed in the reports ranged from 0.5 to 100μM. In order to make further experiments on this effect, two maize callus lines, 87-54 and SAN1, from anther culture in vitro, were chosen. The medium N6 with eight percent sucrose; 1mg/l kinetin and 0.5mg/l 2,4-D, was prepared. For each callus line, 15 dishes of medium were inoculated; five of them were controls without $AgNO_3$, five, with 50μM $AgNO_3$, and the other five, 100μM $AgNO_3$. On the average, each dish had 30 pieces of actively growing callus. They were kept in a temperature range of 65 to 75 F. Six weeks later, friable calli appeared on both callus lines grown on the medium with both 50 and 100μM $AgNO_3$. None was found for those on the control medium. Then the calli were subcultured on the same but freshly prepared medium. Two weeks after transfer, white and compact pro-embryos occurred in many of the calli for those on both 50 and 100μM $AgNO_3$ media. None were observed on the controls. By actual counting of the number of pro-embryos, it was concluded that medium with 50μM $AgNO_3$ was more effective in callus differentiation as well as pro-embryo induction than that with 100μM $AgNO_3$.

COLLEGE STATION, TEXAS
Texas A&M University

RFLP analysis of *opaque2* genotypes in segregating populations

--Srinavas R. Kata, Brian H. Taylor, A. J. Bockholt and J. D. Smith

The Texas A&M University Quality Protein Maize (QPM) breeding program has focused on the development of hard, corneous endosperm forms of elite inbreds that are homozygous *opaque2*. The general approach has been to select inbreds from open pollinated QPM (CIMMYT) and High Lysine (South Africa) populations, which had previously been selected for hard endosperm. These inbreds are used as non-recurrent parents for improvement of seed characteristics of elite lines by backcrossing.

Marker assisted selection procedures are particularly useful in breeding programs involving seed specific, recessive alleles such as *o2*. Under the assumption that a probe derived from the target gene would minimize recombination, we obtained a full length cDNA of the *O2* gene from M. Maddaloni (Bergamo, Italy), and developed an RFLP marker assay to identify polymorphisms tightly linked to the *o2* locus. Under lab conditions, the presence or absence of specific polymorphisms provided genotypic identification of juvenile plants.

To field test the procedures, Tx5855 was crossed to plants from four hard endosperm *o2/o2* populations (POP20, POP21, POP22 and POP23, provided by Dr. Hans Gevers) from South Africa and two CIMMYT QPM populations (POB67 and POB68). The F1 plants were selfed and backcrossed to both the parents, and the resultant F2 and BC1 populations were planted on Texas A&M University Research Farm. Individual seedlings were tagged and leaf tissue was collected from juvenile (4-5 weeks after planting) plants. The leaf samples were individually frozen in liq-

uid nitrogen and stored at -80 C. DNA was isolated from the plants using a CTAB extraction procedure, digested with *Hind*III, size separated on an agarose gel and blotted to polysulfone membrane (Biotrace; Gelman Sciences). The blots were probed and genotypes were identified for individual plants. To compare predicted genotype with the progeny seed phenotype, each plant was selfed and the seed phenotypes were determined using a light box. Genotypic analysis was done for 270 plants in the segregating populations. Then genotypes of individual plants were compared with progeny seed phenotypes. In all the cases where the seed phenotype was clear, the RFLP analysis matched the phenotype prediction. However, the opacity of *o2/o2* kernels varied considerably in different genetic backgrounds, and the visual identification of seed phenotypes was not reliable in some cases. Utilization of RFLP analysis in determining the *opaque2* genotypes was essential in these lines.

Probe testing of several lines (not used in field test), presumed to be *o2/o2* hard endosperm derivatives, revealed that the *o2* allele had been lost. This emphasizes the usefulness of the RFLP probe in tracking the *o2* allele during conversion of established lines of *o2/o2* and vitreous *o2* forms. The results of our field tests indicate that RFLP analyses correctly identified *opaque2* genotypes in segregating populations with 100% accuracy, and that this assay can be used for genotypic identification in different source materials.

Endosperm protein variation in maize "triads"

--Kuanhung R. Lin, A. J. Bockholt, Brian H. Taylor and J. D. Smith

The nutritional advantage conferred by the *o2* allele has been well documented, but deleterious agronomic characteristics associated with opaque grain have inhibited its general acceptance by producers. Most of these characteristics appear to result from the soft, floury endosperm typical of opaque kernels. Consequently, several groups in the U.S., Mexico and South Africa have taken different approaches toward solving this problem by selecting for hard endosperm forms of *o2/o2* lines.

Brian Larkins and his associates have reported that QPM populations developed by CIMMYT have high levels of γ-zein (27kD), and their investigations suggest that γ-zein levels may be more important than total endosperm protein in determining endosperm hardness. By itself, though, an increase in zeins is counter productive, since it reduces the percentage of lysine in endosperm protein.

We have analyzed a series of inbred "triads" to investigate the effects selection for hard endosperm may have on expression of the various solubility fractions of endosperm proteins. A triad represents 3 forms of an inbred line: normal, *o2* and QPM. The *o2* forms were derived by backcrossing W64A *O2* (non-recurrent parent) to the standard inbreds at least 4 times. QPM forms were derived by selecting inbreds from CIMMYT QPM *o2* populations previously selected for hard endosperm, and backcrossing the QPM inbreds (non-recurrent parent) to the *o2* inbreds of each triad 2-4 times with selection for hard endosperm. To the best of our knowledge, the original CIMMYT lines also carried the W64A *o2* allele. Although the triads are far from being isogenic, genetic variation among the forms of a triad should be significantly reduced for genes that are not associated with *o2* or regions related to hard endosperm expression.

Table 1 presents data related to total endosperm protein, protein extracted for albumin, globulin, glutelin and zein solubility fractions, and lysine levels for the B73, Mo17, T224 and T232 triads.

Although variation in total protein and solubility fraction levels was observed both within and among triads, the low levels of albumin and globulin fractions suggest that they have relatively small effects, if any, on endosperm hardness and lysine content. Thus, if non-zein proteins actually contribute to endosperm hardness, it seems probable that this contribution stems from enhanced expression of glutelin proteins.

Table 1. Endosperm protein distribution and lysine levels in inbred triads. Total protein levels are presented as mg prot/g dry weight of endosperm. Solubility fractions are given as % total protein. Lysine levels are given as mole % of total protein.

Triad	Total protein	Albumin	Globulin	Glutelin	Zein	Lysine
B73	85.2	2.5	2.2	37.1	59.6	1.53
B73 *o2*	72.7	8.7	4.0	46.5	46.5	3.20
B73 QPM	81.7	3.3	3.1	44.9	48.7	2.69
Mo17	96.5	1.9	1.7	35.3	61.0	1.29
Mo17 *o2*	85.4	2.9	3.3	45.3	48.6	2.56
Mo17 QPM	98.2	2.9	2.7	48.0	46.4	3.04
T224	84.7	3.2	2.5	29.2	65.2	1.87
T224 *o2*	82.6	4.6	4.2	41.6	49.5	3.86
T224 QPM	94.8	3.0	2.5	39.3	55.2	2.17
T232	82.4	2.5	2.3	35.3	60.0	1.07
T232 *o2*	73.0	3.0	2.6	47.7	46.6	2.27
T232 QPM	81.7	3.0	3.2	46.3	47.5	2.19

Samples of protein for each solubility class were run on 8-25% SDS-polyacrylamide minigels using a Phast System gel electrophoresis unit (Pharmacia LKB) and staining with Coomassie blue. Band intensity, area and molecular weight were measured with a high resolution laser densitometer using Gelscan software (Pharmacia LKB). Relative intensities of selected zein and glutelin bands determined by this method are shown in Table 2.

Table 2. Relative intensities of selected zein and glutelin bands. Minor glutelin bands are not shown. The indicated intensities were measured by laser densitometer scans of Coomassie blue stained protein gels.

	Zein		Glutelin		
Inbred	22kD	27kD	37kD	45kD	75kD
Mo17	0.37	0.35	0.26	0.14	0.33
Mo17 *o2*	0.19	0.29	0.45	0.20	0.35
Mo17 QPM	0.06	0.43	0.48	0.45	0.40
T224	0.39	0.31	0.28	0.20	0.18
T224 *o2*	0.09	0.21	0.45	0.33	0.32
T224 QPM	0.24	0.45	0.44	0.33	0.52
B73	0.25	0.23	0.30	0.15	0.25
B73 *o2*	0.11	0.18	0.46	0.16	0.47
B73 QPM	0.14	0.25	0.55	0.21	0.48
T232	0.35	0.38	0.32	0.18	0.19
T232 *o2*	0.18	0.25	0.44	0.26	0.30
T232 QPM	0.20	0.30	0.45	0.28	0.32

The intensity of the 22kD zein band in each line reflects the amount of α-zein present, while that of the 27kD bands reflects the amount of γ-zein. No 27kD band was detected in the glutelin fraction, indicating that the extraction method used efficiently removed all the γ-zein from the glutelin fraction. Both the Mo17 and T224 QPM lines showed substantially increased levels of γ-zein, while the B73 and T232 QPM lines had only a slight increase, suggesting that in these lines the hard endosperm phenotype does not result from an increase in γ-zein. All of the triads showed higher levels of 37kD, 45kD and 75kD glutelin bands in the *o2* and QPM forms relative to the standard inbred. However, B73 and Mo17 QPM lines had sharply higher intensities of the 37kD and 45kD bands, respectively. These elevated bands may reflect the presence of specific QPM modifier genes in these lines, and might be useful as phenotypic markers for RFLP tagging.

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Further characterization of *En/Spm* transposition in tobacco

--Guillermo H. Cardon, Monika Frey, Heinz Saedler and Alfons Gierl

We have determined the frequency of germinal excision and reintegration, and the frequency of linked transposition of the maize transposable element *En/Spm* in an artificial transgenic tobacco system (Frey et al., EMBO J. 9:4037-4044). In this transposition system, TnpA and TnpD, the two *En*-encoded transacting factors necessary to promote excision of an *I/dSpm* receptor element, are expressed from their cDNAs fused to the CaMV 35S promoter. Two different receptor elements were tested: 1 2.2kb native *dSpm* (*dSpm* standard or *dSpm-S*) and a 4.2kb artificial receptor which carries a *DHFR* marker gene conferring resistance to methotrexate (*dSpm-DHFR*). Excision and reinsertion of these receptors take place with similar characteristics as in the homologous host maize. Excision reporter constructs were made in which the *dSpm* is inserted in the 5' untranslated leader of the *bar* gene, blocking its expression. Germinal revertants could therefore be isolated by seed germination on L-PPT containing medium. *dSpm* excision leading to the generation of *bar* expressing gametes (germinal excision) was measured in three independent single copy transformants of each *bar*-based excision reporter construct. The average frequency of germinal excision (female germline) was 10.1% for *dSpm-S* and 1.3% for *dSpm-DHFR*. The frequency of germinal excision transmitted by pollen seems to be similar. Southern analysis of *dSpm-S* germinal revertants showed that *dSpm-S* reintegrates with high frequency (up to 90%). Generally, several independent transposition events were detected within a single seed capsule. The *bar* excision reporter construct carrying the marked receptor *dSpm-DHFR* allows detection of reinsertion after germinal excision by plating seed on culture medium containing L-PPT and methotrexate. Even though *dSpm-DHFR* seems to undergo loss more frequently than *dSpm-S*, it proved to be a valuable tool for the selection of revertants carrying a transposed element. *dSpm-DHFR* was also instrumental for the estimation of the fraction of transposed elements which reinserted in the proximity of the donor site. The study of the segregation of the L-PPTT^R and Mtx^R markers (empty donor site and transposed *dSpm-DHFR*, respectively) in outcross progeny of 18 independent germinal revertants showed that in tobacco about 44% of the reinsertions take place into linked chromosomal locations. In maize, up to 60% of the transpositions are into linked sites on the same chromosome (P. A. Peterson, pers. comm.). Therefore, similarly to maize, the probability of tagging a gene will be increased if the receptor is inserted at a linked location. All parameters analyzed indicate that this *En/Spm* three component tagging system has the potential of generating insertion mutants. If the components of the system are kept at low copy, the molecular characterization of the mutants and later isolation of the tagged genes should be straightforward.

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A morphological analysis of the *liguleless2-2757* mutant, a new allele at the *lg2* locus

– Katrina Snyder and R. Bertrand-Garcia

In normal maize leaves, the ligule is a landmark structure delineating the blade from the sheath. The ligule is initiated as a result of periclinal divisions (it projects out from the plane of the leaf) and traverses the entire width.

The recessive *liguleless2* mutation was first described by Brink (J. Hered. 24:325, 1933). This spontaneous mutation removes ligule from the lower leaves of the plant and produces only partial ligules in the upper leaves.

We have characterized the phenotype of a new *lg2* allele recovered in a Mutator transposon background. This new *lg2* allele (*liguleless2-2757*) was recovered from a field of heterozygotes (*Lg2/lg2*) in a Mutator background. The new allele was confirmed by Southern analysis and allelism was determined by test crossing to the spontaneous *lg2* mutant (M. Freeling lab unpublished data). The mutant allele removes the ligule from the first two leaves and retards its development in subsequent leaves. When the ligule first appears it is fragmented and only present on the edges of the leaf. In later leaves the ligule progressively becomes more complete but never fully develops. Often the ligule is present in discontinuous segments progressing diagonally down the leaf. Even in the uppermost leaves, where the ligule is almost complete, the ligule is never joined across the mid vein and is often displaced along the leaf.

We have examined the ligules of both wildtype and *lg2-2757* siblings in a B73 background. Leaves 1, 2, 5, 7 and 12 were removed during various stages of leaf development and examined using SEM. Our results show that ligule development is arrested just after the onset of periclinal division in leaves 1 and 2. In addition it appears that blade cells in these leaves are also arrested before fully forming (Figure 1). These blade-like cells are elongated with only slight crenulations and appear to be in an intermediate state of development (between blade and sheath).

Leaves 5 and 7 show a progressive increase in the cellular organization of the blade-sheath boundary, however, these leaves

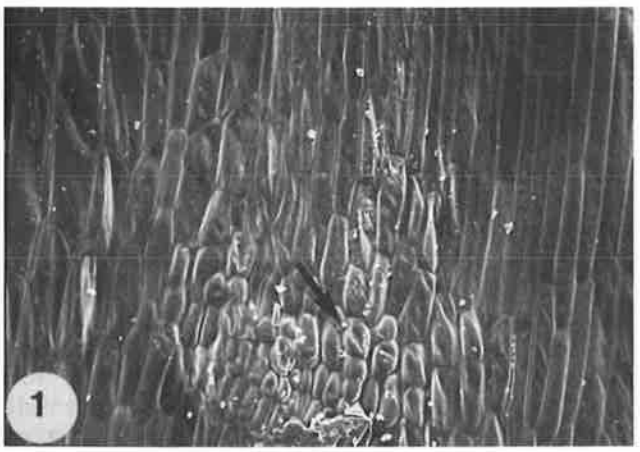

Figure 1. Shows the ligular region of a mature second leaf from a *lg2-2757* homozygote. The arrow shows cells that have been arrested during the onset of periclinal division. Cells surrounding this region are sheath-like in appearance. (190X)

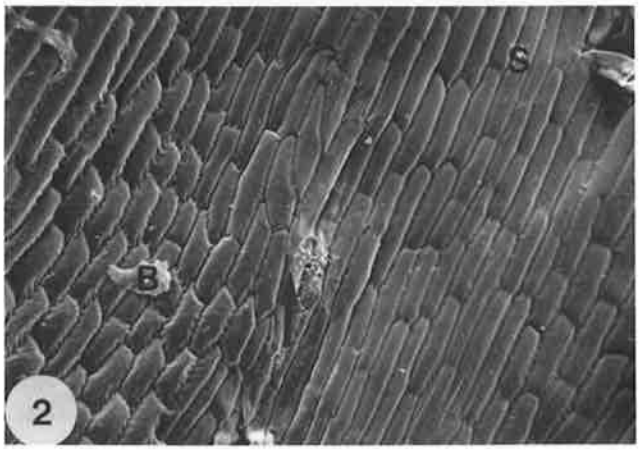

Figure 2. Shows the ligular region of a mature seventh leaf from a *lg2-2757* homozygote. The arrow indicates a transition zone between blade cells (B) and sheath cells (S). This sheath tissue is invading the blade, in a region that would normally give rise to ligule. (260X)

never show a distinct blade and sheath region. Further, these leaves have projections of sheath invading the normal blade tissue (Figure 2).

In the ligular region of leaf 12 the cells are not clearly separated into distinct blade and sheath regions as they are in the wildtype. There are distinct hair and blade-like cells that develop below the ligule. In addition the ligule on these leaves is not entirely normal and has hairs developing on the ligule surface (Figure 3).

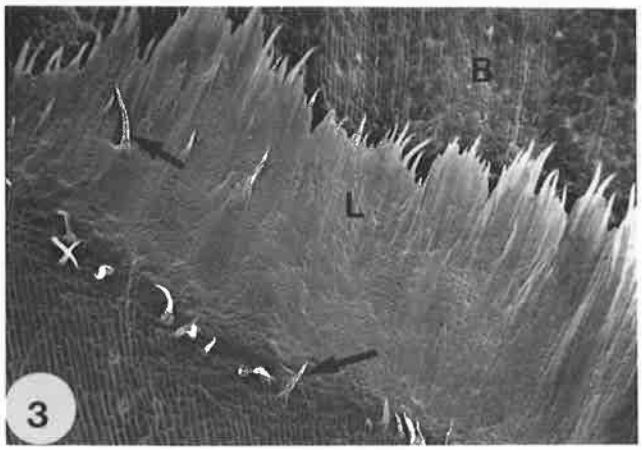

Figure 3. A partially formed ligule (L) on a mature twelfth leaf of a *lg2-2757* homozygote. The arrows show hairs developing both beneath the ligule and on the ligule itself. Those cells producing hair at the base of the ligule appear blade-like (showing crenulations along the margin and a cuboidal shape). (34X)

These results suggest that the *lg2-2757* mutant causes the leaf to lose the ability to establish a normal blade-sheath boundary. The transition from blade to sheath is disrupted both temporally and spatially. These results suggest that the wildtype *Lg2* gene product may play a key role not only in the formation of the ligule but also in setting up the proper boundaries between blade and sheath.

The degree of disorganization may result in fewer cells being able to respond to the ligule signal or a disruption in the signal itself. Ligule development is initiated, even in the lower most leaves, suggesting that a few cells in the first leaves are competent to receive the signal for ligule formation, however, most are not. These smaller leaves mature faster and may have a smaller window in which to see the ligule signal. Later leaves, that take longer to mature may have cells that remain competent longer and therefore (1) have more time to set up a boundary and set up cellular order, and (2) have more cells that remain competent to receive the ligule signal. This would explain why the later leaves show more ligule formation.

We are unsure whether this cellular disorder is caused by temporal factors, spatial factors or both. We are exploring these possibilities by placing the *lg2-2757* allele in various time-to-flowering backgrounds. Preliminary results suggest that there is a difference in both the amount of ligule formed, and the leaves in which it is formed, depending on the plant's time-to-flowering.

Ligule development occurs simultaneously at the normal sheath-blade boundary and in the prong of the *Hsf1-O* mutant

-- Jessica Saberman and R. Bertrand-Garcia

The dominant *Hsf1-O* (Hairy-sheath frayed) mutant of maize has been shown to retard the transition of juvenile cells to adult. One of the most obvious *Hsf1-O* phenotypes is the transformation of leaf blade cells into sheath, resulting in prong-like projections of sheath tissue along the edges of the blade (Bertrand-Garcia and Freeling, Am. J. Bot. 78:747, 1991).

In normal maize leaf development, as basipetal cell differentiation along the leaf occurs, a ligule develops perpendicular to the plane of the leaf separating blade tissue from sheath. Likewise, prongs may have their own ligule growth, at the boundary between sheath and blade, so that a single leaf will have more than one ligule. The development of the prong is spatially separate from that of the normal blade-sheath boundary. This spatial separation of ligular forming regions in *Hsf1-O* mutants presents an excellent model in which to study ligule development and to further understand the nature of the proposed signal for the initiation of ligule growth (Becraft and Freeling, Plant Cell 3:801, 1991).

Plants heterozygous for *Hsf1-O* in a B73 inbred background were observed for prong formation. These plants show the development of prongs, as early as leaf numbers 5-7. In young seedlings, prong initiation can be seen occurring distally, within 1.0cm of the normal sheath-blade boundary. Leaves 5-7 showing one or more prongs were examined under a dissecting microscope to determine the approximate state of prong differentiation. Twenty prong specimens in the earliest stages of development (1.0cm from the sheath-blade ligule) were removed from the leaf along with the normal ligular region. These specimens were fixed, sputter coated with gold and examined by scanning electron microscopy.

Comparison of ligule initiation at the normal sheath-blade boundary and within the prong of the same leaf shows the simultaneous initiation of the ligule at both sites (Figs. 1-4). This finding would support a model of a single ligule signal that originates at the tip of the leaf and diffuses basipetally. Only cells that are competent (at the appropriate developmental stage) to receive and respond to the signal then proceed on to initiate ligule development. If more than one signal existed or if the ligule signal was continuously produced we would expect cells in the prong region to respond first and for ligules in the prong region to be more developed. These results further suggest that cells in the prong, although chronologically older than those in the normal blade-sheath region, are retarded in their development and remain competent to receive the ligule signal. This model affirms previous investiga-

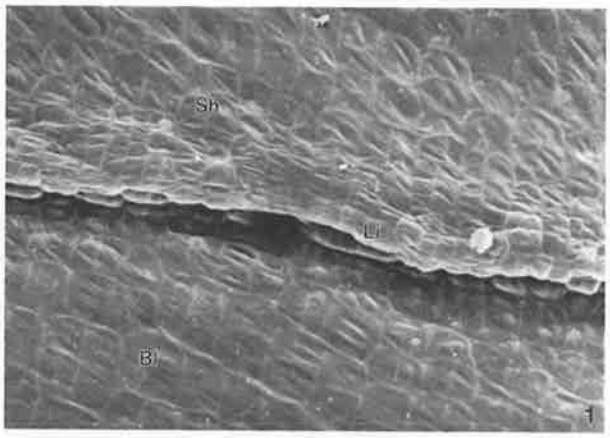

Figure 1. Early ligule (Ll) development at the normal boundary between sheath (Sh) and blade (Bl) on *Hsf1-O* plant, leaf number six. (x460)

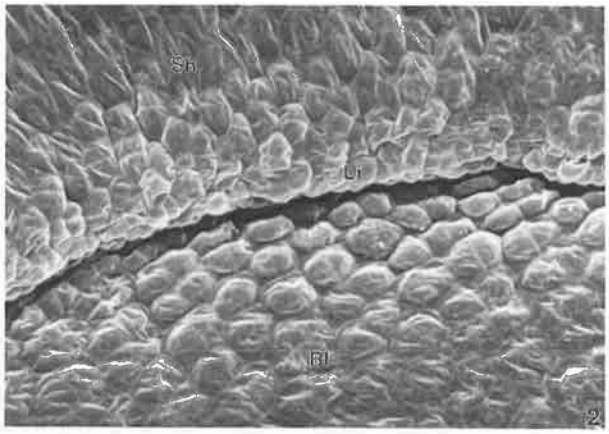

Figure 2. Ligule development at prong area from leaf shown in Figure 1. (x460)

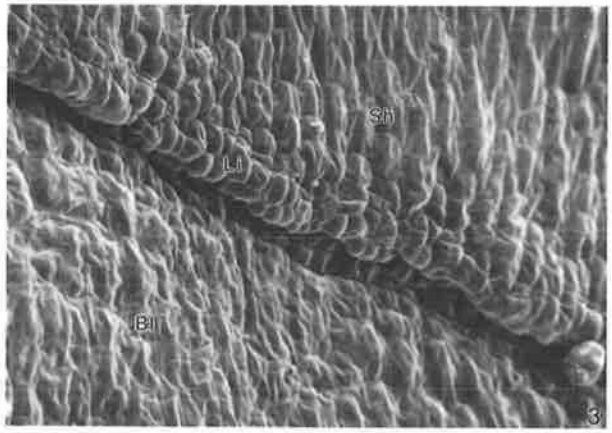

Figure 3. Example of ligule development at the normal sheath-blade boundary in *Hsf1-O*. Figure shows leaf number twelve. (x460)

tions which suggested that *Hsf1-O* is a heterochronic mutant.

Although this study concludes that normal and prong ligule development are initiated simultaneously, it is interesting to note that prong ligules do not always develop fully. It is more likely to observe several stages of maturation along the prong, the most advanced at the center of the transformed region, where the veins of the leaf are seen to break their parallel lines and bend into the prong (Fig. 5). This appears to be the point of origin for both the prong and ligule development along the rim of the blade.

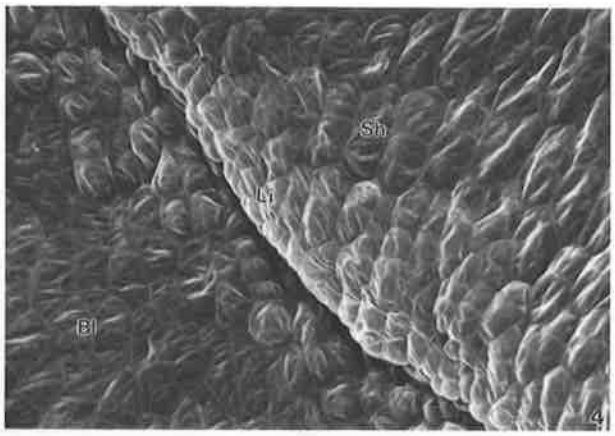

Figure 4. Prong region with ligule development, from leaf shown in Figure 3. (x460)

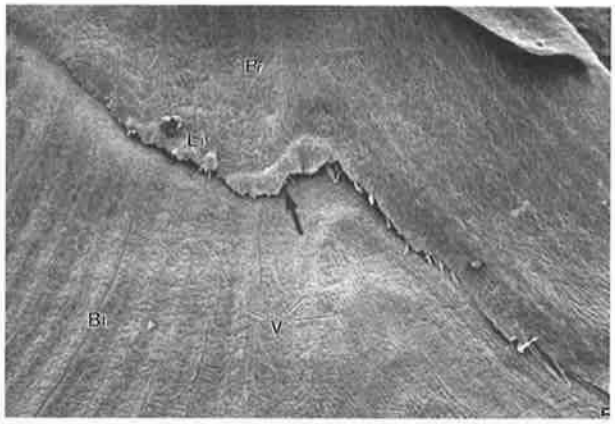

Figure 5. Prong (Pr) growth along the edge of the blade. Note how the typically perpendicular veins (V) "bend" inwards from the blade (Bl) towards the center of the ligule (Ll), where it is most developed. This region (arrow) is the point of ligule initiation in the prong.

The sporadic occurrence of ligule in some prongs and not in others, and the variable maturation of the ligule in prongs, may occur (1) because of temporal separation, the competent cells in the prong may be reaching a state in which they can no longer respond normally to the signal, or (2) because fewer cells in the region can respond. Further studies using this mutant will address questions regarding the level of determination in those cells competent to receive the ligule signal.

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Diagnosing map order of genes with RFLP markers

--Ed Coe

Following are two simple reference tables we use to simplify diagnosis of order for dominant/recessive loci with codominants in F2 progenies. The first table considers a gene locus between two codominants, showing the scoring classes (A, homozygous for the A allele; B, homozygous for the B allele; D, dominant; H, heterozygous; R, recessive), possible events (p, parental; d, double; 1, single in region 1; 2, single in region 2) , the minimum number of crossovers that will yield that class, and alternative order diagnoses suggested by occurrence of classes that are expected to be rare. The F1 from which these F2 products were derived is ADA/BRB.

Class	Events	Min. co	Order Diagnosis
ADA	p,d	0	
ADH	1,2,2/d	1	
ADB	2/2,1/2	2	
ARA	d/d	4	>AAR or RAA
ARH	1/d	3	>AHR
ARB	1/1	2	
HDA	1,2,1/d	1	
HDH	p,1/2,2/2,1/1,d,d/d	0	
HDB	2,2/d,1/d	1	
HRA	2/d	3	>RHA
HRH	1/2,d	2	
HRB	1	1	
BDA	1/1,1/2	2	
BDH	1,1/d,2/d	1	
BDB	d,d/d	2	>BBD or DBB
BRA	2/2	2	
BRH	2	1	
BRB	p	0	

In the accompanying note on interval mapping, arrays of data are displayed with adjacent loci comparable to the above triples vertically in columns rather than horizontally.

The second table is for the alternative order in which the gene locus is adjacent to two codominants, for an F1 of AAD/BBR.

Class	Events	Min. co	Order Diagnosis
AAD	p,2	0	
AHD	1,d,2/d	1	
ABD	1/d,d/d	3	>ADH
AAR	2/2	2	
AHR	2/1	2	
ABR	1/1	2	
HAD	1,2/1,d	1	
HHD	p,2,2/2,1/1,1/d,d/d	0	
HBD	1/2,d,2/d	2	
HAR	2/d	3	>AHR
HHR	2,1/d	1	
HBR	1	1	
BAD	1/1,1/d	2	
BHD	1,1/2,2/d	1	
BBD	2,2/2	1	
BAR	d/d	4	>BRA
BHR	d	2	>BRH
BBR	p	0	

The logical extension of these considerations is to peruse the scores down a longer series of loci, anticipating that, for fairly short intervals, crossovers will result in extended chains of non-crossover constitution (e.g., AAAA, BBBB, BBRB, BBBR, HHHH, etc.) or presumed non-crossovers (e.g., AADA, AAAD, HHDH, HHHD, etc.), frequent one-crossover (e.g., AAAH, BBBH, BRBH, HHHR, etc.), some two-crossover (e.g., AAHR, AAAR, etc.), few double-crossover (e.g., BBHR, BBDB, AAHA, etc.), and rare double-double crossover (e.g., BBAR, etc.) events. These evaluations simply extend to codominance the long-familiar (i.e., Sturtevant) approaches to diagnosis of map order in testcrosses.

Interval mapping of NEP factors on chromosomes 1, 3, 4, 5, 6, 7, 8, and 9

--Ed Coe, Oscar Heredia, Susan Melia-Hancock and Shiaoman Chao

The placements of Naked Eye Polymorphisms at the *sr1, zb4, ts2, p1, as1, br1, f1, an1, gs1* and *bm2* loci with RFLP markers, shown by dotted connecting lines in the 1992 maps (refined this year after re-analysis), are supported by the linkage data presented in the accompanying tabulations. These are scoring data from 4 F2 families provided from the Stock Center collections by Earl Patterson, detailed in 10 segments. The scores, locus by locus and individual by individual, are identified by letters according to the conventions used in MAPMAKER, as follows: A (homozygous for the allele of parent A), B (homozygous for the allele of parent B), H (heterozygous), C (dominant from parent B), D (dominant from parent A) and - (dash; unscored). The volume of mapping data now developing in this laboratory and in others is very considerable. Data in such volume can be offered in hard copy tables such as this, but they will be best documented in the future in database form, where they can be accessed by any user for any desired purpose, and can be merged with other data to develop composite maps. We present the following as examples of systematically organized form for such data, with information about the approach we use in deriving and analyzing the tabulations.

The loci are first arranged in order according to placements on the UMC Core Map, or other RFLP maps and prior information, to derive the best available skeleton order. The array of scores is then sorted in order according to genotypes in the row of scores for the target locus (e.g., *sr1*) on a primary sort, and secondarily for 1 or 2 nearby loci. The sorted array facilitates scanning for matches and mismatches of adjacent loci (crossovers). If scanning indicates serious misplacement of a gene locus, we assess alternative placements and rearrange where needed. These steps yield the best-order diagnosis. The array is then analyzed by MAPMAKER to derive percents of recombination for closest markers, and to compare alternative orders mathematically by differences in LOD scores. Because data for dominant-recessive loci inherently are less robust than data for codominants, LOD score differences are often small, so a statistically based decision is not always possible. In such cases we use the conventional "fewest-

FAMILY 1

LOCUS	SCORES	PCT[1]
bn15.62	HABBBBHBBHHABBHBHBHHHAHHHHHAHHAABHHHAHBAHHHAHHAAHHHBBHHBBAHHHBHHAAHHHHBHAA	
umc164	HABBBBHBBAHABBHBHBHHHAHHHHAAHHAABBHHAHBHHHHAHAAAHHHBBHHBBAHHHBHHHAHHHHHAA	4.9
umc115	BBBBBBHBBB--BB-BBB-HHHHH-AHHH--HHHHH------AAB-AHHHHHAHHHHAHHHH-------HHHH-	26.2
sr1	BBBBBBBBBBBBBBBBBBDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDD	2.8
zb4	BBBBBBBBBBBBDDDDDDBBBBBBDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDDD	13.2
umc76	BB---B-HBBHAHBHA-BHBBBHHAAHHA-AHHHAHHHH--BAA--H------AHHHABHBHHHHAHHHHHH--	17.2
umc162	BBBBBBHBBBHABBHHHBHBBBBHAAHHAHHHHHHAHAHHHHHHAAHAHHHHHHAAHHHABHBHHBHAHHHHHBHH	7.5
umc11	BBBBBBHBBBHABBHHHBHBBBBHHAHHAHHHHHAHHHHHBHHAHBHHHHHHAAHHHABBBHHBHABHHBBBHB	7.8

[1]The percentage of recombination between the locus on that line and the one above it, derived with MAPMAKER.

Comments:

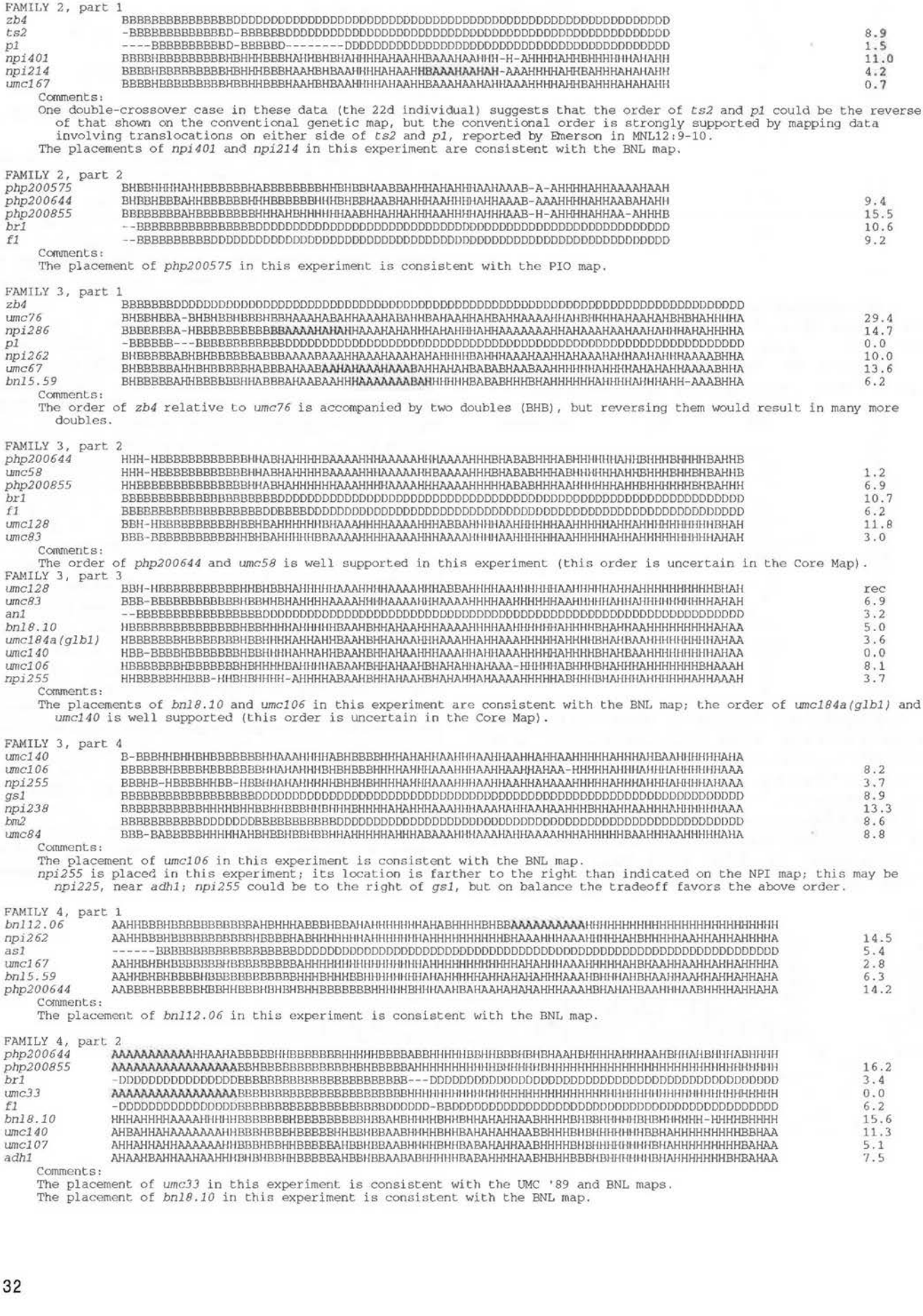
The order of *bn15.62* and *umc164* could be reversed; this order is also uncertain in the Core Map.

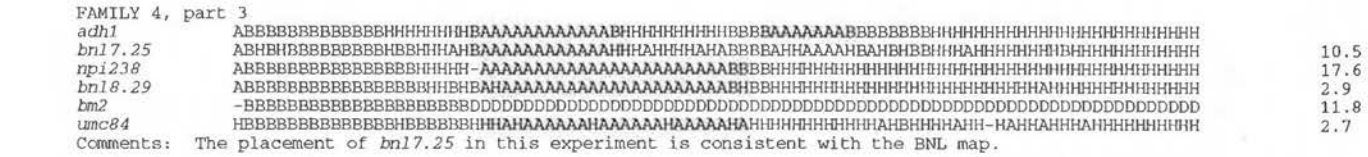
sr1 and *umc115* could be reversed only at the cost of a double exchange.

zb4 and *umc76* could not be reversed without reflecting several more double exchanges.

The placement of *umc162* in this experiment is consistent with that for the UMC '89 map.

The distance from *sr1* to *zb4* may be subject to reduction.


FAMILY 2, part 1
zb4
ts2
p1
npi401
npi214
umc167
8.9
1.5
11.0
4.2
0.7
Comments:
One double-crossover case in these data (the 22d individual) suggests that the order of ts2 and p1 could be the reverse of that shown on the conventional genetic map, but the conventional order is strongly supported by mapping data involving translocations on either side of ts2 and p1, reported by Emerson in MNL12:9-10.
The placements of npi401 and npi214 in this experiment are consistent with the BNL map.
FAMILY 2, part 2
php200575
php200644
php200855
br1
f1
9.4
15.5
10.6
9.2
Comments:
The placement of php200575 in this experiment is consistent with the PIO map.
FAMILY 3, part 1
zb4
umc76
npi286
p1
npi262
umc67
bnl5.59
29.4
14.7
0.0
10.0
13.6
6.2
Comments:
The order of zb4 relative to umc76 is accompanied by two doubles (BHB), but reversing them would result in many more doubles.
FAMILY 3, part 2
php200644
umc58
php200855
br1
f1
umc128
umc83
1.2
6.9
10.7
6.2
11.8
3.0
Comments:
The order of php200644 and umc58 is well supported in this experiment (this order is uncertain in the Core Map).
FAMILY 3, part 3
umc128
umc83
an1
bnl8.10
umc184a(glb1)
umc140
umc106
npi255
rec
6.9
3.2
5.0
3.6
0.0
8.1
3.7
Comments:
The placements of bnl8.10 and umc106 in this experiment are consistent with the BNL map; the order of umc184a(glb1) and umc140 is well supported (this order is uncertain in the Core Map).
FAMILY 3, part 4
umc140
umc106
npi255
gs1
npi238
bm2
umc84
8.2
3.7
8.9
13.3
8.6
8.8
Comments:
The placement of umc106 in this experiment is consistent with the BNL map.
npi255 is placed in this experiment; its location is farther to the right than indicated on the NPI map; this may be npi225, near adh1; npi255 could be to the right of gs1, but on balance the tradeoff favors the above order.
FAMILY 4, part 1
bnl12.06
npi262
as1
umc167
bnl5.59
php200644
14.5
5.4
2.8
6.3
14.2
Comments:
The placement of bnl12.06 in this experiment is consistent with the BNL map.
FAMILY 4, part 2
php200644
php200855
br1
umc33
f1
bnl8.10
umc140
umc107
adh1
16.2
3.4
0.0
6.2
15.6
11.3
5.1
7.5
Comments:
The placement of umc33 in this experiment is consistent with the UMC '89 and BNL maps.
The placement of bnl8.10 in this experiment is consistent with the BNL map.



doubles' criterion, and evaluate for chains of the same constitutions across neighboring loci.(i.e., anticipating that, across short distances, interference means that one crossover is not often followed by another in the same strand). We display up to 3 markers on each side of a target locus when available, to aid visualization of chains of constitutions. Comments are provided where appropriate, following each table. Order of the associated loci is emphasized more than distances in these analyses.

Similar diagnoses provide placements for NEPs on chromosome 3 (*g2, cr1, ys3, pm1, ig1, lg2, na1, a3, et1*), chromosome 4 (*ts5, su1, gl4, o1, tu1, gl3*), chromosome 5 (*a2, bm1, pr1, ys1*), chromosome 6 (*y1, pl1, wi1, pb4, sm1, py1*), chromosome 7 (*ra1, gl1, ij1*), chromosome 8 (*bif1, j1*), and chromosome 9 (*yg2, c1, sh1, wx1, d3, gl15, bk2, wc1, bm4, bf1*). The data will be available in the maizedb database.

Location of *Rld1*

--S. Chao and M. G. Neuffer

The dominant rolled leaf mutant (*Rld1*), for which two separate mutants were reported (MNL64:51), has been tested extensively for location using the full set of *wx* marked translocations. Both mutants *Rld*-1441* and *Rld*-1990* were tested against 23 translocations and the results were negative. From this we concluded that these mutants, which may be alleles, are located in some area of the genome not covered by the *wx1* T set used. The most logical next step was to test the ends of the chromosome arms using the RFLP technique since these are the only areas not covered.

Bulking analysis was used to look for RFLP markers linked to *Rld1*. F2 populations segregating for the *Rld*-1441* and *Rld*-1990* alleles were used. Three genotypes, *Rld1/Rld1*, *Rld1/+*, and *+/+*, can be visibly distinguished in each F2 population. The genomic DNAs of plants having either the *Rld1/Rld1* or *+/+* genotype were bulked in equal amounts, with each bulk containing DNA from 8 to 9 individual plants. RFLP markers from distal ends of 20 chromosome arms were used to screen the bulks. Probe *npi97* has a clearly detected polymorphism between *Rld1/Rld1* bulk and *+/+* bulk for both populations when the DNA is digested with *Hin*dIII. The polymorphism appears as a band present only in *Rld1/Rld1* bulk and not in *+/+* bulk. Probe *npi97* was then used to hybridize F2 individuals to determine the map distance. Since probe *npi97* detects duplicate loci on 9L and 1S (Burr, 1992), flanking markers on both were examined. The results showed that single locus probes *bnl5.62* and *umc157*, which map distal to *npi97* on 1S, have no linkage with the *Rld1* locus; however, linkage between *Rld1* and *npi209* was detected. Probe *npi209* also detects duplicate loci on 9L and 1S. However, no linkage was found by RFLPs detected by *npi209*, *umc157* and *bnl5.62*. Therefore, we conclude that the *Rld1* locus is located on 9L. The map distance is centromere - *npi209* - 12cM - *npi97* - 14cM - *Rld1*. This mapping is based on the results from a *Rld*-1441* mapping population with 116 individuals. Even though the polymorphism detected by *npi97* cannot be easily scored in the *Rld*-1990* population, the location for *Rld1* on 9L is confirmed. Two morphological traits, *Bf1* and *bm4*, are known to be distal to *npi97* on 9L (MNL 1992). The relative distances between *Rld1*, *Bf1*, and *bm4* will be investigated.

A similar mutant, *Ce1*, reported by Chourey and Mouli in 1975 (Genetics 77:s11) has also been tested by Chourey with negative results. A test for allelism of *Rld1* and *Ce1* is in progress.

New mutant designations

--M. G. Neuffer

ad2-2356A: This EMS-induced mutant has fused tassel and upper leaf parts. Leaf and floral tissues above the ear node tend to adhere, often growing together into single tissue. Unlike *ad1*, seedling and juvenile stages are normal. The first six leaves (those laid down in the embryo or supported by the endosperm) are completely normal, suggesting early maternal support provides normalcy. This mutant is designated *ad2*.

Trn1-1597: An EMS-induced dominant mutant, designated *Trn1*, expresses chlorotic and adherent leaf tissue on later leaves as they emerge, which become green and healthy after a few days of exposure to sunlight. This condition results in a smaller plant with torn leaves at flowering. Tests with 17 of 23 *wx* marked translocations available show linkage with all, but the tightest linkage is with those with breakpoints in the long arm of chromosome 9. Backcross data from a normal *wx* heterozygote give a map distance of 16±2.7cM from *wx*. This would place *Trn1* distal to *gl15* on 9L.

Location of *Wrp1*

--M. G. Neuffer

The dominant wrinkled plant mutant (*Wrp1*) discovered by Bockholt and Smith (MNL63:56) was tested for location to chromosome with *wx* marked translocations. Linkages were found on three translocations involving chromosome 2 but not for any of the others.

The data from chromosome 2 are listed below:

		Backcross classes					
Translocation	Breakpoint	Wx Wrp	Wx +	wx Wrp	wx +	Rec.	S.E.
T2-9c	2S.33	28	11	6	28	.23	.05
T2-9b	2L.22	27	9	5	35	.18	.05
T2-9d	2L.83	26	7	5	26	.19	.05

From these data it appears likely that *Wrp1* is located on the long arm of chromosome 2 between *wx1* T2-9b and T2-9d breakpoints.

Les-U957*, a disease lesion mimic mutant with clonal properties

--Elizabeth A. Lee, M. Gerry Neuffer and Edward H. Coe

Les-U957*, isolated from a Mutator screening project, is a dominant lesion mimic mutant whose expression ranges from small necrotic lesions (similar to *Les2*), arranged in a clonal fashion, to large necrotic sectors. Appearance of lesions begins at the 5 to 7-leaf stage, with lesion formation beginning at the tips of newly emerging leaves and progressing towards the base of the leaf. Lesion formation is characterized by small, watery spots that turn

into small necrotic lesions or light green sectors, which will eventually become completely necrotic. Revertant (completely green) sectors are also characteristic of this mutant. Direct exposure of the newly emerging leaf tissue to light appears to be necessary for lesion formation. Depending on genetic background, *Les*-U957* can be maintained as a homozygote. In some backgrounds, such as W23, heterozygous plants will senesce before anthesis. We are currently mapping *Les*-U957* using waxy translocation stocks and examining several environmental/physiological parameters influencing its expression.

CORVALLIS, OREGON
Oregon State University

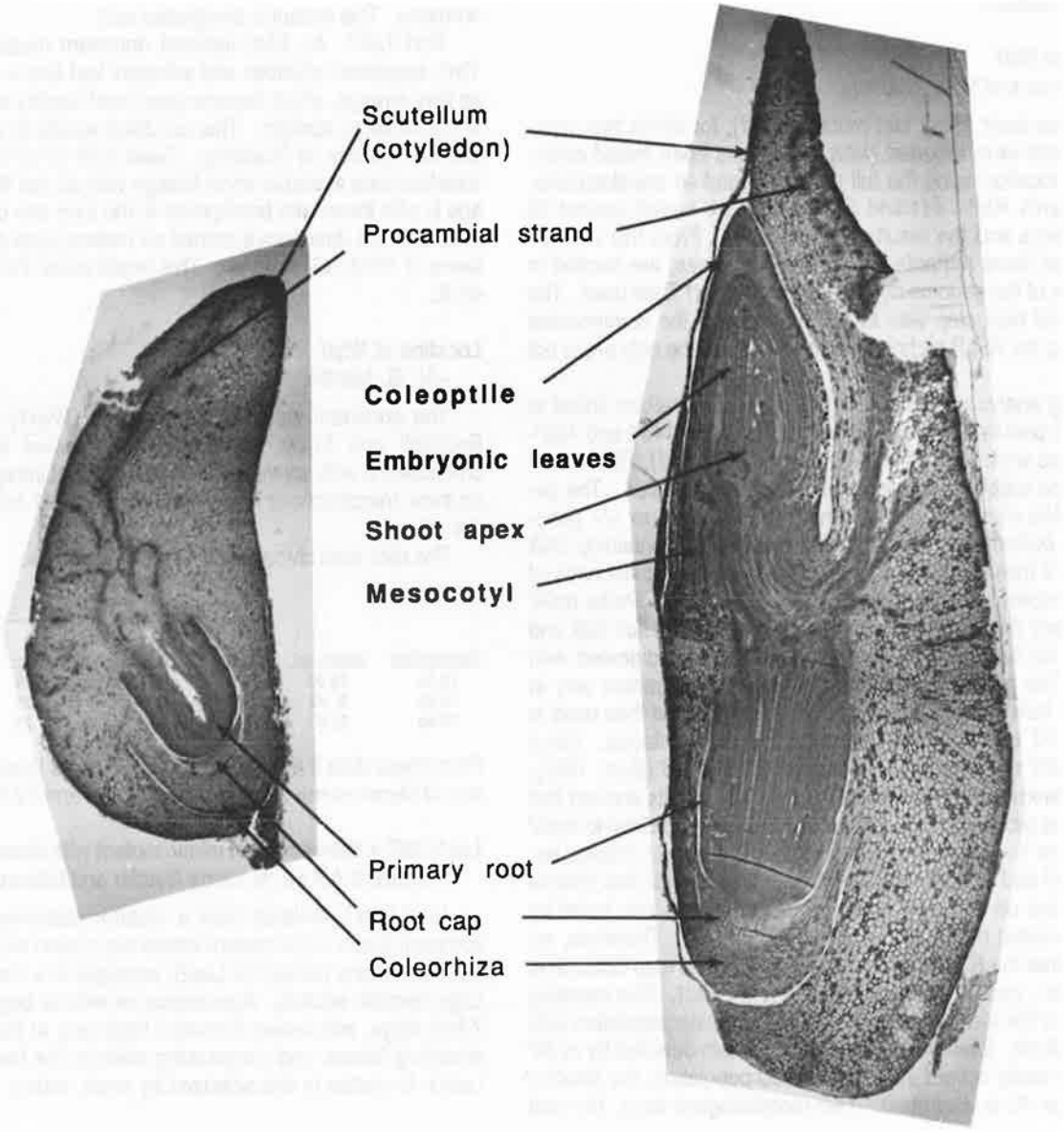
dks8, a mutation specifically eliminating shoot formation during embryogenesis

--John D. Sollinger and Carol Rivin

From a group of maize embryo mutants generated in a Mutator-family transposon line, we have identified one that specifically eliminates the development of the shoot pole during embryogenesis, while the rest of the embryo and the endosperm undergo normal morphogenesis and maturation. The mutant, termed *dks8* (defective kernel shootless-*Mu8*), is an excellent candidate for a pattern formation gene determining the earliest establishment of a functional shoot meristem. We are using descriptive and experimental embryology to characterize the role of this locus in development. Because *dks8* appears to have been created by insertion of a *Mu8* transposable element, we also have the opportunity to clone this gene and determine its product and expression.

We have examined the morphogenesis, growth and protein and lipid complement of homozygous *dks8* mutant kernels over the course of embryogenesis and seed maturation. We find that *dks8* kernels meet the criteria outlined by Jurgens et al. (1991) to distinguish mutants in pattern formation genes from other types of developmental abnormalities:

1. The morphological abnormality is specific. The morphological abnormalities of homozygous *dks8* seeds are confined to components of the shoot system. Serial sections through mature mutant embryos show that there is no trace of leaf primordia, no



coleoptile, no shoot vasculature, and no evidence for a shoot apical meristem. Neither do they reveal any sign of necrosis. The adaxial face of the scutellum is more circular than deltoid and is reduced in length by half, but we suspect that this is a secondary effect of the mutant condition, since the shoot normally acts both as a sink and as a source of developmental cues. Median longitudinal sections of mutant and wildtype embryos are shown in the accompanying figure.

2. The mutant phenotype deviates from wildtype at the time the pattern element is established. The shoot component can first be detected during normal embryogenesis with the formation of the coleoptilar ring. Using light microscopy and SEM, we examined developing *dks8* mutant embryos and found that this feature never forms. Thus the mutant deviates from wildtype shoot development from a very early point in the establishment of that pattern element, suggesting a primary block to the specification of a shoot apical meristem, or to the function of that group of cells.

3. The mutant block should not interfere with the completion of other development events. The *dks8* mutation has no effect on the capacity of the seed to complete other aspects of seed morphogenesis and differentiation. The mutant seeds are small, but otherwise morphologically normal and appropriately pigmented. They acquire a normal constellation of maturation proteins and storage lipids. They are desiccation tolerant and capable of germination of the primary root. Placed on a nutrient medium, this root will grow indefinitely, producing many root branches. However, there is no development of the secondary roots (products of the shoot system in maize). These features contrast sharply with the phenotype of other embryo mutants of maize, which are severely distorted in morphology, blocked from continued development, and unable to germinate.

Genetic studies of the *dks8* mutation show that it is a single gene recessive condition. The *dks8* kernels account for approximately 20% of the seeds on a self-fertilized ear, somewhat below the Mendelian expectation. We have found that the transmission of the *dks8* allele through the pollen is equal to that of the wildtype locus. We expect, therefore, that the reduced percentage of *dks8* homozygotes reflects a reduction in *dks8* transmission through the ovule, a proposition we are now directly testing. It may be that the gene acts in the female gametophyte as well as in the embryo. Complementation tests conducted in our lab and with the help of Guy Farish at North Dakota have thus far shown that the *dks8* mutation is not allelic to other mutants isolated by Bill Sheridan and his co-workers that have abnormalities in embryonic shoot formation.

The *dks8* mutation was isolated from an active Mutator transposon stock. We have conducted Southern blot analyses for co-segregation of various *Mu* elements with this mutant. In a survey of 70 individuals from several lineages and outcrosses, one *Mu8* band consistently co-segregates with the *dks8* allele. We are cloning the *Mu8*-containing fragment to map the *dks8* locus and with the hope that we can determine the product of this gene.

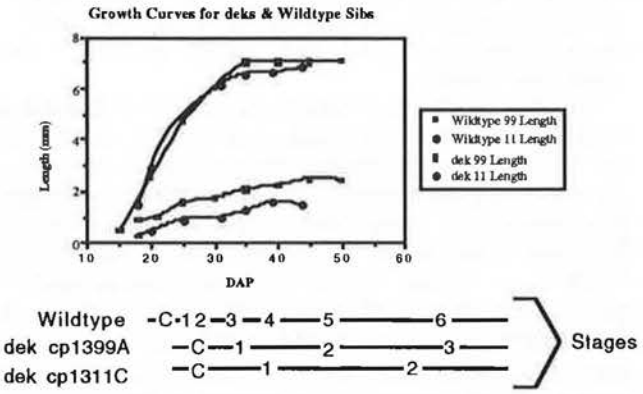
Developmental studies of vigorless *dek* mutants

--John D. Sollinger and Carol Rivin

Early in development maize embryos acquire the capacity to germinate or to mature. During maturation phase embryos accumulate nutritional reserves, gain tolerance to desiccation and become metabolically quiescent. One approach we have taken to increase our understanding of maturation has been to analyze embryo mutants that are blocked from normal passage through this phase. We have been studying a group of defective kernel (*dek*) mutants which were reported by Neuffer et al. (1986) to undergo morphogenetic blocks after forming only two or three leaf primordia. This stage of shoot development corresponds to the time when wildtype embryos enter maturation phase.

We have been investigating the developmental potential of this class of *dek* mutant by characterizing their growth, morphogenesis, accumulation of maturation products, and ability to germinate over the course of ontogeny. We report here the results from our descriptive and experimental studies of two *dek* mutants, *cp*-1311C* and *cp*-1399A*, kindly given to us by Sheridan and Neuffer.

The first question we asked of these mutants was whether or not the mutations represented blocks to continued development. We compared growth (size, fresh and dry weights) and morphological stages of mutant embryos to that of their wildtype counterparts at various time points during embryogenesis. Cumulative growth profiles of *deks cp*-1311C* and *cp*-1399A* and their wildtype sibs (see figure) show that these embryos do not become



developmentally arrested. Instead, *dek* embryo growth and morphogenesis is slow and steady from early in development until metabolic quiescence near full term, rather than arrested. We suspect that these mutants are altered in some component of nutrient uptake or in a metabolic pathway rather than in the maturation process.

These mutants reach morphological stages associated with early maturation events in wildtype embryos, but their timetables of development are not synchronized with the changing maternal environment. We suspected that this situation would allow maternal cues for development to affect the mutant embryos at age- rather than stage-appropriate times, thereby allowing us to distinguish between environmental and seed-autonomous cues. To determine if and when *dek* embryos matured and whether biochemical differentiation remained coupled to growth and morphogenesis, we looked for the accumulation of pigments, storage macromolecules and LEA proteins (proteins associated with dehydration). We compared the results from mutant and wildtype embryos, matched by either morphological stage or age (days after pollination). In both *dek cp*-1311C* and *cp*-1399A* embryos, carotenoids, anthocyanins and the major embryo storage globulin protein, GLB1, accumulated properly according to the size and morphological stage of the mutant embryos. Linkage to morphological stage suggests that cues for the accumulation of pigments and storage proteins are seed-autonomous. In contrast, both *dek*

mutants accumulated the LEA group 3 protein, MLG3, at the same time and in the same amount as their wildtype siblings. The uncoupling of MLG3 accumulation from *dek* embryo morphogenesis suggests that the accumulation of MLG3 is due to a maternal cue.

The acquisition of desiccation tolerance and ability to germinate are additional features of the maturation process. *dek cp*-1311C* and *cp*-1399A* embryos were able to germinate precociously, from 18 days after pollination until full ear maturity (albeit less vigorously than wildtypes) when embryos were cultured in a hormone-free germination media (GM). However, after 60 DAP they were unable to germinate, either as whole kernels or dissected embryos. To test whether *deks* lose their ability to germinate because of an intolerance to desiccation and subsequent imbibition, we used fluoroscene diacetate (0.1%) as a vital stain on ear mature *dek*, wildtype (positive control) and heat-killed wildtype embryos (negative control). The embryos were soaked in water overnight at room temperature, sectioned longitudinally and then viewed under ultraviolet light. *dek cp*-1399A* scutella fluoresced as brightly as the wildtype, while *dek cp*-1311C* scutella fluoresced very little. Desiccation tolerance is gained by wildtype embryos by stage 4 (ca. 30 DAP). Since the *deks* (especially *cp*-1399A*) can withstand maturation drying, they must be desiccation tolerant prematurely with respect to their terminal morphological stages.

To further assess the physiological age of mutant embryos, we studied their behavior in embryo-culture experiments. Immature and mature mutant and wildtype embryos were cultured in GM, or GM plus abscisic acid (ABA) or osmoticum. We have previously shown that wildtype embryos have an age-dependent sensitivity to these cues for maturation. Wildtype embryos at stages 2 and 3 (18-22 DAP) germinate readily in GM alone but are inhibited from germination by both 10μM ABA and 20% sucrose (osmoticum). By 50 DAP the germination of wildtype embryos is unaffected by ABA and only slightly slowed by osmoticum. In contrast, *dek* embryos at 50 DAP (stage 2 or 3) respond to the culture treatments in a stage- rather than age-appropriate fashion -- i.e., both ABA and osmoticum inhibit germination. Therefore, before the *dek* embryos completely lose their ability to germinate following desiccation, their response to culture treatments is stage-appropriate.

GA and the germination of embryos in vivo and in culture

--Connie White and Carol Rivin

The phytohormone abscisic acid (ABA) has been shown to be an important regulator of maize embryo maturation. Excised immature embryos are inhibited from germination and synthesize a complement of embryo proteins in response to culture with ABA. Certain viviparous mutants appear to undergo precocious germination as a result of diminished ABA levels. Because of the strong antagonism between ABA and gibberellic acid (GA) documented in other monocot systems, and because of the known role for GA in the germination of other cereals, we examined the effects of GA and GA biosynthesis inhibitors on germination processes of both wildtype and viviparous embryos in culture and in vivo.

We assayed germinability of embryos excised at stages 2, 3, and 4. Stage 2 and 3 wildtype embryos germinate readily in culture, while stage 4 embryos are quiescent. We asked whether disruption of GA synthesis would alter germination. Germination of stage 2 embryos was relatively unaffected by the inhibitors ancymidol and paclobutrazol, while germination of stage 3 embryos was suppressed in the presence of these inhibitors. Inhibitor-mediated suppression was alleviated by the addition of exogenous GA_3. Stage 4 embryos cultured with exogenous GA_3 germinated readily. Our simplest interpretation of these results is that the presence of GA is required for in vitro germination and that stage-specific variation in germinability and/or sensitivity to GA and GA inhibitors reflects variation in endogenous ABA and GA levels or synthetic capacity.

In addition to affecting germination, these growth regulators also influenced the levels of anthocyanin synthesized in wildtype embryos. Anthocyanin synthesis was enhanced by either ancymidol or paclobutrazol and antagonized by exogenous GA_3. We found that the formation of anthocyanins in cultured embryos required the presence of an *R* allele conditioning embryo pigmentation in vivo.

We tested the effects of altered GA metabolism in two viviparous mutants both in vitro and in vivo. *vp1* (ABA insensitive) and *vp5* (ABA deficient) embryos were excised at various stages and cultured in the presence and absence of GA inhibitors. Germinability of either genotype was relatively unaffected by any treatment. We disrupted GA synthesis in vivo by biochemical and genetic means. In the first protocol, one side of *vp1* ears were treated with either of two commercial preparations of inhibitors, "A-rest" (ancymidol plus surfactant--Dow Elanco) or "Bonzi" (paclobutrazol plus surfactant--ICI UniRoyal) at either 10 or 100 micromolar concentrations at 5 day intervals starting with 6 days after pollination and ending at 26 days after pollination. Other ears were treated with 10 or 100 micromolar GA_3 alone or in combination with 100 micromolar paclobutrazol. The treated ears were examined 50 days after pollination. Compared to the untreated half and to control ears, none of the treatments induced a notable difference in degree or extent of vivipary. Wildtype ears in the same genetic background (W22) were treated in a like manner and similarly showed no altered effects either in kernel maturation or germinability after drying. Our genetic protocol involved planting the F2 generation of a cross between *vp1* plants and *dwarf1* (*d1*) plants. Dwarf plants were both selfed and outcrossed to standard *vp1* stock to test for expression and presence of the *vp1* allele. Self-pollinated ears of *d1/d1*, *vp1/+* plants segregated for vivipary in the expected 3:1 ratio. The extent of vivipary was not dramatically different between selfed ears and ears pollinated by a standard *vp1* stock. Shoot growth was slightly diminished on ears whose mother plant was *d1/d1*. Similar experiments are currently being undertaken on *vp5* mutants.

Our results indicate that while de novo GA synthesis is required for germination of immature wildtype embryos, germination of cultured *vp1* or *vp5* embryos does not depend on the synthesis of this hormone. In addition, expression of vivipary by *vp1* embryos seems unaffected by diminished GA levels in vivo. These differences in sensitivity may indicate that germination of cultured wildtype embryos proceeds along an entirely different pathway than that which occurs with viviparous embryos or alternatively, that our crude methods are obscuring some delicate and perhaps transient GA-mediated regulation of vivipary. Since germination may involve a balance of ABA and GA, experiments to assay the effects of lowered GA levels on *vp5* embryos in vivo should enable us to distinguish between these two possibilities.

Overlapping pathways of ABA-modulated gene expression during embryo maturation

--Connie White and Carol Rivin

Viviparous mutants of maize fail to complete normal embryo maturation and instead precociously germinate on the ear. We have used a differential screen to isolate novel cDNA clones whose expression is reduced or abolished in viviparous embryos. We obtained *Vp1*-requiring cDNA clones by preparing a mid-maturation phase cDNA library and screening it differentially with wildtype and *vp1* cDNA. These messages were then characterized according to their sequence, their expression during normal development and their tissue specificity. The individual effects of *Vp1*, ABA and changing water relations on these mRNAs were examined by measuring message levels in ABA-synthesis deficient mutants (*vp5*) and wildtype embryos in embryo culture, where hormone levels and water uptake can be controlled experimentally. The accumulation of the maturation set of messages was examined in wildtype and mutant development in vivo and compared with that seen in cultured embryos. We also compared two mutant alleles of *vp1* (*vp1* and *vp1-mcwhirter*) that differ in their ability to suppress precocious germination. From these experiments, our clone set can be divided into groups that encompass four overlapping pathways of maturation expression:

Group 1: The most abundant class of messages reaches peak accumulation between 27 and 30 days after pollination. In addition to requiring the *Vp1* gene product, these messages require high levels of ABA for induction. They include the *Glb1* storage protein and a homologue to the wheat LEA gene *Em*. Other messages have sequences unrelated to previously reported genes. Several of the clones of this group detect a small amount of transcript in maturing tassel, but none in leaf, cob or husk.

Group 2: These messages reach peak expression earlier in embryogenesis than Group 1 and they require lower levels of ABA for induction. No homologous sequences were found in genetic databanks.

Group 3: Expressed later in embryogenesis than Group 1, this class requires a high level of ABA. Unlike Group 1 or Group 2 messages, the *vp1-mcwhirter* allele will not substitute for wildtype *Vp1* in the expression of these mRNAs. The *Glb2* storage protein message is in this group.

Group 4: Using a wheat homologue, we isolated a cDNA for maize LEA group 3, a protein associated with dehydration. This message is ABA-inducible, but unlike all the other clones we have examined, it does not also require the *Vp1* gene product. This message peaks in the same time frame as Group 2.

Use of flash tape to solve bird problems in the field

--Carol Rivin

When I first moved to Oregon I had terrible problems with birds scratching out my germinating seedlings. I found that a reflective mylar tape strung in twists between newly planted rows totally alleviated this problem. This tape doesn't interfere with other planting or cultivation tasks. However, the flashing makes folks a little crazy, so we take it down when the plants are large enough to be safe. The tape is cheap, easy to put up and take down, and re-usable if you're careful. I'm often asked for the source of the stuff, so here's where I get it: Nishizawa (USA) LTD, 112 West 9th Street suite 903, Los Angeles, California 90015. Phone: 213-627-7491. Ask for Flash Tape #996215. Last time I bought it, it came in 90 meter rolls at about $.95 each.

DEFIANCE, OHIO
Defiance College

Environmental programming of paramutation in meristems of two and three week old seedlings

--Bernard C. Mikula

In MNL, 1992, I reported, with pictures, that temperature and light conditions applied to two and three week-old seedlings could significantly alter the level of paramutation of the *R* gene when testcrossed at maturity. This report follows up with testcross scores of 80 plants which received light and temperature treatments as two and three week-old seedlings. Growth chamber conditions were those reported in MNL 1992. The seeds used in all lines of Table 1 came from a single ear of an *R R-lst* heterozygote.

Table 1. Testcross scores reflecting changes in paramutant *R*-gene expression following early temperature and daylength treatments of two and three week-old seedlings. LL=constant light, LD=12 hr. light:12 hr. dark.

		Pooled Means		Growth Chamber Environment	
		Earliest	Latest		
Line	n	Pollinations	Pollinations	da. 1-15	da. 16-21
48	8	8.0±3.4	9.3±5.0	LL 22 C	2 LD 22 C-4 LL 32 C
47	7	10.3±2.0	13.5±3.2	LL 22 C	2 LD 22 C-4 LL 22 C
46	6	8.7±3.1	10.9±2.9	LL 22 C	6 LL 22 C
49	9	9.3±1.7	12.5±1.1	LL 22 C	4 LD 22 C-2 LL 22 C
50	6	9.9±4.0	14.4±3.3	LL 22 C	4 LD 22 C-2 LL 32 C
45	8	9.8±2.6	13.4±2.1	LL 22 C	6 LD 22 C
				da. 1-10	da. 11-15
30	6	3.3±.6	3.2±1.5	LL 32 C	5 LL 32 C
31	7	8.2±2.3	9.6±3.8	LL 32 C	5 LL 22 C
26	5	8.7±3.5	9.2±3.2	LL 32 C	5 LD 32 C
27	7	9.3±3.2	10.7±2.5	LL 32 C	5 LD 22 C
28	7	9.2±3.8	9.2±3.6	LL 32 C	2 LD 32 C-3 LL 22 C
29	4	8.8±1.2	10.8±3.2	LL 32 C	2 LD 22 C-3 LL 22 C

Scoring was done by matching 50 kernels from each testcross ear against a set of standard kernels ranging from 0 to 20, colorless to fully pigmented, respectively. Colorless kernels were excluded from computed scores since a colorless paramutated *R* phenotype could overlap colorless *R-lst* or *r* expressions. Scores of testcross ears from pollen sampled from plants in each line are reported as pooled ear means for the earliest and latest pollinations of the plants in that line. The fifth column of Table 1 shows the preliminary conditions in which the seedlings were maintained prior to the tassel inductive conditions (last column) applied in the second or third week depending on temperature. Earlier work (MNL42) had shown that seedlings can be ready for tassel induction a week earlier at 32 C than for those seedlings raised at 22 C. The last column shows the variety of treatment combinations applied during the period tassels are determined. The LL symbol represents constant light; LD represents 12 hr. light:12 hr. dark photoperiod. The number of cycles is represented by a single digit before the LL or LD symbol.

When pollen of single plants was sampled over the seven day period that pollen was shed and testcross scores of the earliest and latest pollen samples are compared in Table 1, the earliest pollen samples produced testcross scores consistently lighter than the latest. A statistically significant difference exists between the scores of the earliest and last pollinations of lines 49 and 45, P= .001 and .02 respectively, the two lines with the smallest standard errors. A wider range of difference is observed in testcross scores between individual plants in lines which were

started in 22 C temperatures and were given light or temperature perturbations in the third week; this is reflected in larger standard errors for each pooled mean. A significant difference (P = .001) exists between the scores of the 30 plants which were started in 22 C and subjected only to 22 C treatments in the third week (lines 45, 46, 47, 49) and those scores of the thirty plants of the last four lines of Table 1 (lines 26, 27, 28, 29, 3l). The greatest range of variation within and between the scores of each line is found in the latest pollinations representing the pollen from the lowest branches of the inflorescence. The gametes from these lowest branches are, therefore, the most likely to undergo a heritable change in paramutant *R*-gene expression following environmental treatment. Testcross scores from pollen sampled from the upper branches, the earliest pollinations, are essentially alike for both temperature conditions.

One of the objectives of this work was to discover experimental operations where short treatment periods could result in significant changes in the testcross scores for *R*-gene expression. Line 48, treated with LL conditions at 32 C for the last four days, shows the largest standard error as well as the lowest score for seedlings started in 22 C LL conditions. Because of the small sample sizes required by limited growth chamber space, inferences regarding other mixed treatments will require further study. Line 30 shows that seedlings held in constant light for 32 C throughout the first 15 days yielded plants at maturity whose testcross *R*-gene pigment scores approached the colorless condition. It is these nearly colorless testcross ears that make the strong case for the paramutant *R*-gene being influenced by the environment. With these same environmental treatments no change has been noted in the *R*-gene expression in the absence of the paramutagenic allele *R-lst*. Nor was it possible to produce this extreme change in paramutant *R*-gene expression maintained in the Wisconsin inbred W22 background. It may be concluded the paramutation process, which involves transposable elements, is responsive to temperature and light conditions. That transposable elements in somatic tissue respond to temperature has been reported for many years for a number of different plants. This is the first report where the variegation associated with transposable elements expressing in gametogenic tissues of maize can be placed under environmental control so that binary (on-off) change in gene expression can be followed in later generations.

That the *R* gene is responding to developmental cues seems likely since floral determination is brought about by the application of light-dark conditions between days 11 and 15 at 32 C or between days 16 and 21 at 22 C. Inbred W22 seedlings held in LL conditions during either of these periods will not be florally induced until removed to the photoperiods of field conditions at the end of their respective LL treatment periods. Plants which received LL conditions have two more nodes when compared with those plants which received LD conditions at 22 C. Increased node number has been consistently associated with lower paramutant *R*-gene scores.

The late R. A. Brink considered that the tassel gave rise to a mosaic of paramutant *R*-gene expressions. Tassel branch mosaics for paramutant *R*-gene expression was confirmed in reports of testcrosses of pollen samples, all made the same day, from five tassel branches of single plants (Cooper, H. B., U. Wis. Thesis, 1964). The Wisconsin experiments used inbred W22 into which an *R* gene, highly selected for "stability", had been backcrossed and maintained through many generations of selection. Using the *R* gene in the inbred W22 background I have found only small differences between early and late pollen collections. In inbred W22 small but statistically significant differences in paramutant *R*-gene expression were found between testcrosses of plants matured from environmentally treated seedlings (Mikula, Genetics 56: 733-742). The small differences required a skeptical attitude until much more control of experimental operations could be found. I feel the data above permit me to relax my skepticism about whether the results are real. My dependence on an *R* gene, considered metastable (mottled), but highly inbred and selected for "stability" of expression, may have been responsible for the lack of responsiveness to early environmental conditions. Logically, why should one use a highly inbred, stabilized, "unstable" gene if instability is what is to be studied? Why was I so blind for so many years? I failed to consider the genetic operations of selection for uniformity of expression which shaped the *R* gene stock in the W22 background with which I was dealing. Stadler was aware and warned of this operational trap long ago.

The work reported in Table 1 makes it possible to consider different conclusions from those proposed by the Wisconsin Laboratory for the paramutant *R* gene in inbred W22 (Genetics 52:407-424). 1) Paramutation changes are not random but depend on environmental conditions of seedlings at the time of tassel determination. 2) A polarity of paramutant *R*-gene expression results from certain environmental treatments: the lower branches produce darker paramutant expressions than the earlier upper branches. 3) The time course of paramutation in the terminal meristem of the main stalk can be fixed within a few days at a developmental stage prior to tassel differentiation. 4) Because paramutation takes place late, just prior to terminal meristem differentiation into tassels, one could expect paramutation from the ears to be different from those of the tassel. 5) Uniformity or variation of paramutation is an expression of the environmental conditions affecting the meristem when seedlings are two or three weeks old depending on temperature and perhaps light.

With increased attention being focussed on methylation of transposable elements a greater emphasis must be placed on experimental operations which can explore developmental events throughout the life history of the maize plant. The extended life cycle of maize compared with shorter cycles of "certain" other plants can be regarded as an asset providing longer developmental windows for experimentally influencing epigenetic events. The early work in plant physiology dealing with biological rhythms and floral induction provides a rich literature worth attention for methods which may influence developmental processes of genetic interest, especially methylation. Since the paramutant *R* gene has been associated with changes in methylation and probably has associated transposed segments of transposed elements, it seems likely this transposed material could provide the transducer which responds to temperature and light. A simple germination test of an *R* gene with plant pigment will show that roots and coleoptiles fail to produce pigment at higher temperatures or when certain colors are filtered out of white light. Are these light and temperature responses the function of transposed material controlling transcription of the *R* gene through DNA modification?

The seeds scored in lines 30, 45 and 47, Table 1, were grown out the following year with no further treatment as seedlings. Figure 1 shows that the differences recorded in the scores of 1991 are maintained the following year, 1992. The four ears on the left of Figure 1 came from a plant represented in line 30, the

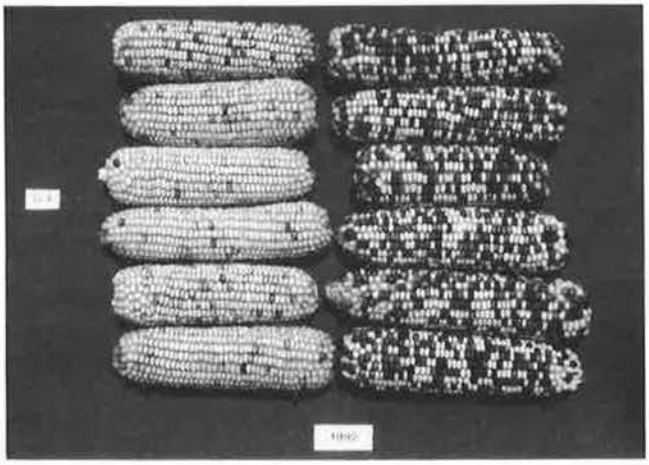


Figure 1. Testcross seeds from Table 1 of Line 30 (left column of ears) and 45 (right column) were grown and testcrossed in 1992. Differences observed in testcrosses the previous year persist in above testcrosses made a year later without further treatment as seedlings.

four ears to the right are representative of plants from lines 45 and 47.

DURHAM, NORTH CAROLINA
Duke University

A cross between *Tripsacum dactyloides* and *Zea diploperennis*
--Mary Eubanks

Tripsacum dactyloides and *Zea diploperennis* have been crossed using standard pollination technique and viable, fertile hybrids obtained. The cross has been effected both ways. With *diploperennis* as female parent, the plant is referred to as Sun Dance, and with *Tripsacum* as female parent, the plant is called Tripsacorn. *Zea diploperennis* seed was provided by Professor Hugh H. Iltis of the University of Wisconsin at Madison. It was collected from populations growing in their natural habitat in Jalisco, Mexico.

In growth habit and overall general appearance, the F1 plants are more like *Z. diploperennis* than *Tripsacum*. Other morphological features are intermediate between the two parents. The hybrid is perennial by means of basal offshoots and underground rhizomes. Plants die back in autumn when temperatures reach ~25 F and produce new growth after the ground thaws in spring. Grown in outdoor nurseries in Mississippi and North Carolina, it has come back every year surviving temperatures as low as 0 F. *Z. diploperennis*, which is also perennial in its native habitat and greenhouse grown, has not survived winters when temperatures have dropped below 25 F.

The *Tripsacum-diploperennis* hybrid produces numerous culms (15-50 when field grown) with some better developed than others. The inflorescences are variable. Some are entirely staminate and appear as tassels at the summit of culms. Some, which are bisexual with staminate flowers subtended by pistillate flowers, more closely resemble the *Tripsacum* inflorescence. Some pistillate ears are a single rowed spike of caryopses completely enclosed in hard shell-like fruitcases, whereas others are highly unusual and have four rows of paired kernels partially enclosed by glumes. These latter ears have distinct interspacing between rachis segments and resemble the oldest archaeological maize specimens from Tehuacán, Mexico.

A unique feature of the *Tripsacum-diploperennis* hybrid is it can be rooted in water or soil from stem cuttings. Furthermore, excised nodal tissue placed on tobacco initiation medium produces callus. Green growth of early shoot formation and roots have developed from the calli.

The chromosome number from root tip counts and study of pollen mother cells at diakinesis ranges from 2n=16 to 2n=24. This indicates chromosome loss, a phenomenon frequently observed in interspecific hybrids, has occurred. There is a range in degree of chromosome pairing as indicated by varying numbers of bivalents and univalents at metaphase and by incomplete synapsis observed at pachytene. Trivalents, as well as chains of 3 and 4, have also been observed at diakinesis and metaphase. Preliminary cytological evidence for *Tripsacum* introgression is further seen in the architecture of the chromosome carrying the nucleolar organizer region. Although not yet verified by comparison of karyotypes from actual plants involved in the cross, it looks like *Tripsacum* chromosome 16. If it is not Tr16, it is *Z. diploperennis* chromosome 6 with a broken terminal end.

The hybrid has a high degree of pollen fertility as measured by scoring 100 pollen grains stained with cotton blue. Tripsacorn is 94.1% fertile and Sun Dance is 92.3% fertile.

There is segregation in F2 progeny with some plants producing 4 to 8-rowed ears and others producing single-rowed spikes. The chromosome number in the F2s is 2n=20 and pollen fertility is 93.4%.

The hybrid is cross fertile with maize. The cross has been effected with hybrid and inbred corn lines, as well as with indigenous Mexican and Andean races. Pollen fertility in these crosses is dependent on which maize lines is used in the cross. For example Zapalote Chico x Tripsacorn is 68.5% fertile and W64A x Tripsacorn is 84.6% fertile.

The unexpected fertility of the *Tripsacum-diploperennis* hybrid and cross-fertility with maize is potentially of great value because it is a natural genetic bridge for transferring *Tripsacum* genes into maize. It provides a mechanism for importing *Tripsacum* genes into maize in one generation by natural breeding techniques. When *Tripsacum* is the female parent in the hybrid, it provides unique opportunity for transferring *Tripsacum* cytoplasmic genes into maize. Benefits to maize observed in field tests, greenhouse plants and laboratory bioassays include heat and drought tolerance, cold tolerance, hybrid vigor, increased standability, resistance to corn rootworm and corn leaf aphid, enhancement of maize tissue culture capability.

Corn rootworm resistance conferred to maize via *Tripsacum* x *Zea diploperennis*
--Mary Eubanks

Two types of bioassays, in petri dishes and in pots, were conducted to determine if rootworm resistance could be imparted to maize via a bridge species derived from crossing *Tripsacum dactyloides* and *Zea diploperennis*. For the bioassays, 1,000 non-diapausing western corn rootworm, *Diabrotica virgifera*, eggs in soil were shipped from French Agricultural Research, Inc., Lamberton, Minnesota, to Durham, NC, under U. S. Department of Agriculture permit number 922762. Plants were infested with newly hatched first instar larvae of western corn rootworm. The larvae were transferred to test containers by lifting with a small paint brush. Two separate petri dish bioassays and three pot bioassays were performed.

For the bioassays, seed from Tripsacorn, i.e. *Tripsacum dactyloides* (female) x *Zea diploperennis* was crossed with four diverse types of maize. The four maize types included: Funk's G4522, a commercial hybrid corn seed; B73 and W64A, two inbred lines; Zapalote Chico, a native Mexican race classified as a prehistoric mestizo derived from ancient indigenous races. Other plants infested with corn rootworm included G4522 x Sun Dance, *Tripsacum*, Tripsacorn and maize controls. Sun Dance refers to *Zea diploperennis* (female) x *Tripsacum dactyloides*.

Petri dish bioassays were employed to screen for antibiosis versus antixenosis. If there is an antibiotic effect, evidence for eating and dead larvae can be seen; if there is an antixenotic effect, larvae can be observed trying to leave the dish. For these tests, 10 grams of top soil sieved through a 1mm mesh screen was placed in a petri dish with 3 to 5 freshly germinated seedlings or, in the case of *Tripsacum*, with a small clonal piece of plant with young roots, and kept moist. The rims of each dish were ringed with petroleum jelly to monitor for any larvae trying to leave the dish. Up to a total of 50 larvae were added to each dish over a three day period. Each treated dish was observed for several days under a dissecting microscope at 60X magnification and behavior recorded.

Results of petri dish bioassays are summarized in Table 1. In all cases, larvae remained on or near the roots, seed and cotyledons, or in the soil. There was no indication of larvae trying to exit dishes; thus, there is no evidence for antixenosis. *Tripsacum*, Tripsacorn, B73 x Tripsacorn, W64A x Tripsacorn, G4522 x Tripsacorn and G4522 x Sun Dance did not show any signs that the roots produce a substance that is a deterrent to the insects. Evidence for antibiosis and tolerance was indicated with Tripsacorn and the hybrids between corn and Tripsacorn tested, whereas there was no evidence for antibiosis or tolerance with the corn and maize x Sun Dance materials tested.

Plants grown in pots were used to screen for evidence of tolerance and/or antibiosis. Lodging is seen in plants that are susceptible to rootworm damage, whereas plants that remain upright and healthy when exposed to rootworms are indicative of tolerance and antibiosis. Root damage was observed and scored according to the Hills and Peters rating scale from 1 to 6 that is widely used in the corn belt to evaluate root damage (J. Econ. Entomol. 64:764-765, 1971). The rating criteria are: (1) no damage or only a few minor feeding scars; (2) feeding scars evident but no roots eaten off to within 1 1/2 inches of the plant; (3) several roots eaten off to within 1 1/2 inches of the plant but never the equivalent of an entire node of roots destroyed; (4) one root node completely destroyed; (5) two root nodes completely destroyed; (6) three or more root nodes destroyed.

When a bioassay was complete, two or three plants were removed from pots, soaked in water, then rinsed with a gentle spray to thoroughly clean roots, and observed under a dissecting microscope for scoring. The mean calculated from the total scores of plants in each category is reported. Tolerant plants may suffer root damage but are capable of regrowth and varying degrees of plant recovery. Well developed secondary root systems are often capable of compensatory growth from damaged crown roots.

In the first pot bioassay, 3 to 5 seedlings (approximately 1 week old), or in the case of *Tripsacum* a small clone with young roots, were planted in potting soil in 10-ounce containers and were grown indoors under artificial grow lights. A total of 70 larvae were added to each container over a two day period and plants were observed for 11 days.

In the second pot bioassay, ≤10 day old seedlings were planted in potting soil in 3 inch peat pots and grown indoors under artificial grow lights. A total of 30 larvae were added to each pot over a three day period. Although most plants were dead within one week, observation of the ones that survived extended over two weeks before plants were sacrificed for root evaluation. For each type, there were a minimum of two plants, and in most cases there were four plants.

In the third pot bioassay, plants were grown under natural light and were 11 to 14 days old at infestation. A total of 30 larvae were added to each pot over two days. Observation was for 11 days before sacrificing plants to score root damage.

Results of pot bioassays are summarized in Table 2. The data indicate that maize x Tripsacorn plants are definitely more resistant to corn rootworm than corn controls and maize x Sun Dance. Antibiosis and tolerance are evidently the mechanisms for resistance inherited from Tripsacorn. All plants sustained some injury to roots. Lodging in corn controls and maize x Sun Dance plants was ≥45° and damage on the Hills and Peters scale ranged from 5 to 6. Although maize x Tripsacorn plants sustained some root

Table 1. Results of petri dish bioassays.

	No. of Larvae	Observations/Comments
Bioassay #1		
Tripsacum	50	Larvae stay on root, some feeding but virtually no damage to roots, larvae not visible after a couple of days
Tripsacorn	50	Some feeding, little root damage
B73 x Tripsacorn	50	Some feeding, little root damage, plants continue to grow
G4522 x Tripsacorn	50	Some feeding, little root damage, plants continue to grow
G4522 x Sun Dance	50	Extensive feeding, plants died
Corn control	50	Extensive feeding, plants died
Bioassay #2		
Tripsacorn	20	Light feeding, some dead larvae
Corn control (W64A)	45	Extensive feeding, plants died
W64A x Tripsacorn	45	Feeding on roots, seed and cotyledons, some dead larvae

Table 2. Results of pot bioassays.

	No. of Larvae	Duration	Root Damage*	Observations
Bioassay #1				
Tripsacum	70	11 days	Not recorded	No sign of damage
Tripsacorn	70	11 days	Not recorded	No sign of damage
B73 x Tripsacorn	70	11 days	Not recorded	No sign of damage
G4522 x Tripsacorn	70	11 days	Not recorded	No sign of damage
G4522 x Sun Dance	70	11 days	Not recorded	Plants died at 6 days
Bioassay #2				
Corn control (W64A)	30	14 days	5.0	Plants died
W64A x Tripsacorn	30	14 days	2.0	Plants weakened
G4522 x Sun Dance	30	14 days	5.0	Plants died
Bioassay #3				
Corn control (Zapalote Chico)	30	11 days	4.0	Lodging (≥45°), leaf damage
Zapalote Chico x Tripsacorn	30	11 days	2.0	Minor leaf damage
Corn control (W64A)	30	11 days	4.0	Lodging (≥45°)
W64A x Tripsacorn	30	11 days	2.3	Plant upright, growing
G4522 x Sun Dance	30	11 days	5.0	Lodging (≥45°), leaf damage
Tripsacorn	30	11 days	1.0	No damage

*Hills and Peters scale (1971)

damage, they remained upright and appeared healthy. There was good development of secondary roots from the damaged crown showing capability for compensatory growth in all maize x Tripsacorn plants examined. One plant each of Zapalote Chico x Tripsacorn and W64A x Tripsacorn from Bioassay #3 was replanted after examination for root damage. Both have completed the growth cycle and produced seed.

Preliminary data from forage analysis of *Tripsacum-diploperennis* hybrid

--Mary Eubanks and Joseph C. Burns

Eastern gamagrass, *Tripsacum dactyloides* (L.), is a highly productive and palatable warm season grass with good prospects as a forage crop. Widespread use, however, has been limited by the following: inadequate seed production, inferior seed quality, difficulties in vegetative establishment, and lack of persistence (Ahring and Frank, J. Range Manage. 21:27-30, 1968). *Zea diploperennis*, discovered on the threshold of extinction in Jalisco, Mexico (Iltis, Doebley, Guzman and Pazy, Science 203:186-188, 1979), probably owes its survival to the fact that it is a very important source of fodder and is maintained as an integral part of a traditional agroecosystem (Benz, Sánchez-Velásquez and Santana M., Maydica 35:85-98, 1990). Since *T. dactyloides* and *Z. diploperennis* have been successfully crossed to produce fertile hybrids that are prolific seed producers and can be propagated vegetatively, efforts are underway to evaluate the *Tripsacum-diploperennis* hybrid for use as a forage crop.

Preliminary results of in vitro digestant and nutri-detergent fiber analyses of *Zea diploperennis*, *Zea diploperennis* x *Tripsacum dactyloides* referred to as Sun Dance, *Tripsacum dactyloides* x *Zea diploperennis* referred to as Tripsacorn, and Sun Dance x *Zea diploperennis*, i.e. the *diploperennis-Tripsacum* hybrid backcrossed to *diploperennis*, are reported here. For analysis of whole plant, leaf, stem and sheath, several stalks with leaves were collected from individual plants of each of the above designations. Plants were grown in an experimental plot at Duke University during the summer of 1992, and material for analysis harvested in late September of the same year. At harvest, all plants were the same age. *Z. diploperennis* was in flower, but the hybrids showed no signs of floral initiation. Therefore, although data are not derived from material at comparable physiological stages and are not useful for precise quantitative comparison of forage quality among specimens, it provides valuable qualitative information about fiber content and palatability. Preparatory to processing, plant tissue was freeze-dried and passed through a 1mm sieve in a Wiley mill. Components analyzed include: (1) NDF (neutral detergent fiber), a measure of cell wall; (2) ADF (acid detergent fiber), cell wall after hemicellulose removed; (3) hemi (hemicellulose), NDF/ADF and digestibility is variable; (4) cell, cellulose in cell wall after lignin and soluble ash have been removed; (5) LIG, lignin in cell wall oxidized and washed out, concentration obtained by obtaining difference of value before and after oxidation; (6) ADF-ash, ash left in ADF residue. The values reported in Table 1 are means of duplicate samples.

Table 1. Forage analyses of *Tripsacum-diploperennis* hybrids.

	NDF	ADF	LIG	ADF-Ash	Cell	Hemi
Leaf						
Zea diploperennis (1-2A)*	58.2	29.0	3.4	1.0	24.6	29.3
Z. diploperennis (3-7) x *T. dactyloides*	61.2	30.1	2.8	1.1	26.3	31.1
Z. diploperennis (2-3) x *T. dactyloides*	58.9	29.3	2.9	1.4	25.0	29.6
T. dactyloides x *Z. diploperennis* (3-3)	60.5	29.2	5.2	0.8	23.2	31.3
Sun Dance x *Z. diploperennis* (3-7)	62.8	31.5	2.9	1.3	27.3	31.3
Sheath						
Zea diploperennis (1-2A)*	66.3	38.8	5.5	1.4	31.9	27.6
Z. diploperennis (3-7) x *T. dactyloides*	70.5	38.9	5.6	1.4	31.9	31.6
Z. diploperennis (2-3) x *T. dactyloides*	66.1	36.1	4.9	1.5	29.8	29.9
T. dactyloides x *Z. diploperennis* (3-3)	67.2	38.0	5.4	0.9	31.7	29.1
Sun Dance x *Z. diploperennis* (3-7)	63.1	34.8	6.0	1.1	27.8	28.3
Stem						
Zea diploperennis (1-2A)*	70.8	46.8	7.9	0.3	38.6	24.0
Z. diploperennis (3-7) x *T. dactyloides*	65.4	41.1	7.1	0.4	33.6	24.4
Z. diploperennis (2-3) x *T. dactyloides*	66.2	42.9	8.0	0.3	34.6	23.3
T. dactyloides x *Z. diploperennis* (3-3)	66.5	41.3	7.0	0.3	34.0	25.2
Sun Dance x *Z. diploperennis* (3-7)	64.1	41.6	6.4	0.2	34.9	22.8
Whole plant						
Zea diploperennis (1-2A)*	76.1	45.2	8.7	1.7	34.9	30.9
Z. diploperennis (2-3) x *T. dactyloides*	76.2	44.7	7.5	2.6	34.5	31.5
T. dactyloides x *Z. diploperennis* (3-3)	74.6	43.0	6.9	2.1	34.0	31.6
Sun Dance x *Z. diploperennis* (3-7)	75.9	44.7	6.3	2.8	35.6	31.2

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A new *Mu* induced mutation whose phenotype is suppressed in *Mu*-inactive tissue

--Michelle Nolasco and Alice Barkan

A *Mu* induced mutation, *hcf106*, was described previously, whose phenotype is modulated by the phase of *Mu* activity (Martienssen et al., EMBO J. 8:1633-1639,1989; Martienssen et al., Genes Dev. 4:331-343, 1990). Plants homozygous for *hcf106* are pale green, non-photosynthetic, and seedling lethal when *Mu* is in its active phase. However, in *Mu*-inactive plants, the mutant phenotype is suppressed, i.e. leaves are dark green, and plants are viable. We have recently identified a new *Mu*-induced mutation whose phenotype responds in an analogous fashion to the phase of *Mu* activity.

psa1 arose in the *Mu* stocks propagated by the Hake and Freeling groups. Mutant seedlings are pale green with very small revertant sectors, and are non-viable due to the absence of the photosystem I complex. Rare plants in families segregating *psa1* have large sectors of dark green tissue in young pale green leaves, with each successive leaf containing an increasing proportion of dark green tissue. This phenotype is very similar to that exhibited by *hcf106* plants in which *Mu* became inactive during somatic development (Martienssen et al., 1990).

To test the possibility that suppression of the *psa1* phenotype in dark green tissue was correlated with a change in *Mu* methylation status, DNA purified from pale green and dark-green sectors of two such plants was cut with *Hin*fI and analyzed by Southern hybridization using a *Mu1* probe. Dark green sectors from these plants contained primarily methylated *Mu* elements whereas pale green sectors from the same plants contained primarily unmethylated *Mu* elements. Fully mutant plants contained only unmethylated *Mu* elements. Therefore, the loss of the mutant phenotype in leaf tissue correlates with increased methylation of *Mu* elements.

Currently, we are testing this correlation between *Mu* methylation and suppression of the mutant phenotype by analyzing more sectored plants. To confirm that the mutant allele is retained in such plants, we will attempt to recover the mutant phenotype by crossing a viable *Mu*-inactive plant derived from a sectored seedling with a *Mu*-active plant heterozygous for *psa1*. In the future, the molecular mechanism will be elucidated that leads to sup-

pression of the mutant phenotype in tissue containing methylated *Mu* elements. It will be interesting to compare this mechanism with that described previously for *hcf106* (Barkan and Martienssen, Proc. Natl. Acad. Sci. USA 88:3502-3506, 1991).

_Mu_-induced nuclear mutations affecting late stages of chloroplast biogenesis: gene nomenclature, screening methods, and progress in mapping and allelism testing

--Alice Barkan, Rodger Voelker, Janet Mendel-Hartvig, Michelle Nolasco, Macie Walker and David Johnson

The phenotype of "high chlorophyll fluorescence" (*hcf*) has been used to identify numerous non-photosynthetic mutants with near normal pigmentation (Miles, Methods in Chloroplast Molecular Biology, 1982). *hcf* mutations are expected to define genes involved in chloroplast gene expression and assembly, as well as structural genes for the various photosynthetic enzymes. Because such mutants are almost invariably slightly pale green, we find it simpler to screen initially for pale green or yellow green mutants that are lethal at the three leaf stage. Subsequent analysis of seedling leaf proteins usually reveals the absence of one or more photosynthetic complexes.

Since many different nuclear-encoded functions contribute to the biogenesis of the photosynthetic apparatus, it is not surprising that such mutants arise at high frequency. In lines harboring active *Mu* transposons, we find approximately 1 new mutant in every 200 F2 families. While such mutants usually do exhibit some degree of increased chlorophyll fluorescence, we have chosen to stop using the *hcf* designation. Instead, we have assigned new gene nomenclatures that reflect more completely the variety of mutant phenotypes. Mutants are grouped into different classes according to the specific nature of the chloroplast defect. The assays used include Western analysis of the major chloroplast proteins, Northern analysis of chloroplast RNAs, and analysis of chloroplast polysomes.

The following mutants arose in the *Mutator* material generated by the Hake and Freeling groups at the University of California. These mutations segregate as single, recessive Mendelian traits. Many are unstable, exhibiting small revertant sectors of dark-green tissue.

***psb* mutants.** *psb* mutants lack the protein components of the photosystem II core complex (PSII). Other chloroplast proteins accumulate to normal levels. We have isolated two independent mutants of this type (*psb1* and *psb2*). Preliminary results suggest that these mutants are not allelic. *psb1* was formerly called *hcf134.*

***psa* mutants.** *psa* mutants lack the protein components of the photosystem I core complex (PSI). Other chloroplast proteins accumulate to normal levels. We have four independent mutants, *psa1*, *psa2*, *psa3* , and *psa4*. Preliminary results suggest that these four mutants are not allelic.

***pet* mutants.** *pet* mutants lack the protein components of the cytochrome f/b6 complex. Other chloroplast proteins accumulate to normal levels when plants are grown in low intensity light ($100\mu E/m^2s$). Plants grown in higher intensity light have a five- to ten-fold reduction of PSII core proteins, suggesting that PSII proteins are lost due to photo-induced damage that occurs in the absence of the cytochrome f/b6 complex. *pet1*: uncovered by TB-8Lc; formerly called *hcf121. pet2*, *pet3, pet4*, *pet5*: preliminary results suggest that these four mutants are not allelic to one another.

***crp* mutants.** *crp* mutants have unique defects in chloroplast RNA processing. *hcf38* also falls into this category (Barkan et al., EMBO J. 5:1421-1427, 1986). *crp1*: fails to accumulate monocistronic *petB* and *petD* mRNAs and lacks cytochrome f/b6 proteins; formerly called *hcf136. crp2*: fails to degrade excised group II introns; formerly called *hcf143.*

***cps* mutants.** *cps* mutants have global defects in chloroplast protein synthesis, as revealed by a decrease in the levels of all thylakoid membrane complexes and RUBISCO, as well as a decreased association of all chloroplast mRNAs with polysomes. Chloroplast mRNAs are unaltered in these mutants. *hcf7* (isolated by Don Miles from the Neuffer collection) also falls into this category (AB, manuscript submitted). *hcf7*, *cps1*, and *cps2* mutants define three separate complementation groups. *hcf7*: uncovered by TB-1La; this mutation causes a defect in the processing of the 16S rRNA. *cps1*: two independent alleles were obtained (*cps1-1* and *cps1-2*); the phenotype of *cps1-2* is the more severe. *cps2*: uncovered by TB-6Lc; formerly called *hcf133*; this mutation is unusual in that it causes a 20-fold decrease in RUBISCO and only a two-fold decrease in thylakoid membrane complexes.

***cgx* mutants**. *cgx* mutants have global defects in chloroplast gene expression. These mutants have reduced levels of all thylakoid membrane complexes as well as RUBISCO, but chloroplast mRNAs and rRNAs are normal in size and abundance. This group is distinguished from *cps* mutants in that most or all chloroplast mRNAs are associated with polysomes. We have two independent mutants of this type, *cgx1* and *cgx2.*

***tha* mutants**. *tha* mutants have defects in thylakoid membrane assembly. These mutants lack more than one thylakoid complex but have normal levels of RUBISCO. The missing chloroplast-encoded components appear to be synthesized normally but fail to accumulate. *hcf106* also falls into this category (Barkan et al., 1986; Martienssen et al., EMBO J. 8:1633-1639, 1989). *tha1*: causes a 10-fold reduction in PSII proteins, and a 5-fold reduction in PSI and cytochrome f/b6 proteins; the level of the ATP synthase is unaffected. *tha*2: causes a 10-fold reduction in the cytochrome f/b6 complex, a four-fold reduction in PSI and PSII, and a three-fold reduction in the ATP synthase.

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The *pl* gene undergoes paramutation

--Garth I. Patterson, Etienne Kaszás, Karen Cone, E. H. Coe, Jr. and Vicki Chandler

The expression of the biosynthetic genes of the anthocyanin pathway in maize is regulated by the genes *pl*, *c1*, *b*, and *r*. Two of these genes, *r* and *b*, are known to undergo paramutation. Paramutation can be simply defined as follows: one allele of a gene heritably alters the other allele when the two alleles are present in a heterozygote. For example, in *b* paramutation, when *B'* and *B-I* are present in a heterozygote, the *B'* allele changes *B-I* into *B'*, so that only *B'* alleles are transmitted. In this report, we describe the isolation and characterization of an allele of *pl* that causes paramutation. This allele, which we call *Pl'-mahogany* (*Pl'-mah*), changes the *Pl-rhoades* (*Pl-rh*) allele into *Pl'-mah*, so that only *Pl'-mah*

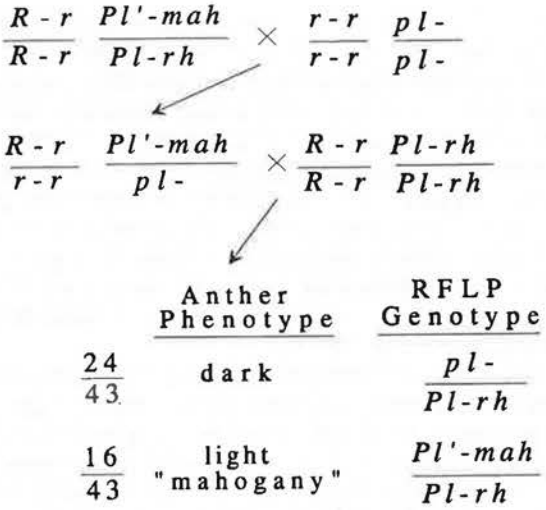
alleles are transmitted from a *Pl'-mah/Pl-rh* plant. (A note on nomenclature: *Pl-rhoades* is the standard *Pl+* allele in our stocks. The phenotype produced by this allele is not unusual--according to the old nomenclature, this allele would simply be known as *Pl*).

An unusual variant was isolated in a family of genotype *B-I/B-I, R-r/R-r, Pl-rh/Pl-rh* (W23 background). The expected phenotype for this genotype is intense color in sheath, culm, and husk due to the action of *B-I* and *Pl-rh*, and intense purple color in the anthers due to the action of *R-r* and *Pl-rh*. The unusual variant had color in all of these tissues, but was different in two ways. First, the variant had less total pigment in each tissue. Second, the formation of pigment was more "sun-red", that is, the formation of pigment was substantially less in tissues not exposed to the sun. For example, inner husks of the variant were mostly green, whereas inner husks of the normal plant had substantial purple color. We call this variant phenotype "mahogany". This isolation is not a unique event; plants with a similar phenotype are seen occasionally in our *B-I/B-I, R-r/R-r, Pl-rh/Pl-rh* (W23 background) stock. We have not carefully measured the frequency, but we estimate that a few percent of these plants have this phenotype.

Initial crosses indicated that one of the genes in this stock was undergoing paramutation. The variant plant was thought to be mutant for either *B-I, R-r,* or *Pl-rh* alleles, but, since it was a self progeny of *B-I/B-I, R-r/R-r, Pl-rh/Pl-rh*, the plant should also have had at least one normal copy of each of these genes. When the variant was crossed to various genotypes, however, 100% (60/60) of the progeny had the mahogany phenotype, i.e. were light-colored and sun-red. None of the progeny had the normal phenotype that should have been seen if the original variant plant was due to a simple dominant mutation. The failure of a normal allele to segregate when it is heterozygous with certain alleles is a hallmark of paramutation.

We suggest the following hypothesis for the mahogany phenotype. The original variant had a new *pl* allele--*Pl'-mah.* The genotype of the variant plant was *B-I/B-I, R-r/R-r, Pl-rh/Pl'-mah*. The *Pl'-mah* allele produces a lighter, more sun-dependent phenotype, and is dominant to *Pl-rh*. Paramutation in this plant would change *Pl-rh* into *Pl'-mah*, therefore, all progeny of the variant would receive the *Pl'-mah* allele. This hypothesis explains the fact that all progeny of the variant plant had the mahogany phenotype.

Two lines of experimentation support this hypothesis. First, the mahogany phenotype segregates with the *pl* locus, as shown in the figure. The variant plant, of putative genotype *Pl'-mah/Pl-rh*, was crossed to a *pl/pl* tester, to produce *Pl'-mah/pl* heterozygotes. The *r-r* allele indicated in the figure gives the same anther phenotype as the *R-r* allele. *Pl'-mah/pl* plants were crossed to *Pl-rh/Pl-rh* plants, and the progeny were grown and classified by anther phenotype. 24/43 progeny had uniform purple anthers, indicative of a *Pl-rh/pl* genotype, 16/43 progeny had light anthers (mahogany phenotype), indicative of a *Pl'-mah/Pl-rh* genotype, and 3/42 plants had anthers with intermediate color, and were of uncertain genotype. This segregation of approximately 1:1 is that predicted by the hypothesis that the mahogany phenotype is caused by a *Pl'-mah* allele that can suppress a normal *Pl-rh* allele. To verify that the segregation of phenotypes was correlated with *pl* segregation, we used *pl* probes on Southern blots to determine the genotypes of the plants. The *Pl-rh* and *pl* alleles can be distinguished on Southerns, but the *Pl'-mah* and *Pl-rh* alleles are indistinguishable with the enzymes tested to date. *Pl'-mah/Pl-rh* can be distinguished from *pl/Pl-rh*, since the *pl* probe hybridizes



to two bands in the latter, and only one in the former. The Southern analysis demonstrated that the 24 plants with uniform purple anthers were, in fact, of *Pl-rh/pl* genotype, and the 16 plants with light anthers were, in fact, of *Pl'-mah/Pl-rh* genotype. The three plants with an intermediate phenotype were *Pl'-mah/Pl-rh* genotype. Thus, 40/40 plants with a clear phenotype had the genotype predicted by the hypothesis that the mahogany phenotype is due to *pl* paramutation.

The other evidence that the mahogany phenotype is due to *pl* paramutation is that the change in phenotype is correlated with a change in *pl* expression. Siblings that were either *Pl'-mah/Pl-rh* or *pl/Pl-rh* were grown, tissue collected and total RNA prepared. Husk and anther tissue from plants with a mahogany phenotype had substantially less *pl* message than similar tissues from plants with a normal phenotype. In these same samples, the amount of *b* and *r* message was similar in all plants, regardless of whether the phenotype was mahogany or normal.

Thus, we have seen a novel anthocyanin phenotype that is linked with the *pl* locus and associated with changes in *pl* expression. We propose that this novel phenotype is due to a *pl* mutation, which we call *Pl'-mah*. This mutant has an unusual property, in that, when *Pl'-mah* is heterozygous with *Pl-rh*, only *Pl'-mah* is transmitted. This exclusive transmission is a hallmark of paramutation, indicating that *pl* should be added to the short list of maize genes that undergo paramutation. We hope that further work on this system, and comparison of results from this system with *b* and *r* paramutation will shed light on this interesting phenomenon.

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Further characterization of the double mutant *opaque2 brittle2*

--Winfried Hetz, Klaus Grasser, Peter Südbeck, Günter Feix and Curt Hannah

C. Y. Tsai et al. previously generated a set of double mutants by combining the *o2* mutant with several starch-modified or starch-deficient mutants and studied the influence of the concomitant presence of these two types of mutations on the zein protein SDS PAGE pattern of alcohol soluble proteins of mature

kernels (Tsai, Larkins and Glover, Biochem. Genet. 16:883-896, 1978). While the majority of the analysed double mutants did not show any major effect on the zein protein PAGE pattern, a dramatic change in the composition of alcohol soluble protein was observed in the case of the *o2 bt2* and *o2 sh2* double mutants. The authors found no protein at the SDS PAGE running positions of the normally predominant 19 and 21kd zein proteins and observed instead a great number of proteins of larger and smaller sizes. As one of the causes for the observed changes of the protein pattern with the striking absence of 19 and 21kd size proteins, the authors proposed an effect of the elevated levels of RNAses detected in the endosperm of this material.

In line with our interest in studying assumed interconnections between the storage protein and starch syntheses in the developing endosperm, we looked again at the *o2 bt2* mutant kindly given to us by Charles Tsai and were particularly interested in the question of whether a transcriptional effect is involved in the mutant behaviour. Towards this analysis we grew plants under controlled conditions in a phytochamber and analysed the zein protein SDS PAGE pattern of proteins isolated from kernels collected at two day intervals from the 9th to the 39th day post pollination, confirming basically the previous observation of Charles Tsai's group. We then isolated the predominant protein band of larger than 21kd size and identified it clearly as the 27kd zein protein by a sequence analysis of its 16 N-terminal amino acids. If analysed over development of the endosperm, the 27kd protein appeared at the 14th day post pollination and disappeared again after 28 days, indicating a breakdown of the synthesized protein normally stably accumulating in wildtype material. In Northern experiments with RNA prepared from various endosperm stages, no RNA coding for the 19 or 21kd zein proteins could be detected while RNA specific for the 27kd protein was present from the 15th to the 28th day, indicating a severe synergistic influence of the two deficient *o2* and *bt2* genes on zein gene transcription. It should be remembered that so far no mutants or mutant combinations with a complete deficiency in the synthesis of the major zein proteins of 19 and 21kd have been identified.

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_Ds_ revertant alleles from _sh2-m1_ show a high frequency of precise excision

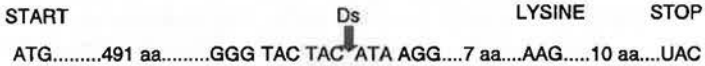
--Michael J. Giroux, Ronald J. Okagaki and L. Curtis Hannah

We are currently analyzing wildtype revertants of a mutable *Ds* containing allele, *sh2-m1*. Revertants from this allele have been shown to differ in their allosteric properties for the enzyme ADPglucose pyrophosphorylase (AGP).

AGP in maize is encoded by the *shrunken2* (*sh2*) and *brittle2* (*bt2*) genes. AGP is believed to be the rate limiting step in starch biosynthesis and is an allosteric enzyme activated by 3-PGA and inhibited by PO_4.

The site of *Ds* insertion in the *sh2-m1* allele is in a region important for the regulation of AGP in plants. Previous data have shown that revertants from the *sh2-m1* allele differ in their allosteric properties. The *Ds* element in *sh2-m1* is located 9 amino acids from a lysine residue important in the regulation of the spinach AGP enzyme (Figure, part A). This region is also highly conserved among different plant species. The *Ds* is only 20 amino

A



B

Revertant Sequence	Frequency
GGG TAC TAC ATA AGG	12/27
GGG TAC TAC TAC ATA AGG	11/27
GGG TAC TAC CCT ATA AGG	2/27
GGG TAC TAC CGTACT ATA AGG	1/27
GGG TAC TAC TTACTA ATA AGG	1/27

acids from the 3' end of the SH2 protein in the gene's last exon.

The DNA sequence of the revertant alleles at the site of *Ds* insertion was characterized in 27 revertant alleles by direct DNA sequencing of PCR products. The PCR products were amplified with primers flanking a 400 base pair region surrounding the site of *Ds* insertion. Both strands of the PCR products were sequenced and the revertant sequence data is summarized in the Figure, part B.

Approximately 50% of the revertants (12/27) contain no *Ds* footprint. Of the remainder, roughly 50% (11/27) contain a footprint of TAC (tyrosine). This likely represents a duplication of host sequences during repair synthesis following excision of the *Ds* element.

Several exceptional sequences were also noted. Two of the 27 revertants sequenced contain a footprint of CCT. One revertant has the added sequence CGTACT and one, TTACTA. These sequences are not explained by current models of transposable element transposition in plants. These models postulate footprints consisting of a portion of the target site duplication or a wildtype sequence. While most of our sequenced revertants are explained as portions of the duplicated host sequence or perfect excisions, several are not. The high frequency of perfect excisions also contradicts evidence that the perfect excision of transposable elements is rare. However, this may reflect the importance of this region of the *Sh2* gene to AGP activity. This is supported by the fact that each of the germinal revertants analyzed restores the wildtype open reading frame.

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Suppression-of-mutability in _bz1-m13::dSpm_ derivative alleles

--Ron J. Okagaki and Oliver E. Nelson, Jr.

Over the last 10 years the mutable allele *bz1-m13::dSpm* has increased our understanding of transposable elements. Here we report on a collection of putative derivative alleles from *bz1-m13::dSpm* that apparently arose in the absence of an autonomous *Spm* element. These alleles have an unusual pattern of inheritance similar to one reported by Victor Raboy and Oliver E. Nelson, Jr. (MNL63:44-45, 1989), which we are calling suppression-of-mutability. Excision in *bz1-m13::dSpm* is frequent and early in kernel development when *Spm* is present, giving a coarsely variegated phenotype, but excision in these change-of-state alleles is delayed giving either a profuse pattern (many small revertant sectors) or a late pattern (few small revertant sectors). Progeny from plants carrying a suppressed allele and *Spm* then segregate for

the change-of-state phenotype and the coarsely variegated *bz1-m13::dSpm* phenotype. Four *bz1-m13::dSpm* change-of-state alleles, *CS-A*, *CS-B*, *CS-C*, and *CS-E*, have been followed for two generations, and results from a third generation are being tabulated.

The starting materials for these experiments were *Sh1 bz1-m13::dSpm/sh1 bz1*, no-*Spm* seeds. One copy of the *bz1-m13::dSpm* allele gives pigmented kernels in the absence of *Spm*. Plump purple seeds were planted, and there was no evidence of *Spm* activity in these seeds. Resulting plants were crossed by a *sh1 bz1/sh1 bz1*, +*Spm* tester. Most ears from these crosses had occasional change-of-state kernels, and these kernels represented *Spm* induced events. A few ears segregated for a change-of-state (CS) phenotype. We infer that these events producing new phenotypes occurred in the previous generation. Hence, these new alleles were probably created in the absence of *Spm*, although a transient activation of cryptic *Spm* elements cannot be excluded. Seeds were taken from segregating ears and retested with the *sh1 bz1/sh1 bz1*, +*Spm* tester, and ears that continued to show their change-of-state phenotype were crossed with a *sh1 bz1*, no-*Spm* tester to isolate the allele.

Suppression-of-mutability was noticed in test crosses between change-of-state alleles and recessive testers. Figure 1 diagrams suppression-of-mutability in a cross between *Sh1 CS-E/sh1 bz1*, +*Spm* and the *sh1 bz1/sh1 bz1*, no-*Spm* tester. Both profusely spotted kernels, *CS-E*, and coarsely variegated kernels, *bz1-m13::dSpm*, were recovered. The linked marker *sh1* was used to follow the recessive *bz1* allele. Kernels carrying a change-of-state allele are *Sh1/sh1* and plump, while shrunken kernels, *sh1/sh1*, are homozygous *bz1*. Table 1A presents results from the four alleles studied. A sample of seed from two ears were counted, and there were no apparent differences due to the direction of a cross.

In *CS-E* half of the kernels are bronze colored and shrunken (*sh1 bz1/sh1 bz1*, +/-*Spm*). One-quarter of the kernels are plump and purple (*Sh1 CS-E/sh1 bz1*, -*Spm*). Remaining kernels are expected to be *Sh1 CS-E/sh1 bz1*, +*Spm*. All of these kernels are expected to be plump with profuse variegation, but roughly equal numbers of profusely variegated plump kernels and coarse variegated plump kernels were counted. *CS-C* gave a similar result; a deficiency of plump purple kernels (*Sh1 CS-C/sh1 bz1*, no-*Spm*) may be due to the presence of more than one *Spm* element in this line. It was decided to focus on these two alleles.

$$\frac{Sh1\ \ CS\text{-}E}{sh1\ \ bz1}\ +Spm\ \times\ \frac{sh1\ \ bz1}{sh1\ \ bz1}$$

Phenotypes	Genotypes		Ratio
shrunken, bronze	$\frac{sh1\ \ bz1}{sh1\ \ bz1}$	+/- *Spm*	4
plump, purple	$\frac{Sh1\ \ CS\text{-}E}{sh1\ \ bz1}$	-*Spm*	2
plump, profuse	$\frac{Sh1\ \ CS\text{-}E}{sh1\ \ bz1}$	+*Spm*	1
plump, coarse	$\frac{bz1\text{-}m13?}{sh1\ \ bz1}$	+*Spm*	1

Figure 1. Suppression-of-mutability.

Table 1. Segregation of change-of-state alleles in test crosses.

A: Generation 1: *Sh1 CS-*/sh1 bz1*, +*Spm* X *sh1 bz1/sh1 bz1*.

Phenotypes[1]:	*sh bz*	*Sh Bz*	*Sh* coarse	*Sh* late	*Sh* profuse	# ears
Alleles:						
CS-A	89	65	38	--	16	2 ears
CS-B	181	91	32	--	71	2 ears
CS-C	129	13	60	58	--	2 ears
CS-E	116	61	23	--	37	2 ears

B: Generation 2: *Sh1 CS-*/sh1 bz1*, +*Spm* X *sh1 bz1/sh1 bz1*.

Phenotypes[1]:	*sh bz*	*Sh Bz*	*Sh* coarse	*Sh* late	*Sh* profuse	# ears
Alleles:						
CS-C:late	209	13	132	130	---	4 ears
CS-C:coarse[2]	342	121	120	---	1	4 ears
CS-C:coarse[3]	307	69	105	1	---	4 ears
CS-E:profuse	410	62	144	---	166	6 ears
CS-E:coarse[2]	372	130	167	6	---	5 ears

[1]A sample of approximately 100 seed was counted per ear.
[2]Coarsely variegated seed that arose from late or profuse variegated kernels.
[3]Coarsely variegated seed that arose from coarsely variegated kernels.

Suppression-of-mutability was observed in the next generation. *CS-C* and *CS-E* again produced both change-of-state and *bz1-m13::dSpm* phenotypes in approximately equal numbers (Table 1B). This behavior was restricted to kernels with the change-of-state phenotype. Coarse variegated seeds from *CS-E* or *CS-C* gave rise to coarse variegated progeny.

Determinants of this phenomenon might reside in three places. The autonomous *Spm* element could be responsible, although the pattern of inheritance would be difficult to explain. Second, a trans-acting dominant modifier could give the observed phenotype and the pattern of inheritance. A trans-acting modifier of *Spm* activity has recently been reported (M.G. Muszynski and P.A. Peterson, 34th Annual Maize Genetics Conference, 1992). Finally, there could be strand-specific modification to *bz1-m13::dSpm* as suggested by V. Raboy and O.E. Nelson, Jr. (MNL63:44-45, 1989). These possibilities are being explored in genetic tests currently in progress.

The first test brought a known *Spm* element into the change-of-state lines. If autonomous *Spm* element(s) in the change-of-state lines are modified, then a normal *Spm* element should restore the coarse variegated phenotype. The second test used *sh1 bz1/sh1 bz1* kernels from test crosses; these kernels should segregate for modified-*Spm* elements or trans-acting modifiers if they exist. Crossing these plants with *bz1-m13::dSpm*, no-*Spm* would give rise to ears segregating for the change-of-state and coarse phenotypes. This is the test used by Raboy and Nelson to demonstrate that suppression-of-mutability was a property of their alleles. Crossing *CS-C*, +*Spm* and *CS-E*, +*Spm* with *bz1-m13::dSpm*, no-*Spm* was the final test. If kernels showing both coarse and late or profuse variegation appear, then suppression-of-mutability is a property of the *bz1* locus.

Two additional tests are in progress. First, the *Sh1 Bz1* class of kernels from test crosses with change-of-state alleles have been planted. These kernels contain *CS-C* and *CS-E* but lack *Spm*. Crossing these kernels with the *Spm* tester line may tell us if the presence of an active *Spm* element is necessary to maintain suppression-of-mutability. Second, *wx1-m8* is being introduced into these lines to allow us to monitor mutability at a second locus. Existing data indicate that the *dSpm* element in *wx1-m8* is identical to the element in *bz1-m13::dSpm*. Results of these genetic tests and future molecular experiments should provide insight into mechanisms suppressing transposable element activity.

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An optimized procedure for protein extraction and 2-D electrophoresis of maize embryo proteins

--Guy Farish and William F. Sheridan

In our laboratory, we have optimized procedures for extracting maize embryo proteins, and their subsequent analysis using 2-D electrophoresis.

Our protein extraction is a modification of the methods of Laemmli (1970) and O'Farrell (1975). Isolated embryos were crushed in liquid nitrogen and boiled for 5 minutes in 300µl of a buffer containing 62.5mM Tris-HCl pH 6.8, 5% SDS, 5% β-mercaptoethanol, and 10% glycerol. The sample was then spun for 3 minutes in a microfuge and the supernatant recovered. This extract was precipitated with 9 volumes of acetone at -20 C for 2 hours, the precipitate spun down and dried. The precipitate was resuspended in 20µl of lysis buffer containing 20mM Tris-Hcl pH 7.6, 5mM $MgCl_2$, 1% Nonidet P-40, and 50µg/µl leupeptin. This suspension makes a thick slurry which was incubated on ice for 15 minutes with 1µl of 2µg/µl Dnase I and 1µl of 1µg/µl Rnase. After nuclease treatment, 25mg of urea and 40µl of sample buffer (2% Nonidet P-40, 2% ampholytes, 5% BME, and 9.5M urea) were added to the sample. The samples were then stored at -80 C.

Samples were assayed for protein content by taking a 10µl aliquot and precipitating it with 9 volumes of acetone at -20 C. This precipitate was redissolved in 0.1N NaOH, a standard Lowry assay performed and the samples read in a spectrophotometer at 750nm. This avoided the interference problems that most protein assays have with samples containing high concentrations of urea or detergents. A sample from a single mature embryo contained on average 14µg/µl protein.

Isoelectric focusing was performed using 2.0mm internal diameter glass tubes and gels were poured to 15cm in length. This large size allowed good resolution of 50-100µg of total protein. We used a 3:1 ratio of ampholytes (.75ml pH 5-7, .25ml pH 3-10) using Bio-Rad Biolyte ampholytes. Samples were focused for 20 hours at 500 volts plus 4 hours at 800 volts for a total of 13,200 volt/hours.

Second dimension separation was performed on uniform 12.5% polyacrylamide gels at pH 8.8 which are 16 x 18cm x 1.5mm in size. Gels were run 4-5 hours at 75mA/gel using constant current.

These conditions have given us excellent resolution and good reproducibility. Gels are commonly silver stained or blotted onto nitrocellulose.

Recombinational mapping data for five translocation breakpoints on the short arm of chromosome one and four translocation breakpoints on the long arm of chromosome three

--Don Auger and William F. Sheridan

We are modifying a set of forty-five reciprocal translocations and five pericentric inversion stocks so that they will be useful for transposon tagging with the *Ac* element. The usefulness of these stocks will be enhanced by their detailed genetic and cytogenetic characterization. Presented here are data from nine recombinational mapping experiments to determine the location of several reciprocal translocation breakpoints. The first five experiments tested the location of the breakpoint in chromosome arm 1S and involved the five translocations: T1-3(5597); T1-3(5982); T1-4b; T1-5(6899); and T1-3k. The other four experiments test the location of the breakpoint in chromosome arm 3L and involved the four translocations: T1-3(8995); T1-3k; T1-3(5597); and T1-3(5982).

Plants which are heterozygous for reciprocal translocations show 50% gamete abortion. All plants in this study were scored for semisterile or fully viable pollen. In all mapping experiments the parental translocated chromosome carried dominant alleles at the mapping loci and the parental normal chromosomes carried with them the recessive alleles. Genetic recombination with the breakpoints would bring the recessive traits into coupling with the translocation breakpoint (T). The corresponding locus on the normal chromosome is indicated as N. For these experiments T can be considered as dominant to N. In the tables the loci are shown in the inferred order. The parental (P) class is always presented first. Crossover regions are numbered left to right.

Tables 1 through 5 show the experimental design and the recombination data for the four translocation breakpoints on chromosome arm 1S. The genetic mapping markers were *zb4* (*zebra4*) and *P* (*pericarp color*). Homozygous recessive *zb4* plants have zebra cross banding on seedling leaves. The *P* locus conditions both pericarp and cob color and four different alleles were used in these experiments. *P-rr* produces a red cob and a red pericarp; it is dominant over all other alleles. *P-vv* displays a variegated red and white pericarp and cob. *P-wr* conditions a white (actually clear) pericarp and a red cob. *P-ww* is recessive to all the other *P* alleles and when homozygous both the pericarp and the cob are white.

Tables 6 through 9 show the data and experimental design for mapping the 3L breakpoints. Homozygous recessive *ts4* (*tassel seed4*) plants have a compact tassel containing both pistillate and staminate florets. Plants which are homozygous *lg2* (*liguless2*) possess reduced ligules and auricles. Kernels which are homozygous for recessive *a1* (*anthocyaninless1*) have colorless aleurones.

Although the order of loci can usually be determined from these data (there was no recombination between T and *lg2* in T1-3k), the observed genetic distances between the breakpoints and

Table 1. Linkage of T1-3(5597) with *zb4* and *P*. The cytological breakpoints are 1S.77 and 3L.48. The backcross was T *Zb4 P-wr*/N *zb4 P-ww* x N *zb4 P-ww*/N *zb4 P-ww*.

a) P:	T *Zb4 P-wr*	43	82
	N *zb4 P-ww*	39	
b) CO1:	T *zb4 P-ww*	7	22
	N *Zb4 P-wr*	15	
c) CO2:	T *Zb4 P-ww*	3	4
	N *zb4 P-wr*	1	
d) CO1&2:	T *zb4 P-wr*	0	0
	N *Zb4 P-ww*	0	
		Total=108	

% recombination: T-*zb4* (b+d)=22 or 20.4%±3.9%; *zb4-P* (c+d)=4 or 3.7%±1.8%

Conclusion: The breakpoint (T) on 1S is distal to *zb4*.

Table 2. Linkage of T1-3(5982) with *zb4* and *P*. The cytological breakpoints are 1S.77 and 3L.66. The backcross was *Zb4* T *P-rr**/*zb4* N *P-ww* x *zb4* N *P-ww*/*zb4* N *P-ww*.

a) P:	*Zb4* T *P-rr*	143	252
	zb4 N *P-ww*	109	
b) COI:	*zb4* T *P-ww*	26	46
	Zb4 N *P-rr*	20	
c) CO2:	*Zb4* T *P-ww*	11	17
	zb4 N *P-rr*	6	
d) CO1&2:	*zb4* T *P-rr*	2	3
	Zb4 N *P-ww*	1	
		Total=318	

*One of four families (N=53) had *P-wr* as the dominant *P* allele.
% recombination: T-*zb4* (b+d)=49 or 15.4%±2.0%; *zb4-P* (c+d)=20 or 6.3%±1.4%

Conclusion: The breakpoint (T) on 1S is distal to *zb4*.

Table 3. Linkage of T1-4b with *zb4* and *P*. The cytological breakpoints are 1S.55 and 4L.83. The backcross was *Zb4 P-vv** T/*zb4 P-ww* N x *zb4 P-ww* N/*zb4 P-ww* N.

a) P:	*Zb4* T *P-vv*	250	453
	zb4 N *P-ww*	203	
b) CO1:	*zb4* T *P-ww*	2	5
	Zb4 N *P-vv*	3	
c) CO2:	*zb4* T *P-vv*	0	5
	Zb4 N *P-ww*	5	
d) CO1&2:	*Zb4* T *P-ww*	2	3
	zb4 N *P-vv*	1	
		Total=466	

*One of four families (N=101) had *P-wr* as the dominant *P* allele.
% recombination: *zb4*-T (b+d)=8 or 1.7%±0.6%; T-*P* (c+d)=8 or 1.7%±0.6%

Conclusion: The breakpoint (T) on 1S lies between *zb4* and *P*.

Table 4. Linkage of T1-5(6899) with *zb4* and *P*. The cytological breakpoints are 1S.32 and 5S.20. The backcross was *Zb4 P-wr* T/*zb4 P-ww* N x *zb4 P-ww* N/*zb4 P-ww* N.

a) P:	*Zb4 P-wr* T	51	83
	zb4 P-ww N	32	
b) CO1:	*zb4 P-wr* T	3	4
	Zb4 P-ww N	1	
c) CO2:	*zb4 P-ww* T	1	3
	Zb4 P-wr N	2	
d) CO1&2:	*Zb4 P-ww* T	0	0
	zb4 P-wr N	0	
		Total=90	

% recombination: *zb4-P* (b+d)=4 or 4.4%±2.2%; *P*-T (c+d)=8 or 3.3%±1.9%

Conclusion: The breakpoint (T) on 1S is proximal to *P*.

Table 5. Linkage of T1-3k with *zb4* and *P*. The cytological breakpoints are 1S.17 and 3L.34. The backcross was *Zb4 P-wr* T/*zb4 P-ww* N x *zb4 P-ww* N/*zb4 P-ww* N.

a) P:	*Zb4 P-wr* T	127	237
	zb4 P-ww N	110	
b) CO1:	*zb4 P-wr* T	5	9
	Zb4 P-ww N	4	
c) CO2:	*zb4 P-ww* T	11	25
	Zb4 P-wr N	14	
d) CO1&2:	*Zb4 P-ww* T	1	2
	zb4 P-wr N	1	
		Total=273	

% recombination: *zb4-P* (b+d)=11 or 4.0%±1.2%; *P*-T (c+d)=27 or 9.9%±1.8%

Conclusion: The breakpoint (T) on 1S is proximal to *P*.

Table 6. Linkage of T1-3(8995) with *ts4 lg2* and *a1*. The cytological breakpoints are 1S.49 and 3L.06. The backcross was T *Ts4 Lg2 A1*/N *ts4 lg2 a1* x N *ts4 lg2 a1*/N *ts4 lg2 a1*

a) P:	T *Ts4 Lg2 A1*	49	96
	N *ts4 lg2 a1*	47	
b) CO1:	T *ts4 lg2 a1*	1	3
	N *Ts4 Lg2 A1*	2	
c) CO2:	T *Ts4 lg2 a1*	16	39
	N *ts4 Lg2 A1*	23	
d) CO3:	T *Ts4 Lg2 a1*	25	60
	N *ts4 lg2 A1*	35	
e) CO1&2:	T *ts4 Lg2 A1*	0	0
	N *Ts4 lg2 a1*	0	
f) CO1&3:	T *ts4 lg2 A1*	1	3
	N *Ts4 Lg2 a1*	2	
g) CO2&3:	T *Ts4 lg2 A1*	2	4
	N *ts4 Lg2 a1*	2	
h) CO1,2&3:	T *ts4 Lg2 a1*	0	1
	N *Ts4 lg2 A1*	1	
		Total=206	

% recombination: T-*ts4* (b+e+f+h)=7 or 3.4%±1.3%; *ts4-lg2* (c+e+g+h)=44 or 21.4%±2.9%; *lg2-a1* (d+f+g+h)=68 or 33.0%±3.2%

Conclusion: The breakpoint (T) on 3L is proximal to *ts4*.

Table 7. Linkage of T1-3k with *ts4 lg2* and *a1*. The cytological breakpoints are 1S.17 and 3L.34. The backcross was *Ts4* T *Lg2 A1*/*ts4* N *lg2 a1* x *ts4* N *lg2 a1*/*ts4* N *lg2 a1*.

a) P:	*Ts4* T *Lg2 A1*	71	148
	ts4 N *lg2 a1*	77	
b) CO1:	*ts4* T *Lg2 A1*	11	30
	Ts4 N *lg2 a1*	19	
c) CO2:	*Ts4* T *lg2 a1*	0	0
	ts4 N *Lg2 A1*	0	
d) CO3:	*Ts4* T *Lg2 a1*	29	46
	ts4 N *lg2 A1*	17	
e) CO1&2:	*ts4* T *lg2 a1*	0	0
	Ts4 N *Lg2 A1*	0	
f) CO1&3:	*ts4* T *Lg2 a1*	6	13
	Ts4 N *lg2 A1*	7	
g) CO2&3:	*Ts4* T *lg2 A1*	0	0
	ts4 N *Lg2 a1*	0	
h) CO1,2&3:	*ts4* T *lg2 A1*	0	0
	Ts4 N *Lg2 a1*	0	
		Total=237	

% recombination: *ts4*-T (b+e+f+h)=43 or 18.1%±2.5%; T-*lg2* (c+e+g+h)=0 or 0.0%±0.0%; *lg2-a1* (d+f+g+h)=59 or 24.9%±2.8%

Conclusion: The breakpoint (T) on 3L lies between *ts4* and *lg2*.

Table 8. Linkage of T1-3(5597) with *ts4 lg2* and *a1*. The cytological breakpoints are 1S.77 and 3L.48. The backcross was *Ts4 Lg2* T *A1*/*ts4 lg2* N *a1* x *ts4 lg2* N *a1*/*ts4 lg2* N *a1*.

a) P:	*Ts4 Lg2* T *A1*	59	124
	ts4 lg2 N *a1*	65	
b) CO1:	*ts4 Lg2* T *A1*	14	25
	Ts4 lg2 N *a1*	11	
c) CO2:	*ts4 lg2* T *A1*	0	0
	Ts4 Lg2 N *a1*	0	
d) CO3:	*Ts4 Lg2* T *a1*	25	42
	ts4 lg2 N *A1*	17	
e) CO1&2:	*Ts4 lg2* T *A1*	1	2
	ts4 Lg2 N *a1*	1	
f) CO1&3:	*ts4 Lg2* T *a1*	4	6
	Ts4 lg2 N *A1*	2	
g) CO2&3:	*ts4 lg2* T *a1*	0	1
	Ts4 Lg2 N *A1*	1	
h) CO1,2&3:	*Ts4 lg2* T *a1*	0	0
	ts4 Lg2 N *A1*	0	
		Total=200	

% recombination: *ts4-lg2* (b+e+f+h)=33 or 16.5%±2.6%; *lg2*-T (c+e+g+h)=3 or 1.5%±0.9%; T-*a1* (d+f+g+h)=49 or 24.5%±3.0%

Conclusion: The breakpoint (T) on 3L lies between *lg2* and *a1*.

Table 9. Linkage of T1-3(5982) with *ts4*, *lg2* and *a1*. The cytological breakpoints are 1S.77 and 3L.66. The backcross was *Ts4 Lg2* T *A1*/*ts4 lg2* N *a1* x *ts4 lg2* N *a1*/*ts4 lg2* N *a1*.

a) P:	*Ts4 Lg2* T *A1*	47	97
	ts4 lg2 N *a1*	50	
b) CO1:	*ts4 Lg2* T *A1*	19	49
	Ts4 lg2 N *a1*	30	
c) CO2:	*ts4 lg2* T *A1*	5	11
	Ts4 Lg2 N *a1*	6	
d) CO3:	*Ts4 Lg2* T *a1*	5	11
	ts4 lg2 N *A1*	6	
e) CO1&2:	*Ts4 lg2* T *A1*	1	1
	ts4 Lg2 N *a1*	0	
f) CO1&3:	*ts4 Lg2* T *a1*	5	5
	Ts4 lg2 N *A1*	0	
g) CO2&3:	*ts4 lg2* T *a1*	1	3
	Ts4 Lg2 N *A1*	2	
h) CO1,2&3:	*Ts4 lg2* T *a1*	0	0
	ts4 Lg2 N *A1*	0	
		Total=177	

% recombination: *ts4-lg2* (b+e+f+h)=55 or 31.1%±3.5%; *lg2*-T (c+e+g+h)=15 or 8.5%±2.1%; T-*a1* (d+f+g+h)=19 or 10.7%±2.3%

Conclusion: The breakpoint (T) on 3L lies between *lg2* and *a1*.

the adjacent loci are understated. When translocations are heterozygous with normal chromosomes there is generally a reduction in crossing over near the breakpoints.

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New meiotic mutations isolated from *Mu* and *Ac* stocks

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The goal of this experimental work was the isolation of new tagged mutations of maize meiotic genes. For this purpose 584 families of 20 kernels each were planted from self-pollinated ears produced with active Mutator stocks. In addition 150 families of 20 kernels each were planted from self-pollinated ears of *Ac* stocks. The *Ac* material was produced in the previous generation by planting kernels that were selected on the basis of containing a transposed *Ac* element. These materials were planted in the field in Grand Forks in 1992 and checked for segregation for male sterility.

We have isolated 75 families from the *Mu* background belonging to 15 independent sources and 15 other unique families from the *Ac* background that segregated male sterile plants. Twenty kernels from each of these families were planted in the Hawaii winter nurseries. The young tassels of 6-15 plants were sampled in 56 families derived from the *Mu* background which belonged to 10 independent sources and in 8 families from the *Ac* background. Tassels were fixed in Farmer's fixative (3:1), and all sampled plants were grown to flowering. During pollen shedding the phenotype (fertile/sterile) of each plant was checked in the field, and microsporocytes from fixed tassels of each sterile plant were examined under the light microscope.

Five new meiotic mutations were identified: four induced by *Mu* and one by *Ac*. One of the *Mu* background families segregated sterile plants with an ameiotic pattern of meiosis, eight families (selfed sibs) segregated sterile plants with a severe desynaptic phenotype. Three families (selfed sibs) segregated sterile plants with meiocytes stopped in different stages of meiosis from prophase I onward. In spite of the complete formation of the pollen envelope, we could easily identify at what stage was meiosis stopped. Finally, two families (selfed sibs) segregated sterile plants with abnormal segregation of sister chromatids at the second meiotic division. Meiosis of sterile plants was normal before metaphase II and anaphase II and exhibited no visible abnormalities prior to these stages. The meiotic phenotype of these sterile plants was similar to that described in *Drosophila melanogaster* mutation *S 332a* (Davis, MGG 113:251, 1971; Goldstein, Chromosoma 78:79, 1980).

The one family from the *Ac* background segregated sterile plants with abnormally condensed chromosomes at metaphase I. This phenotype was the same as that described for the dominant meiotic mutation designated as *Mei 025* (Golubovskaya, Adv. Genet. 26:149, 1989).

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Effect of cadmium on in organello mitochondrial DNA, RNA and protein synthesis

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Studies on molecular biology of mitochondrial DNA (mtDNA) may elucidate the role of this organellar genome in the whole genetic system of the maize cell. Increasing concentrations of Cd in the industrial environment have drawn attention to the toxic effect of this heavy metal ion on properties of the cell genetic apparatus. Little is known about the influence of Cd on the genetic system of mitochondria. In this connection it appears reasonable to examine the effects of various concentrations of Cd on activities of DNA, RNA and protein synthesis in isolated mitochondria.

The mitochondria were isolated from 3-day-old etiolated maize seedlings (hybrid Krasnodarsky 362 ATV) by a standard method of differential centrifugation. Mitochondrial protein was determined by the Lowry method. The DNA was synthesized in mitochondria according to the method of Schegget and Borst (Biochim. Biophys. Acta 95:235-248, 1971) with the use of ^{32}P-dATP (specific radioactivity is >111 PBq•mol^{-1}). The conditions of the RNA synthesis in mitochondria were essentially similar to those described elsewhere (Carlson et al., Curr. Genet. 11:151, 1986). The kinetics of the mtRNA synthesis was registered by ^{32}P-UTP (specific activity is >74 PBq•mol^{-1}. The protein synthesis in mitochondria was registered with ^{3}H-labelled amino acids (Forde et al., PNAS 75:3841-3848, 1978). Cadmium was added as $CdCl_2$ to concentrations of 10 to 250μM. The kinetic data were obtained from at least 3-4 experiments.

In the course of our investigations on the effect of cadmium on in organello mitochondrial DNA synthesis, we observed that Cd concentrations of 10 to 150μM had a slight stimulatory effect on this kind of mitochondrial genome activity, while 250μM resulted in a significant decrease of ^{32}P-dTTP incorporation in the mtDNA (Fig. 1).

Figures 2 and 3 show the influence of various Cd concentrations on the activities of RNA and protein synthesis in isolated mitochondria. All Cd concentrations concerned of 10μM to 250μM proved to be inhibitory in a dose-dependent manner.

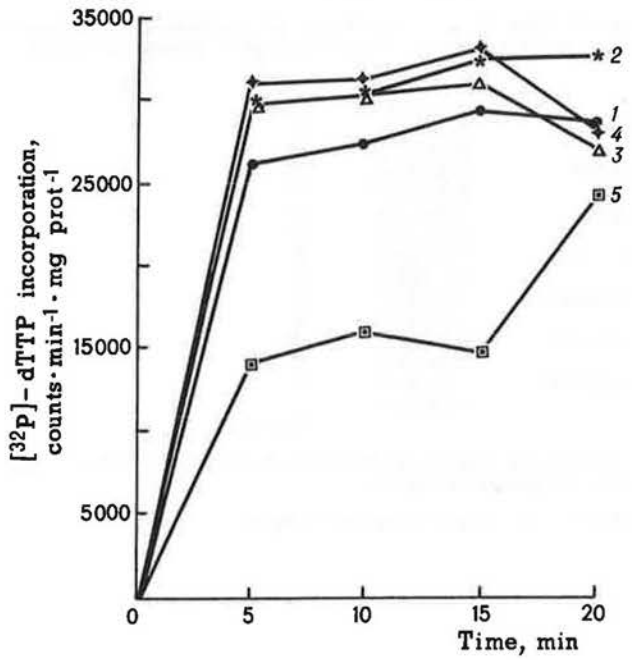

Figure 1. Kinetics of mtDNA synthesis in isolated mitochondria in the presence of different concentrations of Cd. 1, control; 2, 10μM Cd; 3, 50μM Cd; 4, 150μM Cd; 5, 250μM Cd.

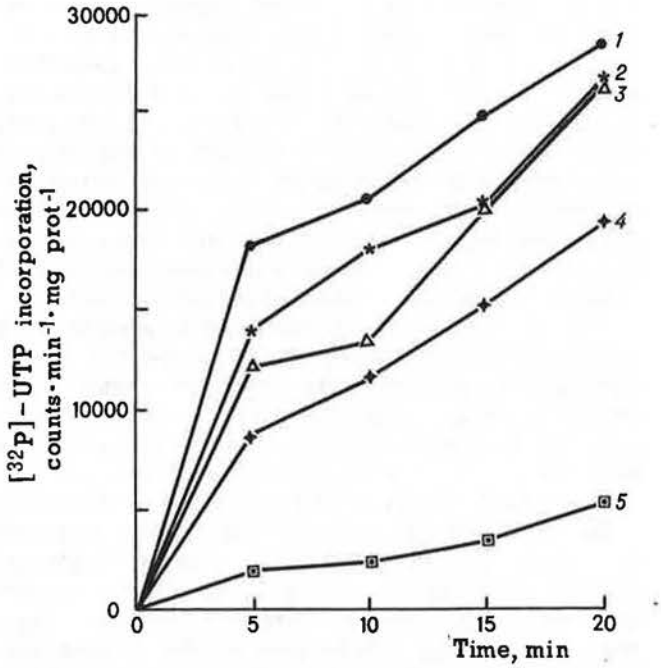

Figure 2. Kinetics of mtRNA synthesis in isolated mitochondria in the presence of Cd. The designations are the same as in Figure 1.

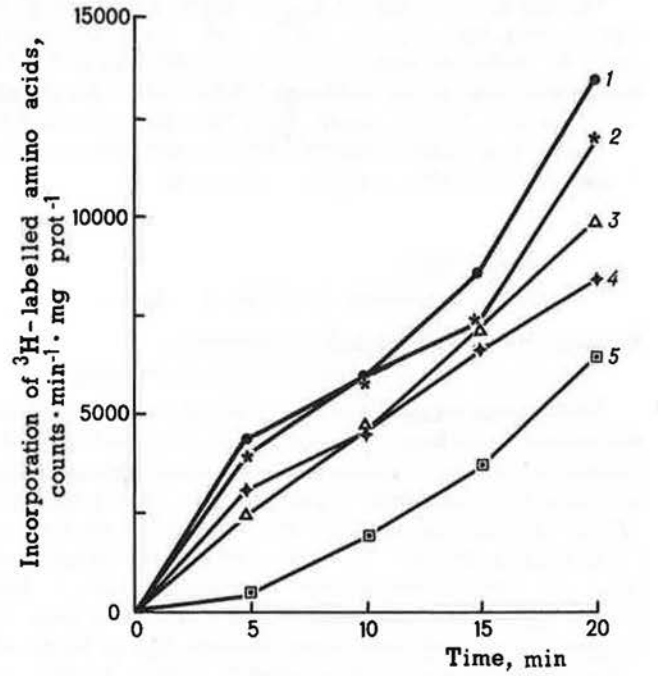

Figure 3. Kinetics of protein synthesis in isolated mitochondria in the presence of Cd. The designations are the same as in Figure 1.

Page et al. (J. Environ. Qual. 1:288-291, 1972) reported the uptake of cadmium from solutions by corn and other crops. Concerning the effect of various concentrations of cadmium on the genetic system of plants cells, Hirt et al. (Planta 179:414-420, 1989) showed that Cd concentrations of 100-150μM stimulated growth of cell cultures of *Nicotiana tabacum* L., whereas all other concentrations were inhibitory. Interestingly, RNA and protein syntheses were responsible for the revealed stimulation of cell culture growth. Our data suggest that low Cd concentrations of 10μM to 250μM could affect the mitochondrial genetic processes. The Cd concentration of 250μM had the most pronounced inhibitory effect on all three types of mitochondrial macromolecule synthesis. The apparent similarities between the inhibitory effect of Cd on mitochondrial RNA and protein syntheses imply that Cd affects primarily the rate of mtRNA synthesis and hence the effective concentration of genetic templates available for protein synthesis. The data obtained illustrate high sensitivity of the mtDNA to the deleterious effect of this heavy metal ion. We propose that the mitochondrial genome of corn seedlings is possibly one of the targets of cadmium toxic effects on the plant cell.

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Knob DNA in relation to combining ability

--Sajjad R. Chughtai, Habib I. Javed, Haq Nawaz Malik, M. Aslam and Dale M. Steffensen

Knobs are large blocks of highly repeated (heterochromatic) DNA found at fixed chromosomal locations of maize and its close relatives in the Maydeae. Knob DNA is composed of a tandemly arranged 180-base pair repeat. Knob frequency is negatively correlated with latitude and altitude. Different races (and even inbreds) possess different knob compositions.

Knobs have extensively been utilized for characterizing the races of maize. They have been shown to be associated with a large number of agronomic features of maize, including maturity and yield. Several studies indicate that knob frequency is closely associated with combining ability of maize genotypes. Generally, the best hybrids result from crossing high (or medium)-knob genotypes with knobless or low-knob genotypes. Interestingly, Reid's Yellow Dent and Lancaster Sure Crop, two germplasms with maximum contribution to hybrid corn in the U.S.A., both originated from crosses between high-knob southern dents and low-knob northern flints.

Recently, Crossa, Taba and Wellhausen (Crop Sci. 30:1182-1190, 1990) published extensive data on hybrids among 25 Mexican races of maize. Over 300 hybrids were evaluated at 3 different altitudes in Mexico during 1963 and 1964. Similarly, extensive data on the knob composition of maize from the Americas have been published by McClintock et al. (Chromosome Constitution of Races of Maize, Colegio de Postgraduados, Chapingo, Mexico, 1981). We have combined these yield and knob data to see if knob constitution is related to hybrid performance. A summary of these analyses is presented here.

At high altitude the top 40 hybrids have, on the average, more heterozygous (5.93) than homozygous (3.66) knobs, while the reverse is true for the bottom 40 hybrids (8.33 homozygous and 3.53 heterozygous knobs) (Table 1). At low and medium elevations, the top 40 hybrids have a higher number of homozygous (7.85 and 6.75) than heterozygous (3.55 and 3.63) knobs, while the reverse is true for the bottom 40 hybrids (5.4 and 4.90 heterozygous, and 2.25 and 3.60 homozygous knobs, respectively). In other words, knob heterozygosity is directly related to hybrid vigor. Thus, it is the knob condition (homozygous or heterozygous) which is related to combining ability rather than the frequency of

knobs.

Among the top 40 hybrids, those between lowland and midland (LxM) races at low (48%), MxM at medium (35%), and MxH at high altitude (67%) were the most frequent (Table 2). Among the bottom 40, MxH (55%), HxH (32%) and LxL (43%) at these altitudes, respectively, were the most frequent. Thus, the racial composition of the top 40 hybrids is different from those of the bottom 40 at each altitude.

Table 1. Mean yield and number of homozygous and heterozygous knobs in hybrids among Mexican races of maize.

		Number of knobs[A]		
Altitude (m) of test site	Hybrid ranking	Homozygous	Heterozygous	Yield[B] (mg/ha)
High (2249)	Top 40	3.66	5.00	6.02
	Bottom 40	8.33	3.53	1.52
Medium (1800)	Top 40	6.75	3.63	7.77
	Bottom 40	3.60	4.90	3.48
Low (1300)	Top 40	7.85	3.55	5.92
	Bottom 40	2.25	5.40	2.52

[A]The knob data have been computed on the basis of knob composition given by McClintock et al. (1981).

[B]The yields for the top 40 hybrids are from Crossa et al. (1990) while those for the bottom 40 are from their unpublished data.

Table 2. Racial percent composition of hybrids among Mexican races of maize.

		Racial composition (%) of hybrids[A]					
Altitude (m) of test site	Hybrid ranking	LxL	LxM	LxH	MxM	MxH	HxH
Low (1300)	Top 40	20	48	10	15	7	0
	Bottom 40	0	0	8	2	55	35
Medium (1800)	Top 40	3	25	12	35	25	0
	Bottom 40	13	13	13	2	27	32
High (2249)	Top 40	0	3	20	5	67	5
	Bottom 40	43	38	7	5	7	0

[A]The races were classified as lowland (L), midland (M) and highland (H) on the basis of the highest yield obtained at a location by Crossa et al. (1990).

These data also indicate that highland races combined well only at the high altitudes (in 92% of the top 40 hybrids) but not at low and medium altitudes (in 98% and 72% of the bottom 40 hybrids, respectively). The lowland races combined well at the low altitude (in 78% of the top 40 hybrids) but not at high altitude (in 88% of the bottom 40 hybrids). The midland races combined well at all locations, being involved in 70%, 85% and 75% of the top 40 hybrids at low, medium and high altitudes, respectively. That midland races combined well at high altitude suggests the potential utilization of the subtropical maize material in temperate regions. This may be of great interest to maize breeders in the U.S.A. and other temperate regions of the world. However, in such hybrids the other parent would essentially be a low-knob genotype adapted to the cool environments. The observation that highland races combined poorly at low and medium altitudes suggests that temperate material may not be useful for hybrid production in the tropics and subtropics.

The question is, how can we explain the relationship between knob condition and hybrid performance? Or in other words, why are knob heterozygotes adapted to the highland areas, and knob homozygotes to the lowland and midland areas? Our studies (S. R. Chughtai, Ph.D. Thesis, University of Illinois, 1988) showed that knob homozygosity delays plant development. The maturity of the hybrid coincides with the knobless genotype regardless of the knob number and maturity of the other parent. This indicated the absence of the delay effect of the knob in the heterozygous state. We hypothesized that knob DNA affects development by controlling the expression of the bracketing genes by a cis-acting position effect. Thus, knob heterozygotes, due to their early maturity (like the low-knob genotypes), are well adapted to the temperate environment where the growing seasons are short and the temperatures are low. However, they are not adapted to the tropical and subtropical environments since, because of their early maturity, they cannot avail themselves of the full length of the long growing season. Knob homozygotes, on the other hand, are adapted to such warmer climates due to their late maturity because they can avail themselves of the full length of the growing season. Due to their late maturity, they are, however, not adapted to the cooler climates where low temperatures delay plant development and knob homozygotes are unable to mature during the growing season.

In cooler climates, the early maturity of the knobless or low-knob genotypes can be coupled with the high yield of the late-maturing high-knob genotypes. Thus the linkage between maturity and yield is broken. Apparently, this is the secret behind the success story of hybrid maize production in the U.S.A. and other temperate regions. This may also explain the relatively limited success of hybrid maize in tropical and subtropical regions of the world. The tropical and subtropical maize varieties are generally intolerant to inbreeding. We believe that selection of early vigorous plants in the tropics is over-emphasized. This leads to selection of knob heterozygotes and ultimately of low-knob genotypes which are poorly adapted to the warm climates. In these areas, selections for acceptably late maturing plants should be emphasized to ensure knob homozygosity (Chughtai and Steffensen, SABRAO J. 21:21-26, 1989).

The new evidence strongly supports earlier contentions (T. A. Kato Y., Mass. Agric. Exp. Sta. Bull. No. 635, 1976; S. R. Chughtai and D. M. Steffensen, Maydica 32:171-187, 1987) that knob heterochromatin plays an active (though indirect) role in the adaptation of maize to its environment. Thus, there is a great need for considering knob heterochromatin in breeding and improvement of maize adapted to different regions of the world.

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Research on selected high quality protein hybrids

--Z. X. Liu, S. F. Jia, Q. F. Guo, J. F. Xu and L. M. Wang

Better normal maize inbred lines were crossed with *o2* (floury endosperm) inbred lines, by means of inbreeding, backcross and many other methods, to develop new and excellent agronomic characteristics in *o2* inbred lines. In order to overcome such defects of *o2* lines as lower grain yield and floury endosperm, the lines selected were crossed with high quality protein maize populations, which carry the *o2* gene with modified endosperm texture. After several generations of inbreeding and selection of hard endosperm, we obtained maize inbred lines with high quality protein and modified endosperm texture, which make them indistinguishable from other normal flint lines.

The best lines obtained have an average lysine content of from 0.35% to 0.42% in whole kernels, which is an increase of 75% to 110% over normal lines. The protein content is from 8.7% to 11.2% and similar to normal lines. For example, the protein content of QI205 is 10.2% and its lysine content is 0.4% in whole kernels. It has outstanding agronomic characteristics and has been used in the production of hybrids.

We bred high quality protein maize hybrids with not only higher lysine content, but also higher grain yield. For instance, LU-

DAN203 was crossed by two excellent *o2* lines (ZHONGXI091 and QI205). The hybrid has a higher grain yield of 8,500 kg/ha in general, and the maximum grain yield will reach more than 12,750 kg/ha. In 1990 and 1991, average grain yields of LUDAN203 were 8,325 and 8,355 kg/ha respectively in the state regional test across 18 experimental sites, with increases of 21.8% and 18.5% over normal maize hybrid ZHONGDAN2 (6,833 and 7,065 kg/ha) as check. The normal maize hybrid once obtained the highest invention prize of our country. Protein and lysine content in whole kernels of LUDAN203 are 9.8% and 0.40%. It also has excellent agronomic characteristics and has received wide utilization. Recently, the planted area of this variety has reached more than 6,000 hectares.

A significant feeding effect of the hybrid was obtained when LUDAN203 was used as feed for hens and pigs in 1991. The daily increase of gross weight and the rate of egg production of test hens rose by 20% and 26.8% respectively over normal maize check hybrid (YEDAN4). The daily increase of gross weight of test pigs rose by 35% over the normal maize check hybrid.

The results obtained indicate that in the *o2* genetic background it is effective to select high quality protein maize hybrids with modified endosperm texture (hard endosperm) so as to obtain higher grain yield. The effectiveness is higher in flint maize than in dent maize due to the fact that in the former it is easier to develop the modified endosperm texture kernel than in the latter.

Nevertheless, we believe the characteristic of multiple rows of the ear is important to increase kernel number, and to obtain higher grain yield in the selection process. For example, LUDAN203 has more than 800 kernels per ear. This is an easy way for the intercession of the negative correlation between high quality and high grain yield.

In addition, the lysine content in kernels has been taken into account in selecting lines of the high quality protein and modified endosperm texture. As a matter of fact, there is still room for improvement in our work. We hope to develop high-yielding hybrids with high quality protein and hard endosperm in the near future.

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Two new alleles to male-sterile mutants *ms2* and *ms8*

--M. R. Trimnell and M. C. Albertsen

Two new male-sterile mutant alleles were identified in elite proprietary breeding material. MCA observed a selfed progeny row segregating for male-sterile plants at our research station at Garden City, KS, in 1987. We grew remnant seed in Johnston, IA, in 1988 and observed a segregation of 11 male fertiles to 6 male steriles. Two male-sterile plants were sibbed with two male-fertile plants in the row, resulting in the following:

Ear #1	7 male fertiles	7 male steriles
Ear #2	12 male fertiles	1 mostly fertile (shedder)

The 7 male-sterile plants were testcrossed with known heterozygotes of the male-sterile mutants listed in Table 1. Progenies were grown in Johnston in 1989. At least 15 plants were observed per row. As shown in the table, there was a segregation of 10 male fertiles to 10 male steriles among the *ms2* testcross progeny. Our designation for this new allele is *ms2-GC87A*.

The other allele was observed in our Hawaii winter nursery in 1987 by Mr. George Peverly. He observed a selfed progeny row segregating for male sterility from our research station at York, NE. The male-sterile plants were crossed with A632, and progenies were grown in Johnston in 1988. All plants were male fertile, so self pollinations were made. The next summer, progeny from two ears were grown with the following results:

Ear #1	13 male fertiles	2 male steriles
Ear #2	17 male fertiles	1 male sterile

We decided to conduct the allelism tests even though the X^2 value was marginal (4.45; .05> P >.025). Only three male-sterile plants could be used in testcrosses, so fertile plants were selfed to determine heterozygosity and testcrossed with male steriles from known male-sterile plants. These progenies were grown in Johnston in 1989, and results are given in Table 1. At least 15 plants were observed per row. Allelism was observed when *ms8* was used as a heterozygous male. Segregation was 9 male fertiles to 8 male steriles. Our designation for this new allele is *ms8-YK87A*.

Table 1. Testcrosses made to two spontaneous male-sterile mutations occurring in elite proprietary breeding material.

Known male sterile	*ms2-GC87A*	*ms8-YK87A*
ms1	All Fertile	All Fertile
ms2	10 fert : 10 ster	All Fertile
ms5	All Fertile	--
ms7	All Fertile	--
ms8	All Fertile	9 fert : 8 ster
ms9	--	All Fertile
ms10	--	All Fertile
ms11	--	All Fertile
ms12	All Fertile	--
ms13	--	All Fertile
ms14	All Fertile	--
ms17	--	All Fertile

Description and mapping of the tassel-less (*tls1*) mutation

--M. C. Albertsen, M. R. Trimnell and T. W. Fox

Woodworth (J. Hered. 17:405-411) published a description of a "barren-sterile" mutant in 1926. This mutant was described as exhibiting an "inhibition...of sex, since neither tassel nor ear is produced". Because neither a tassel nor an ear was produced, it was described as being both "barren" and "sterile". The mutant was designated *bs1*. The mutation was never mapped and seed stocks apparently were lost. MCA was given seed stock for a similar mutant in 1976 by Dr. Robert Brawn, then of Funk Seeds International. Dr. Brawn had referred to the mutant as "tasselless". This mutant was derived from Minn Syn 3 and segregated mostly as a single gene recessive. In its original nuclear background, plants homozygous for the mutation were distinguishable at the 4-8 leaf stage. These plants appeared more pubescent than normal sibs and the leaves had a leathery look and texture, which seemed to impart an olive-green cast to the plants. In other nuclear backgrounds, e.g. A632, these differences are greatly diminished. Homozygous mutant plants generally do not develop tassels, although ear shoots are produced. Generally, however, no ears develop within the husk tissue. Mutant expression is variable, especially among progeny from selfing a heterotic cross. Tassel-like structures can be obtained that vary from having only a couple of spikelets to having a more complete looking tassel. Ear development also can vary from a structure on which only a few kernels develop to a structure that resembles a 'Strawberry Pop' type of ear. The amount of ear development is not necessarily correlated with the amount of tassel development, nor vice versa.

We chose to designate this mutation as tassel-less, *tls1*. It is most likely the same mutation as that described by Woodworth, as

his J. Hered. photographs and description match our mutant exactly. Still, without the original seed stocks, we could not make the allelism crosses. We chose to call the mutation *tls1* because it seemed generally to best describe the most striking feature of the mutation, that of a missing tassel.

We crossed homozygous mutant plants that had been selected to produce small 'Strawberry Pop'-type of ears as female with an A-B interchange series. F1 progenies were grown in our Hawaii nursery in 1991. Although all chromosome arms were not covered, we determined that the likely location for the mutation was on chromosome 1L (Table 1). We subsequently interval mapped this

Table 1. Location of *tls1* to chromosome 1L using homozygous TB-1Lc crossed onto homozygous *tls1* plants selected for ear development.

No. hypoploid plants	No. *tls1* hypoploids	No. normal plants
11	11	80

mutant using the following RFLP probes on chromosome 1: *bnl8.10*, *bnl7.25*, *bnl8.29*, *php15058*, *php20557*, *bnl6.32*. Twenty normal sibs and 12 *tls1* plants were bulked by phenotype. Differential polymorphisms were observed with probes *bnl8.29*, *php15058*, *php20557*, and *bnl6.32*. This agrees with the A-B interchange results and locates the mutation to the distal one third of chromosome 1L.

Disease lesion mimics

--Guri Johal* and Steve Briggs
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Disease lesion mimics are a class of mutants that promote the production of discrete leaf lesions in the absence of obvious stress, injury or disease on the plants. These are named disease lesion mimics because each mutation causes symptoms that resemble some known pathological condition of maize (Walbot, Hoisington and Neuffer, in Genetic Engineering of Plants, eds. Kosuge et al. p. 431, 1983). Both spontaneous and mutagen-induced cases of recessive and dominant lesion mimic mutations have been reported. A majority of these mutations are dominant. Nothing is known about the nature of these mutations, but it is likely that they represent defects in either the plant's recognitional system or in mechanisms that regulate symptom development during infection.

Lesion mimics constitute excellent models for plant cell-cell interactions. Three issues can be directly addressed by cloning and characterizing lesion genes: how are signals initiated, propagated, and terminated? The large number of mutants in maize suggests that there may be pathways for lesion development. This should be a fruitful avenue for genetic investigation. While such studies are of a basic nature, they have the added value of potentially elucidating the process of infectious disease development.

We are particularly interested in *lethal leaf spot* (*lls1*), a recessive mutation which mimics the symptoms produced by race 1 of *H. carbonum* on susceptible maize (Ullstrup and Troyer, Phytopathology 57:1282, 1967). The expression of *lls1* is developmentally programmed; there is a progression of lesion formation, beginning first in the most mature and advancing toward the youngest tissues. Lesions first appear near the tips of the oldest leaves about three weeks after seedling emergence and then spread and enlarge to kill the plant just before or shortly after pollen shed. Thus, the developmental distribution of lesions on the plant, as well as the development and appearance of individual lesions, mimics closely disease caused by *H. carbonum* race 1.

A project has been initiated to tag *lls1* with Mutator (*Mu*). Both the targeted and random-mutagenesis approaches were taken. Seven *Mu*-induced alleles (including two that are mutable) have been isolated. These mutants have been advanced three generations and are now ready to be subjected to cosegregation analysis.

In the course of this and the *Hm1* cloning project, 16 cases of a dominant lesion mimic mutation, designated *Les*-2552*, were recovered from Mutator lines. Phenotypically they all look alike, and four of them have been shown to be allelic. This mutation differs from *lls1* in that the lesions are arrested in their growth while still small (~1mm). A number of these *Les*-2552* alleles have been advanced 3-4 generations to reduce the number of background *Mu* elements.

Both *lls1* and *Les*-2552* are cell-autonomous. Somatic clonal sectors were observed either as forward mutations or reversion events for both mutations. These sectors may be the result of somatic transposition of *Mu* during development. A cloning strategy based on the use of these mutant sectors is under investigation. Briefly, the *Mu*-hybridization pattern of DNA from a mutant sector is compared with the DNA from adjacent wildtype tissue. Any extra band in the mutant DNA may represent the *Mu* insertion responsible for the mutation. Using this approach, a *Mu1* hybridizing 6.0kb fragment was identified from the *lls1-1424* mutant and association of this restriction fragment with the mutant allele was confirmed by cosegregation analysis in progeny derived from this mutant. To incite early (large) revertant sectors, which will be critical for the strategy outlined above, both *lls1* and *Les*-2552* mutants have been crossed with Ginny Walbot's 'big spot' Mutator line carrying *bz2-Mu2* (MNL65:96, 1991).

Altered HC-toxin reductase activity in transgenic BMS cultures

--Bob Meeley, Keith Lowe, Dave Songstad, Guri Johal , Joanie Phillips and Steve Briggs

In maize, tolerance to the cyclic peptide HC-toxin is the basis of resistance to the fungal pathogen *Cochliobolus carbonum* Nelson race 1. The toxin is required by the pathogen for successful colonization of susceptible maize tissues. The resistance gene *Hm1* (located on 1L) encodes the enzyme HC-toxin reductase, which inactivates HC-toxin by carbonyl reduction. Due to the action of HCTR, resistant maize lines are 100-fold more tolerant to HC-toxin, and are able to avoid infection by *C. carbonum*. HC-toxin induces a variety of biological responses in maize. In callus cultures, effective doses of HC-toxin cause necrosis and cell death. This toxin is being tested for use as a selectable marker in maize transformation. Nearly all commercially relevant maize lines carry the *Hm1* allele, therefore our selection method relies on over-expression of HCTR activity.

The *Hm1* gene has been cloned and sequenced. A vector containing the *Hm1* cDNA fused to the CaMV 35S promoter was used in conjunction with a second plasmid containing the *BAR* (herbicide "Basta" resistance) and *GUS* (β-glucuronidase) genes. BMS cotransformants containing these constructs were generated by microprojectile bombardment. HC-toxin reductase activities were measured in 22 independent Basta-resistant transformants and 4 controls (transformed with *BAR* only). Relative to the controls, over-expression of HCTR activity was detected in 19 out of 22 transformants. The range of HCTR expression was grouped into classes: transformants expressing 10- to 20-fold higher HCTR

activity (3), 5- to 10-fold higher (9), and 2- to 5-fold higher (7). Two transformants had no detectable increase in HCTR activity. The final transformant from the group of 22 had a five-fold reduction in HCTR activity compared to controls. This transformant is a candidate for the phenomenon of co-suppression.

Representatives from each transformant class, including the co-suppressed colony and controls, were tested for sensitivity to HC-toxin in a callus bioassay. Samples (50mg) from each colony were suspended in MS salts containing Basta (5µg/mL), and varying doses of purified HC-toxin. After several days in suspension at 30 C, the toxin response was evaluated by necrosis, a reduction in the suspension culture fresh weight, and a loss of *GUS* activity. Based on these criteria, transformants displayed tolerance to elevated levels of HC-toxin, consistent with the findings from the HCTR assays. The co-suppressed colony was considerably more sensitive to HC-toxin than the controls. Presently, HC-toxin is being tested as the primary selection agent for a fresh set of 35S-*Hm1* transformants.

Cloning gibberellic acid biosynthetic genes from maize using transposon tagging

--Robert Bensen, Guri Johal, Pat Schnable* and Steve Briggs
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Andromonoecious dwarfs, phenotypic for GA-deficient and GA-insensitive plants, have been identified in Mutator and *Ac-Ds* lines. Allelism tests demonstrated *an1, d1,* and *d5* mutants to be among the identified dwarfs. A co-segregating DNA fragment which contains *Mu2* was observed in the dwarf *an1-891339*. This fragment was cloned and a 2.6kb flanking sequence and subcloned. Both genomic and cDNA clones were selected using the flanking sequence as a probe. The allelic deletion mutant, *an1-6923*, lacks DNA homologous to either the flanking sequence or the selected cDNA. Northern analysis suggests a 3.5kb mRNA for the gene. Sequence analysis of the genomic and cDNA clones indicates that *Mu2* is inserted within an exon or at an intron-exon border. With 2.5kb of the message sequenced a number of introns have been identified, but no homology to any published sequence is evident.

Is our 'heartbreaker' a 'tourist'?

--Guri Johal, Pam Close and Steve Briggs

'Heartbreaker' is the designation given to an insertion found in one of the tagged *Hm1* mutants. This mutant, *hm1-1062::dHbr*, was isolated during directed-mutagenesis of *Hm1* by Mutator and was the first allele to be characterized in detail. A *Mu1*-hybridizing band was found to co-segregate with the mutant allele and was cloned. However, when mapped at higher resolution, it was found to be present ~5cM away, on the proximal side, from the *Hm1* gene. This disheartening result led us to designate the *hm1-1062::dHbr* mutant as the heartbreaker mutant and the agent (an insertion) that caused this mutation, a 'heartbreaker'.

The *Hm1* gene was eventually cloned with the help of other mutations. Southern blot hybridizations of the *hm1-1062::dHbr* DNA with a gene-specific probe identified a small insertion in the 3' end of the gene. A 3.1kb *Xho*I fragment containing the insertion was cloned and sequenced. A 314bp insertion, absent in the progenitor allele, was recognized in the mutant allele. The overall structure of this insertion was reminiscent of a transposable element but lacked homology with any known transposable element systems. This insertion has a 14-17bp imperfect terminal inverted repeat and a flanking three base pair duplication. The database search located a highly homologous (>90% identity), 314bp sequence present 3' of the wildtype *waxy* gene (thanks to Alfons Gierl for providing the complete sequence). A similar but diverged sequence was also found in the first intron of the *P* gene.

A highly repetitive pattern was observed on Southern blots of maize DNA but no cross hybridization was detected with sorghum and *Arabidopsis* DNA. A cDNA library from B73 was probed with 'heartbreaker' and 30 different cDNA clones were isolated. DNA restriction and hybridization analyses have suggested these clones to be unique and probably represent 'heartbreaker' co-transcripts since all the clones contain at least one of the TIRs.

Recently, a new transposable element has been isolated in Sue Wessler's laboratory which identified a new transposable element system, named 'tourist' (Bureau and Wessler, Plant Cell 4:1283, 1992). Our 'heartbreaker' seems to belong to this system. Out of the 14 bases that constitute the 'tourist' TIR, 11 are identical in 'heartbreaker'. In addition, like 'tourist', 'heartbreaker' causes a three base pair duplication of the target site and has the same target site specificity, i.e., TAA. However, the size and internal sequences are completely different in these two element families.

Vestigial glume : tunicate interaction

--Guri Johal and Steve Briggs

Two dominant mutations in maize, *vestigial glume* (*Vg1*) and *tunicate* (*Tu1*), affect the same plant part (glumes) but in opposite ways. In *vestigial glume*, the glumes are much reduced in both the male and female inflorescence. In addition, the *vestigial glume* mutant is liguleless.

Tunicate, on the other hand, greatly increases the size of glumes on both the male and female spikelets resulting in complete encasement of kernels. The ligule is not affected.

A double heterozygote of *vestigial glume* and *tunicate* was constructed to find how these two mutations interact. The phenotype of the double heterozygote is intermediate between the two extremes exhibited by either mutation but is still quite different from the normal plant phenotype. On the ear, *Tunicate* seems to have the upper hand. The kernels are still half to fully covered depending upon where they are on the ear. Kernels near the butt of the ear have larger glumes than the kernels located near the tip of the ear. In tassels, the effect of *vestigial glume* seems to dominate. The glumes are about half the size of wildtype and, as a result, the anthers tend to protrude out of the spikelets. In contrast, the ligule is completely suppressed in the double mutant. Clearly, these mutations are antagonistic to each other only in the inflorescence and the action of *Tu1* appears to be restricted to the inflorescence. To gain further insight into this interaction, stocks containing different doses of *tunicate* and *vestigial glume* are being constructed.

Identification of new male sterile-silky gene(s)

--Pamela Close* and Steve Briggs
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The silky (*si1* or *ms-si*) mutation was first reported in 1933 by A. C. Fraser. Ears are phenotypically normal except for the presence of up to three additional silks around the base of each kernel. These silks persist long after fertilization of the ear. The additional silks arise from the tips of three "paddle-like" protuber-

ances which develop in a whorl around the kernel. These "paddles" are carpel-like in appearance. The whorl is within the first flower of the spikelet and appears to be at the position where stamens would develop if maize produced a perfect flower. Silks are present on the tassels, which are typically male-sterile. No seed is produced by the tassel.

We have observed at least two new silky mutations within our active Mutator lines. These mutants are of independent origin and are phenotypically indistinguishable from the *si1* (*ms-si*) tester stock obtained from the Maize Cooperation Stock Center. Allelism tests repeated over two seasons indicate that neither of these two mutations is allelic to *si1*. We are currently testing whether they are allelic to each other. Expression of both ear and tassel phenotype of one of the new silky mutations varies considerably with genetic background.

The new silky mutations are currently being screened for co-segregating Mutator-containing restriction fragments.

To investigate gene interactions, we are making crosses to combine the new silky mutations with other mutations affecting the development of the maize flower. Since gene duplication is common in maize, *si1* and the new silky mutation(s) may encode the same function. Alternatively, two genes have been identified in *Arabidopsis* (*apetala-3* and *pistillata*) which cause a similar transformation of the stamen whorl to carpel. It is possible that *si1* and the new silky mutations may be functional homologs of these genes.

Bar gene as a selection marker for maize transformation

–Z. Zhao, K. Lowe and W. Marsh

The *Bar* gene has been widely used as a selectable marker for plant transformation. Transgenic cells and plants expressing this gene are resistant to the herbicides Basta (registered in Europe), Bialaphos (registered in Japan) and Ignite (registered in the USA).

A reliable selectable marker is essential for the recovery of transgenic plants at the present time. When a transgenic plant is crossed with a non-transgenic plant, segregation of the transformed gene(s) in the next generations is expected. A screenable selectable marker linked to an agronomic gene(s) can make the identification of transformed progeny efficient and fast. The data presented here indicate that the *Bar* gene is ideal for this purpose. Large or small scale screening to identify transformants can easily be done in greenhouse and field settings. Herbicide screening has been used to successfully identify transformants, and these results have been confirmed by Southern analysis.

The herbicide Basta is considered a non-selective herbicide, and is toxic to all non-transgenic plants. To determine the correct dose of Basta, seedlings from three maize genotypes (B73, Mo17 and W22) were treated at the 3-5 leaf stage. The seedlings were sprayed with different concentrations of Basta solution and scored for herbicide damage as shown in Figure 1. From these results, it has been noticed that three major factors, genotype, herbicide concentration, and the environment, influence the response of maize plants to Basta.

Based on these results, seedlings carrying the *Bar* gene, grown as above, were sprayed with Basta solutions ranging from 4.5 L/ha. to 8 L/ha. These seedlings were resistant to all of the treatments (Fig. 1).

Seed was produced from *Bar*-containing plants pollinated with non-transgenic pollen representing three independent transformation events (making up three families). The original plants representing these three events indicated varying levels of *Bar* enzyme activity, from high (1,518 units) to low (635 units, control=476 units). However, the resistant seedlings representing these three transformation events (families) showed equal resistance to these herbicides at all concentrations. When older plants (7-10 leaves) were sprayed in the same manner occasional lesions were observed but these did not affect plant development. *Bar*-containing adult plants painted with 1% Basta solution to an area of their leaves usually showed very good resistance with minimal damage, correlating to *Bar* enzyme activity. However, painting 3 inches of a leaf tip of a non-transgenic adult plant with 1% Basta solution will result in necrosis of the painted area or in some cases death of the entire plant.

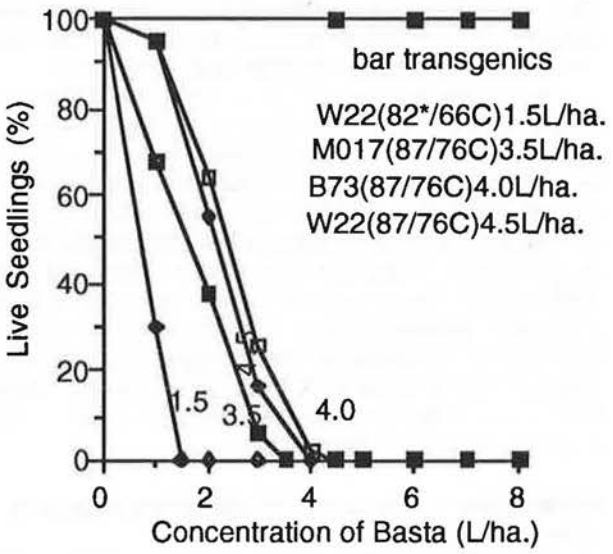

Figure 1.

Since young plants (3-5 leaf stage) carrying the *Bar* gene are completely resistant to herbicide spraying we recommend screening plants at this stage of growth.

To confirm the reliability of herbicide screening for identification of *Bar* containing plants, genomic Southern hybridization was done on randomly selected plants segregating for the *Bar* gene. Southern analysis (using the *Bar* coding region as a probe) from a random sampling of 45 out of 1447 segregating seedlings confirmed 33 out of 45 plants contained the *Bar* gene, and only these plants were resistant to Basta.

In summary, the herbicide spraying (Basta/Ignite) of young maize seedlings is a very dependable and economic method that can successfully identify *Bar*-containing plants.

Standardization of Pioneer Hi-Bred RFLP probe nomenclature

--Debra L. Blair

We have recently begun an effort to standardize the nomenclature of biological material from Pioneer Hi-Bred to ensure that it

is consistent with the rules developed by the Maize Nomenclature Committee. We have made every effort to keep this system both informative and easy to use--two things not always easy to unite! We will be adopting a three letter prefix for all material. The first two letters "ph" denote Pioneer Hi-Bred. The third letter denotes the type of material being described: for example, a third letter of "p" will be used to denote cloned DNA. We will be establishing a glossary for all third letter codes in 1993 and will submit it to the 1994 Newsletter.

We find it is necessary to alter the probe names used on our previously published maps to accommodate this nomenclature standardization. We will no longer use the designator "pio", but will now use "php". We have also dropped the central 0 (zero) in our number to help streamline the name. By making these two changes, we hope to keep our naming convention consistent with that agreed upon by the scientific community, while keeping any necessary changes intuitive and at a minimum. The new RFLP probe names are given in the accompanying list.

OLD NAME	NEW NAME	OLD NAME	NEW NAME	OLD NAME	NEW NAME	OLD NAME	NEW NAME
PIO060005	*php06005*	*PIO200075*	*php20075*	*PIO200644*	*php20644*	*PIO200809*	*php20809*
PIO060007	*php06007*	*PIO200501*	*php20501*	*PIO200646*	*php20646*	*PIO200810*	*php20810*
PIO060011	*php06011*	*PIO200503*	*php20503*	*PIO200654*	*php20654*	*PIO200813*	*php20813*
PIO060012	*php06012*	*PIO200508*	*php20508*	*PIO200655*	*php20655*	*PIO200816*	*php20816*
PIO100002	*php10002*	*PIO200509*	*php20509*	*PIO200661*	*php20661*	*PIO200817*	*php20817*
PIO100005	*php10005*	*PIO200511*	*php20511*	*PIO200668*	*php20668*	*PIO200818*	*php20818*
PIO100007	*php10007*	*PIO200518*	*php20518*	*PIO200674*	*php20674*	*PIO200821*	*php20821*
PIO100012	*php10012*	*PIO200521*	*php20521*	*PIO200675*	*php20675*	*PIO200827*	*php20827*
PIO100014	*php10014*	*PIO200523*	*php20523*	*PIO200679*	*php20679*	*PIO200831*	*php20831*
PIO100016	*php10016*	*PIO200527*	*php20527*	*PIO200681*	*php20681*	*PIO200832*	*php20832*
PIO100017	*php10017*	*PIO200528*	*php20528*	*PIO200682*	*php20682*	*PIO200835*	*php20835*
PIO100025	*php10025*	*PIO200530*	*php20530*	*PIO200684*	*php20684*	*PIO200838*	*php20838*
PIO100033	*php10033*	*PIO200531*	*php20531*	*PIO200689*	*php20689*	*PIO200841*	*php20841*
PI0100040	*php10040*	*PIO200533*	*php20533*	*PIO200690*	*php20690*	*PIO200843*	*php20843*
PIO100059	*php10059*	*PIO200536*	*php20536*	*PIO200701*	*php20701*	*PIO200844*	*php20844*
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PIO120006	*php12006*	*PIO200548*	*php20548*	*PIO200713*	*php20713*	*PIO200852*	*php20852*
PIO120026	*php12026*	*PIO200554*	*php20554*	*PIO200714*	*php20714*	*PIO200853*	*php20853*
PIO150005	*php15005*	*PIO200557*	*php20557*	*PIO200715*	*php20715*	*PIO200854*	*php20854*
PIO150012	*php15012*	*PIO200558*	*php20558*	*PIO200719*	*php20719*	*PIO200855*	*php20855*
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PIO150018	*php15018*	*PIO200563*	*php20563*	*PIO200725*	*php20725*	*PIO200870*	*php20870*
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PIO200035	*php20035*	*PIO200597*	*php20597*	*PIO200789*	*php20789*	*PIO200904*	*php20904*
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PIO200044	*php20044*	*PIO200603*	*php20603*	*PIO200793*	*php20793*	*PIO200907*	*php20907*
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PIO200062	*php20062*	*PIO200626*	*php20626*	*PIO200804*	*php20804*		
PIO200071	*php20071*	*PIO200640*	*php20640*	*PIO200808*	*php20808*		

Updated Pioneer Hi-Bred maize RFLP linkage map

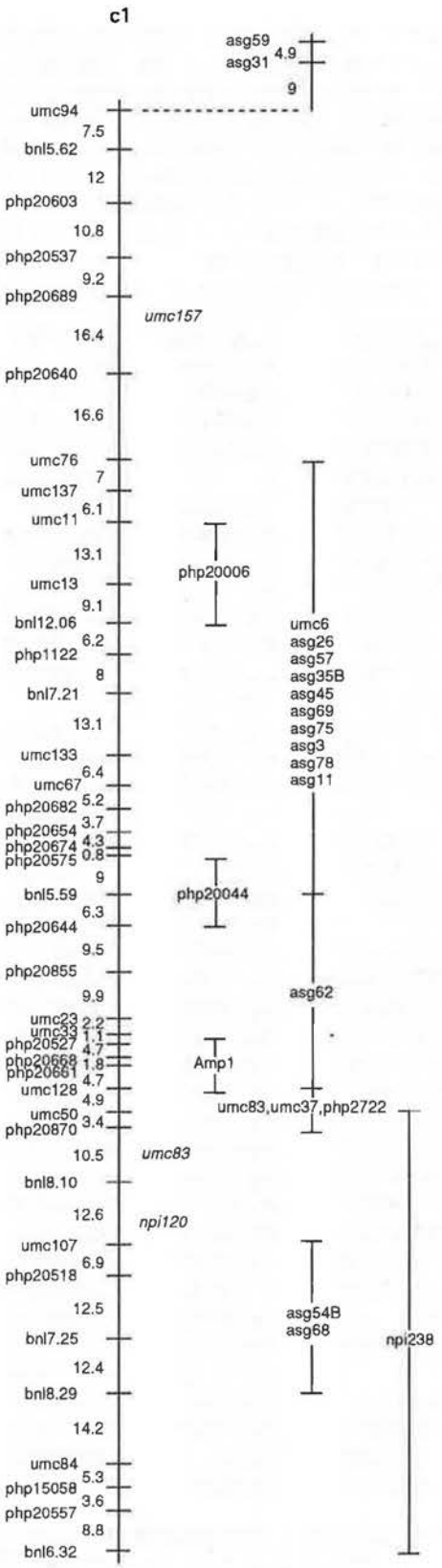
--D. Grant, D. Blair, T. Owens, M. Katt and W. Beavis

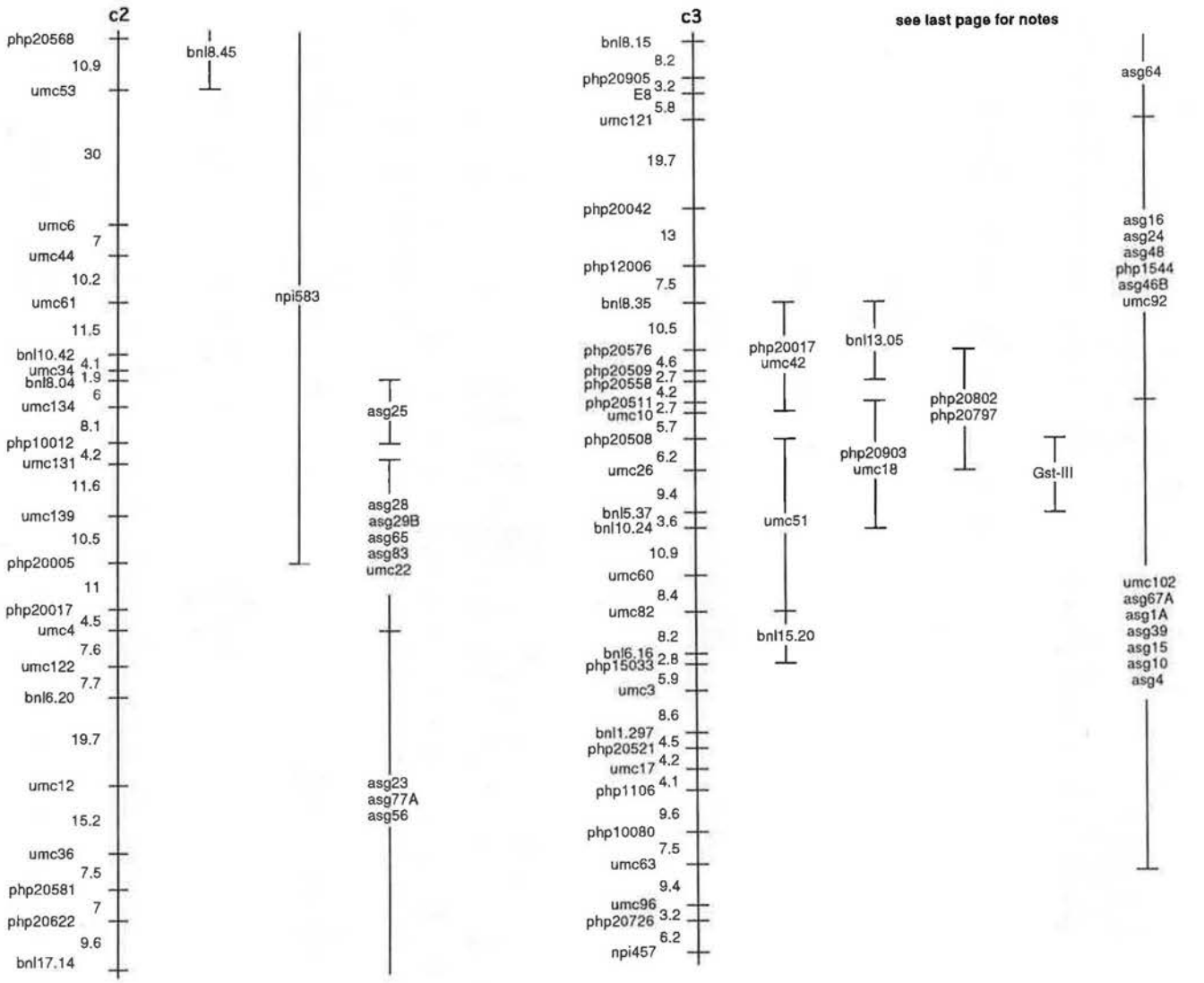
We are currently doing genetic mapping in several segregating maize populations. Maps for some of these crosses, B73/Mo17, B73/G35, K05/W65, and J40/V94, were previously reported in this Newsletter (Beavis and Grant, 1991). The sizes of these populations range from approximately 100 to 250. Maps were created using MAPMAKER (Lander et.al.,1987) and composite maps were created using the log likelihood statistic as described by Beavis and Grant (Theor. Appl. Genet., 1991).

These maps include newly added probes, the changes resulting from our efforts to correct discrepancies caused by previously undetected scoring errors, and the name changes described in our

accompanying Newsletter article. Scoring errors were revealed and analyzed using data integrity procedures developed at Pioneer Hi-Bred. The left-most map of every chromosome is a composite created using probes that have segregation scores in two or more populations. No marker order discrepancies were detected between these populations. The maps to the right of this are based on probes with segregation scores in only one, though not necessarily the same, population. The endpoints of the map intervals for these markers indicate flanking markers from the composite map that were used in mapping these markers.

The probes used came from Ben Burr (bnl), University of Missouri (umc), Asgrow/UpJohn (asg), Pioneer Hi-Bred (php) and several other outside researchers. Probes with the designator "npi" are originally from Native Plants Incorporated, but are now owned and distributed by Pioneer Hi-Bred.

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Gst-III
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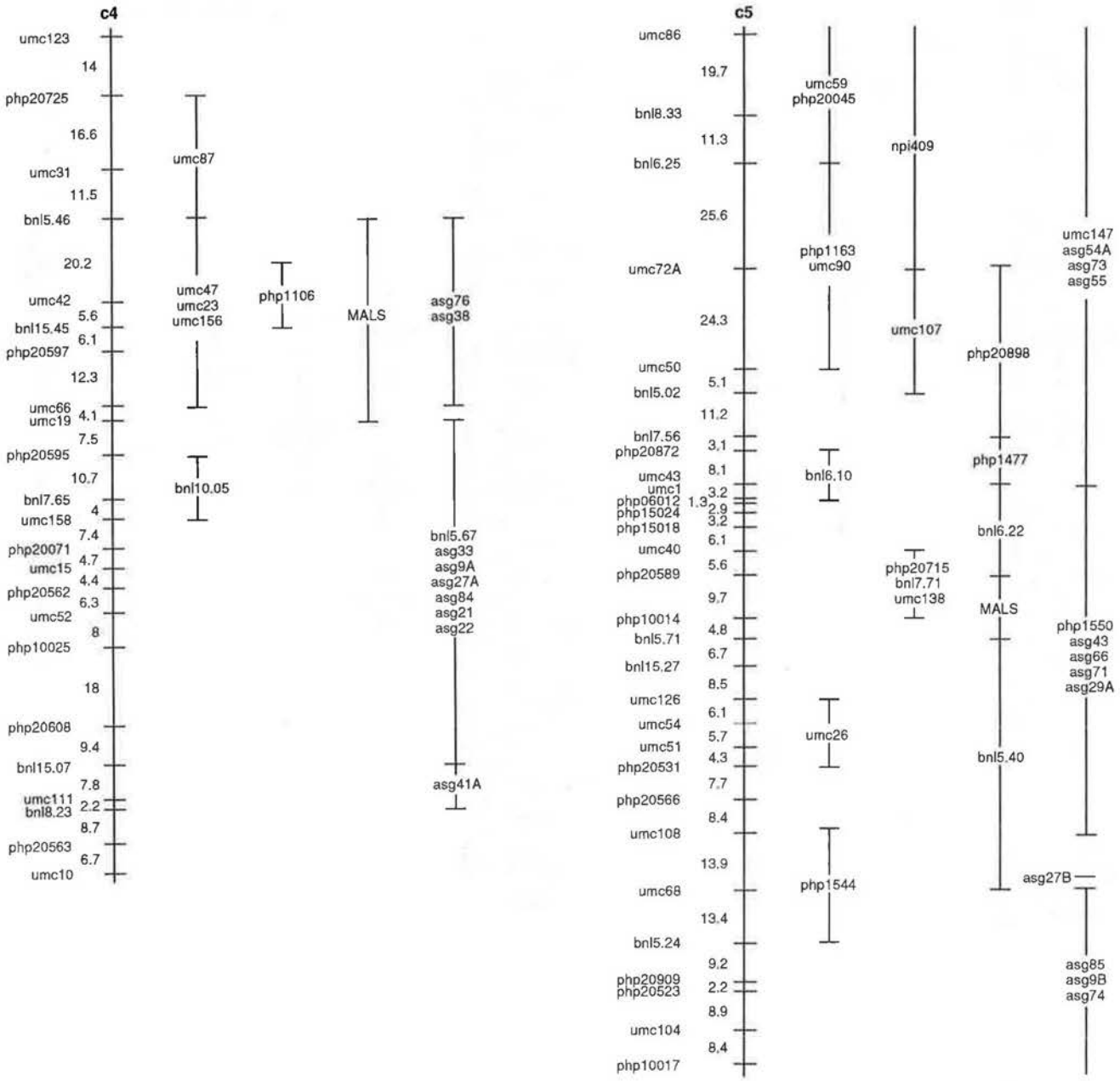
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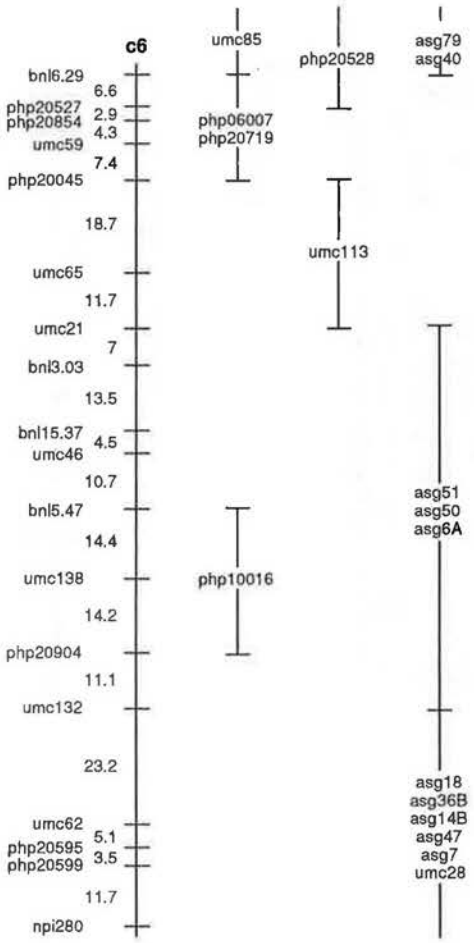
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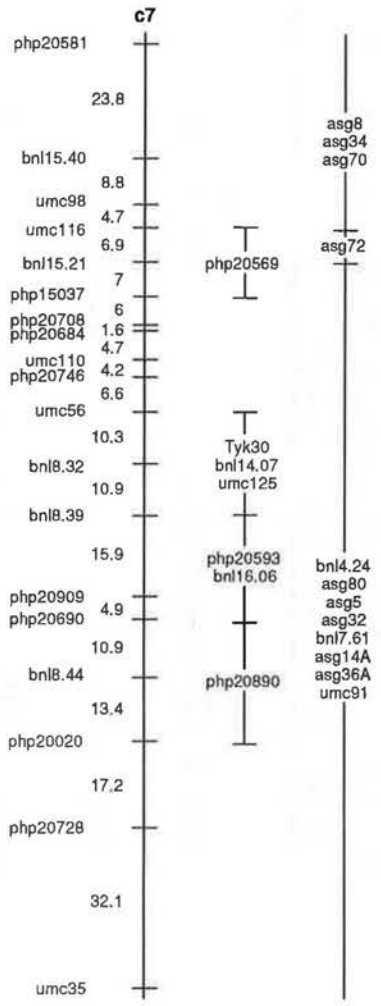
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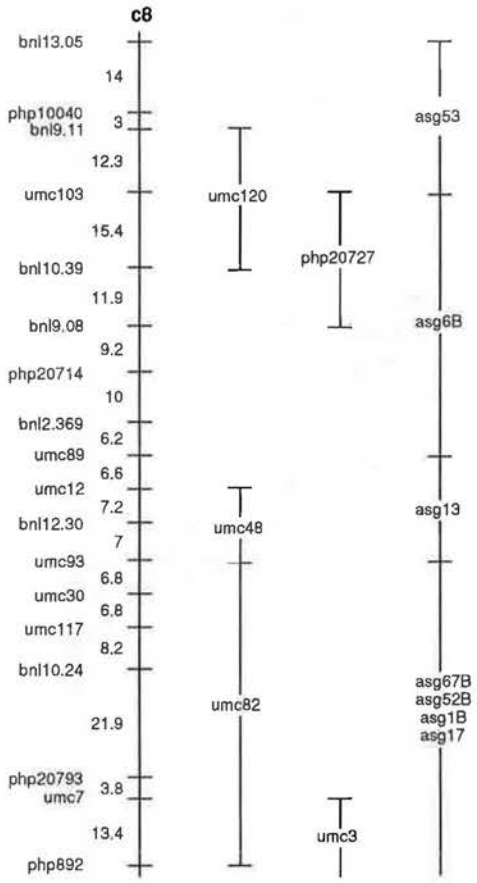
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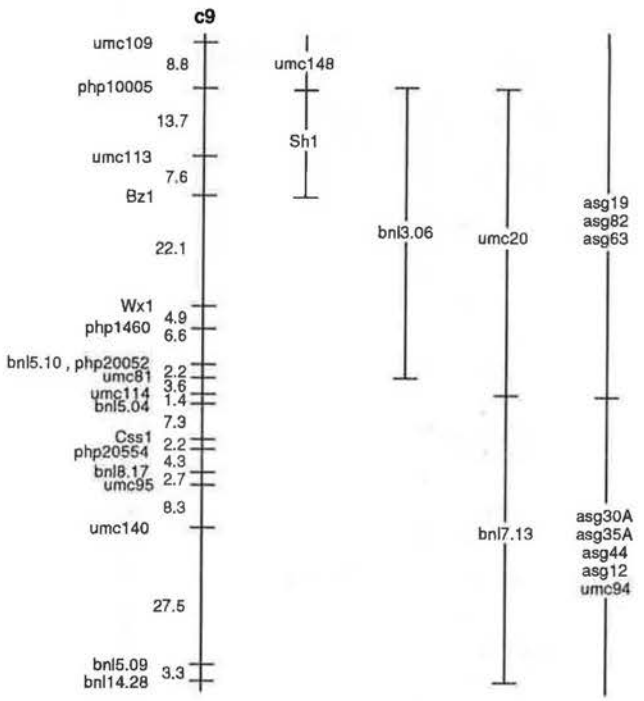
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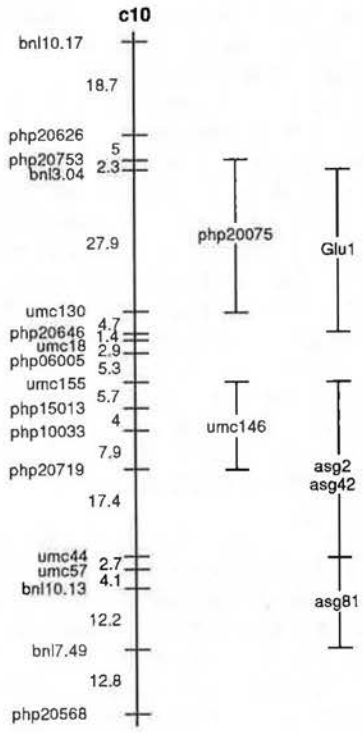

Notes:
1) For each chromosome, the left map is a composite based on data from two or more populations per probe and the maps to the right of this are based on data from one population per probe.
2) The markers in italics to the right of the chromosomes are based on recent mapping data; there are no flanking markers shown as the map position is only approximately known.
3) Data was originally compiled and map drawn on 8/21/92, nomenclature edited 1/6/93.

Cycling parameters for RAPD in maize

--Emily Chin and Stephen Smith

RAPD technology developed by Williams, Tingey, and Rafalski (Nucleic Acids Res. 18:6531-6535, 1990) is being used as a tool to generate molecular markers. We have successfully applied this technology to maize and are able to obtain clear reproducible data. To optimize clarity and reproducibility of profiles, we investigated the effects of different cycling parameters in order to develop a set of routinely applicable and reliable protocols for RAPD in maize. DNA is extracted (Jhingan, Meth. Mol. Biol. 3:15-22) from lyophilized leaf flour taken from 2 week old seedlings. Details of the protocol are given below.

The most expensive component of the reaction is the polymerase enzyme. Using varying amounts of Taq, ranging from 0.5 units to 2.0 units per reaction (with 20ng genomic DNA), and annealing temperatures of either 35 or 37 C, we established an optimum concentration of 0.8 units of Taq per a 25µl reaction. Too much Taq was undesirable because it resulted in high background presumably caused by non-specific binding during annealment. Too little Taq did not allow any visibly detectable amplification.

We have used annealing temperatures of 31, 33, 35, 36, 37, 39, and 41 C each with one unit of Taq for 45 cycles; no differences for the moderate to brightly fluorescing bands (with EtBr) could be seen. We also found no differences for these bands between annealment at 35 C and Touchdown RAPD from 42 C ramping down to 36 C.

Another component of the reaction mix we investigated was the concentration of dNTP mix. A high concentration of readily available dNTP theoretically should allow for more efficient incorporation of bases during primer extension. We tried 0.3, 0.6, 0.9, and 1.2mM dNTP per reaction. Our results indicated that a concentration of 0.6 dNTP was sufficient to generate clear and reproducible profiles.

Since the number of cycles used in the amplification process significantly determines the total duration of profile generation via PCR, we established the minimum number of cycles that would be necessary to generate clear and reproducible profiles. After trying amplification with 25, 30, 35, 40, and 45 cycles we found that 40 cycles could be used most reliably and efficiently.

Our current protocol for RAPDs in maize is as follows:

1) DNA extraction (Jhingan, Meth. Mol. Cell Biol. 3:15-22);
2) Reaction mix: 2.5µl 10X buffer, 0.6µl 10mM dNTP, 0.8 unit Taq polymerase, 5.0pM primer (10mer), 20.0ng DNA template (buffer is 67mM Tris-HCl pH 8.8, 16.6mM $(NH_4)2SO_4$, 6.7mM $MgCl_2$, 10mM DTT, 0.01% NP-40, 0.01% Tween-20);
3) Cycling profile is
   a) initial denaturation of DNA into single strands by 94 C 3.00 min;
   b) 40 cycles of:
      i) 94 C 1.00 min;
      ii) annealing at 37 C 1.00 min;
      iii) extension at 72 C 2.00 min;
   c) 72 C 7.00 min, then hold at 4 C until gel loading or store frozen prior to gel loading;
4) Electrophoretic conditions - run on 2% NuSieve 3:1 agarose gel after adding loading dye to each sample.

RAPD primers used in analysis of the genome appear to be highly specific

--Emily Chin and Stephen Smith

Primers used with the RAPD procedure are assumed to perhaps have only a partial homology to the target sequences. However, we have evidence to show that, at least in maize, this homology appears rather to be quite precise.

Firstly, it is usual to employ a less stringent annealing temperature (e.g. 35 C) in RAPD analyses in order to allow for potential mismatches between primer and target sequences. However, we have used Touchdown RAPD with annealing temperature initially at 42 C and reducing to 31 C. This procedure resulted in the same amplification profile as when compared to the profiles for samples run under annealment at 35 C alone.

Secondly, we have performed a series of RAPD amplifications using different annealing temperatures. The annealing temperatures were 31, 33, 35, 36, 37, 39, and 41 C. The amplification products were all run on 2% agarose gels and no differences for the moderately to brightly fluorescing bands could be noted. This range of annealing temperatures would be expected to encompass a very wide range of stringencies for a 10mer.

Thirdly, we synthesized a series of 11 nucleotides each 10 bases in length. Pairs of these primers differed by only a single nucleotide from each other, whilst maintaining the purine pyrimidine ratio. These primers were used to amplify, via the RAPD procedure, nine maize inbred lines that collectively encompassed a broad range of Corn Belt genetic diversity. Observation of the RAPD profiles showed that varying a single base anywhere on the primer, irrespective of whether it was toward the 3' or 5' end, resulted in completely different banding profiles when applied to

the same corn inbred line. One might have expected, if annealment occurred with less than at least a fairly complete homology between primer and target sequence, that substitution of single bases in the primer would sometimes not be consequential with respect to the outcome of the amplified products. We never found this to be the case, thereby demonstrating a degree of specificity of primers in respect to the target sequence.

Direct amplification by RAPDs of fresh leaf disc tissue

--Emily Chin and Stephen Smith

Most of the RAPD analyses done to date require a carefully calculated concentration of extracted genomic DNA as the target source. Using three inbred lines of maize, we were able to directly amplify and generate the same DNA profiles from fresh leaf tissue as were obtained using extracts made from lyophilized leaf tissue from two week old seedlings. Leaf discs collected from three-four week plants were analyzed using both conventional PCR and with RAPD. Leaf discs were immersed directly into the amplification cocktail without additional attempts at extracting DNA. DNA of the same inbred lines extracted from lyophilized leaf flour was also amplified under the same PCR or RAPD conditions in order to compare banding profiles obtained directly from leaf discs versus the more conventional source of DNA.

Amplifications obtained via "classical" PCR using a pair of primers gave clear and repeatable banding profiles regardless of whether the DNA had been extracted from lyophilized flour or whether the reaction occurred instead in the presence of an immersed fresh leaf disc. With RAPD, banding profiles obtained from reactions that had DNA from lyophilized leaf flour also gave clear profiles. However, with fresh leaf discs as the source of DNA, the RAPD method did not result in any visually obvious bands.

For each of the samples that had utilized a fresh leaf disc as the potential source of target DNA, an aliquot was taken following one round of amplification to then initiate a sequential and second round of amplification. Following two rounds of amplification for the PCR reactions, there were several bands that were either not present or only faintly present following the first round of amplification. However, with RAPD, the second round of amplification resulted in clear and distinct banding profiles that were very similar to those obtained from a single round of amplification when DNA extracted from lyophilized leaf flour had been the target source. These results showed that the binding of primers to target DNA in a RAPD reaction could be less efficient than with PCR. However, once a primer-target is established, then amplification can occur with high fidelity. Fresh leaf discs, therefore, could be used to generate RAPDs but two sequential rounds of amplification would then be necessary.

Polymorphism revealed by RAPD is highly repeatable

--Emily Chin and Stephen Smith

There have been concerns about the reproducibility of banding profiles generated by RAPDs. These concerns have brought into question the reliability of the method to provide useful profile data either for genetic or varietal identification studies. With 200 primers (10mers from Operon Tech.), we have screened nine inbred lines of maize that collectively encompass a broad degree of genetic diversity for the Corn Belt. Using an extremely high stringency of selection, namely the presence of a few clear, well defined, and easily scorable bands per DNA/primer complex, and the ability to discriminate among at least 50% of the inbreds in this initial screen, we were able to identify 71 primers that are to be preferred for varietal identification in maize. This list of primers will be made available upon request.

An integral component of this screening process was to check the reproducibility of the banding profiles. DNA was extracted from each inbred line in duplicate and each DNA sample was then amplified twice giving four amplification products for each DNA/primer complex. Duplicate amplifications were either performed on the same thermocycling unit but on different days or on different thermocyclers during the same day. Visual comparisons of the profiles showed a very high degree of repeatability with only some of the faintly fluorescing bands showing presence and absence variation. The moderately to highly fluorescing bands were constant across replicates for each of the preferred primers against all maize genotypes that were included in the screening process.

One possible explanation for the sometimes observed lack of repeatability for bands that show a low level of fluorescence could be that they represent instances of inefficient priming, possibly due to a poor sequence homology between the primer and target site on the genomic DNA. These sites would then not be able to compete as effectively in comparison to other sites that have 100%, or near to 100%, sequence homology. A lack of repeatability for the faint bands could possibly be overcome, or at least reduced to a minimum, by increasing the stringency of the annealing conditions. Such a goal was a major component in the derivation of our current RAPD conditions for maize (see separate report by Chin and Smith, this Newsletter).

Once an ability to repeatably generate RAPD profiles has been established, it is then necessary to evaluate that capability for practical and routine purposes of varietal identification. In order to achieve that goal, it will be necessary to arrive at some objective criteria for the scoring and databasing of those profiles and to more thoroughly evaluate the repeatability of profile generation. Therefore, we are now directing the focus of our research into more objectively measuring the repeatability of molecular weights and fluorescent intensities generated by RAPDs. DNA extractions and amplifications have each been performed in duplicate by two laboratory personnel. RAPD profiles have been scanned by digital camera and recorded using Bioimage software and hardware.

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Associations among inbred lines as revealed by RFLPs and by a thermocycling amplification methodology, Amplified Fragment Length Polymorphisms (AFLPs)

--J. S. C. Smith , M. Zabeau and S. Wright

Forty-eight publicly available inbred lines of maize were profiled using 58 RFLP clones each directed against a single restriction enzyme digest. A total of 370 bands were scored across all inbred lines. Genetic distances were calculated and cluster analy-

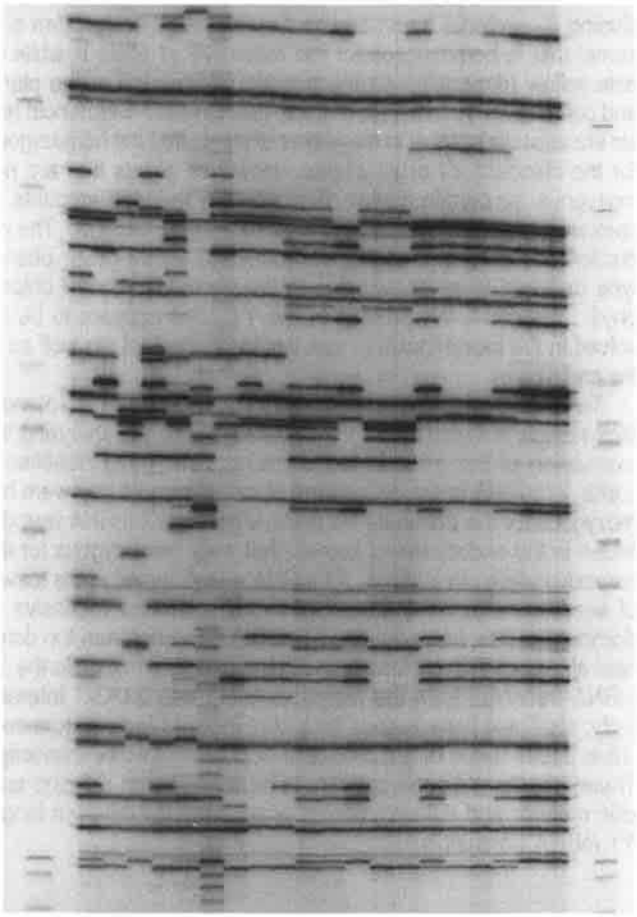

Figure 1. Detail from AFLP profiles of 20 maize samples including both inbred lines and hybrids for a single primer. Molecular weight ladders are arrayed on either side of the maize lanes.

sis was performed as described by Smith et al. (Theor. Appl. Genet. 80:833-840, 1990). These same inbred lines were also profiled by AFLPs. This is a methodology that takes advantage of DNA amplification technology but which differs from both 'classical' PCR and single random primer methods such as RAPD, AP-PCR and DAF. The AFLP method generates multi-banded profiles that can reveal extensive polymorphism among inbred lines of maize (Fig. 1). (The technology is currently patent pending; for further details, readers are recommended to contact Dr. Marc Zabeau, Keygene, Agro Business Park 90, P.O. Box 216, 6700AE Wageningen, The Netherlands, FAX (31)837024939.) Across 44 of these inbred lines (4 lines had missing data) a total of 135 AFLP bands were identified. Each inbred was scored for the presence or absence of each band; genetic distances and clusters were generated as described above. Associations among inbreds on the basis of RFLPs and AFLPs are presented in Figures 2 and 3, respectively. Both methodologies showed a clustering of inbred lines that were related by pedigree into similar groupings. However, associations among these groupings showed some differences. These data are preliminary in that additional RFLPs and AFLPs could be scored. However, they demonstrate that DNA profiles generated either by a probe based technology or by a DNA amplification technology both provide evidence of similar associations when inbreds are related by pedigree. With regard to aiding in a more complete comprehension of genetic diversity with

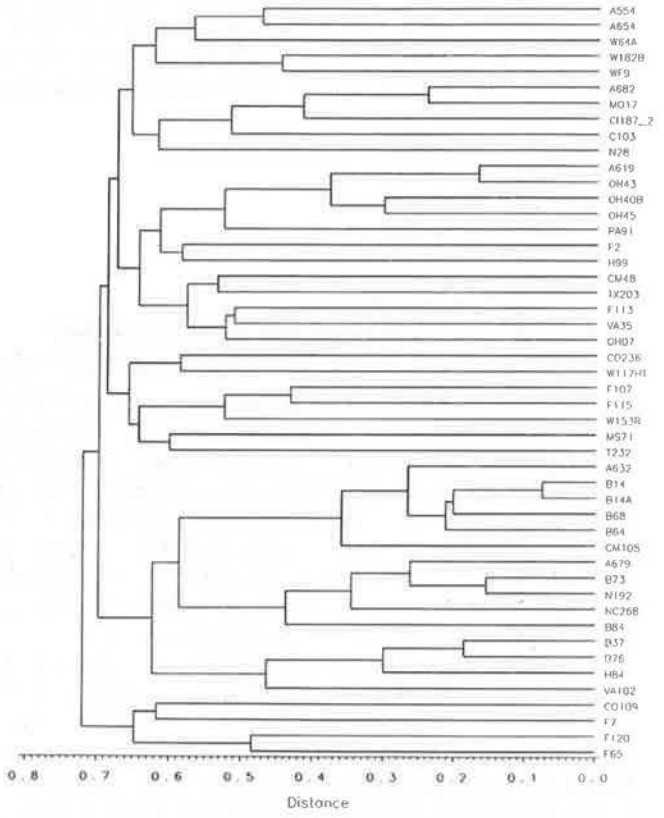

Figure 2. Associations among 48 inbred lines as revealed by cluster analysis of RFLP data.

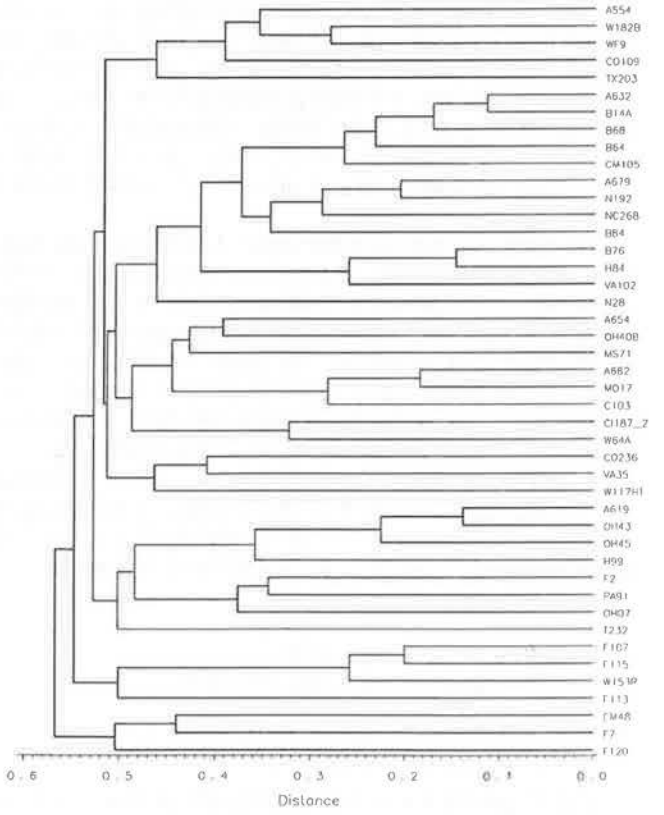

Figure 3. Associations among 44 inbred lines as revealed by cluster analysis of AFLP data.

regard to the assignation of inbred lines within heterotic groups, their further classification within these groups, and correlation of these data with performance data, AFLPs appear to offer researchers valuable and possibly fresh insights compared to RFLPs.

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Interval mapping a new dominant male-sterile mutant, *Ms42*

--M. C. Albertsen, T. W. Fox, M. R. Trimnell and M. G. Neuffer

One of the dominant male-sterile mutations that MGN identified from his EMS pollen mutagenesis research was designated *Ms*-2082*. This dominant male sterile exhibited extremely poor penetrance in both Mo17 and B73 as there was a shortage of male-sterile plants in crosses that should have segregated 1:1 for fertility vs. sterility. Previous experience with A632 suggested that male-sterile mutants often segregated more as expected in this background. We backcrossed *Ms*-2082* into A632 and observed 1:1 segregation ratios after a few backcrosses.

Genetically mapping a dominant male sterile can be tedious. We decided to utilize RFLP interval mapping as a way to determine the chromosome arm location of *Ms*-2082*. Leaves were collected from 30 male-sterile plants and 30 male-fertile plants, and were bulked according to fertility. DNA was CTAB-extracted; digested with *Bam*HI, *Eco*RI, and *Hin*dIII, and transferred to Duralon-UV. Membranes were probed sequentially with 23 probes from 17 of the 20 chromosome arms. Probe *bnl6.25* (chromosome 5S) gave polymorphisms across all three enzymes, with *Ms*-2082* exhibiting unique bands as well as bands in common with the bulked fertiles. Probe *umc50* (chromosome 5S) also gave polymorphisms with *Eco*RI and *Hin*dIII. It showed a new band in the steriles, as well as bands in common with the fertiles. Although all chromosome arms are not yet covered, the differential probe hybridizations are strongly indicative that the gene is located on the short arm of chromosome 5.

We propose to designate *Ms*-2082* as *Ms42*, as it now segregates as a single dominant gene and we have reasonable mapping information on it. Although *Ms44* is currently the highest numbered male-sterile mutant, there are several gaps in the numbering of male-sterile mutations. We decided to fill in these gaps where we are certain that no previous designation exists. There are currently only two other dominant male sterile mutants in maize that are described and that have available seed stocks. These are *Ms41* and *Ms44*. A third dominant male sterile, *Ms21*, has been lost. All existing dominant male-sterile mutations have been derived from EMS mutagenesis. *Ms41* and *Ms42* are from EMS pollen mutagenesis, and *Ms44* is from EMS seed mutagenesis.

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***Y1* gene expression in the endosperm and leaves**

--Brent Buckner and Claravon Mathews

The *Y1* gene is involved in the biosynthesis of carotenoids in maize. The endosperm of a kernel that contains one or more dominant *Y1* alleles is yellow due to the accumulation of carotenoids including β-carotene, lutein and zeaxanthin. The endosperm of a kernel that is homozygous for the recessive *y1* allele is white or pale-yellow (depending on the genetic background of the plant) and contains very low levels of these carotenoids. Carotenoid levels are essentially equal in the leaves of plants that are homozygous for the standard *Y1* or *y1* alleles. However, plants that are homozygous for certain alleles of *y1* contain reduced amounts of carotenoids in their leaves, as well as in their endosperm. The reduction of carotenoids in the leaf results in a light-green phenotype (referred to as pastel) due to the photooxidation of chlorophyll. Therefore, the product of the *Y1* gene appears to be involved in the biosynthesis of carotenoids in the leaf, as well as in the endosperm.

To understand how the *Y1* gene is involved in carotenoid biosynthesis in maize endosperm and leaves, we are analyzing the expression of this gene in these tissues. We have identified a 1.8Kb *Y1* mRNA in the endosperm of maize kernels that were homozygous for the dominant *Y1* allele, while no *Y1* mRNA was detected in the endosperm of kernels that were homozygous for the recessive allele. In addition, *Y1* mRNA was detected in the leaves of seedlings that were homozygous for either the recessive or dominant alleles, however, the *Y1* mRNA transcript from the dominant allele is 1.8Kb (as was found in the endosperm), while the *Y1* mRNA transcript from the recessive allele was 2.0Kb. Interestingly, seedlings homozygous for a *Mu3*-induced temperature-sensitive pastel allele of *y1* contain a 1.7Kb *Y1* mRNA transcript. Therefore, the *Y1* gene appears to be regulated in a tissue specific manner and different alleles of *y1* code for different length *Y1* mRNA transcripts.

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Cytogenetic localization of the *y1*, *l10*, *l12*, *ms1* and *si1* loci

--Brent Buckner and Donald S. Robertson

We have produced plants heterozygous for reciprocal translocations involving different breakpoints in the same two chromosomes ("overlapping translocations"). When these plants undergo meiosis one-fourth of the gametes produced should be deficient for the chromosomal region between the interchange breakpoints. A megagametophyte receiving a large deletion will probably abort. However, megagametophytes with small deficiencies might possibly be viable and if they are fertilized by sperm carrying a recessive allele for a trait located within the deficient chromosomal segment, the resulting sporophytes should be hemizygous for the recessive trait.

There were several reasons why we initiated these studies. We were interested in generating plants that contain a small interstitial hemizygous region for the long arm of chromosome 6 including the *y1* locus. During our attempt to isolate the *Y1* gene, the DNA from these plants was analyzed by DNA blot hybridization analysis using maize genomic sequences that were putative clones of the *Y1* gene as hybridization probes. If a clone was found to be in the hemizygous state in these plants it demonstrated that the sequence was closely linked to the *y1* gene and, therefore, the clone was further characterized. We also produced these plants to test

if a deletion in chromosome 6, including the chromosomal segment containing *Y1*, was viable in the megagametophyte. In addition, these studies have allowed us to begin to compare and correlate the cytogenetic, classical and molecular maps of maize for a small region of 6L.

We produced plants that are heterozygous for 8 different combinations of overlapping translocations having breakpoints in 6L (Table 1). When these plants were pollinated by plants that were homozygous or heterozygous for several different recessive *y1* alleles, white kernels were observed in six different overlapping translocation combinations. In several crosses the allele uncovered was a recessive temperature-sensitive pastel allele of the *y1* gene. Seedling tests confirmed that the plants possessed the pastel phenotype. In no case did the number of white kernels approach one-fourth of the kernels on the ear. Typically we observed from 1 to 12 kernels per ear. In addition, the white kernels were reduced in size when compared to sibling nonwhite kernels. Our observations suggest that megagametophytes that possess deficiencies for the segment of 6L containing the *Y1* gene are viable, however, these deficiencies reduce their fitness.

Table 1. Combinations of translocations tested.

Translocation	6L Breakpoints	*Y1* Uncovered
T6-9e/T6-9(043-1)	0.18/0.36	yes
T6-9(6270)/T6-9(043-1)	0.19/0.36	yes
T6-9(6270)/T6-9(6019)	0.19/0.27	no
T6-9(6019)/T6-9(0431)	0.27/0.36	yes
T4-6(055-8)/T4-6(8428)	0.25/0.28	no
T4-6(055-8)/T4-6(6623)	0.25/0.31	yes
T4-6(8428)/T4-6(6623)	0.28/0.31	yes
T6-10b/T6-10d	0.12/0.16	yes

Breakpoints are those listed by Longley (USDA ARS 34:16, 1961).

The DNA from plants derived from white kernels of two different combinations of overlapping translocations were analyzed by DNA blot hybridization analysis using the *Y1* gene as a hybridization probe. These plants were found to be hemizygous for the *Y1* gene (data not shown).

Results of our studies using overlapping translocation combinations in which interchange points were between chromosome 6 and chromosome 4 or 9 place the *y1* gene in the region between positions 0.28 and 0.31 on 6L [T4-6(055-8)/T4-6(6623)]. The largest interstitial hemizygous region that uncovered the *y1* gene was delineated by positions 0.18 and 0.36 on 6L [T6-9e/T6-9(043-1)]. However, overlapping translocation combinations in which the interchange points were between chromosome 6 and 10 place the *Y1* gene between positions 0.12 and 0.16 on chromosome 6L (T6-10b/T6-10d). Because the positions estimated for *Y1* using the 4-6 and 6-9 overlapping translocations are consistent, it would suggest that the estimations of the T6-10b and T6-10d interchange points may be in error.

Several other loci on 6L were also uncovered. The *l10*, *l12*, *ms1* and *si1* loci were uncovered by overlapping translocation combinations T6-9e/T6-9(043-1). In addition, *l12* was also uncovered by overlapping translocation combinations T6-9(6019)/T6-9(043-1) and T4-6(8428)/T4-6(6623). Since *l12* is the most proximal of those analyzed, these data suggest that these loci, which span approximately 4 map units on 6L, all map to the region between positions 0.28 and 0.36 on 6L.

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The *Y1* gene codes for phytoene synthase

--Brent Buckner, Phillip San Miguel and Jeffrey L. Bennetzen

The *Y1* gene is involved in the biosynthesis of carotenoids in maize. We have sequenced a 1.4Kb cDNA and approximately 6Kb of genomic DNA for a *Y1* allele (a full length cDNA of the *Y1* allele is expected to be approximately 1.8Kb in length). The cDNA sequence has good homology (64% identity, 75% similarity at the amino acid level) to the tomato gene *pTOM5* which codes for phytoene synthase. Phytoene synthase is the enzyme that catalyzes the condensation of two molecules of geranylgeranyl pyrophosphate to yield phytoene, the first C_{40} carotenoid synthesized by plants. In addition, the deduced amino acid sequence of the *Y1* gene product also exhibits good homology to the gene product of the *crtB* genes of *Erwinia uredovora* (31% identity and 48% similarity) and *Rhodobacter capsulatus* (29% identity and 52% similarity). The *crtB* gene product is also believed to be a phytoene synthase. Therefore, we conclude from these strong homologies that the *Y1* gene of maize codes for phytoene synthase.

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***Cg2* macromutation linkage data**

--N. V. Krivov and V. N. Lysikov

Four semidominant heterochronic mutations, the localization of which is well known, *Cg1* (Singleton, MNL23:7, 1949; Galinat, MNL26:51, 1952), *Tp1* (Lindstrom, Hered. 16:135, 1925; Weatherwax, Hered. 20:325, 1929), *Tp2* (Peterson, MNL33:41, 1959; Horovitz, MNL37:148, 1963), and *Tp3* (Beckett, J. Hered. 69:27, 1978; Poethig, Genetics 119:959, 1988) have originated spontaneously. They are responsible for the ancestral phenotype of the shoot vegetative morphology and the transformation of reproductive structures into leaves. The analyses of the phenotypes of *Tp1*, *Tp2* and *Tp3* (Poethig, 1988) demonstrate that the traits displayed during the juvenile phase are a part of the genetically controlled developmental programme and support the hypothesis (Galinat, MNL40:102, 1966) that this phenotype presents a defect in the transition from the juvenile to mature phase during the shoot development.

Like already known heterochronic mutations, the *Cg2* macromutation was supposed to be located in the 3rd, 7th and 10th chromosome. With this aim crossings with Mangelsdorf's marker, as well as with *d1* at 3S-44, *ra2* at 3S-49, *lg2* at 3L-101, *o2* at 7S-16, *ra1* at 7S-24, *gl1* at 7L-32, *v5* at 7L-36 and *g* at 10L-50, were carried out. Only *ra2* and *lg2* showed linkage with *Cg2* (Table 1).

However, due to the significant excess of phenotypically normal plants, + +, + *ra2* and + *lg2*, which was, apparently, caused by the

Table 1. *Cg2* + +/+ *ra2 lg2* self-pollination.

Parental		Crossover		Total	% recombination
Cg2 +	+ *ra2*	*Cg2 ra2*	++		
338	261	73	470	1142	39.2±2.7
Cg2 +	+ *lg2*	*Cg2 lg2*	++		
335	203	76	528		46.5±2.9

Table 2. *Cg2 +/+ ra2* self-pollination.

Parental		Crossover		Total	% recombination
Cg2+	*+ ra2*	*Cg2 ra2*	*++*		
239	65	61	55	420	29.9±2.8

presence of a recessive gene that suppresses *Cg2* in the marker line *ra2 lg2*, we doubted whether the linkage observed refers only to *Cg2-ra2* and *Cg2-lg2*. It is not less probable that the linkage observed can refer both to the pairs marker-gene suppressor and *Cg2*-suppressor. Moreover, the excess of *ra2* carriers, which is probably connected with the *ra2* display increases on the background of this macromutation, confusing the picture even more. Nevertheless, a chance occurrence has helped to answer this question unequivocally. A marker line carrying *ra2* and containing no suppressor gene was found. Now it became obvious that the linkage marked earlier, undoubtedly, referred to *Cg2-ra2* and *Cg2-lg2* (Table 2).

A test for allelism between *Cg2* and *Cg*

--N. V. Krivov

Ninety various loci which affect morphogenesis have been identified and described in maize (Sheridan, Annu. Rev. Genet. 22:353-85, 1988), among them two semidominant mutations, *Cg* at 3S-35 (Singleton, Amer. Nat. 85:88-96, 1951; Galinat, MNL26:51, 1952; Poethig, MNL62:98, 1988) and *Tp3* on 3S (Poethig, Genet. 119:959-973, 1988), that have an extremely strong pleiotropic effect. Atavistic changes in the shoot vegetative morphology occur in the carriers of these mutations, and it is surprising that they are both located on the 3rd chromosome.

Is *Cg2* the allele of *Cg* or *Tp3*? A complete genetical affinity could not occur. Firstly, because as a rule, any mutational event in any locus is an unique phenomenon and reverses, as a rule, do not exactly restore the primary function of this locus. Secondly, because of the origin, and mainly due to the fact that the genetic background was not similar. The *Cg2* macromutation originated among many M3 plants as a single plant after the exposure of the line VIR-44 pollen to irradiation with gamma-rays from Co-60 at a dose of 1500 rads, and pollination with a mixture of pollen from different neighbouring maize lines. The *Cg* macromutation is known to originate spontaneously in plantings of Lincoln sweet corn hybrid (Singleton, MNL21:6, 1947). And finally, while the expression of *Tp1* is suppressed by increasing doses of its wildtype allele, the expression of *Tp2* is essentially indifferent to the wildtype gene dose. These response patterns suggest that *Tp1* has an antimorphic function, while *Tp2* has a neomorphic one. Although this does not necessarily mean that these genes are functionally distinct, it seems unlikely that antimorphic and neomorphic mutations of the same function would have phenotypes as similar as those of *Tp1* and *Tp2* (Poethig, 1988).

To test for allelism the subline isolated in the progeny of the macromutation of corngrass, and *Cg* from the All-Union Institute of Plant Growing (St. Petersburg) collection (*Cg2/Cg2* x *Cg/+*), was used. Thus, in case of no allelism of these two macromutations in F1 the heterozygote of two types *Cg2 +/+ Cg* and *Cg2 +/+ +* should be expected. In fact, in F1 of 113 maize plants 56 had a strong mutant corngrass phenotype and resembled *Cg*, while the rest had a weakened mutant *Cg2* phenotype, as they didn't tiller and differed from the normal maize in tassel (side branchlets in the tassel were absent).

In F2 it was expected on self-pollination that the plants with a strong mutant corngrass phenotype (*Cg2 +/+ Cg*), if *Cg* and *Cg2* are allelic, would have progeny represented only by the carriers of the corngrass macromutation and if they are not allelic, corngrass and phenotypically normal plants will appear in the ratio approximately 15:1, and that self-pollination of plants with a weak mutant phenotype (*Cg2 +/+ +*) would give the segregation of *Cg2* to normal of 3:1 respectively. In fact, the plant progeny with a strong mutant expression has segregated in the ratio 3:1 (Table 1).

Table 1. Segregating progeny resulting from selfing plants (*Cg2 +/+ Cg*) with strong mutant corngrass phenotype.

Family	Norm	*m(+)*	*Cg*	*Cg2*	*m(Cg2)*	Total	X^2 1:3
286	15		15		6	36	5.3*
287	7		40	3		50	3.2
288	10	2	15	1	1	29	4.1*
289	5	5	28	9	1	48	0.4
290	10	2	21	4	3	40	0.5
291	15	2	37	5	5	64	0.1
292	21	4	23	6	9	63	7.2*
Total	83	15	179	28	25	330	3.9*

*The deviations are reliable at the significant level of 0.05%.

On the self-pollination of *Cg2 +/+ Cg* the appearance of phenotypically normal plants in a quantity larger than was expected theoretically suggests that the suppressor is more probably connected with the recessive gene (or genes), which contains *Cg/+* in the genome, as both F1 and F2 plants with a strong mutant corngrass phenotype resemble *Cg*. These two phenotypically similar mutations located in one chromosome are likely to have different functions and the mutant "corngrass" phenotype is displayed only in those genotypes where *Cg* is present at least in one dose. The variation of gene suppressor dose, as well as *Cg2* or *Cg* macromutations tested, probably resulted in the fact that in nearly half of the families the part of the phenotypically normal plants was significantly higher than can be expected (Table 2). Undoubtedly, the F1 phenotypical discreteness and the appearance of the phenotypically normal plants on selfing heterozygotes *Cg2 +/+ Cg* suggest that *Cg2* and *Cg* macromutations are not allelic to each other.

Table 2. Segregating progeny resulting from selfing plants (*Cg2 +/+ +*) with weak mutant corngrass phenotype.

Family	Norm	*m(+)*	*Cg*	*Cg2*	*m(Cg2)*	Total	X^2 1:3
296	22	4		27	7	60	10.7*
297	29	6		24	11	70	23.3*
298	42	5		14	6	67	72.8*
299	17			50	4	71	0.04
300	34	2		36	2	74	22.0*
301	35		7	52	7	101	5.0*
302	19		1	45	6	71	0.1
303	6		1	20	8	35	1.1
304	11		1	24	1	37	0.4
305	10	2		24	2	38	0.9
306	9	1		25	2	37	0.1
307	14			31	1	46	0.7
308	12	2		32	1	47	0.6
309	13		2	33	3	51	0.01
310	13	1		22	2	38	2.8
311	6	1		9	1	17	2.4
312	2			9	5	16	1.3
313	21			18	1	40	16.1*
314	25			19		44	23.7*
315	13			13		26	8.7*
316	8			10	1	19	3.0
317	29			17	3	49	30.5*
318	27			23	4	54	18.0*
319	24		3	12	5	44	20.5*
Total	441	24	15	589	83	1152	154.0*

*The deviations are reliable at the significant level of 0.05%.

Note: As M. D. Golubovsky suggests, plant class *m*(+), in which most shoots are normal phenotypically, at the time of segregation estimation were referred to as "norm" as distinguished from *m(Cg2)*, in which most shoots have a mutant phenotype and were referred to as "corngrass".

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Line 613/2 as a source of a high frequency of spontaneous diploidization in corn

--E. R. Zabirova, O. A. Shatskaya and V. S. Shcherbak

In mass induction of autodiploid lines diploidization of haploid plants becomes one of the problems. Traditionally this is realized by treating the seedlings with 0.12% colchicine solution. In this case a frequency of diploids induced usually ranges from 20 to 30%. Sometimes this value is as high as 50%. However, it is known that the lines passing through a haploid state show a higher percent of diploids. Considering this and the fact that colchicine treatment is a labour consuming process we started searching for a new approach, i.e. a new source of a high frequency of spontaneous diploids. It was found that 33% of the plants originating from 613/2 line in a haploid state became spontaneous diploids (Table 1), in contrast to the lines of the Corn World Collection

Table 1. Frequency of spontaneous diploids in some corn lines.

Line	Number of haploids	Number of pollinated ears	Frequency of diploids (%)
613/2 C4	114	38	33.3
620/1 C1	26	3	11.5
A344	72	3	4.2
A663	26	1	3.8
Kr 123 CO	20	0	0.0
P2	8	0	0.0

where no spontaneous diploidization occurred. The selected line was obtained after 4 cycles of selection, i.e. it had passed four times through a haploid state. It seems involvement of the line as a source of a high frequency of diploids into crosses may in part solve the problem of mass induction of haploid lines.

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Meiotic studies in native races from northwestern Argentina

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Several previous studies presented cytogenetical evidence that maize and related species with 2n=20 chromosomes have an amphiploid origin, and five was postulated as the basic number of the genus *Zea* (Molina and Naranjo, 1987; Naranjo et al., 1990, 1991; Poggio et al., 1990). The principal evidences are: 1) Secondary association of bivalents at meiosis and a maximum of five groups of two bivalents each was frequent at diplotene-diakinesis; 2) formation of two spindles with five bivalents each at metaphase-anaphase I.

In maize most of the studies were done on several lines and improved populations cultivated in the "Instituto Fitotécnico de Santa Catalina (IFSC)". We have just begun cytological studies in six native races from northwestern Argentina. All six races showed the presence of two groups of five bivalents at diplotene-metaphase I in most of the studied cells (Fig. 1B, C, D). On the other hand, secondary association of bivalents, with a maximum of five groups of two bivalents each was frequent at diakinesis-metaphase I (Fig. 1A). These observations indicate that these phenomena are general and not restricted only to commercial lines and intraspecific hybrids of maize. In these races there is a variable frequency of B-chromosomes (see Chiavarino et al., this volume). The presence of B-chromosomes does not alter the disposition of the two groups of five bivalents each since the extra chromosome joins one of the two groups or, in a few cases, rests outside of the metaphase plate. In some cases a very slight asynchrony in the meiotic behavior between both groups of five bivalents each was detected. This asynchrony, which is very strong in alloplasmic lines of *Zea* (Poggio et al., 1992) supports the hypothesis that the groups of five bivalents each belong to different genomes (Poggio et al., unpubl.).

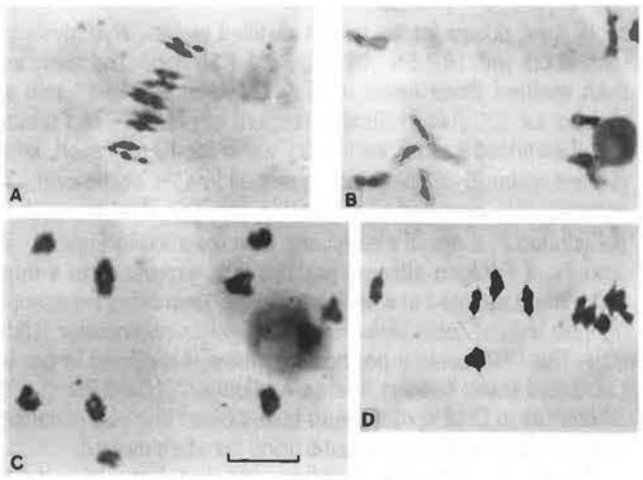

Figure 1. A-B: "Altiplano" race. A: Metaphase I, secondary association; B: Diplotene, two groups of 5II each. C: "Negro" race, diakinesis, two groups of 5II each. D: "Amarillo de ocho" race, metaphase I, two groups of 5II each. The bar=10µm.

Genome size in six races of maize from northwestern Argentina

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The genus *Zea* exhibits intra- and interspecific variation in DNA content (Price, 1988). Rayburn et al. (1985) found a significant negative correlation between DNA content and latitude and a significant positive correlation between genome size and the amount of heterochromatin in North American inbred and open pollinated lines of maize. Tito et al. (1991) found a positive relationship between genome size and the interval from germination to flowering in several races and lines of maize.

In the present investigation the 2C DNA content of several races from northwestern Argentina is examined. In this report we present information on range of DNA content variation and discuss the origin of this variation.

DNA content was measured in telophase nuclei (2C) of the root apex of germinating seeds. Roots of 0.5-1cm length were fixed in 3:1 (alcohol: acetic acid) for 1-4 days. After fixation

roots were rinsed for 30 min in distilled water. Hydrolysis was carried out with HCl 5N at 20 C + 2 for 30 min. The roots were then washed three times in distilled water for 10-15 min 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 min each, kept in distilled water (5-15 min) and squashed in 45% acetic acid. The coverslip was removed after freezing with CO_2 and the slide was dehydrated in absolute alcohol and then mounted in Euparal. The amount of Feulgen staining per nucleus, expressed in arbitrary units, was measured at a wavelength of 570nm using the scanning method with a Zeiss Universal Microspectrophotometer (UMSP 30). The DNA content per basic genome, expressed in pg, was calculated using c-tester line as a standard (2C= 5.78pg). The differences in DNA content were tested by an analysis of variance and comparisons between means using Scheffe's method.

There is interracial variation, ranging from 2C= 4.31pg (Altiplano) to 2C=8.8pg (Negro), which represent the lowest and highest values so far obtained and consequently the greatest range. The percent difference (51%) is the biggest found in maize. Other authors report Gaspe flint with 2C= 4.9 pg as the lowest value (Rayburn et al., 1985) and Zapalote chico with 2C=6.74pg (Laurie and Bennett, 1985), SC6 with 2C=6.87pg (Tito et al., 1991), and Tuxpeno with 2C=7.71pg (Guillin et al., 1991) among the highest values. The intraracial variation found in the Argentine races is 11-35% (Table 1).

Two sources of variation of DNA content were detected in these races. One is the presence of B-chromosomes and the other

Table 1. DNA content and B-chromosomes in six native races of maize from northwestern Argentina.

Taxa	DNA (2C) XE.±S.(pg)	No. nucleus	2n
"Altiplano"	4.31±0.30 a(1)		20
	5.00±0.53 b		20
	5.44±0.15 b	70	20
	5.62±0.25 b		20+2B
"Amarillo de Ocho"	5.49±0.29 bd	20	
	5.65±0.52 bd	41	20
	6.67±0.30 bd		20
"Capia blanco"	5.24±0.18 b		
	5.64±0.33 b		20+1B
	5.74±0.20 b	96	20+1B
	5.77±0.22 b		
	5.86 ±0.32 b		
"Capia rosado"	5.62±0.18 b		20+1B
	5.77±0.31 b		
	6.08±0.46 b	114	
	6.87±0.56 d		20+1B
	6.89±0.49 d		20+4B
	8.13±0.81 e		
"Pisingallo"	5.63±0.53 bd		
	5.68±0.40 bd		
	5.89±0.49 bd		20+1B
	5.91±0.30 bd		20+1B
	6.11±0.61 d		
	6.17±0.41 d	174	20+1B
	6.25±0.45 d		
	6.37±0.23 d		20+3B
	6.42±0.40 d		
	6.51±0.38 d		20
	8.65±0.71 e		20+4B
"Negro"	5.99±0.25 d		
	6.21±0.47 d		20
	6.22±0.18 d		
	6.47±0.33 d	117	
	6.95±0.76 e		
	7.86±0.80 e		
	7.96±0.72 e		
	8.80±0.78 e		20+1B

(1) Average values with the same letters are not significantly different.

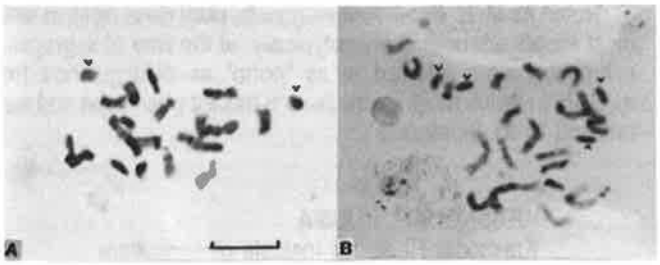

Figure 1. Mitotic metaphase. A: "Pisingallo" 2n=20+2B's. B: "Amarillo de ocho" 2n=20+4B's. Arrows show B-chromosomes. The bar=10µm.

is the polymorphism for repetitive DNA. Porter and Rayburn (1990) did not find a correlation between B-chromosomes and DNA content in twelve maize populations from Arizona. In the races studied in the present work the presence of B-chromosomes (Fig. 1) did not always increase the value of DNA content (Table 1). The DNA content and the determination of chromosome number was made in the same individual. A possible explanation for the observed results is the presence of polymorphism for repetitive DNA in A-chromosomes, which could mask the differences in total DNA content due to variation in the number of B-chromosomes. Differences observed in number of C+ bands in A-chromosomes among individuals of Negro race supported this hypothesis.

B-chromosomes in six Argentine races of maize

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Populations with extra chromosomes, termed B-chromosomes, are frequent in maize. These chromosomes have accumulation mechanisms which involve nondisjunction at a very high frequency in the second pollen mitosis. Pollen grains with B-chromosomes preferentially fertilize the egg cells.

In this investigation we analyze the meiotic behavior of B-chromosomes in several Argentine maize races, which have not been investigated cytologically. These races are: "Altiplano", "Amarillo de ocho", "Capia rosado", "Capia blanco", "Pisingallo" and "Negro".

In "Altiplano", individuals with 0, 1 and 3 B-chromosomes were found. In individuals with 2n=20+1B the B was outside the metaphase plate during metaphase I in 76% of the studied cells and migrated precociously to one pole during anaphase I. In the remaining 24% of the cells the B-univalent split the two chromatids and migrated to both poles during anaphase I. "Altiplano" individuals with 2n=20+3B's showed in diakinesis the configuration 10 II + 1 III (58% of cells) or 11 II + 1 I (42% of cells). In MI the frequency of cells with B's forming trivalents was 36%. In cells with B's forming 1 II + 1 I, the bivalent shows normal migration without separation from A-chromosomes while the univalent tends to move precociously to one pole at anaphase I.

In "Amarillo de ocho", only one individual with 2n=20+2B's was studied. The two B's formed one positive heteromorphic bivalent in pachytene-diplotene. In metaphase I it was indistinguishable from A-bivalents and has normal migration.

In "Capia rosado" the meiotic behavior of individuals with 2n=20+1B was analyzed. The B-univalent remains outside the plate in metaphase I, and in anaphase I moves precociously to one pole (Fig. 1A, B) or splits its chromatids, showing normal migra-

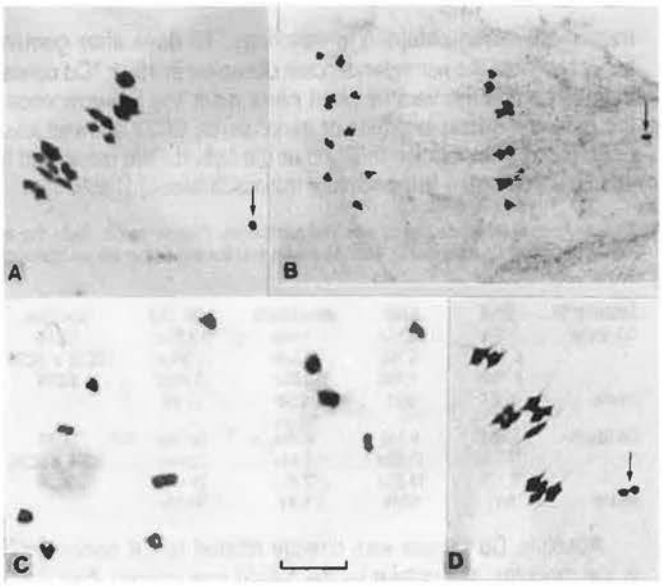

Figure 1. A and B: "Capia rosado"; A: metaphase I with 10 II + 1 B; B: anaphase I. C and D: "Capia blanco"; C: metaphase I with 11 II (20+2B's); D: diakinesis with 11 II. Arrows show B-chromosomes. The bar=10µm.

tion.

In "Capia blanco", individuals with 2n=20+2B's were studied. The most frequent configuration was 11 II and the B's migrate normally, together with the A-chromosomes (Fig. 1C, D). In some cells 10 II + 2 I was observed.

"Pisingallo" has 2n=20, 20+2B's and 20+4B's individuals. The 20+2B's show a meiotic behavior similar to that described for "Capia blanco". The 20+4B's showed 12 II. One of the extra bivalents shows a normal migration without separation from A-chromosomes, while the other migrates to one pole. Very few cells show 1 quadrivalent but the composition of this quadrivalent and its migration has not yet been studied.

In "Negro" one individual of the seven studied has 2n=20+2B. It showed in metaphase I 11 II or 10 II + 2 I. As in the other cases the B's formed bivalents segregating normally and the univalent moved precociously to the poles at anaphase I. This individual showed 2n=20 chromosomes in mitosis of root apex.

Our data indicated that, in the Argentine races, the meiotic behaviour of B-chromosomes to suppress meiotic loss is similar to that described by Carlson and Roseman (1992).

It is very likely that the presence of B-chromosomes modifies the inheritance of biochemical and morphological characters of agronomic importance, and need to be taken into account in plant breeding programs.

Cryptic homoeologies in the hybrid *Zea perennis* x *Z. mays* ssp. *mays* (2n=30) revealed by treatment with dilute colchicine solution

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Poggio et al. (Theor. Appl. Genet. 79:461-464, 1990) show the existence of cryptic homoeology in maize and *Z. perennis* (2n=40) using pre-zygotene colchicine (0.5 x 10^{-4}M) pretreatment. Under this treatment maize forms 1 to 5 quadrivalents and *Z. perennis* increases the frequency of quadrivalents. These results support the amphiploid nature of maize proposed by Molina and Naranjo (Theor. Appl. Genet. 73:542, 1987) and suggest that the species have homoeologous genomes (*Z. mays* ssp. *mays*, AmAm BmBm; *Z. perennis*, ApApAp'Ap' Bp1Bp1Bp2Bp2) which fail to pair, probably due to the presence of Ph-like genes (Poggio et al., ibid.). In *Z. perennis* (2n=40, ApApAp'Ap' Bp1Bp1Bp2Bp2) the number of quadrivalents increased from 5 to 10, revealing some cryptic homologies within the Ap and particularly within Bp genomes. The absence of octovalents shows the lack of homology between them.

Naranjo et al. (MNL65:74-75, 1991; submitted) pretreated with colchicine several stems of *Z. diploperennis* (2n=20, AdAd BdBd) and found no quadrivalents. Therefore, no noticeable homologies between Ad and Bd genomes were detected. In the *Z. diploperennis* x *Z. perennis* F1 hybrids (2n=30, ApAp'Ad BdBp1Bp2; and 2n=40, ApAp'AdAd Ap1Ap2AdAd) the frequency of multivalents increased. In the 2n=30 hybrid the frequency of trivalents increased from 25% in the untreated material to 90% in the treated one. In the 2n=40 hybrid, the quadrivalent frequency increased from 46% to 70%. Hexavalents or octovalents were not observed in these hybrids. These results show greater homologies between Bp and Bd genomes than those detected in untreated materials.

To complete the analysis of genome relationships we have studied one artificial F1 hybrid *Z. perennis* x *Z. mays* ssp. *mays*: 2n=30 (ApAp'Am Bp1Bp2Bm). 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 (over 154 cells studied). In addition, quadrivalent and hexavalent formation was observed in 43% of the cells. According to the homologies detected between Am and Bm genomes in treated maize material, the occurrence of IV and VI in this hybrid would be predicted.

The authors acknowledge Ing. E. A. Bernatené for the technical assistance in the treatment and handling of the plants. They also thank Dr. N. Jouve for the facilities offered at the Department of Cell Biology and Genetics of the Alcala de Henares University (Spain) where the slides and the cytological observations were performed.

2-hydroxyethyl disulfide effect on spindle and chromosomes

--M. C. Molina and E. A. Bernatené

Cytological evidence of the basic number x=5 in the genus *Zea* were obtained by Molina and Naranjo (TAG 73:542-550, 1987) and Naranjo and Molina (MNL61:62-63, 1987).

One observation was the formation of a double spindle with 5 II each. From the (C-banding) analysis of the chromosome distribution in each spindle it was concluded that they do not distribute at random. The formation of quadrivalents up to a maximum number of 5 IV was also observed when treating *Zea mays* with a diluted solution of colchicine. Similarly an increase in the number of quadrivalents up to a maximum of 10 IV was detected when treating *Z. perennis* and hybrids between *Z. perennis* x *Z. diploperennis* (2n=40) with the same solution (Poggio et al., TAG 79:461-464, 1990; Naranjo et al., Res. XXI Cong. Arg. de Genet., C. del Uruguay:62, 1990).

The protein called tubulin is the basic structural unit of the spindle. This protein frequently appears as dimers linked by S-S linkages of 40-80 A size. The mitotic apparatus only disrupts with compounds able to break the S-S linkages, for example 2-hydroxyethyl disulfide or with urea which breaks the hydrogen linkages (Zimmerman, p. 159 in The Cell in Mitosis, Levine, ed., Academic Press, New York, 1963).

With the purpose of studying in further detail the spindle and the chromosome distribution in the genus *Zea*, the cultivar "Ever Green" was treated with two different 2-hydroxyethyl disulfide diluted solutions ($1x10^{-4}$M and $2x10^{-4}$M) during 1991, whilst immature tassels of "Ever Green" and "Colorado Klein" were treated with three diluted solutions ($2x10^{-4}$M, $3x10^{-4}$M and $4x10^{-4}$M) during 1992. All treatments were practiced by putting the tassels into diluted solutions during 18 hours (keeping the submerged portion of the stem in absolute darkness). Thereafter tassels were washed by placing them in pots with tap water for 7 hours. Untreated tassels were cut simultaneously and were placed into tap water for 25 hours. Later on, treated and untreated tassels were fixed in 3:1 (absolute alcohol:acetic acid) solution and kept in a refrigerator until their study. Anthers were squashed in 2% acetic haematoxylin (Nuñez, 1988).

When comparing the treated and untreated tassels, it was seen that 2-hydroxyethyl disulfide concentrations at $1x10^{-4}$M and $2x10^{-4}$M did not affect any cells. Higher concentrations ($3x10^{-4}$M and $4x10^{-4}$M) showed significant differences at diplotene, diakinesis and metaphase I. 65% of the cells in metaphase I showed a clear differentiation in two spindles with 5 chromosomes each. In the remaining 35% of the cells this configuration was not clear. In some cells the apparent formation of two spindles was observed, with 8-9 chromosomes in one of them and only 1-2 chromosomes in the other.

A 5 II association into a group of compact appearance was observed. In others 5 II were distributed in a circle. This characteristic kept or was deeply observed in prometaphase I.

A remarkable difference never observed before in the untreated material is the appearance of a high number of quadrivalents, up to a maximum of five. This effect is similar to that produced when treating "Colorado Klein" with colchicine diluted solutions ($0.5x10^{-4}$M).

Other effects were the chromosome contraction and sticking among themselves, the differentiation amongst chromosomes inside each group being sometimes difficult. High numbers of univalents and endomitosis were also observed.

Cadmium toxicity during germination

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Numerous data from a number of sources have demonstrated that different cultivars of the same plant show differences in their Cd absorption characteristics. Different plant parts (leaves, stems, roots) accumulate different amounts of Cd (SCOPE 31:141, 1987).

Concentration of Cd in soils varied widely. Elevated values of 10mg/kg soil or more were found.

During 1991 (MNL66:61, 1992) and 1992 we have studied some characters during germination of maize grains in controlled conditions without soil to assess the comparative behaviour of the progeny and the progenitors treated with elevated amounts of Cd. As in 1991, we have employed two flint type inbred lines, SC66 and SC75, and the hybrid SC66 x SC75. Grains of similar weight (244.5mg) were individually put into vials with cotton-wool and 2ml of distilled water with 0, 44.5 or 89μM of Cd^{2+} (as Cl^-) and were grown at 28 C.

Relative mean Cd concentrations (μg Cd g DW^{-1}) and dry matter diminution obtained in seedlings, 10 days after germination, presented the same tendencies observed in 1991: Cd concentrations among the various plant parts mark the pre-eminence in the roots in relation to grains or aerial parts; SC75 showed lesser DM diminution in relation to SC66 or the hybrid. We measured the absolute Cd uptake (μg cadmium into each tissue) (Table 1).

Table 1. Absolute Cd uptake (μg) 10 days after germination. Control: no Cd. N=5. For each parameter (root, grain, aerial parts, total DM) means with the same letter are not statistically different (P=0.05).

Treatments	Root	Grain	Aerial parts	Total DM	Genotype
Cd 45μM	2.99e	4.74e	4.92b	12.65d	SC66
	6.70bc	5.74d	5.54b	17.98bc	SC66 x SC75
	6.12cd	6.64c	3.06c	15.82cd	SC75
means	5.27	5.71	4.50	15.48	
Cd 89μM	5.68d	9.74b	6.74a	22.16b	SC66
	11.72a	13.62a	7.44a	32.78a	SC66 x SC75
	7.12b	13.22a	2.28c	22.62b	SC75
means	8.17	12.19	5.49	25.85	

Absolute Cd uptake was directly related to Cd concentration in the medium. Cd uptake by the hybrid was greater than Cd uptake by SC66.

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Characterization and expression of a gene encoding a third member of the 18kDa HSP family in an inbred

--Manish Raizada, David B. Walden and Burr G. Atkinson

All organisms possess genes whose expression is up-regulated by heat shock/stress and code for a group of proteins referred to as the heat shock proteins (HSPs). Unlike animal HSPs, the majority of HSPs synthesized in plants appear to consist of a complex group of low molecular weight proteins with M_rs ranging from 15,000 to 30,000. Although the size and complexity of the low molecular weight HSPs vary among plant species, proteins of approximately 18kDa comprise the most prominent HSP class in maize, and, much like their counterparts in other organisms, are considered to be encoded from a complex, multigene family. In previous reports (Goping et al., MNL64:79-80, 1990; Plant Mol. Biol. 16:699-711, 1991), we described the genetic information from cDNAs encoding two different members of this family (cMHSP18-9 and cMHSP18-3). In this communication, we report the isolation and characterization of cDNA and genomic clones encoding information for a third member of this HSP family (g/c MHSP18-1).

A comparison of the sequence in the genomic clone with that obtained from the cDNA clone establishes that no introns are present in the gene encoding this 18kDa HSP. The 5' untranscribed region of gMHSP18-1 contains three, paired (decanucleotide), exact heat shock element (HSE) consensus sequences (all within an area of 62 nucleotides), and two putative TATA boxes. One TATA box is located 44 nucleotides upstream from the most 5' HSE and the other is located 18 nucleotides downstream from the most 3' HSE. The cDNA clone (cMHSP18-1) is 780 nucleotides long and contains a 465 nucleotide open reading frame (ORF) terminated by a TGA codon, flanked by a 5' untranslated region (UTR) of 58 nucleotides and a 3' UTR of 257 nucleotides containing a complete, animal-like polyadenylation signal sequence, AATAAA, 21 nucleotides upstream from the polyadenylated site.

A comparison of cMHSP18-1 with the two other 18kDa HSPs

in this same maize inbred reveals that although the 3' and 5' UTRs share little identity (34-54%), the ORF of cMHSP18-1 shares 94.6% identity with the ORF in the other 18kDa HSP mRNAs. Analyses of the amino acid sequence derived from the putative ORF of cMHSP18-1 predict that its protein product contains 154 amino acids, and has an M_r of 17,027 and an isoelectric point (pI) of 8.4. Although the M_r and pI predicted for the protein of cMHSP18-1 result in a protein slightly smaller and much more basic than the other characterized 18kDa HSPs in this inbred, the derived amino acid sequence of cMHSP18-1 shares 93% identity with the one derived from cMHSP18-3 and 89% identity with the one derived from cMHSP18-9.

Northern and dot blot hybridization analyses, using a DNA fragment specific (as shown by Southern hybridization analyses) for cMHSP18-1 (see Atkinson et al., this volume) with RNAs isolated from plumules and radicles of control (25 C) and heat-shocked (41 C) 5-day-old maize seedlings, substantiate that the expression of mRNA transcripts encoded from this gene are up-regulated during heat shock. Two-dimensional (IEF/SDS) polyacrylamide gel electrophoretic separation of the protein product transcribed and translated from cMHSP18-1, and of the protein product translated from hybrid-selected mRNAs, demonstrate that the nucleotide sequence in the ORF of g/cMHSP18-1 represents a gene which encodes another member of the 18kDa HSP multigene family in this inbred line.

The expression of the 18kDa HSP genes is independent and stage-specific during microsporogenesis and male gametophyte development

--Burr G. Atkinson, Robert A. Bouchard, J. Roger H. Frappier and David B. Walden

In previous reports (Goping et al., Plant Mol. Biol. 19:679-711, 1991; Atkinson et al., Develop. Genetics, in press; Raizada et al., this volume), we described the genetic information from genomic and cDNA clones encoding three different members of this family (designated as g/cMHSP18-1, g/cMHSP18-3 and cMHSP18-9) in an inbred line (Oh43). In this communication, we describe (1) the preparation of DNA fragments from these clones which contain nucleotide sequences which are either common to all of the characterized members of the 18kDa HSP family or specific for each of the 18kDa HSPs we characterized in this family, and (2) the use of these DNA fragments as probes to assess the expression of these genes during microsporogenesis and gametophyte development in Oh43.

We excised and subcloned, into pBluescript II SK$^-$, a 0.342kb *Pst*I/*Spe*I DNA fragment (scMHSP19-9-2) from the open reading frame (ORF) of cMHSP18-9 to use as a probe for a sequence common to all of the 18kDa HSP sequences we characterized in Oh43; it shares 90% identity with a sequence in both g/cMHSP18-1 and g/cMHSP18-3. We also excised and subcloned, into pBluescript II SK$^-$, DNA fragments from the 3' untranslated regions (UTRs) of each of the 18kDa HSP sequences we characterized in Oh43, and used them as probes specific for each of these 18kDa HSP sequences. These specific probes include a 0.242kb *Bst*XI/*Xho*I DNA fragment (scMHSP18-1-1) from the 3' UTR of cMHSP18-1, a 0.139kb *Hin*dIII/*Eco*RI DNA fragment (scMHSP18-3-3) from the 3' UTR of cMHSP18-3, and a 0.235kb *Sal*I/*Eco*RI DNA fragment (scMHSP18-9-3) from the 3' UTR of cMHSP18-9. Southern blot hybridization analyses confirmed the commonality of the scMHSP18-9-2 probe and the specificity of each of the other probes.

Northern and dot blot hybridization analyses with these 18kDa HSP probes, using RNAs isolated from plumules and radicles of control (25 C) and heat-shocked (42 C) 5-day-old maize seedlings, confirmed their ability to recognize mRNAs encoding the 18kDa HSPs in this inbred. Since studies in our laboratories (Frappier et al., in Stress Protein and the Heat Shock Response, Cold Spring Harbor Press, p.53, 1991; Atkinson et al., Develop. Genetics, in press) and other laboratories (Dietrich et al., Plant Physiol. 96:1268-1271, 1991) have reported an enhanced expression and/or accumulation of the 18kDa HSP mRNAs in the male gametophyte of maize in the absence of heat stress, we elected to assess the levels of mRNAs encoding specific members of this 18kDa HSP family during microsporogenesis and gametophyte development in Oh43. Our results, from Northern and dot blot hybridization analyses, demonstrate that mRNA transcripts encoding different members of this 18kDa family are expressed and/or accumulate independently, in a stage-specific manner during microsporogenesis and male gametophyte development. For example, mRNA transcripts recognized by g/cMHSP18-1 are predominant early in microsporogenesis (mid-prophase I through meiosis II), those recognized by cMHSP18-9 are most abundant in the binucleate stage of the gametophyte, and those detected by g/cMHSP18-3 do not appear to accumulate significantly at any stage of microsporogenesis or gametophyte development. These observations imply that the stage-specific expression of genes encoding particular members of this family results from gene-specific regulation during these phases of male gametophyte development, rather than from an overall activation of the heat shock or stress response. Moreover, these results suggest that particular members of this 18kDa HSP family may serve specific functions in the normal development of maize meiotic cells and/or the male gametophytes they produce.

Characterization of a cDNA encoding an 80kDa HSP and its expression in somatic and male gametophytic tissue

--J. Roger H. Frappier, Robert A. Bouchard, David B. Walden and Burr G. Atkinson

When an organism is exposed to an environmental stress, such as hyperthermia, the organism responds in a very predictable manner. The most striking aspect of the cellular response is the synthesis of a select set of proteins, the so-called heat-shock proteins (HSPs). Until recently, these highly-conserved HSPs were considered to solely protect the cell/organism from the damaging effects of an external stressor. However, an increasing number of reports have demonstrated that the expression of these HSPs is not restricted to a stress response, but their expression also appears to be developmentally regulated (reviewed in Lindquist and Craig, Ann. Rev. Genetics 22:631-637, 1988; Nagao and Key in Culture and Somatic Cell Genetics of Plants, Vol. 6 pp. 297-328, 1989). We have previously reported on the stage-specific expression of the low molecular weight HSPs (Atkinson et al., Dev. Genetics, in press) during microsporogenesis and male gametophyte development. We report here, the isolation and characterization of a partial 80kDa sequence from maize, designated scMHSP80-6-4, and on its expression in somatic and male gametophytic tissue.

A comparison of the 1887 base pairs in scMHSP80-6-4 with the Genbank database revealed that this sequence was in fact re-

lated to known HSP 80 genes. This sequence showed 80.6% identity with a rice HSP82A gene (Van Greusegen et al., unpublished) and 74.4% identity with the *Arabidopsis thaliana* HSP81-1 (Takahaski et al., unpublished). Since scMHSP80-6-4 is a cDNA clone, while the rice and *Arabidopsis* sequences are genomic clones, the homology was, as expected, restricted to exon 3 of HSP82A and exon 4 of HSP81-1. Further inspection revealed that scMHSP80-6-4 is lacking approximately 500 nucleotides downstream from the putative ATG start codon. A 587 nucleotide *Xho*I/*Pst*I fragment from scMHSP-6-4 was subcloned into pBluescript II SK$^-$ (designated as scMHSP80-6-5) and used in further studies of the maize HSP 80 gene(s).

Northern hybridization analyses with scMHSP80-6-5 of total RNA isolated from radicles and plumules of control (25 C) and heat-shocked (42.5 C) 5-day-old etiolated 0h43 maize seedlings, revealed the presence of a 2.6kb heat-inducible transcript. Furthermore, dot-blot hybridization analyses revealed that transcripts from the HSP 80 gene(s) are present at elevated levels (as compared to the level in control plumule RNA) throughout microspore development with peak accumulation in anthers containing early-to-midprophase I microsporocytes. The detectable amount of HSP 80 RNA is reduced in mature pollen. The developmental regulation of HSP 80 has previously been reported in diverse organisms such as yeast (Kurtz et al., Science 231:1154-1157, 1986) and mammalian male germ cells (S-J. Lee, Mol. Cell Biol. 10:3239-3242, 1990). It has been shown that the 80kDa gene family is divided into heat-inducible members (HSP 80), as well as developmentally regulated members (heat-shock cognate or HSC 80; Koning et al., Plant Physiol. 100:801-811, 1992).

In order to further characterize the expression of specific 80kDa family members in maize, screening of a genomic library was undertaken. A number of clones have been isolated and are presently under scrutiny. Furthermore, Southern blot hybridization analysis with scMHSP80-6-5 of different maize inbreds was performed in order to determine the number of family members present in this monocot. It is hoped that gene-specific probes for the 80kDa family may be found in order to clearly assess developmental expression during microsporogenesis.

Characterization and expression of genes encoding different polyubiquitin mRNAs

--Ling Liu, David B. Walden and Burr G. Atkinson

Ubiquitin is a remarkably conserved 76-amino acid-containing protein found in all eukaryotic cells examined. In eukaryotes, ubiquitin is encoded by a multigene family and the active ubiquitin monomer originates from post-translational cleavage of two different forms of a polyprotein precursor. One form of the precursor consists of a number of direct repeats of ubiquitin (i.e. polyubiquitin), and the other form consists of monoubiquitin linked via its C-terminus to a small, unrelated protein.

In this communication, we characterize the structure and expression of the sequences in genomic and cDNA clones encoding different polyubiquitin genes in an inbred line (0h43). One of the clones, g/cMub1, encodes an mRNA transcript containing seven tandem repeats of a nucleotide sequence for a ubiquitin monomer; the last repeat contains an extra, glutamine-encoding codon before the stop codon. The 3' UTR is 190 nucleotides long and contains 2 partial polyadenylation signals located 78 and 22 nucleotides upstream from the polyadenylated site. The gene for this polyubiquitin contains a 1005 nucleotide intron which, like one found in the sunflower (Binet et al., Plant Mol. Biol. 17:395-407, 1991), has its 3' splice site just before its ATG start codon. Another clone, g/cMub9, encodes an mRNA transcript containing five tandem repeats of a ubiquitin-encoding sequence; in this case, the extra codon in the last repeat encodes tyrosine. The 3' UTR of g/cMub9 consists of 206 nucleotides and contains a complete, animal-like polyadenylation signal 20 nucleotides upstream from the polyadenylated site. Since, at this time, the cDNA we have for this gene lacks a 5' UTR, we are unable to document whether or not its 5' UTR is also interrupted by an intron.

We have characterized the expression of these genes in radicles and plumules from control (25 C) and heat-shocked (42.5 C) 5-day-old seedlings by Northern and dot blot hybridization analyses of poly(A)$^+$ RNAs isolated from the total cellular RNAs and from the polyribosomal RNAs. The nucleotide sequences used for these analyses included a sequence from the ORF of g/cMub1 (scMubC1-1; a 0.473kb *Xho*I/*Sac*I-excised DNA fragment) common to both genes, and a sequence from the 3' UTR of each gene which is specific for each gene (MubC1-1, a 0.204kb PCR-generated DNA fragment specific for g/cMubC1; and scMubC9-3, a 0.224kb *Acc*II/*Eco*RI-excised DNA fragment specific for g/cMubC9). Results from the Northern and dot blot hybridization analyses establish that the mRNA transcripts corresponding to the sequence in g/cMubC1 is up-regulated in both the total RNA and polyribosomal RNAs during heat shock, while the level of the mRNA transcript corresponding to g/cMubC9 is not affected in either case by heat shock. These differences in the expression of these genes in response to heat shock clearly indicate the need for using gene-specific probes when assessing the expression of mRNA transcripts originating from different members of this multigene family.

Characterization and expression of genes encoding two different ubiquitin-fusion protein mRNAs

--Ling Liu, David B. Walden and Burr G. Atkinson

In this communication, we characterize the sequence and expression of two different ubiquitin fusion-protein genes, MubG7 and MubG10, in an inbred line (0h43). Both of the genes contain an ORF of 468 nucleotides which encodes for a polyprotein consisting of a ubiquitin monomer and an unrelated extension (fusion) protein consisting of 79 amino acids. While the ORFs of these genes share 95% identity, the first 300 nucleotides in the 5' and 3' flanking regions share only 30% and 46% identity, respectively. The derived amino acid sequence of the ubiquitin moieties encoded from MubG7 and MubG10 are identical to each other as well as to those derived from the maize polyubiquitin genes characterized elsewhere in this Newsletter (Liu et al.). The derived amino acid sequence of the fusion-proteins encoded from MubG7 and MubG10 differ by 5 amino acids. Both fusion-proteins are highly basic (pI ~10.8), and both contain sequence motifs for protein localization to the nucleus as well as zinc-finger structures for nucleic acid binding.

The expression of mRNA transcripts encoded from these genes was investigated in radicals and plumules from control (25 C) and heat-shocked (42.5 C) 5-day-old seedlings (0h43) by Northern and dot blot hybridization analyses. Poly(A)$^+$ RNAs, isolated from the total cellular RNAs and from polyribosomal RNAs, were probed with a DNA fragment from the fusion-protein sequence which is common to both of these genes (scMubG10-C-2; a 0.323kb *Nar*I/*Bgl*II-excised DNA fragment from MubG10 which

shares 95% identity with fusion-protein sequence in MubG7). Results from the Northern and dot blot hybridization analyses, using poly(A)$^+$ RNAs from the total cellular RNA, support the contention (Christensen and Quail, Plant Mol. Biol. 12:619-632, 1989) that the level of mRNA transcripts for these fusion-proteins does not change during heat shock. However, when poly(A)$^+$ RNAs from the polyribosomes of these tissues were similarly analyzed, the results disclosed that there is a marked depression in the levels of the fusion-protein mRNA transcripts associated with the polyribosomes from tissues undergoing heat shock. These studies demonstrate that the genes encoding the maize ubiquitin fusion-proteins are not heat shock-inducible genes. Moreover, the decrease in the amount of their transcripts on polyribosomes during heat shock implies that the synthesis of these proteins is, in fact, depressed by heat shock.

The expression of polyubiquitin and ubiquitin-extension genes is independent and stage-specific during microsporogenesis and gametophyte development

--R. A. Bouchard, J. R. H. Frappier, Ling Liu, B. G. Atkinson and D. B. Walden

Recent work employing cloned probes which represent members of the small heat shock protein genes of maize has shown that transcripts from some members of this stress-inducible gene family also accumulate in maize microsporogenic tissues during prophase and later stages of male gametophyte development (Bouchard and Walden, MNL 1990; Dietrich et al., Plant Physiol. 96:1268, 1991; Atkinson et al., this Newsletter). In light of the results with this stress-induced gene family, we have examined the developmental induction of other heat shock gene families during this important developmental sequence.

One such family is comprised of various genes that produce transcripts encoding the ubiquitin polypeptide. Maize cDNA or genomic clones representing a number of these genes have recently been isolated and characterized (Liu, PhD. Thesis, U. Western Ontario, 1991.; Liu et al., this Newsletter). We have used probes derived from these Mub (Maize-ubiquitin) clones to follow the overall accumulation of ubiquitin-encoding RNAs across maize microsporogenesis and gametophyte development by RNA-dot hybridizations. The RNAs used in these experiments were prepared from premeiotic tassels, staged and sorted maize anthers, and pollen, along with control and heat-shocked plumules of five-day seedlings. Our results indicate that there is in fact strong accumulation during development of RNA transcripts representing members of the ubiquitin gene family in maize. In addition, we find that this accumulation of transcripts is modulated independently for gene family members encoding two distinct types of ubiquitin polypeptides: polyubiquitins and "ubiquitin-fusion" proteins.

Because of the high evolutionary conservation of the ubiquitin ORF, a DNA fragment representing this region from the clone cMubC1 was used to determine the aggregate abundance of all transcripts encoding this polypeptide. The results indicate that the collective abundance of transcripts encoding the ubiquitin polypeptide itself is elevated throughout the entire developmental sequence relative to what is seen in either control or heat-shocked somatic tissue, with its apparent maximum late in development, after the haploid mitosis producing the binucleate gametophyte.

In order to compare this aggregate accumulation with that seen for individual members of the Mub gene family, subfragments of clones representing gene-specific regions were employed. Results obtained with probe MubC1-3, which represents the 3' untranslated region of a polyubiquitin gene, show a two-fold heat induced accumulation on polyribosomes in somatic tissue (Liu et al., this Newsletter). Across microsporogenesis and gametophyte development, the transcript of this gene shows an increase which parallels that seen for ubiquitin-encoding transcripts in general, but which appears to be even more pronounced at the end of the sequence, in anthers containing mature unshed pollen and in freshly shed pollen.

A contrasting pattern was found using a probe scMubG10-E, representing the 5' untranslated region of a particular type of ubiquitin gene, one encoding what is called a "fusion" or "extension" protein. This class of ubiquitin polypeptides consists of a single ubiquitin polypeptide segment fused to a distinctly different polypeptide containing a zinc-finger region; in yeast this portion of the protein has been shown to be a ribosomal component while the ubiquitin unit to which it is attached is required for efficient biogenesis of the ribosomes (Finley et al., Nature 338:394, 1989). Transcripts from this gene show their highest abundance at the beginning of the male developmental sequence, in premeiotic tassel and in anthers containing early microsporocytes. Interestingly, in addition to this independence in developmental regulation relative to MubC1, there is also a difference in accumulation of these transcripts on polyribosomes in somatic tissue during heat shock, where MubG10 transcripts show a decline in response to heat shock (Liu et al., this Newsletter). The high level of fusion-protein transcripts found in early maize PMC development (prior to and during prophase) may be significant in relation to the major turn-over of ribosomes that has been observed during meiotic prophase in other plant systems (Porter et al., J. Cell Sci. 62:177, 1983).

The maize genome also contains at least one polyubiquitin encoding gene, g/cMubC9, whose transcripts exhibit neither an increase nor a decrease in accumulation on polyribosomes of somatic tissue during heat shock (Liu et al., this Newsletter). The developmental modulation of transcripts detected by a probe specific for the 3' untranslated region of this gene is distinct from either of the others. Indeed, the accumulation of this transcript appears to parallel the pattern of aggregate abundance seen with the ubiquitin ORF probe (cMubC1).

The results of these studies indicate that, in addition to their heat shock regulation, transcripts from specific members of the ubiquitin gene family do exhibit developmental modulation during male meiosis and gametophyte development in maize. Moreover, specific members of this gene family encoding different types of ubiquitin-containing polypeptides show independent regulation. These observations are very analogous to the patterns emerging for members of the sHSP (small heat shock protein) family (Atkinson et al., this Newsletter). Programmed expression of specific gene family members may well prove to be a general feature of stress-gene families during this key sequence of development.

The production and characterization of HSP 18 polyclonal antibodies and their use as immunocytological probes in radicles

--Greg M. Brothers, Burr G. Atkinson, Dalia Kudirka and David B. Walden

The number of stressors reported to induce the expression of heat shock proteins (HSPs) in plants is extensive (Rees, MNL60:92, 1986; MNL61:69, 1989; Plant Physiol. 90:1256-1261,

1990). In many cases, the synthesis of HSPs has been implicated in the acquisition of stress tolerance. Further evidence suggests that some of the HSPs are developmentally regulated (Bouchard and Walden, MNL64:122, 1990; Atkinson, this issue). In spite of a concerted effort to understand the heat shock response little is known yet about the role these proteins play within the cell. We report on the preparation of polyclonal antibodies against the HSP 18 family and preliminary results on the intracellular localization of these proteins in maize radicles.

The low molecular weight 18kDa family of HSPs was isolated and purified from plumules of etiolated, 4 day-old Oh43 seedlings which had been heat-shocked at 42 C for three hours (as described by Baszczynski, Can. J. Genet. Cytol. 28:1076-1087, 1986). The 18kDa peptides were separated on 7.5%-17.5% gradient SDS polyacrylamide slab gels, eluted and concentrated. A small aliquot of each concentrated protein sample was electrophoresed on 1-D slab gels and samples containing only 18kDa peptides were pooled. An aliquot of the pooled peptides was separated further by 2-D IEF-SDS PAGE and visualized by Coomassie Blue staining and fluorography. The pooled peptides were then emulsified in complete Freund's adjuvant and injected subcutaneously into rabbits. Injections and boosts were performed as described previously by Rees (1989). Crude sera was purified by chromatography on a protein A sephadex CL-4B column (Pharmacia). The IgG-containing column fractions were tested for their antigenic specificity by immunoblotting using nitrocellulose and a Bio-rad alkaline phosphatase conjugate substrate kit to detect antibody binding. As well, immunoprecipitations of radiolabelled proteins extracted from heat-shocked and nonheat-shocked radicles was carried out according to the method of Baszczynski (1986), and immunoprecipitated peptides were analyzed by 2-D PAGE and fluorography. The resulting fluorograms and Western blots demonstrated the specificity of the IgG for the 18kDa family of HSPs. The HSP 18 antibodies showed no detectable cross-reactivity with other heat shock or nonheat shock peptides.

For the purpose of immunocytological localization, heat-shocked and nonheat-shocked Oh43 root tips were cut into 1mm cubic sections and fixed for one hour in 2% gluteraldehyde and 1% osmium tetroxide in 0.2M sodium cacodylate (pH 6.8) at 4 C. The fixed roots were embedded in Epon/Araldite. Gold sections were cut and placed on 400 mesh nickel grids or formvar/cabon-coated slot grids. Sections were treated with sodium periodate, blocked with 1% blotto (in PBS) and incubated with 1:10 IgG:PBS for 1 hour at room temperature. After rinsing in PBS the grids were incubated in protein A-gold conjugate for an hour, rinsed in PBS then ddH_2O and viewed on a Phillips CM 10 electron microscope at 60kV. Background level of colloidal gold labelling was determined by incubating serial sections either with preimmune serum or immune serum preabsorbed with 18kDa peptides (see Table 1). Pictures were taken, at 15,000 times magnification, of cortex root cells 1 to 2mm from the root tips. Cells of approximately equal size were chosen from three different heat-shocked and nonheat-shocked roots. Whole cell colloidal gold counts revealed that nonspecific labelling was significantly low (see Table 1), with nonspecific binding appearing randomly throughout the cell.

A definite pattern of HSP 18 distribution was evident in the heat-shocked root cells. Gold particles were clustered within the cytoplasm in groups ranging in size from 12 to 30 particles. This cluster distribution was consistent with observations made by autoradiographic detection of HSPs (Neumann, Eur. J. Cell Biol. 334:254-264, 1984). The clustering has been interpreted as representing the heat shock granules reported and characterized by Nover (Mol. Cell Biol. 3:1648-1655, 1983). A number of other organelles showed high levels of gold labelling (see Table 2). Label was detected in both the nucleus and nucleolus. Gold was also evident close to and on the E.R. and golgi membranes. We found no other cellular structures with a significant increase in the amount of detected HSPs during heat shock. However, increased levels of label were observed along short regions of the plasma wall, primary cell wall and across the middle lamella. Gold was found in discrete, irregular groupings along the cell wall, suggesting that there may be movement of HSPs between cells via the plasmodesmata.

Table 1. Whole cell quantitation of gold particles in heat-shocked and nonheat-shocked (control) root cells. The number of particles counted in control cells incubated with immune serum was assigned a value of 1.

Treatment	Pre-immune	Serum immune	HSP absorbed immune
Heat Shock	0.4±0.08*	36.7±6.1	0.8±0.43
Control	0.5±0.20	1±0.61	0.4±0.16

*mean of three cells ± standard deviation

Table 2. Distribution and relative quantity of the HSP 18 family within the cell. The numbers of gold particles within an organelle range from none (-) to greater than 30 particles per square µm (++++).

Organelle	Heat Shock	Control
cytoplasm	++++	-
nucleus	++	+
nucleolus	++	+
vacuole	++	++
mitochondria	-	-
golgi	+++	-
endoplasmic reticulum	+++	-
plasma membrane	++	-
cell wall	+	-
middle lamella	++	-

These primary results suggest that, after three hours of heat shock, HSP 18 peptides distribute in a predictable pattern within the cytoplasm and that the HSPs may be transported intercellularly.

Non-radioactive detection of single copy sequences in maize

--Dan Maillet, Kelly Jo Bates and Alex Richman

Recent advances in non-radioactive nucleic acid labelling and detection techniques have made it possible to detect single copy sequences on Southern blots of maize genomic DNA. We have adapted the digoxigenin based protocol, provided by Boehringer Mannheim (BM), for the detection of maize RFLPs. This contribution identifies changes or clarifications we have made to the steps in the BM protocols. (The steps identified below correspond to those presented in the BM protocols.)

DNA labelling (see DIG DNA labelling and detection kit protocol). We label (step 4) 200ng of DNA for 20 hours to ensure a high ratio of labelled to non-labelled fragments. The probe is precipitated (step 7) overnight at -70 C.

Southern Transfer. We load 0.8% agarose gels , 150ml, 11 by 14cm with 15-20µg of digested genomic DNA per lane and separate by electrophoresis at 20-40 volts in a model H5 gel tank (BRL). DNA is transferred under neutral conditions, as specified in Molecular Cloning (Sambrook et al., 1989), to BM positively charged nylon membranes and cross linked for 3 min in a UV Stratalinker 1800 (Stratagene).

Hybridization and stringency washes (see Lumigen PPD protocol). Wet blot in 2 X SSC. Prehybridize blots in plastic bag with 20ml of hybridization solution for 2 hours at 42 C with mild agitation. Hybridize (step 2) overnight (approx. 16 hours) with 5 to 6ml of hybridization solution containing 10ng per ml of a denatured DIG labelled probe. The probe is denatured by boiling in a water bath for 10 min and quickly cooled in salt and ice.

Chemiluminescent detection. Incubate (step 5) for 2 hours in 100ml of buffer 2. Wash (step 8) 6 X 5 min with 100ml of washing buffer. Drain substrate solution (step 12) and store at 4 C in dark for reuse (up to 5 times), then reseal the bag (step 13).

Comments. The protocols provided by Boehringer Mannheim resulted in luminographs that could be scored; however, background levels were often unacceptably high. Increased washes after incubation with the antibody was the most important change with respect to lowering background. A longer block in buffer 2 also decreased background. Plastic forceps should be used when handling the membrane. For all steps, use a clean plastic box (21 x 14 x 6cm) unless the volume required is lower than 20ml; in which case a sealed plastic bag may be used. If blots are to be reprobed, they must not dry out at any time. Following stripping, blots can be stored in redistilled sterile water at 4 C.

We have reprobed blots 5 times and have not noticed a decrease in signal strength or an increase in background. Using the Boehringer Mannheim protocols with the modifications outlined in this article we have detected single copy sequences (see Figure 1) in order to determine the strain distribution pattern for a 2kb genomic probe (see the following article). We are interested in correspondence with others using the DIG system or variations of this method.

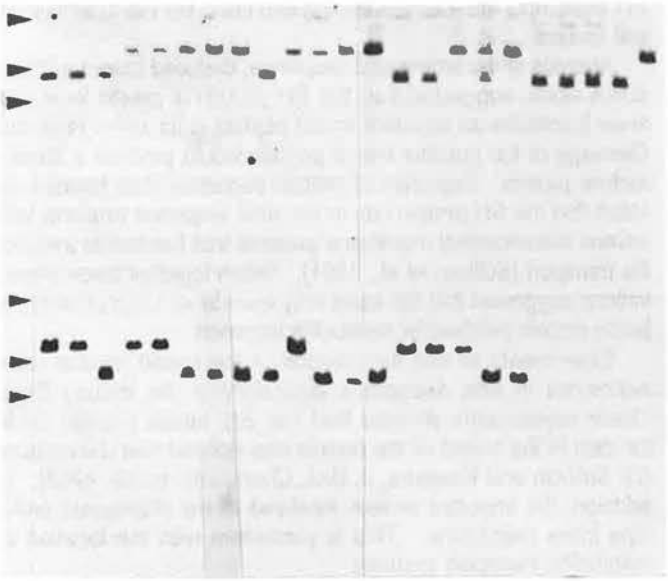

Figure 1. Southern blot analysis using the DIG detection system reveals the strain distribution pattern for a 2Kb genomic probe in the Co x Tx recombinant inbred family. Standard markers of 23130, 9416, and 6557bp are indicated by arrow heads.

Recombinant inbred mapping of a ubiquitin fusion protein gene

–Dan Maillet, Burr Atkinson and David B. Walden

In this communication we describe the mapping of a specific ubiquitin fusion protein gene, MubG7. Inspection of the genomic sequences obtained for two Oh43 ubiquitin fusion-protein genes indicated that the nucleotide sequence 5' to the open reading frame in these genes share little identity (approximately 30%; see Liu et. al., this Newsletter). We excised a 2.0kb, 5'-DNA fragment, designated as scMubG7-J, with *Eco*RI/*Xba*I from a 6.2kb insert in a genomic clone containing MubG7, and assessed the specificity of the fragment by Southern blot hybridization analysis. These analyses confirmed that this DNA fragment recognizes a single copy ubiquitin fusion-protein gene and, therefore, this DNA fragment was used to map this gene in maize.

Southern blots of the genomic DNA extracted from the members of the recombinant inbred families T X CM, and Co X Tx, provided by B. Burr (Brookhaven National Laboratory), were probed with the sequence scMubG7-J, using the digoxigenin (DIG) DNA labelling and detection kit (Boehringer Mannheim), employing the modifications given by Maillet et. al. (this Newsletter). From the resulting luminographs the strain distribution pattern (SDP) was determined for this probe, which will be referred to as uwo1(scMubG7-J) on the RI linkage map. Comparison of the SDP for this sequence with the SDPs for all sequences contained within the RI database revealed very close linkage between our probe and two RFLP markers, *npi438* and *umc7*, allowing uwo1(scMubG7-J) to be assigned a location on the long arm of chromosome 8 at position 92.

Callus induction from various genotypes

--Kelly Jo Bates

Independent callus culture lines were generated from over 5000 immature embryos, ranging from 0.8mm to 6.0mm in length. Callus cultures of the following genotypes were established on both MS (Murashige and Skoog) and N6 callus induction media: inbreds A188, A632, B37, B73, CO159, F2, K55, M14, Mo17, N28, Oh43, TANA, Va26, W23, W64a; an Ontario flint; and the hybrid Seneca 60. Embryo age ranged from 9-18 days post pollination; the cultures were maintained at 26 C with a 18h light/6h dark cycle. Callus was characterized according to established (Type I and Type II) criteria (Torné, 1984), or a third class, Type III. Type III consisted of a very distinct sponge-like texture, with an amber to brown appearance.

Table 1. Types of callus observed for the genotypes over a 5 month period of culture.

I	II	III	I & II	II&III	I&II&III
B37	TANA	A632	A188	N28	M14
CO159	Va26	F2	Seneca 60		Mo17
		K55	W64A		
		Oh43	Ontario Flint		
		W23			

Table 2. Preferable media for inducing callus based on responses over a 5 month period of culture.

MS	N6	BOTH
B73	B37	A188
Mo17	M14	CO159
N28		SENECA 60
W64A		TANA
A632		VA26
Ontario Flint		F2
		K55
		Oh43
		W23

Regenerated plants from callus over a 5 month period were obtained from: A188, A632, B73, CO159, K55, M14, N28, Oh43, Seneca 60, TANA, W23, W64a; and an Ontario flint. Observations on callus growth are summarized in Tables 1 and 2.

An extensive RFLP analysis is underway on samples of callus and regenerated plants.

MADISON, WISCONSIN
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Corngrass (_Cg_) increases susceptibility to common rust and European corn borer

--B. G. Abedon and W. F. Tracy

Cg, when fully expressed, greatly alters the phenotype of the plant, often making plants carrying the allele unrecognizable as corn. Over the years we have observed that *Cg* increases susceptibility to many diseases and pests. To test this observation we performed two trials. In trial 1 we evaluated the effects of *Cg* on susceptibility to common rust (*Puccinia sorghi*). Seed of five inbreds segregating for *Cg* were planted in three replications. Plants were inoculated with spores of common rust and later evaluated for per cent leaf area destroyed by common rust. Averaged over all reps and inbreds the *Cg* plants had significantly more rust damage (51%) than did their normal counterparts (21%). The difference was consistent over all inbreds but the magnitude of difference was greatest in the more susceptible inbreds. We also did a feeding study with second instar European corn borers (*Ostrinia nubilalis*) in which the insects were offered a choice of *Cg* leaf tissue vs. normal leaf tissue. The data are not fully analyzed, however when averaged over reps and inbreds *Cg* leaf tissue was consumed more rapidly than the normal tissue. Thus in addition to its many effects on plant development, *Cg* affects host-parasite interactions for both insects and fungi.

Systemic effects of *Vg*

--W. F. Tracy

Vestigial glume, *Vg*, reduces the length of the glume in both the pistillate and staminate spikelets. It also has been reported to reduce ligule size. To investigate other effects of *Vg* we measured plant and ear height in eight inbreds that are near-isogenic for *Vg*. All *Vg* plants measured were heterozygous *Vg*. In extreme cases *Vg* reduces the size of the tassel, thus we measured plant height from the ground to the ligule of the upper most leaf and ear height from the ground to the ligule of the leaf subtending the ear. In each inbred *Vg* plants were shorter than their normal siblings (Table 1). *Vg* plants averaged over all 8 inbreds were only 82cm tall while their wildtype counterparts were 104cm. The most extreme difference was in the inbred Wh8716 in which the *Vg* plants were only 66% the height of the wildtype plants. Ear height was the same for both types. The stalks of many, if not most, of the *Vg* plants were twisted above the ear-bearing node. We have also observed that ear size is reduced by *Vg* in many inbreds and we will be performing yield trials next summer to test this observation.

Table 1. Plant and ear heights from *Vg* and wildtype plants from 8 inbreds near-isogenic for *Vg*.

	plant height (cm)		ear height (cm)	
Inbred	*Vg vg*	wildtype	*Vg vg*	wildtype
Crsh2 13	100*	127	58	64
Crsh2 19	93	110	61	67
Crsh2 913	66*	85	28	30
Crse 12	110*	130	52	57
Wh9261	71*	95	35	38
Wh9235	68*	95	30	31
Wh8716	60*	90	35	40
Wh8450	87*	103	45	46

*=significantly less than wildtype

Pee Wee rediscovered?

--W. F. Tracy

Reduced1 (*rd1*) was discovered as a mutation in the sweet corn inbred P39. The *rd1* plants were selfed and the resulting inbred was named C30. C30 is approximately 75-80cm tall while P39 is 115-120cm. C30 flowers 5-7 days earlier than P39. Lachman (JASHS 97:347-350, 1971) reported the existence of a mutation from C30; Pee Wee, believed to be allelic to *rd1*. As the name suggests Pee Wee was even smaller than C30. However, Pee Wee was lost. In 1992, in an F3 derived from the cross P39 x C30 there was the expected segregation of *Rd1* and *rd1* plants but also there were plants 45cm tall. Unfortunately these plants were male sterile but they were sibbed with *rd1* plants within the same family. If this new mutation is allelic to *rd1* this is the second time that this allelic series has developed within P39/C30 indicating that *Rd1* may be unstable in this inbred background.

The *brittle1* locus encodes an amyloplast membrane protein

--Thomas D. Sullivan and Oliver E. Nelson, Jr.

Mutations in the *brittle1* (*bt1*) locus result in a collapsed endosperm phenotype with reduced starch deposition, but the primary defect of mutant *bt1* alleles is not known. To provide tools for determining the function of the *Bt1*-encoded protein, we have isolated molecular clones of the locus using a transposable element-tagged allele (Sullivan et al., Plant Cell 3:1337, 1991). As expected from the endosperm-specific defect of *bt1* mutants, Northern blots probed with the cloned *Bt1* sequence show that *Bt1* transcripts are found in endosperm cells, but not in embryo or leaf tissues.

Analysis of the amino acid sequence, deduced from a wildtype cDNA clone, suggested that the *Bt1* protein is plastid localized, since it contains an apparent transit peptide at its amino terminus. Cleavage of the putative transit peptide would produce a 38.6Kd mature protein. Searches of protein sequence data bases indicated that the *Bt1* protein has amino acid sequence similarity with several mitochondrial membrane proteins that function in metabolite transport (Sullivan et al., 1991). Taken together these observations suggested that the locus may encode an amyloplast membrane protein involved in metabolite transport.

Experiments to test the function of the transit peptide were performed in Ken Keegstra's laboratory in the Botany Dept. These experiments showed that the *Bt1* transit peptide could function in the import of the protein into isolated pea chloroplasts (Li, Sullivan and Keegstra, J. Biol. Chem. 267:18999, 1992). In addition, the imported protein localized to the chloroplast envelope inner membrane. This is consistent with the location of metabolite transport proteins.

To confirm that the *Bt1* protein is an amyloplast membrane protein, the subcellular location of the protein was investigated. Antibodies against the carboxy-terminal 56 amino acids of the *Bt1*-encoded protein were obtained by immunizing a rabbit with a

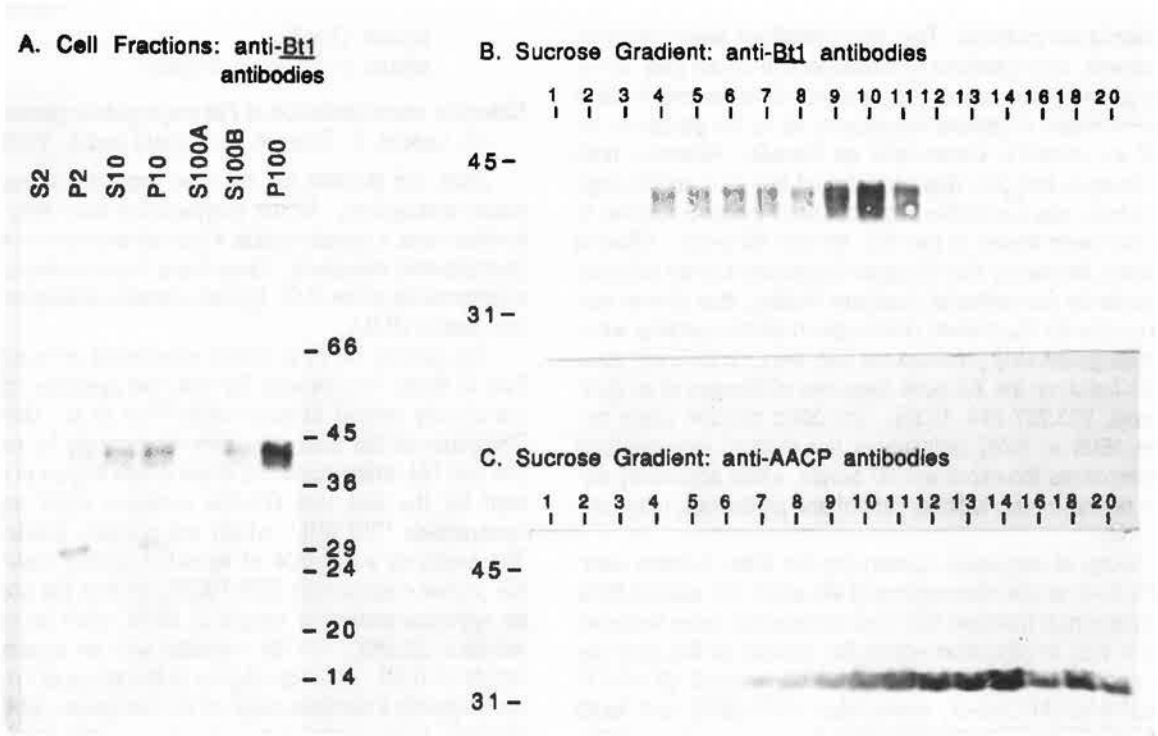

Figure 1. Western blots of endosperm cell fractions. Blots were probed with the primary antibody as indicated on the panels. Secondary antibody was phosphatase-linked goat-anti-rabbit IgG, and the blots were stained using NBT/BCIP. A. Following homogenization, sequential centrifugation of extracts yielded supernatant (S) and pellet (P) fractions from the 2,000, 10,000 and 100,000 xg steps (2, 10 and 100, respectively). Each lane contains 2mg of protein. B. Sucrose gradient fractions are labeled from the top of the gradient. An equal volume from each fraction was loaded. C. A parallel gel was run with the same fractions as used in Panel B.

fusion protein produced in *E. coli*. The fusion protein was produced from a plasmid containing a restriction fragment from the *Bt1* cDNA clone inserted downstream of the glutathione-S-transferase gene in the plasmid pGEX-2T (Smith and Johnson, Gene 67:31, 1988). Following purification by affinity chromatography, the anti-*Bt1* antibodies react with specific proteins on Western blots of endosperm extracts. These proteins are missing in mutant extracts from the *bt1-R* allele (data not shown).

In the summer of 1992, self-pollinated W64A endosperms were harvested (20 days after pollination) and processed to isolate the microsomal membrane fraction using the protocol of Gardner et al. (Physiol. Plant. 69:541, 1987). This fraction should be enriched in amyloplast membranes. In Figure 1A fractions obtained at the different centrifugation steps were probed on a Western blot with the anti-*Bt1* antibodies. The microsomal membrane fraction (i.e., the 100,000 xg pellet or P100) is enriched for the protein detected by the anti-*Bt1* antibodies. (Actually, Western blots appear to contain more than one *Bt1* protein band, with apparent molecular weights ranging from 38 to 42Kd. The basis for this pattern is unknown.)

The P100 was further fractionated by centrifugation through a sucrose density gradient. The gradient fractions were also probed on Western blots (Fig. 1B and C). The *Bt1* proteins are found in fractions 4 through 11 in the gradient (Fig. 1B). These fractions also contain the carotenoid pigments of the amyloplast membranes (data not shown). In Figure 1C, a parallel blot of the gradient fractions was probed with antibodies against the mitochondrial membrane ATP/ADP carrier protein (AACP; Hallermayer et al., Eur. J. Biochem. 81:523, 1977). Although there is overlap between the fractions containing *Bt1* and AACP, the distribution of the two proteins is clearly different. The migration of *Bt1* protein with the carotenoids and in different fractions from a mitochondrial membrane protein suggests that the *Bt1* protein is located in the amyloplast membrane.

From these experiments, it seems likely that the *Bt1* protein functions as a metabolite transporter. Further experiments will be necessary to determine its metabolite specificity. These results indicate that the deficiency for a starch granule-bound oligosaccharide synthase activity, reported earlier (Pan and Nelson, MNL59:105, 1985), is not the primary lesion of *bt1* mutations.

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A PCR-based assay for the *wx-c* allele of maize

--Dave Zaitlin

Many elite maize dent inbred lines have nearly isogenic counterparts that have been "converted" to the waxy kernel phenotype by introgression of the recessive *wx* gene through conventional backcross breeding. The efforts by many industrial corn breeders to accomplish this have been driven by the increasing market for waxy grain as animal feed and by the industrial demand for cornstarch composed solely of amylopectin. To effect a rapid conversion of Agrigenetics starchy inbreds to waxy, we required a simple, precise and very fast assay that would allow us to distinguish the mutant gene from the wildtype gene. Additionally, the assay of choice had to be essentially non-destructive, as we wanted to perform it on very young maize seedlings, and had to be able to unequivocally identify *Wx/wx* individuals. While not as inexpensive as I/KI staining of kernels, the polymerase chain reaction (PCR) meets all of the above criteria.

Wessler and Varagona (PNAS 82:4177-4181, 1985) used molecular techniques to determine the structure of the *Wx* locus in

a collection of *wx* mutants. This study identified twelve different genetic events, both deletions and insertions, that can give rise to the waxy phenotype in maize. Discussions with commercial maize breeders revealed a general uncertainty as to the particular allele(s) of *wx* carried in commercial *wx* inbreds. However, both George Sprague and Sue Wessler believed that *wx-c*, which originated in China, was a possible candidate since it does not revert to *Wx* and has been known in the U.S. for over 80 years. While in Dr. Wessler's laboratory Ron Okagaki discovered a small deletion, undetectable by conventional Southern blotting, that almost certainly accounts for the mutant phenotype in plants carrying *wx-c*. Dr. Wessler generously provided me with the (unpublished) position of this lesion on the *Wx* gene sequence of Klosgen et al. (Mol. Gen. Genet. 203:237-244, 1986). The 30bp deletion spans nucleotides 1606 to 1636 (relative to the start of transcription) and encompasses the exon7-intron7 border, which apparently disrupts the normal in vivo splicing pattern and produces a non-functional mRNA.

The 500bp of sequence surrounding the 30bp deletion averages 51% G+C, unlike other regions of *Wx* which can exceed 85%. Two primers which matched this G+C composition were designed to direct in vitro amplification across the position of the gene sequence encompassing the deletion. Primers wx#2 (5'-ACCTCAAGAGCAACTACCAG-3'; nucleotides 1567-1576) and wx#3 (5'-CTTCATCCAGTTGATCTTCCGG-3'; nucleotides 2000-1979) were synthesized and purified at Epicentre Technologies in Madison, WI. A typical 50µl amplification reaction contained 200-250ng of maize DNA, the primers at a concentration of 1µM each, dATP, dCTP, dGTP and TTP at 0.2mM each, 1 unit of *Taq* DNA polymerase, and the buffers and salts as specified by the enzyme supplier (Perkin-Elmer Cetus). Sequence amplification was effected by cycling the reactions 30 times at 94 C for 1 minute, 55 C for 1.5 minutes and 72 C for 3 minutes. As predicted from the gene sequence, a DNA fragment of 433 base pairs was amplified from genomic DNA isolated from the inbreds C123, W64A, Oh43, Va35, and MBS501. DNA from a maize stock carrying the *wx-c* reference allele (from O. E. Nelson) and C123*wx*, W64A*wx*, Oh43*wx* and MBS501*wx* directed synthesis of a 403bp fragment in the PCR, however. Mixing experiments and amplifications with DNA extracted from known *Wx/wx* heterozygotes showed that the 433 and 403bp fragments are clearly separable on agarose gels of 1.2-1.5% (w/v) in standard Tris-acetate electrophoresis buffers. The notion that *wx-c* has been widely incorporated into commercial dent corn inbreds is also supported by the finding that DNA from plants homozygous for the mutant waxy alleles *wx-B*, *wx-B1*, *wx-B6*, *wx-K*, *wx-M*, and *wx-Stonor* (see Wessler and Varagona, 1985) all support amplification of the 433bp fragment in the PCR.

Coupled with a technique for extracting total DNA from very small amounts of plant tissue (Zaitlin, DeMars and Ma, Genome, in press, 1993) the PCR-based assay for detection of *wx-c* should be valuable for accelerated conversions of starchy inbreds to waxy. The breeder can simply screen 10-20 seedlings after each backcross and save those that are *Wx/wx* for the next generation. Fewer individuals need to be carried along and, after 4 or 5 cycles, conventional RFLP can be used to identify the plants that most closely resemble the recurrent parental line. These are then selfed and the resulting seedlings subjected to PCR to find the *wx/wx* plants.

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Molecular characterization of *Zea mays* glutelin genes and proteins
--B. Lazzari, F. Sparvoli, L. Bernard and A. Viotti

Zeins and glutelins are the most important storage proteins of maize endosperm. All the polypeptides belonging to these two families have a signal peptide which allows them to enter the E.R. (endoplasmic reticulum). Once there, these proteins are stored in enlargements of the E.R., typical of maize endosperm, called protein bodies (P.B.).

The glutelin family is mainly composed of four polypeptides. Two of them, G1-204 and G1-164, are cysteine-rich (6.6%) and are closely related to each other (Prat et al., Gene 52:41-49). The genes of this subfamily code respectively for polypeptides of 204 and 164 amino acids and share a high degree of homology, except for the fact that G1-204 contains eight repeats of the hexapeptide "PPPVHL", which are partially deleted in G1-164. The presence in G1-204 of repeated proline triplets decreases the protein migration in SDS-PAGE, so that the polypeptide has an apparent molecular weight of 28Kd, while its real molecular weight is 22.8Kd. G1-164 migrates with an apparent molecular weight of 16Kd. The high degree of homology of G1-164 and G1-204 suggests a common origin of the two genes, which could have diverged during evolution from a unique progenitor.

The other two glutelin genes, called G2 and G3, are methionine-rich (11% and 20%, respectively) and code for polypeptides of apparent molecular weight of 15 and 10Kd. These polypeptides are also known as β and σ zeins (Pedersen et al., J. Biol. Chem. 261:6279-6284; Anderson, Kirihara et al., Mol. Gen. Genet. 211:477-484).

The complete cDNA sequences of the four genes have been amplified by PCR from total endosperm RNA using specific primers and have been used as probes in Southern blotting experiments. Total DNA extracted from different maize lines (W64A, A69Y and W22) and tissues and digested with different restriction enzymes has been loaded on agarose gels and blotted to Hybond N+ membranes.

Hybridization revealed that there is no difference concerning G1, G2 and G3 patterns between 2n and 3n tissues (embryo and endosperm, respectively), while there is polymorphism in the different maize lines. Southern blotting analysis has also been performed to determine the number of copies per haploid genome of these genes. As G1-204 and G1-164 cross-hybridize, due to their high homology, their copy number has been determined using only the 3' end as a probe, which is almost identical in the two genes: data show that there are between 5 and 10 copies per haploid genome of genes belonging to the G1 subfamily (G1-204 + G1-164).

The same experiment has been carried out with G2 and G3 probes: G2 seems to be present in 1-2 copies, and G3 is present in 2-3 copies per haploid genome.

The importance of glutelins as storage proteins is mainly due to their high sulphur content, especially if we consider that zein, which is the most important family of storage proteins in maize, has a very low content of methionine and cysteine.

A new allele of *sr2* isolated in the progeny of regenerated plants

--M. L. Racchi, G. Todesco, A. Chiusi* and T. Chiari*
*IAO, MAE, Florence

By screening ears originally derived from the selfed progeny of regenerated plants, we isolated two mutants with margins of leaves white-striped, resembling *sr2* in their striping pattern. A complementation test was then run making each mutant heterozygous with *sr2* and scoring for presence of striped leaf phenotype in the mature plant as a proof of allelism between the mutant and *sr2*. This test led to the identification of one additional allele at the *sr2* locus. Reciprocal crosses of the mutant (coded as L468) with *sr2* gave rise to striped plants thus confirming the allelism of the mutant with this locus.

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University of Milan

Genetic variability of maize GST and relation to herbicide tolerance

--M. Sari Gorla, S. Ferrario, L. Rossini and C. Frova

Metabolic detoxification is probably the most general major mechanism involved in plant tolerance to herbicides. In particular, the glutathione (GSH)/glutathione-S-transferase (GST) system appears to be widespread among plants and to confer protection against toxic chemicals by catalysing the conjugation of glutathione to an electrophile center of hydrophobic molecules by means of the SH group. Species tolerant or susceptible to a wide spectrum of herbicides (S-triazines, acetoanilides, thiocarbamates) are characterized by high and low levels of GST respectively, and an increase in specific GST activity in response to some herbicide treatment has been reported in a few cases. Thus the system appears to be involved in the determination of plant tolerance to these classes of molecules.

The system is complex: in maize, at least 3 different GSTs (GST I, II and III) have been detected. They are all functionally dimers and two of them (I and III) have been molecularly characterized. The genetic characterization of the system, however, is far behind. In particular, no information is available about the genetic variability existing within species with regard to the efficiency of the system and of its components.

We have analyzed GST expression by gel electrophoresis in different tissues of several maize inbred lines. Fig. 1a illustrates the enzyme electrophoretic profile in 4 tissues of inbred H99, while the results from 9 genotypes have been pooled together in Fig 1b. On the basis of these zymograms, four GST activity zones (A, B, C, D) have been identified on the gels: some are common to all tissues, while others are found only in the scutellum. Taking into account also the relative band intensities in the tissues analyzed, the presence of two different sets of genes is hypothesized, one expressed mainly in roots and pollen and one in the scutellum. Moreover, although the multiplicity of the protein species observed could be due to the expression of many genes as well as to posttranslational modifications and random association of different monomeric molecules, the analysis of the enzymatic variants and of their combination in different genotypes suggests that at least five genes control GST isozymes in maize.

In order to explore the existence of genetic variability for the efficiency of the system to detoxify EPTC (a thiocarbamate) and Alachlor (a chloroacetanilide), we measured GST activity, basal

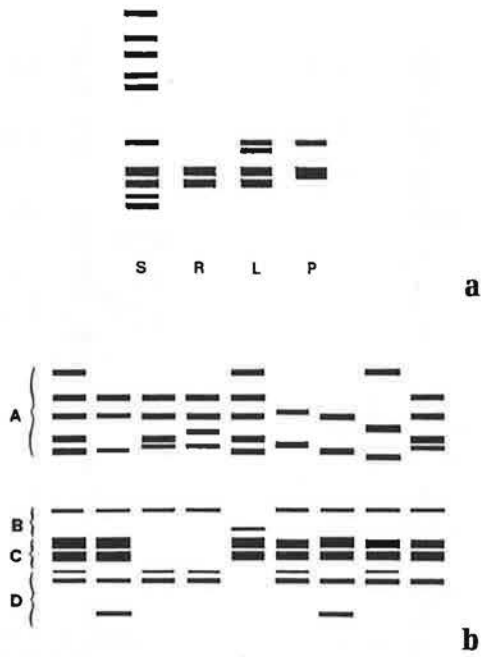


Figure 1. a: Schematic representation of GST isozyme profile in scutellum (S), roots (R), leaves (L) and pollen (P) of inbred H99. b: GST variants expressed in different tissues (pooled data) of nine genotypes.

and after herbicide application, in roots, leaves, scutella and pollen of different genotypes. A wide variability in the basal level was observed in all tissues. Moreover, even though the levels of activity were not equal in all tissues (very high in pollen, scutella and roots, lower in leaves), they were correlated in the different tissues.

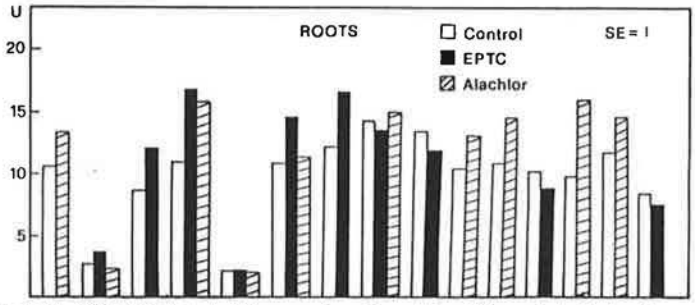


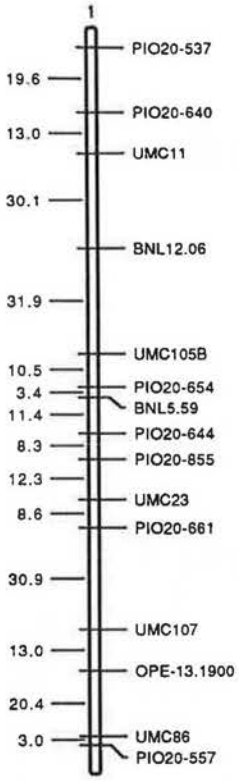
Figure 2. GST activity in roots of fifteen genotypes in control conditions and after treatment with EPTC or Alachlor. SE=standard error.

In roots (Fig. 2), an increase of GST activity was detected after herbicide treatment, in almost all the lines in response to Alachlor, only in some in response to EPTC. A similar pattern was observed in leaves, while scutella and pollen do not seem to respond to herbicide application.

A novel RFLP linkage map from an F2

--M. Enrico Pè, Raffaella Greco, Graziana Taramino, Luca Gianfranceschi, Renato Tarchini and Giorgio Binelli

We present here the linkage data from the analysis of 150 plants of an F2 generation obtained from inbreds B89 and 33-16. The RFLP clones used were from Pioneer Hi-Bred Intl. (pio), Brookhaven Natl. Lab. (bnl) and University of Missouri-Columbia (umc). A total of 160 have been screened on the parents to detect polymorphism using *Eco*RI, *Bam*HI and *Hin*dIII as restriction enzymes. 95 polymorphic clones have been chosen to be used for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13.0
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3.4
BNL5.59
11.4
PIO20-644
8.3
PIO20-855
12.3
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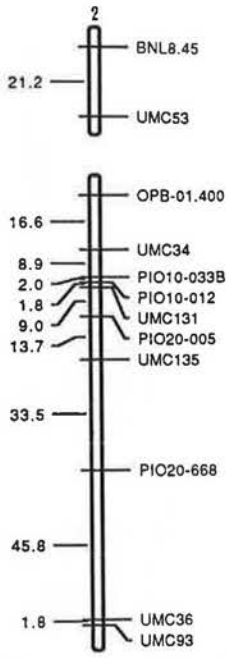
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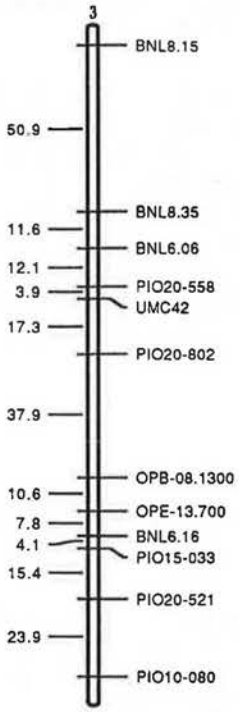
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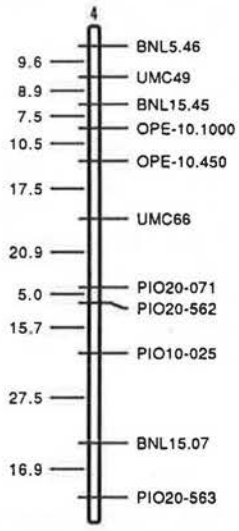
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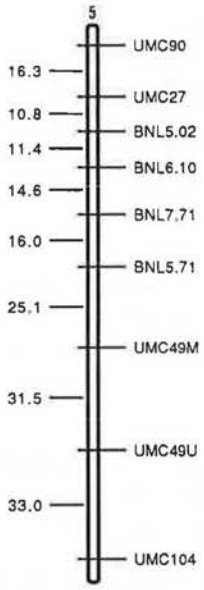
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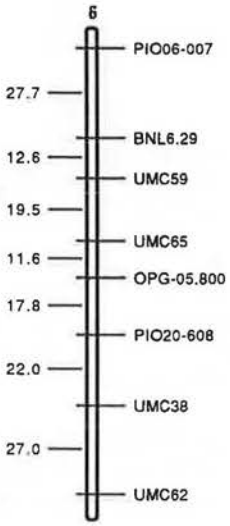
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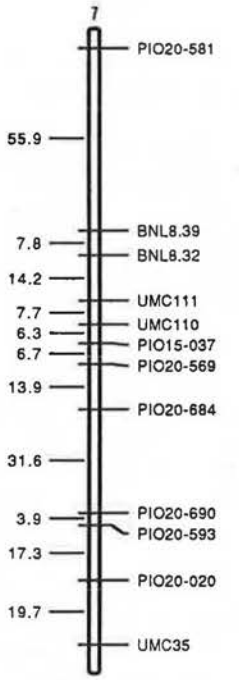
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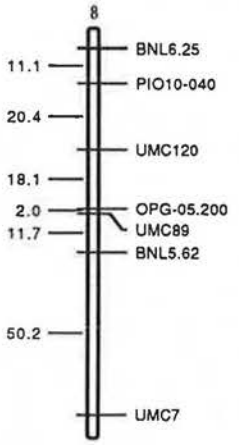
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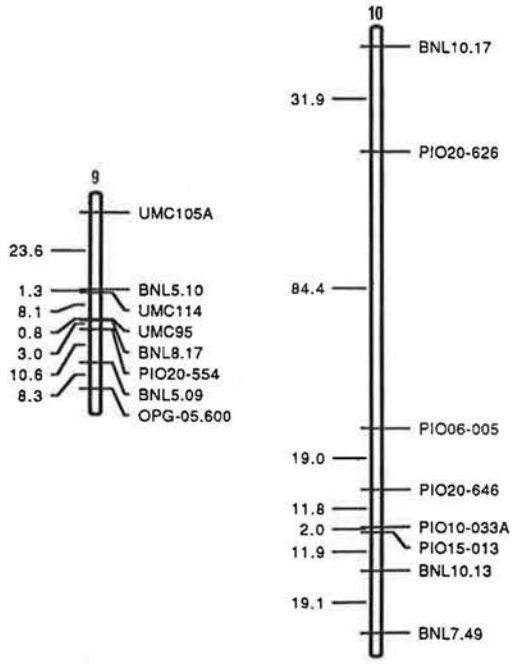


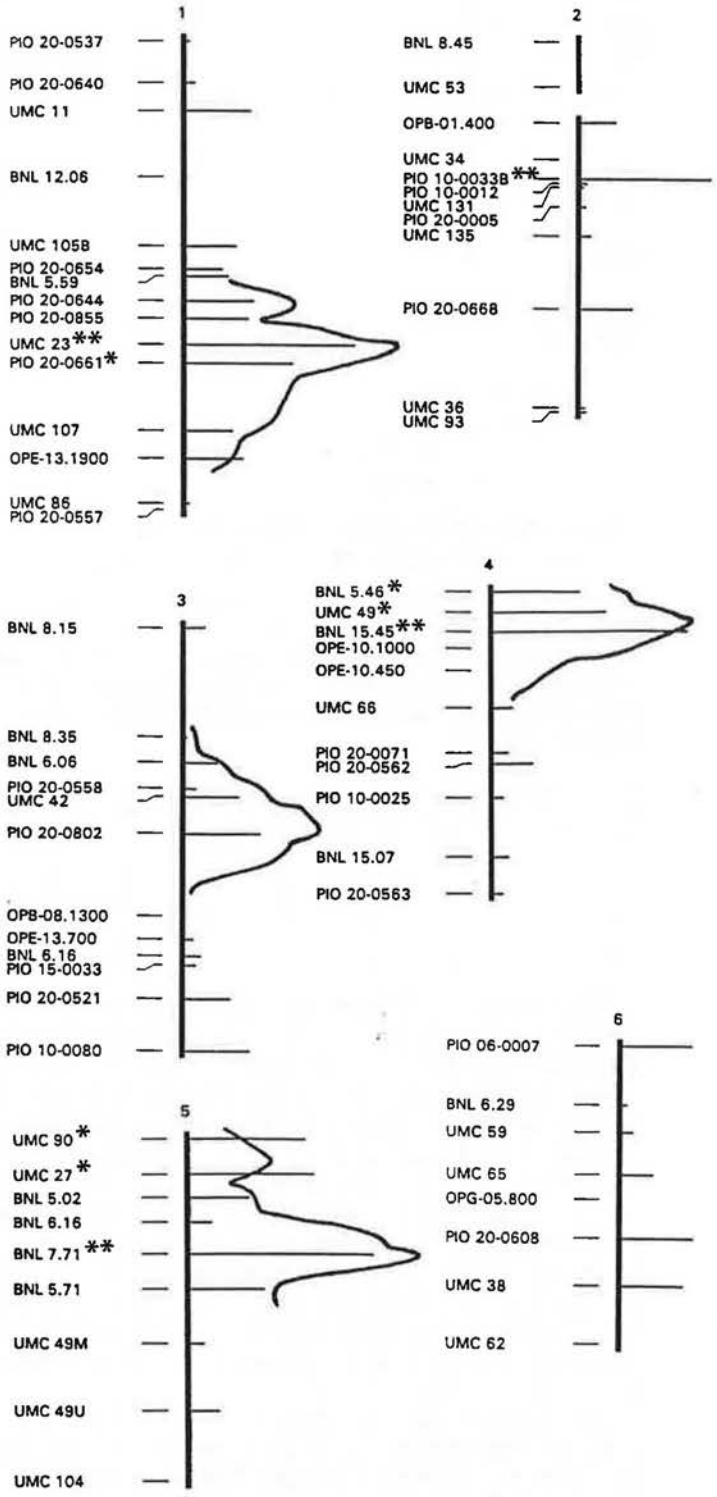
Figure 1. RFLP and RAPD linkage map of maize, based on recombination data from an F2 generation of 150 plants. Genetic distances are in cM (Kosambi's function).

the F2 characterization, trying to avoid overlapping or too large intervals between the clones. 10 RAPD markers (commercially available 10-mers from Operon Technologies) have also been used. The estimate of recombination frequencies and the determination of linkage groups were performed by MAPMAKER. The order and the distance between markers are in accordance with the published maps, with some exception: *pio200668* (on chromosome 1, in our map on chromosome 2), *umc36* (7,2), *umc93* (8,2), *umc49* (2,4), *pio200608* (4,6), *umc111* (4,7), *bnl6.25* (5,8), *bnl5.62* (1,8). Chromosome 2 is split into two groups, due to the presence of a large region where we could not find polymorphic markers. The total genetic distance spanned by our markers is 1539cM (Kosambi's function), with an average distance between clones of 14.5cM. The original data set of the molecular typing of the F2 is available upon request.

Genomic regions controlling stalk-rot resistance

--Giorgio Binelli, Luca Gianfranceschi, Renato Tarchini, Raffaella Greco, Graziana Taramino and M. Enrico Pè

The F3 families obtained by selfing the F2 plants used for the production of the genetic map described in the companion report have been analysed for resistance to infection of *Fusarium graminearum*, the conidial form of *Gibberella zeae*, one of the agents of stalk-rot. 112 F3 families were grown according to a Randomized Block Design: fifteen plants per plot were injected with the spore suspension one week after anthesis and 40 days later harvested and split longitudinally. The character measured was the infected area/total area ratio in the inoculated internode (INF). Both regression analysis between each RFLP and RAPD locus and the INF character and interval mapping (using MAPMAKER/QTL) were used to localize genomic regions involved in resistance to *Gibberella zeae*. The results are indicated in Figure 1 where each marker is represented by its R^2 value while the likelihood surface obtained by interval mapping is shown limited to the significant regions. The concordance between the two methods is remarkable, with the exception of clone *pio100033b* (chromosome 2) significant by regression but not by interval mapping and of the region between *umc42* and *pio200802* (the opposite). The results obtained show that at least four genomic regions exist in the maize genome involved in the determination of resistance to stalk-rot.



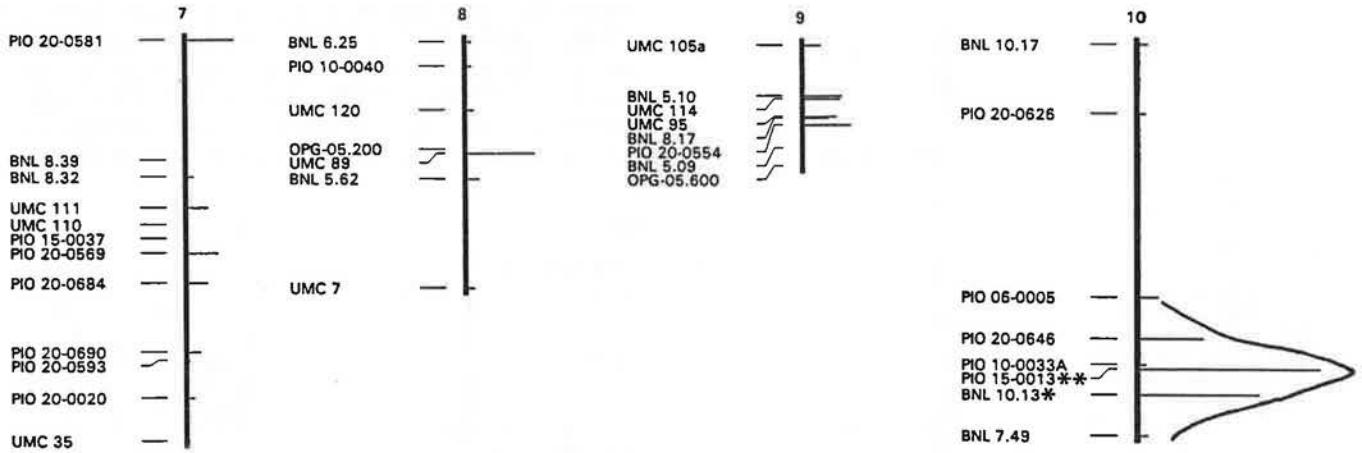


Figure 1. Localisation of putative QTLs controlling resistance to *Gibberella zeae* in maize. Horizontal bars indicate degree of correlation between RFLP loci and the character in terms of R^2. *=p<0.05; **=p<0.01. The continuous line represents the likelihood surface as obtained by interval mapping of QTLs.

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Analysis of new gibberellin sensitive dwarf mutants

--G. Gavazzi, G. Todesco, M. Galbiati and T. Helentjaris

In a previous note (MNL64:86) we presented data regarding the identification of several recessive dwarf mutants and preliminary results on their complementation pattern to the known *d* mutants, affecting a specific block in the gibberellin (GA) pathway. We have extended these results and completed the complementation test (Table 1).

Table 1. Complementation of new dwarf isolates (*d**) with known dwarfs (*d*). Designations + and - stand for complementation or lack of complementation, as established, for each combination, on the basis of several crosses (from 10 to 25).

	d-2*	*d*-3*	*d*-4*	*d*-6*	*d*-7*	*d*-8*	*d*-9*
d1	-	+	+	+	+	+	+
d2	+	+	+	+	+	+	+
d3	+	+	+	+	-	+	+
d5	+	+	+	+	+	+	+

Of the seven new *d** isolates, two appear allelic to known *d* mutants (*d*-2* allelic to *d1* and *d*-7* allelic to *d3)*, while the remaining five identify new genes affecting growth. They all appear responsive to subministration of exogenous GA, applied at a concentration of 10^{-5}M, and they all look alike in terms of plant growth. However, *d*-8* is lethal even after prolonged GA subministration, while *d*-9* is distinguishable from the others for its reduced pigmentation in leaves and its more stunted growth. We don't yet know whether these five mutants represent different lesions on the pathway to GA, but the RFLP linkage analysis together with the results of inter se complementation seem to indicate that these mutants represent five different genes involved in GA metabolism. Information on linkage and inter se complementation so far obtained is given in Table 2.

Two comments seem pertinent here: 1) The RFLP mapping of *d*-3* gave unexpected results. To find segregating polymorphism the mutant was outcrossed to inbred T232 and the F1 selfed. Segregation of the dwarf phenotype in the F2 was surprisingly low, five mutants out of 100 seedlings scored, a segregation

Table 2. Genetic information about the new dwarf mutants.

Dwarf isolates	Genetic information
d-2*	allelic to *d1*
d-3*	RFLP mapping: 1S and 4L close to the centromere
d-4*	complementing to the other *d** isolates
d-6*	RFLP mapping on 5S
d-7*	allelic to *d3*
d-8*	complementing to *d*-2, d*-3, d*-4, d*-9*; lethal at about 15 days after germination
d-9*	complementing to *d*-3, d*-4, d*-6, d*-8*; TB-A mapping on chromosome 6L

expected as a result of duplicate factors. Indeed the results seem to support this hypothesis, since the two regions on 1S and 4L, where positive correlation was found between the mutant and polymorphism, are known to share duplicated genes. 2) Lethality associated with *d*-8* is also unexpected since previous work (Phinney, in The Biosynthesis and Metabolism of Plant Hormones, 1984) has proved that genetic lesions in the GA pathway do not cause lethality. The possibility that *d*-8* is affecting a very early step in the biosynthesis seems unlikely because such a mutation should be associated with pleiotropic effects that are not observed in homozygous *d*-8* seedlings.

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R-plasmid homology regions in mtDNA of the RU maize line possessing low copies of R-plasmids

--E. V. Kuzmin and N. V. Lavrova

RU cytoplasm found in several Latin American indigenous maize lines (D. H. Timothy et al., Maydica 28:139-149, 1983) can be distinguished by the presence of two mitochondrial linear plasmids--R1, 7.4kbp and R2, 5.4kbp--that are thought to be ancestors of S-plasmids. There is no extensive published data so far concerning the organization of integrated copies of R-plasmids present in the mitochondrial chromosome of RU lines. In the course of screening of different local maize lines from Latin America we have found a Bolivian line possessing free R-plasmids present in a comparable copy number to that of ubiquitous N-plasmid. This cytoplasm differs from Enano RU cytoplasm where both R-plasmids

are present in several times higher copy numbers than N-plasmid.

Chromosomal mtDNA of the above-mentioned Bolivian line (Inst. of Plant Industry intermediate cat. No. 3888) was electrophoretically purified from free R-plasmids and blot-hybridizations were performed with the following probes: terminal inverted repeat (IR) of R-plasmids; ORF3 of S1; ORF1 of S2; ORF2 of S1/S2; 5kb-repeat of N-type mtDNA. The results of this analysis showed that this type of RU cytoplasm is very similar to N. Its mitochondrial chromosome contains only one incomplete integrated copy of each R-plasmid adjacent to the 5kb-repeat. The organization of the R2-homology region is indistinguishable from N-type mtDNA, but R1 is integrated in the opposite direction relative to the 5kb-repeat. Both R1 and R2 integrated copies are comparable in size with free R1 and R2 but possess only one IR. No complete integrated R-plasmids with one free linear end similar to S-plasmid integrates in S cytoplasm were found. It could be due to low R-plasmid copy number and resultant low effectivity of homologous recombination between free plasmid IR and IR-part of 5kb-repeat.

If only integrated copies of R-plasmids can be transcribed then line 3888 would possess ORF1 and ORF3 but no ORF2 transcripts in its mitochondria, which is opposite to the situation in other RU lines where ORF2 transcript can be detected readily (P. L.Traynor and C. S. Levings III, Plant Mol. Biol. 7:255-263, 1986). This fact could be one of the causes of low R-plasmid copy number. Another cause--the influence of particular nuclear background--cannot be ruled out. The experimental proof of this prediction is now going on.

The authors are grateful to the Maize Division of the Institute of Plant Industry in St. Petersburg for the generous gift of seeds.

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Peroxidase isozymes revisited

-- Emil E. Khavkin and M. V. Zabrodina

Numerous temporal and spatial changes in peroxidase expression have been documented previously by Brewbaker et al. (J. Hered. 76:159, 1985) who employed flat PAGE with lithium-borate/citrate buffer systems. We found that vertical PAGE, with alkaline Davis, acidic Reisfeld and especially with neutral Taber and Sherman buffer systems, provided better resolution of peroxidase electromorphs. Besides, several previously unsurveyed elite inbreds and additional tissues (especially those of seedlings) were included in our study. Peroxidase loci are designated below according to the nomenclature by Brewbaker et al.

Anodal peroxidases. The slowest electromorphs were produced by the *px7* and *px8* loci, and we could not always discriminate these isozymes properly. Among investigated inbreds, intermediate *px7* electromorphs prevailed, though more than one band could be found in several genotypes. *px8* was expressed in most tissues investigated, with the exception of endosperm and anther, and its activity in pollen sometimes was rather low. The highest *px8* expression was found in young leaves. A two-banded pattern of this zone was observed in most genotypes. Inbreds C103 and Va35 exhibited faster electromorphs than most other genotypes, and both fast and slow allelomorphs were present in ICA L223 and WF9.

px3 was the most universally expressed locus, yet in anther its activity was sometimes too low to be detected. There were two or three allelic variants of this isozyme: fast in A239, B14, B37, B73, C103, Oh43, Va35 and W64A, apparently intermediate in Tx601 and slow in A188 and ICA L223, and this evidence did not always comply with the scores by Brewbaker et al. A double-band pattern was found in most inbreds. We also found several distinct minor bands of peroxidase activity between *px8* and *px3* zones with wider tissue specificity than the root-specific product of *px11* described earlier in this region of electropherograms.

The weakly stained *px6* zone was easily discerned in embryo axis, leaf and root, and traced in scutellum. More mobile zone in this region could be *px10* product, though tissue specificity of this zone and *px10* did not completely coincide.

A double-banded *px2* pattern was found exclusively in already mature pollen; we were not able to trace this isozyme even in the pre-shedding anthers. Two allelic forms were found among screened inbreds: the fast one in A188, A344, A619, A632, A654, A682, B89, Sd41 and 517/3, and the slow one in A677, A681, B91, Pa91 and W23, while both allelomorphs were present in several A632 and A654 plants.

Mobile bands expressed by *px9* were present in all embryo tissues and were especially active in the root as compared to the leaf. *px12* product was found only in the root. *px12* exhibited allelic polymorphism (apparently a faster band in Oh43 and Va35, and a slower band in A188, B37, C103 and ICA L223).

Cathodal peroxidases. The two most spectacular features of the cathodic pattern were the complete absence of peroxidase staining in mature pollen and a characteristic band in the endosperm and scutellum that apparently was not described previously. It was more mobile than the *px1* product. The expression of this fast electromorph in the scutellum rapidly increased from the 1st to the 6th day of germination and slowly declined later, while in endosperm the decrease was already evident by the 3rd day of germination.

With the exception of pollen, *px1* was the second most common peroxidase locus producing heavily stained double bands in seedling tissues. In addition to data by Brewbaker et al., active *px1* bands were found in the endosperm and scutellum, and weaker but quite distinct expression was characteristic of the anther. In the scutellum this band manifested rapid growth of activity in the course of germination, while in the endosperm and leaf it degraded as seedling growth progressed. A fast *px1* allele prevailed (A188, A239, B14, B73, ICA L223, Oh43, Tx601 and apparently WF9) over intermediate (Va35) and slow (C103, W64A and apparently B73) alleles. Upon tissue vs. genetic variability in the *px1* pattern, we suggest that this zone comprises the products of at least two loci producing different combinations of fast and slow electromorphs.

The next zone towards the cathode, with two bands well-resolved in acidic gel, was apparently produced by *px5*. This isozyme was monomorphic in our sample of inbreds, very active in the axial tissues of the seedlings, including leaf, as well as in different tassel tissues, but it stained weakly in endosperm. Contrary to published evidence, we did not observe *px5* expression in mature pollen.

The *px4* pattern of most tissues consisted of two bands, sometimes poorly resolved in neutral gel. This locus was extremely active in shoot and root tissues of the seedling, less prominent in

scutellum and tassel tissues, and practically silent in endosperm. We could not confirm the data on allelic polymorphism reported earlier.

In acidic gel, axial tissues of the seedlings displayed one more cathodal peroxidase band of little or zero mobility, and we are inclined to relate this band to *px7*. The band was absent from endosperm and scutellum as well as from tassel tissues. In alkaline and neutral buffer systems this band moved slowly towards the anode (see above).

Brewbaker et al. had already described tandem (dimorphic) bands in *px3*, *px6* and *px8* patterns. Two or more bands of monomeric peroxidases in most other loci investigated (*px1*, *px4*, *px5* and especially evident in *px2* and *px9*) suggest that this phenomenon can be more common, presumably resulting from gene duplication. Another frequent phenomenon, two allomorphs present in the same inbred individual, presents a more difficult problem.

Heritable changes of anodal esterase and peroxidase patterns in A188 somaclones

--Emil E. Khavkin, M. V. Zabrodina, Yu. I. Dolgykh* and Z. B. Shamina*

Earlier (MNL65:88) we reported numerous quantitative and qualitative deviations from the standard pattern in anodal esterases and peroxidases in plants regenerated from tissue cultures of A188 immature embryos. Some loci were affected by somaclonal variation more often and to a greater extent than the others, and the tissue-specific loci seemed to exhibit this variation more readily than the constitutive loci.

Two further generations of these somaclones were obtained by selfing, and their isozyme patterns were surveyed. Several subfamilies (especially 11-4, 27-5 and 54b-7) were found to maintain and gradually intensify some of the previously described somaclonal variations. The most spectacular among them were: (1) the changes in the zone between *e4* and *e9* esterases: appearance and gradual activation in the leaves of several bands including characteristic "scutellum-specific bands", and the loss from the roots of several bands localized in the same zone of the esterase spectrum; and (2) the emergence of the most mobile anodal peroxidase band (apparently the root-specific product of *px12*) in the leaves. Several loci manifested considerable heritable changes in the level of their activity as compared to the standard (the initial inbred plants). The most prominent example was *px3* activation in the roots of somaclones progressing in their progeny.

In agreement with earlier data, no significant changes were found in cathodal peroxidase spectra.

Development of NaCl-resistant callus culture and regenerants

--S. N. Larina*, Yu. I. Dolgykh* and Z. B. Shamina*

A rapid and specific procedure was developed to screen salt-resistant cell cultures and to obtain plants with salt-tolerant phenotype.

Cell cultures were initiated from immature embryos of A188 inbred 10-11 days after pollination, cultivated in 1% NaCl-supplemented MS growth medium, and subcultured every 30 days.

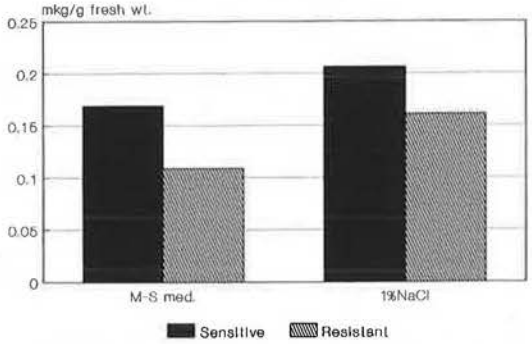
NaCl considerably decreased callus viability (Table), and by the 6th subculture salt-sensitive explants were virtually eliminated. Viable cells were salt-resistant.

Subcultures	1	2	3	4	5	6
Viability, % growing calli	13.0	2.95	0.97	0.28	0.04	0.004

NaCl-resistant embryogenic callus from every subculture was used for regeneration. Eight regenerants were produced after one, 23 after two, and 4 after three months of cultivation. Regenerants were weaker and did not differ substantially from the initial plants in their morphology. About half of the regenerants were branched and had 2-4 shoots.

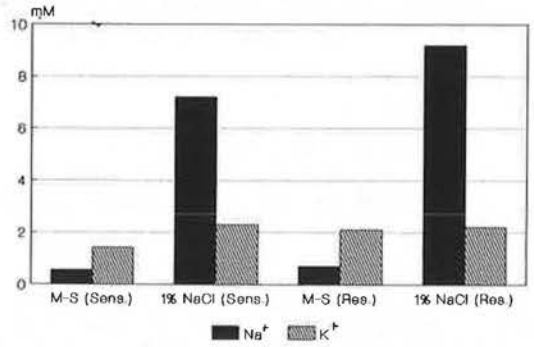
After three subcultures, NaCl-resistant calli were analyzed for their content of osmoticals. Resistant and sensitive cell lines were cultivated in the standard and 1% NaCl-supplemented MS medium for 7 days and extracted for determination of sugar and monovalent ion concentrations. Sensitive cells contained more soluble sugars (Fig. 1), both in the standard and the selective media,

Fig.1.Soluble sugars content



however in the resistant cells salt stress stimulated sugar accumulation by 25%. Na content differed inconsiderably in two cell lines when grown in the absence of NaCl (Fig. 2), the salt stress

Fig.2.$Na^+$and K^+content



increased Na content in both cell lines, however, the resistant line accumulated 1.3 times more Na than the sensitive line. Two lines did not differ substantially in their K content under salt stress.

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Meiotic abnormalities in NEH maize

--M. Kumar and J. K. S. Sachan

A total of 40 landraces of the NEH region of India have been screened by pachytene analysis. Nagaland collections N-24, N-29, N-37 and N-44 exhibited various meiotic abnormalities. In some of these strains univalents as well as bivalents were present at

metaphase I. Abnormal segregation was observed at anaphase I in these as well as a few other NEH strains. Besides these, laggards, precocious segregation, stickiness, micronuclei and pentads instead of normal tetrads were also observed.

As per Prakken's (1943) classification, the Nagaland collections showing desynapsis are medium strong mutants, showing a high number of univalents and a high frequency of segregation abnormalities. The desynapsis has been shown to be under genetic control (Koduru and Rao, 1981), as well as under the control of temperature and physiological factors or nutrient levels. In this context, it is worth mentioning that these NEH maize strains were grown in New Delhi, far away from their native habitat. Markedly different climate and very high temperature in the growing season could have induced these synaptic variants in these strains that are possibly carrying the genes for synaptic mutants. Such synaptic variants have been reported earlier in Sikkim Primitive strains N-3 and N-4, which are also from Nagaland (Dash et al., 1986), as well as some maize varieties being grown in Indian plains (Sarma, 1983).

Occasionally laggards were also observed during anaphase I. The micronuclei observed during late telophase I must be due to these laggards. Micronuclei leading to polyad formation have also been observed in triticale (Gobran, 1980).

The pollen grains carrying meiotic abnormalities appeared to be non-functional as was evident from the constant 2n=20 chromosome count in all these strains. Absence of seed sterility observed in these strains can be explained due to normal megasporogenesis and subsequent fertilization by normal pollen grains.

Stickiness of chromosomes at anaphase I has been consistently observed in the present study. Such adhesion between two or more chromosomes occurs due to a recessive monogenic mutation (Beadle, 1937) in maize.

Abnormal meiotic patterns, the presence of additional RNA and in some cases a second nucleolus were earlier reported in a Sikkim Primitive strain (Peeters, 1982) growing in a place away from its natural habitat.

A cross-shaped structure, formed by the fusion of centromeres at pachytene, was consistently observed in maize strains of Sikkim and Meghalaya. However, subsequent meiotic events at metaphase I were found to be normal, hence the possibility of translocation heterozygotes was ruled out.

Such centromeric fusion has been reported earlier in NEH maize. Randomness of this centromeric fusion was evident from the involvement of any of the bivalents in it. The association between centromeres of non-homologous chromosomes was found to be less frequent in the present study.

Cytoplasmic effect on chromosome pairing in maize-teosinte hybrids

--M. S. Ramesha and J. K. S. Sachan

Observations on chiasma frequency and chromosome configurations of the maize-teosinte hybrids involving *Zea diploperennis*, *Z. luxurians*, *Z. mays* ssp. *parviglumis* and some primitive and advanced races of maize, under different cytoplasmic backgrounds, were made to assess the degree of cytoplasmic divergence and nucleo-cytoplasmic interactions on chromosome pairing.

All the maize-*Z. diploperennis* hybrids showed highly significant reciprocal effects. There was a great reduction in chiasma frequency along with a significant increase in the frequency of rod bivalents and univalents. However, the frequency of ring bivalents reduced significantly in the cytoplasmic background of *Z. diploperennis*. Besides this, there was also an increase in the frequency of cells with chromosomal abnormalities. Maize-*Z. luxurians* hybrids showed reciprocal effects as reflected by reduction in chiasma frequency and increase in the frequency of univalents, however, their magnitude was relatively low. Maize-*Z. mays* ssp. *parviglumis* hybrids did not show any reciprocal effects.

The results indicated that the cytoplasmic divergence between maize and *Z. diploperennis* was maximum, followed by *Z. luxurians* and *Z. mays* ssp. *parviglumis*. The probable factors responsible for cytoplasmic effects could be the time imbalance in the condensation of the maternal and paternal chromosomes of a pair, maybe as a result of some nucleotypic changes brought about by primitive and divergent cytoplasm of teosinte. Further studies on the precise mechanisms of such changes could be helpful in resolving the mystery of origin and evolution of both maize and teosinte.

In vivo propagation in maize-teosinte hybrids

--V. V. Pandit and J. K. S. Sachan

The phenomenon of in vivo propagation of plantlets from male spikelets in maize-teosinte hybrids was reported earlier by H. K. Singh (MNL56:152, 1983). In our studies, in vivo development of plantlets from tassel spikelets occurred with variable expressivity and penetrance depending upon the parents involved in the crosses. The outer glumes of the spikelets remain normal and the inner glume forms the first leaf. The anther development is suppressed in crosses involving maize and *Zea mays* ssp. *parviglumis*, whereas development of anther is normal in maize-*Z. diploperennis* and maize-*Z. luxurians* crosses. The plantlets in situ grow into mature plants with height ranging from 40-120cm. The plantlets were generally tripsacoid with female spikelets resembling teosinte. When these plantlets are transplanted at an early stage directly into the field, they grow into normal plants. These transplants (R1) also showed the ability of in vivo regeneration. The R2 progenies also exhibited this phenomenon. The frequency of embryoid formation and subsequent regeneration of plantlets in vivo in the R2 generation was comparable to that of R1 and F1 generations. Analysis of F2 progenies suggests that it is a polygenic trait. Both the cytological and morphological features indicated the sporophytic origin of plantlets.

In vitro culture studies of glumes showed that somatic cells are embryonically determined very early in tassel development. Glumes cultured five days after tassel initiation developed into a whole plant in the medium devoid of hormones. Glumes of F1s cultured in the callusing medium (MS + 2mg/l 2,4-D), showed a high frequency of embryogenic callus which was slightly yellowish, tough with tiny nodules on the surface and contained small oval cells with prominent nuclei and dense cytoplasm (Ann. Bot. 66:497-500, 1990) as compared to the parents. The cross between CM111 and *Z. diploperennis* produced a very high frequency of embryogenic callus (88.2%) and showed regeneration on hormone free MS medium. This demonstrated that there is endogenous alteration in the auxin-cytokinin balance which is a critical factor for embryo initiation. Occurrence of this phenomenon of in vivo regeneration in maize x teosinte opens up considerable scope for genetic manipulation and helps in studying the regulation of plant development.

***R-marbled* as a transposable element system**

--B. M. Prasanna and K. R. Sarkar

R-marbled (*R-mb*), a 'pattern' allele at the *R* locus, gives a

characteristic aleurone variegation with irregularly shaped, coarse colored sectors on a colorless background. This allele has not been studied extensively so far. We have made an attempt to understand the genetic mechanisms behind the pattern formation and variability in *R-mb* and how this allele differs from its sister alleles, *R-nj* and *R-st*.

Wide variation was observed in the extent of anthocyanin pigmentation of *R-mb* kernels in the same stock background. Elaborate progeny testing of different marbled scores (score 1, only one colored spot, to score 6, almost fully colored) revealed that variations in *R-mb* expression cannot be solely attributed to developmental effects. On the basis of distinct segregation profiles, *R-mb* could be categorized into four classes: very light spotting, light spotting, medium spotting and heavy spotting. Intensity of pigmentation also varied in some marbled kernels (dark, pale etc.) and these differences persisted upon selfing.

At least two doses of *R-mb* are necessary for the characteristic spotted phenotype. The marbled pattern expressed in only 1.5 percent of the *R-mb r r* kernels, with a reduced intensity of pigmentation. Analysis of mean visual scores of *R-mb R-mb R-mb*, *R-mb R-mb r* and *R-mb r r* endosperm genotypes by Student's t-test showed significant differences in aleurone pigmenting potential. Such a drastic dosage effect appears to be a characteristic feature of only *R-mb* among the pattern alleles, since *R-nj* (Kumar and Sarkar, Indian J. Exp. Biol. 24:270-273, 1986) and *R-st* (Ashman, Genetics 45:19-34, 1960) were found to express even in a single dose. It is to be confirmed whether this effect is dependent only on the transmission of *R-mb* by the pollen parent.

We also found that *R-mb* reverts at a high frequency to the fully colored form in both somatic and germinal tissue. Different classes of mutants with discordant endosperm-embryo phenotypes were obtained. Only germinal reversions (*R-mb-->R-sc*) were found to be stably transmissible. Reversion of *R-mb* to *R-sc* appears to occur at a slightly higher frequency than that of *R-st* to *R-sc* (18.54×10^{-4} and 17.0×10^{-4}, respectively).

There were no dominance-recessive relationships observed among the pattern alleles. *R-mb*, in crosses with *R-nj* or *R-st*, gave rise to progeny with expression of both the patterns on the same kernel (nj+mb or mb+st). In the reciprocal crosses, the marbled phenotype expressed only in 1.5 to 1.7 percent of the cases, confirming the earlier observation on the single dose effect of *R-mb* on aleurone pigmentation. An interesting situation arose in the crosses of *R-mb* and *R-nj*; in F2, there is a preponderance of the Navajo phenotype. The possibility of contamination is excluded since kernels borne on heterozygous *R-mb R-nj* ears showing both Navajo and marbled kernels were planted and selfed. The influence of a maternally accumulated signal that can 'preset' the expression of *R-nj* might account for the preponderance of the Navajo phenotype. We observed that the interaction of *R-mb* with *R-st* was not identical to that of *R-mb* with *R-nj*. Occurrence of *R-mb/R-st* kernels with discordant endosperm-embryo phenotypes strongly indicated somatic and germinal instability at *R-mb* and *R-st*.

For an explanation of the phenomena concerning spotting in *R-mb*, it would be necessary to postulate the influence of a tranposable genetic element, on the basis of the following observations: 1) mutations within *R-mb* might result in the appearance of coarse, colored spots in aleurone as well as changes in color intensity and these changes can occur independently; 2) both somatic and/or germinal reversions to wildtype can occur at a very high rate, and 3) only germinal reversions from *R-mb* to *R-sc* were found to be transmissible. The phenomenon of excision of the controlling element at *R-mb* might explain the size, shape and location of the colored spots in aleurone. Chang and Neuffer (J. Hered. 78:163-170, 1987) reported earlier that *R-mb* behaves like *R-st* except that the timing is much earlier. Observations in our study showed that although R-mb shares some similarities with *R-st*, the distinct genetic behavior of this allele might be attributed to the influence of a specific controlling element. Our original *R-mb* genetic stock did not carry *Ac*, *Spm* or *Mu* transposable element families. Further work is being carried out to ascertain the nature of the controlling element influencing the marbled pattern.

The significance of the silk attachment region in the expression of certain *R* alleles

--B. M. Prasanna and K. R. Sarkar

The pigmentation patterns of *R-st*, *R-mb* and *R-nj* are attractive from a developmental viewpoint, as the anthocyanins are distributed specifically in the outermost differentiated aleurone layer of the endosperm. We analyzed the onset and progression of anthocyanin pigmentation in *R-st*, *R-mb*, *R-nj:Illinois*, *standard R-r*, *R-sc* (*ex. R-mb*) and *R-mb/R-nj* in the summer seasons at New Delhi from 1989 to 1992. All these alleles were in the same stock background.

The pattern alleles exhibited diversity in the onset and mode of pigment progression. Pigments could be visually detected on the 11th day after pollination (DAP) in the aleurones of *R-st*, *R-mb*, *standard R-r* and *R-sc* (*ex. R-mb*), while *R-nj* recorded a conspicuous delay (18th DAP). *R-st* and *R-mb* pattern formation reflected the systematic (clonal) development of the aleurone. In *R-st*, the colored spots mostly showed groups of pigmented cells in squares (4 or 16 cells) or rectangles (2, 8 or 32 cells), confirming the observations by Coe (in Maize Breeding and Genetics, ed. D. B. Walden, pp. 447-459, 1978) that divisions during clonal development of aleurone occur in alternating perpendicular planes. The colored spots in *R-mb* showed a large number of uniformly dark cells with clearly defined borders. The *R-nj* allele revealed a very unique mode of pigmentation. We observed that in *R-nj:Illinois*, pigmentation started from the tip of the immature kernel, but only from a specific site, the silk attachment region. Anthocyanin pigments diffused gradually from this site in a typical sun ray-like manner towards the periphery of the crown. Similarly, in both *standard R-r* and *R-sc*, pigmentation occurred first in the cells surrounding the silk attachment region, but progressed in a wave-like manner.

The differences in the pattern formation of *R-mb* and *R-nj*, representing clonal and aclonal systems, could be clearly visualized in the *R-mb/R-nj* heterozygotes. When *R-mb* was used as a female parent in crosses with *R-nj*, kernels with full action of both the alleles, that is, a typical Navajo pattern along with marbled sectors in the aleurone, could be obtained. In kernels where both *R-mb* and *R-nj* expressed, the marbled spots appeared first on the 11th DAP and intensified by the 18th DAP, while the Navajo coloration just started from the silk attachment region on the 18th DAP.

Our study indicated that the Navajo pattern might not be solely due to the delayed onset, as was evident by the clearcut differences in the manner of pigment expression in *R-nj* and other *R* alleles. The important questions are - How are only the cells around the silk attachment region endowed with pigment-produc-

ing potential in the Navajo aleurone, although all the aleurone cells have the same genetic constitution? Why does diffusion-based pigmentation occur only in *R-nj* and not in *standard R-r*? It would be tempting to predict that for the pigmentation to start only from the silk attachment region, a signalling source might be responsible. However, when the *R* gene is under the control of a transposable element (as in *R-st* or *R-mb*), the response might be lacking, leading to the appearance of pigmented clones anywhere on the kernel. At present, we know little about how the activity of such signalling sources is controlled and the molecular nature of the 'regulators' in *R-nj*, *R-st* and *R-mb*. Wilson (in Maize Breeding and Genetics, ed. D. B. Walden, pp. 405-419, 1978) noted that important metabolic events like starch synthesis, protein body formation and transport start from the silk attachment region of the kernel. It is apparent that this site has some significance in the anthocyanin pigmentation of certain *R* alleles, as in other important cellular processes in the developing endosperm.

_R-mb:cc_–a derivative from _R-mb_ with a developmentally programmed anthocyanin pattern

--B. M. Prasanna and K. R. Sarkar

The *R-mb* kernels usually display a spotted phenotype with three important features: (i) no restriction on the number of colored sectors, and different spotting types (very light, light, medium and heavy) with varying extents of pigmentation can be obtained; (ii) pigmented spots (results of random excision events) can occur anywhere on the aleurone, with no specificity in arrangement, and (iii) colored spots have well-defined boundaries, but are irregular in their shape.

We came across some exceptional kernels with altered spatial pigmentation, on *R-mb* selfed or testcross cobs, with the following features: (i) the colored spots on a colorless aleurone background were arranged in an orderly and precise manner, originating from the germinal side and extending to the crown and the abgerminal side of the kernel; (ii) colored sectors were in the form of concentric rings or stripes and seldom irregular in shape; (iii) spots appeared in a symmetrical manner from either side of the scutellum or restricted to one-half of the kernel, and (iv) the flow region on the abgerminal side of the kernel might show irregularly shaped spots (as in *R-mb*), with the rest of the kernel showing the characteristic spotting arrangement. For convenience in presentation, these kernels will be hereafter referred to as *R-mb:cc* (marbled in concentric circles).

The genetic behavior of *R-mb:cc* was largely identical to that of *R-mb*, evidenced by a drastic dosage effect on aleurone pigmenting potential when transmitted in a single dose through the pollen parent. Homozygous *R-mb:cc* kernels with three doses of *R-mb:cc* gave rise to kernels with concentric colored spots in high proportion (92.5%), followed by the colorless kernel class. When transmitted through the pollen parent, a very low percent (1.3%) of *R-mb:cc* phenotypic class was recorded.

Four generations of selfing the *R-mb:cc* kernels with plantings at different locations (New Delhi and Hyderabad) showed consistency in the spatial pattern. The *R-mb:cc* ears frequently showed completely colorless kernels as well as fully colored revertants. Discordant endosperm-embryo phenotypes, like mb:cc endosperm with colored scutellum and colored endosperm with colorless scutellum, were also noticed in frequencies higher than the spontaneous mutation rate. Preliminary observations indicated that further categorization is possible within *R-mb:cc*. Ears with homogeneous expression of light, medium or heavily striped kernels could be obtained. We have studied the pigmentation onset and progression in *R-mb:cc*. As in *R-mb* and *R-st*, visual manifestation of anthocyanin pigmentation first occurred on the 11th day after pollination (DAP). Later on, pigmentation became more intense with little change in the basic pattern. Therefore, in *R-mb:cc*, the spatial pigmentation pattern appears to be determined at an early stage in the aleurone formation.

R-mb:cc faithfully transmitted its characteristic phenotype with concentric arrangement of spots, when crossed with *R-nj* or *R-st*. As in the case of *R-mb*, there were no dominance-recessive relationships among the three alleles and there was a preponderance of Navajo phenotype in the *R-mb:cc/R-nj* and *R-nj/R-mb:cc* genotypes. Reciprocal cross differences also confirmed the dosage effect of *R-mb:cc* on aleurone pigmentation.

The study showed that the *R-mb:cc* was germinally transmissible and had similarities in genetic behavior with *R-mb* from which it was derived. Mutations from recessive to dominant (self-colored revertants) occurred at high frequency, variation within the basic pattern (both in the degree of spotting and the intensity of pigmented spots) and discordant endosperm-embryo phenotypes warrant the basic assumption that the *R-mb:cc* phenotype is under the control of a transposable genetic element. Although we do not yet have any clearcut evidence as to how *R-mb:cc* originated and how the striking regularity in the arrangement of colored spots occurs, it might be instructive to consider the plausible mechanisms, using the information from the well-studied transposable element systems in maize.

In maize, there were no earlier reports of pigmentation pattern in a concentric manner from the germinal side of the kernel, either in the unstable alleles at *R* or other anthocyanin biosynthetic loci. Analogous, however, is the case of two *En* alleles, *En-crown* and *En-flow* (Peterson, MNL40:64, 1966) which respond quite specifically to different parts of the aleurone tissue. Similarly, Doerschug (Theor. Appl. Genet. 43:182-189, 1973) found a *Dt* element that caused restriction of element activity in the kernel. A *Uq-flow* phenotype was recovered by Peterson (in Gene Structure and Function in Higher Plants, eds. G. M. Reddy, E. H. Coe, 1983), where the transaction of the element was restricted to the basal portion of the developing endosperm. These examples typify only restriction of the element activity to specific regions, but no restriction is placed on the arrangement or shape of a colored spot within a region. In contrast, studies carried out on mutable pericarp, characterized by the presence of red stripes on a white pericarp background, revealed that the phenotype is due to transposition of *Mp* (=*Ac*) at the *P* locus (Fedoroff, in Developmental Genetics of Higher Organisms, ed. G. M. Malacinski, pp. 97-125). The stripes are wider at the base of the kernel and come to a narrow point at the silk scar region of the crown. Fedoroff (1988) described that the differences in the shape of the characteristic stripes in the pericarp (a maternal tissue with a growth pattern different from that of the endosperm) are determined by the pattern of cell divisions within the tissue. It should be noted here that the stripes in the *Ac*-influenced variegation pattern 'radiate' from the silk scar region of the kernel, unlike the concentric spots in *R-mb:cc*. It would be interesting to consider, in this context, certain *pallida* alleles in snapdragon. The *pal-33*, *pal-32*, *pal-15* and *pal-41* cause altered patterns of spatial pigmentation in the flower, when compared to other *pal* alleles. To explain this, Coen et al. (in Temporal and Spatial Regulation of Plant Genes,

eds. D. P. S. Verma, R. B. Goldberg, pp. 632-82, 1988) proposed that the wildtype *pal* promoter contains a set of sequences that respond to diverse regulatory signals spatially arranged as a pre-pattern in the flower to generate specific patterns. They hypothesized that novel patterns are produced by mutations that change the interpretations of pre-pattern by modifying the affinity of the *pal* promoter for different regulatory molecules. If the colored spots in *R-mb:cc* are results of excision events during kernel ontogeny, the pattern of excision of the controlling element in only certain cell lineages originating from the germinal side might be under the influence of a developmental signal or host factor. Levy and Walbot (Science 248:1534-37, 1990) demonstrated that the timing of transposable element excision can be controlled by the host. We cannot, at present, rule out other plausible mechanisms by which *R-mb:cc* pattern is regulated, like DNA methylation.

Rooting and establishment of plantlets from longitudinal half plumule of mature embryo in vitro

--Pranab Gayen, Jasbir Kaur Madan and K. R. Sarkar

Recovery of mature, fertile plants from the in vitro culture of maize shoot apical meristem is known (Bommineni et al., Plant Cell Tissue Organ Cult. 19:225-234, 1989). There is also a report about recovery of two mature, fertile plants from the single embryonic axis of immature embryo through microsurgery in vitro (Bommineni, MNL66:98, 1992). We have now obtained success in recovering plantlets from longitudinally bisected embryo and plumule parts of the mature embryo of maize.

Mature seeds of the inbred MG209 were soaked for 60 h and the embryos scooped out from the seed. The embryos were then sterilized for 4-5 min in 0.1% mercuric chloride and washed three times with sterile water. A single medial longitudinal incision was made through the embryonic axis with the help of a sterile razor blade under sterile conditions. The two halves of the embryo or of the plumule only (excluding the radicle part from the middle of the hypocotyl) were separated and placed along the slant of the medium, keeping approximately half of the cut surface inserted into the medium and with the plumule side facing the top of the test tube (placement-A). The medium was comprised of the usual MS salts plus casein hydrolysate (1g/l), inositol (100mg/l), sucrose (3%) and agar (0.7%) with IAA (2mg/l) or without rooting hormone.

In another set (placement-B) two types of explants were placed vertically on the medial surface with the same composition of media. Data on root initiation and plantlet establishment after 10 days of inoculation under white fluorescent light are presented in Table 1. Significant difference in rooting was observed between media with or without rooting hormone. Placement-A was better than placement-B over the two types of media. Half plumules produced normal plantlets; however, half embryos registered a greater degree of success. In placement-B, roots formed were weaker and the number of roots less than in placement-A.

Table 1. Rooting and plantlet establishment from embryonic axis and plumule half of maize.

		Placement-A				Placement-B			
		a	b	c	d	a	b	c	d
Media without rooting hormone	Half-embryo	62	34	39	32	48	12	22	20
	Half-plumule	58	21	35	30	43	9	16	13
Media with rooting hormone	Half-embryo	59	47	53	49	50	19	27	24
	Half-plumule	64	46	50	45	60	21	26	19

a=No. of half embryos/plumules cultured
b=No. of embryos/plumules rooted after 4 days
c=No. of embryos/plumules rooted after 10 days
d=No. of transferrable plantlets with proper root and shoot growth

Placement-A - Explants placed along the surface of the medial slant
Placement B - Explants vertically inserted into the medium

Orientation of mature embryos on culture medium influences callus induction

--Jasbir Kaur Madan, Ashutosh Pandey, Pranab Gayen and K. R. Sarkar

Immature embryo has been used extensively to obtain embryogenic callus and regeneration in maize tissue culture. Utilization of mature embryos for callus induction has also been reported (Wang, Plant Cell Rep. 6:360-362, 1987). For callus induction from immature embryo, the placement fashion of the embryo on the medial slant plays an important role. The plumule-radicle side of the immature embryo, when placed downward, gave a greater frequency of induction and amount of callus induction than the scutellar side placed downward (Green and Phillips, Crop Sci. 15:417-421, 1975).

When mature embryo is placed facing the medium, the plumule starts to germinate quickly, pushing the explant up and disconnecting it from the medial surface, thereby restricting the callus induction. Hence, placement of embryo on the medium plays a critical role in successful cultures. An efficient mode of placement of the embryo to enhance callus induction frequency was identified in our study.

The callus induction medium was MS medium supplemented with 2,4-D 2mg/l, casein hydrolysate 1g/l, inositol 100mg/l, sucrose 3% and solidified with 0.7% agar. The pH was adjusted to 5.8 before autoclaving.

Sixty mature seeds per replication of seven inbreds with two replications were used for this experiment. Five different embryo placements (Table 1) were taken into consideration.

The culture was kept under dark conditions at 26±1 C for 14 days and callus induction frequency was scored visually. The results indicate that the embryo when placed laterally, half inserted into the media (T5), showed better callus induction frequency and callus growth as compared to other treatments for all the inbreds studied. When the data on callus induction frequency over the inbreds for different treatments were subjected to DMRT ranking, it was observed that lateral placement was distinctly superior to the other four orientations.

Table 1. Callus induction frequency of mature embryos from different inbreds with different embryo placements.

	Callus induction frequency (%)*				
Inbreds	T1	T2	T3	T4	T5
3783	66.10	66.65	52.78	58.81	87.91
3786	68.36	70.84	59.09	67.39	86.41
4526	38.39	46.64	47.34	29.16	60.41
4603	21.42	15.00	24.94	11.17	47.51
4532	31.73	46.73	33.32	19.20	59.09
4493	16.44	33.51	16.32	32.77	53.56
4604	12.49	23.04	30.00	11.57	38.52
Mean	33.70b	43.20b	37.68b	32.86b	61.91a

*Each entry is the mean of two replications.
T1=Embryo half inserted vertically in the medium; plumule downward
T2=Embryo half inserted vertically in the medium; radicle downward
T3=Embryo flat on the medium; plumule-radicle axis touching the medium
T4=Embryo flat on the medium; plumule-radicle side upward
T5=Embryo placed laterally, half inserted in the medium; plumule-radicle axis touching the medium
a,b=DMRT ranks

NORTHFIELD, MINNESOTA
Biotechnology Innovation

Bigger backcross bang for the buck

--Ed Weck

In a backcross population, as in any breeding population, individuals have a fixed genomic composition. In order to speed backcross conversions using molecular markers, we must measure the number of recurrent parent alleles of each individual. One constraint of molecular marker analysis, however, is the high cost.

There are two ways to reduce total analysis costs; either reduce the cost per analysis or reduce the total number of analyses. As technical breakthroughs occur, the cost per analysis will decrease. Reduction of total analysis costs should maintain the same selection intensity while analyzing only a subgroup of the population. I present a stepwise procedure, like a taxonomic key for species identification or the qualitative analysis of inorganic chemicals, for reducing the total number of data points required in marker-assisted backcross conversions.

The binomial distribution explains the behavior of populations in which there are only two classes of information, such as molecular marker data from a backcross population. Backcross population data from 4 unlinked markers are binomially distributed in 5 classes (0, 1, 2, 3 and 4 recurrent parent alleles) just as backcross data from 40 unlinked markers are binomially distributed in 41 classes (0, 1, 2 . . . 40 recurrent parent alleles). After an initial analysis with 4, 6 or 8 markers, what progress can be made by selecting the individuals with the highest percentage recurrent parent? A theoretical population of 250 individuals was analyzed with 4, 6 and 8 binomially distributed (unlinked) markers. The results of a 40 marker analysis, after initial selections with 4, 6, and 8 markers, are shown in Figure 1. The best individuals (greater than 50% recurrent parent) selected after analysis with 4, 6, or 8 markers and further analysis with 36, 34, or 32 markers produced the black, stippled or lined curves in Figure 1A, 1B and 1C. The populations of individuals selected after 4, 6, or 8 markers are all skewed toward higher number of recurrent parent alleles of a 40 probe distribution. Selection after this limited analysis eliminated the worst members (lowest percentage recurrent parent) of the population and pushed the distribution (slightly) toward the higher percentage recurrent parent. It seemed amazing that selection after only 4 markers could decrease the number of individuals analyzed and still provide a majority of the best (highest percentage recurrent parent) individuals after a total of 40 analyses.

Based on the previous result, it seemed possible to analyze a backcross population incrementally and select the majority of the best individuals, without being required to analyze every individual with all 40 markers. Consecutive selections in 5, 10, and 15 marker increments are shown in Figure 2. The best individuals (from a 250 plant binomial distribution) were selected at 3/5, 4/5, and 5/5 recurrent parent alleles and analyzed with an additional 5 markers. This distribution is shown in Figure 2A as the black bars. Individuals from the 6/10, 7/10, 8/10, 9/10, and 10/10 recurrent parent classes were selected and analyzed with an additional 5 markers. The result of this binomial analysis is shown in Figure 2B as the black bars. Individuals from the 9/15, 10/15, 11/15, 12/15, 13/15, 14/15, and 15/15 classes were selected and an additional 5 marker binomial was run. The predictions for these selections are shown in Figure 2C. This procedure identifies more than 80% of the individuals with >13/20 recurrent parent alleles but requires only half of the analysis.

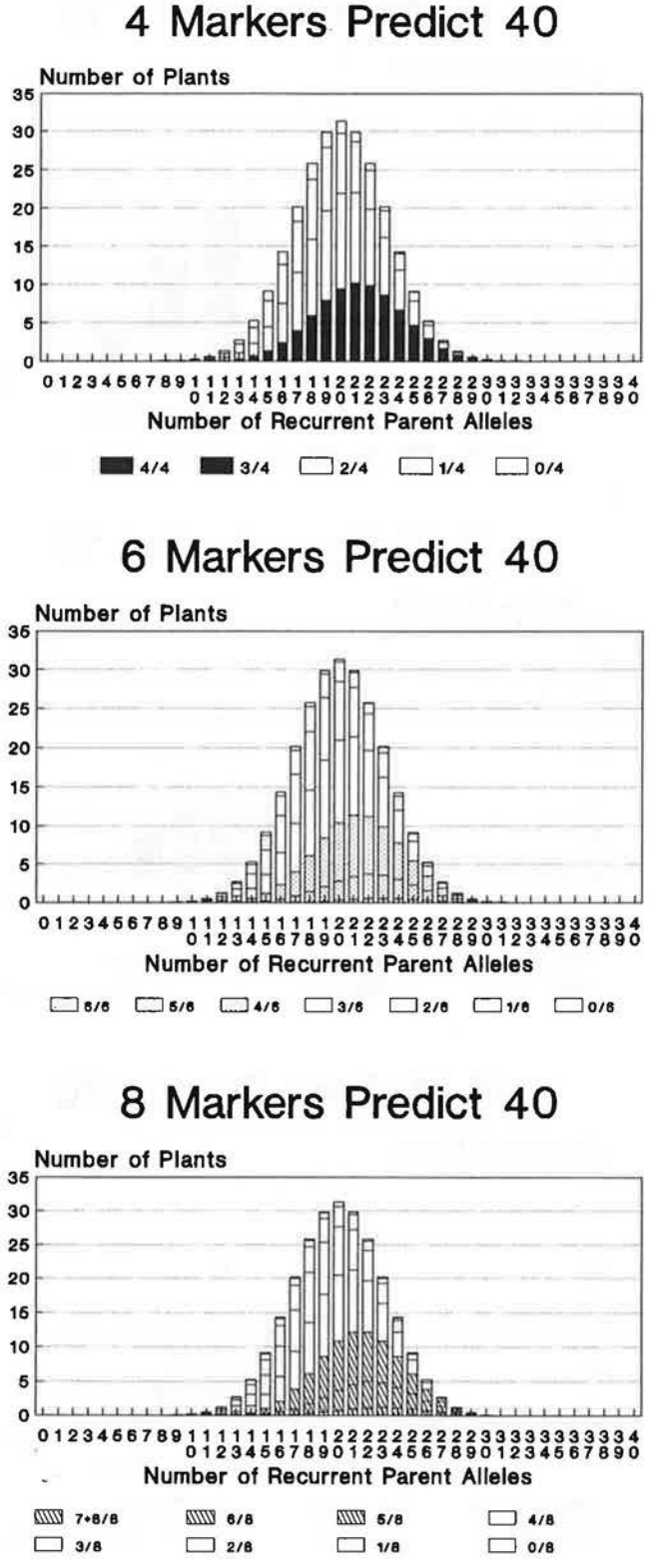
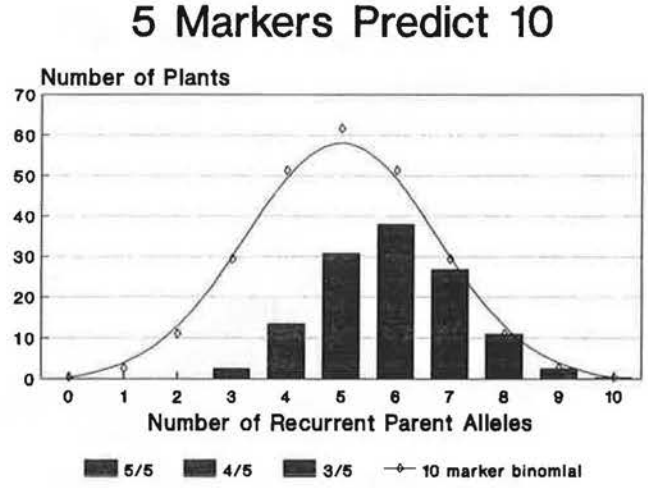
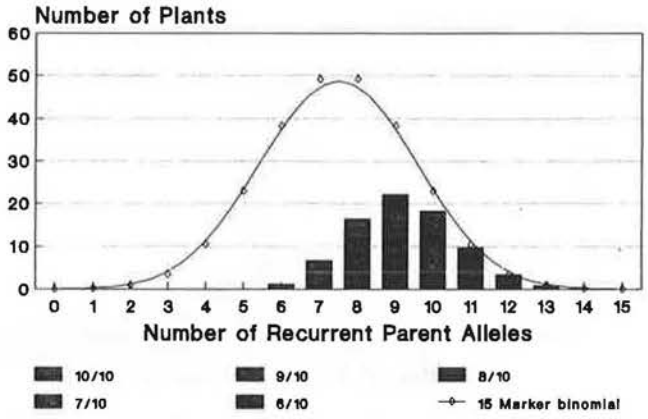


Figure 1.

The attainable progress in a backcross conversion is dependent on the number of plants in the population. Once the number of plants has been selected, theoretical progress is fixed and success is predicated on an accurate measurement of recurrent par-





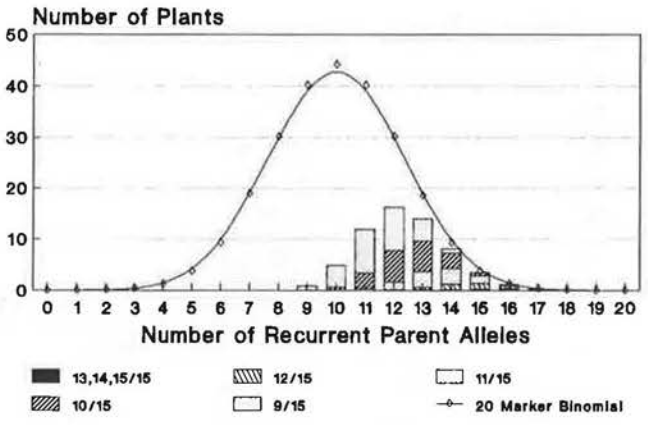


Figure 2.

ent percentage. If a major goal in plant breeding is to eliminate the "losers," this stepwise procedure does that rapidly.

The use of the stepwise selection procedure presented here can reduce the cost of marker-assisted backcross conversions. The total number of individuals analyzed is reduced by sequential selection of the best individuals (2575 vs. 5000 analyses in this example). Additional selection steps can be incorporated if a higher percentage of the "best" individuals is required. This molecular analysis of backcross conversions is better suited to RAPDs (Tingey/McClelland) than to RFLPs because of the ability to set up each PCR experiment individually (no reusable nylon membrane required).

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A study of a new source of *Bg*

--V. V. Kotemiak

Kernels with variegated endosperm structure in which flint sectors were alternating with opaque sectors were found in a selfed generation of hybrid Zpl2077/54-14 *o2* x SP168 *o2*. In 1988 S2 forms of this hybrid (further referred to as 3449 *o2*) originating from variegated seeds were crossed with 346 *o2* and 502 *o2* lines, which possessed standard opaque endosperm. Most of the F1 kernels were variegated, a small part of them were phenotypically normal. Similar results were obtained when the original source of variegation was selfed. This made it possible to suppose the presence in 3449 *o2* of a regulatory factor, in the presence of which the responding recessive *o2* allele reverts to normal type.

Further investigations show that this regulatory factor is not either of the known regulatory elements *Ac* and *Spm*. Thus F2 seeds from crosses between strains with the responding *o2* allele and *wx-m7* and *pg14-m-En* showed no somatic instability at the *o2* locus. In 1990 it became possible to carry out the test for allelism with the regulatory element of the *Bg-rbg* system described by Salamini (MGG 179:497, 1980). The F1 of 3449 *o2* with *o2-m(r) bg* gave fully variegated ears and ears with variegated and a few normal kernels (0.3-4.1%). Plants originating from variegated seeds gave in the F2 normal, variegated and opaque kernels. The ratio of sum of normal and variegated kernels to opaque did not differ significantly from 3:1 (Table 1), indicating allelism of the regulatory element present in 3449 *o2* to the *Bg* element. Taking into account a positive test for allelism, the regulatory element present in 3449 *o2* was designated as *Bg-3449*.

Table 1. Endosperm phenotypes on ears derived from variegated seeds of the cross 3449 *o2* x *o2-m(r)bg*.

		Endosperm phenotype		
Ear number	Normal	Variegated	Opaque	Chi-square 3:1
91-5260-1	15	257	86	0.18
91-5260-2	4	297	86	1.59
91-5260-3	2	182	63	0.03

While studying (346 *o2* x 3449 *o2*) x 346 *O2* and (502 *o2* x 3449 *o2*) x 502 *O2* crosses it was established that *Bg-3449* is not linked to the responding *o2* allele. All plants with this genotype were selfed and crossed (as pollinator parents) with opaque testers 346 *o2* and 502 *o2*. As was expected one half of the progenies obtained (57 of 112) segregated normal, variegated and opaque kernels. For 30 of 57 selfed ears the ratio of normal, variegated and opaque kernels did not differ significantly from 12:3:1 (a nonsignificant deviation of the ratio of sum of normal and variegated to opaque kernels from 15:1 was observed on 51 ears). When the same plants were crossed to opaque testers the ratio of normal, variegated and opaque kernels equal to 2:1:1 was significant for 42 ears. Besides ears segregating normal, variegated

and opaque kernels, and ears segregating normal and opaque kernels, two ears were found with normal kernels only. The possibility of formation of them will be discussed below.

When selfed on plants originating from variegated seeds, there were no ears observed with clusters of revertant kernels, which may indicate that reversion of the receptive *o2* allele in the presence of *Bg-3449* takes place in postmeiotic mitotic divisions during mega- or microsporogenesis, which is a distinctive feature of the *o2-m(r)-Bg* system of controlling elements (Montanelli et al., MGG 197:209, 1984). At the same time the possibility of reversions of the responding *o2* allele during sporophyte development seems not to be excluded. First of all it is necessary to mention the complication of classification of kernels on ears of the original source of instability because variegated kernels were mostly of a coarse type of variegation and had a flint crown. Besides this, as was mentioned above selfed progenies of genotypes (346 *o2* x 3449 *o2*) x 346 *O2* gave 2 ears with normal kernels only. Further analysis of these kernels showed that they were homozygous for normal allele *O2* and did not display *Bg* activity. The formation of these ears may be explained by reversion of the responding *o2* allele to normal during development of the sporophyte before meiosis. Another possible explanation of this phenomenon may be the reversion of the receptive *o2* allele in the presence of *Bg-3449* in postmeiotic mitotic divisions during mega- or microsporogenesis in combination with at least one of the following events: i) inactivation of the regulatory element, the possibility of which was reported by Salamini et al. (Heredity 49:111, 1982), or ii) nonreplicative transposition of *Bg-3449*.

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Genetic maps of visible and RFLP markers in the vicinity of *Tp1* and *Tp2*

--Deverie Bongard-Pierce, Mark Dudley and Scott Poethig

Restriction fragment length polymorphism (RFLP) maps of the regions around *teopod1* (*tp1*) and *teopod2* (*tp2*) were constructed using probes obtained from the University of Missouri (umc), Brookhaven National Laboratories (bnl), Native Plants Incorporated (npi) and Pioneer Hi-Bred Intl. (php). RFLPs in the vicinity of *Tp1* were mapped in the following testcross: *Gl1 Tp1 Sl/gl1 tp1 sl* x *gl1 tp1 sl*. *gl1* and *sl* are the most tightly linked mutations flanking *Tp1*. 254 progeny from three related families were scored for recombination between these loci and 55 individuals that were recombinant for the visible markers were subjected to RFLP analysis. These recombinants provide the following map: *gl1*--8.7--*umc116*--3.9--*php20569*--0.8--*Tp1*--1.6--*bnl15.21*--6.3--*php15037*--0.4--*sl*. These data confirm previous results (R.S. Poethig, MNL63:101) concerning the order of *gl1*, *Tp1* and *sl* but provide a significantly different value for the distance between *Tp1* and *sl*. In each of the three related families, *Tp1* and *sl* exhibited approximately 8% recombination; by contrast, in previous experiments these genes were separated by 3% recombination. It should be noted that our data place *bnl15.21* and *umc116* in an inverted position relative to *php20569* compared to the positions of these loci in the most recent maps from the Brookhaven Lab and Pioneer Hi-bred. However, the distances we observed are comparable to those measured by these two groups.

RFLPs in the vicinity of *Tp2* were mapped using the test cross: -- *Tp2 R-r /Ds tp2 R-scm; C/C* x *P-VV; r;c*. 40 recombinants between *Ds* and *R-* were observed in 290 individuals. All of these recombinant plants as well as 65 non-recombinant plants were scored for the segregation of RFLPs in this region. Two RFLPs, *php20719* and *umc163*, exhibited no recombination with *Tp2* in this cross. In order to map these probes, 615 progeny from the cross *Tp2 G/tp2 g* x *tp2 g* were scored for recombination between *Tp2* and *G* and the resulting 12 recombinants were subjected to RFLP analysis. This cross allowed *php20719* to be positioned 0.8cM distal to *Tp2*. Because none of the *Tp2--g* recombinants were recombinant for *umc163*, the position of this marker is still in doubt. Our current map of the region around *Tp2* is as follows: *php15013*--11.8--*umc155*--5.7--*npi264*--1.9--*Ds* --1.4--(*Tp2, umc163*)--0.8--*php20719*--1.1--*g*--11--*umc44a*--1.4--*R*.

Deletions of *Tp2*

--Mark Dudley, Deverie Bongard-Pierce and Scott Poethig

Tp2-x2 and *Tp2-x16* are X-ray-induced revertants of *Tp2* which are not transmissible through pollen and are transmitted through the egg at a reduced frequency. Both of these revertants are missing the *php20719* band present in the parental *Tp2 g* stock, indicating that they are deletions. The *Tp2*-proximal *npi264* and *umc155* loci are not deleted in *Tp2-x16*. We have yet to analyze these markers in *Tp2-x2*.

The breakpoint of TB-10La

--Mark Dudley and Scott Poethig

The TB-10La translocation uncovers *g*, a recessive mutation approximately 2cM distal to *Tp2*, but does not uncover any recessive mutations proximal to *g*. Consequently the breakpoint of TB-10La relative to *Tp2* is unclear. RFLP analysis demonstrates that the TB-10La breakpoint is located between *npi264* and *umc155*. Since these RFLPs are both proximal to *Tp2*, the TB-10La breakpoint is proximal to *Tp2*.

Vegetative and reproductive development in *leafy1* and early flowering plants

--Hilli J. Passas* and Scott Poethig
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Traditionally, the development of the aerial shoot has been defined by the ability of the plant to flower. In most cases, the transition from the non-flowering, juvenile to the adult phase, in which flowering is possible, cannot be determined with certainty until the plant actually produces flowers. Changes in the vegetative morphology often occur concomitantly with the transition to reproductive maturity and have been used as indicators of this transition; yet, conclusive proof of such correlation is lacking. In order to investigate the relationship between the changes in vegetative and reproductive development, we examined the expression of traits indicative of each phase in early and late flowering lines of the inbred A632. The early flowering line A632E was derived by Don Shaver by introgressing genes from Gaspe Flint. Our preliminary observations suggest that the early flowering phenotype of this line is conditioned by no more than two semidominant genes. The late flowering line carried the dominant mutation *Lfy1*, also obtained from Don Shaver. In maize, the presence of epicuticular wax and epidermal hairs is specific to the juvenile and adult vegetative phases, respectively, while the total number of leaves pro-

duced by the plant correlates with flowering time. Table 1 shows that both lines modify the total leaf number without affecting the expression of the vegetative markers. This suggests that the transition to reproductive maturity and changes in the vegetative development proceed independently of each other.

Table 1. Effect of *Lfy1* and early flowering on placement of vegetative and reproductive organs in A632.

Genotype	Last leaf with wax	First leaf with hairs	Ear node	No. of husk leaves	No. of leaves between ear and tassel	Total number of leaves
Lfy1/+, +/+	10.1±0.2	4.6±0.2	15.9±0.4	11.4±0.7	14.8±1.0	30.7±1.1
+/+, +/+	10.0±0.1	4.7±0.2	14.0±0.2	9.8±0.7	6.1±0.0	20.0±0.2
Lfy1/+, E/+	9.6±0.3	4.9±0.3	14.1±0.5	11.9±0.8	13.8±1.0	27.7±1.3
+/+, E/+	9.3±0.3	4.9±0.2	12.6±0.4	9.6±0.9	5.8±0.3	18.3±0.5
Lfy1/+, E/E	9.7±0.2	5.4±0.3	12.1±0.3	10.6±0.5	10.5±0.5	22.5±0.6
+/+, E/E	9.5±0.3	5.3±0.3	10.8±0.4	9.5±0.7	5.3±0.2	16.1±0.4

Additionally, we were interested in the effect of these mutations on the relative positioning of the reproductive organs. Most inbreds produce five to six leaves between the ear and the tassel, regardless of the number of leaves generated by the plant overall. This constancy has led to the idea that the placement of the reproductive organs relative to each other is coordinately regulated (W.C.Galinat in Corn and Corn Improvement, p. 22, 1988). Such developmental regulation could involve one organ determining the position of the other. Alternatively, coordination could be accomplished by one reproductive stimulus determining the placement of both organs. In the latter scenario the reproductive stimulus would cause the plant to produce two independent shoots, one terminating in a female inflorescence (the ear), the other one in a male inflorescence (the tassel). The number of leaves on both shoots would thus be correlated as long as there is no change in an organ-specific sensitivity to such a stimulus.

To test these possibilities we studied the placement of the ear and the tassel in the lines described above. Our results confirm previously published findings (D. L. Shaver, Proc. Ann. Corn Sorghum Res. Conf. 38:161-180). In A632E the ear node and the total leaf number is reduced without a significant reduction in the number of husk leaves and only a small but significant decrease in the distance between the ear and the tassel. This is consistent with a role of the early flowering factor in A632E in a general reproductive program, to which all floral organs respond equally. In contrast, the principal effect of *Lfy1* is to increase the number of leaves between the ear and the tassel. The effect on the position of the ear and on the number of husk leaves is small but significant. Thus, in *Lfy* plants all lateral buds develop normally, whereas the apical meristem remains vegetative longer than in wildtype. This makes it unlikely that the ear position is determined by the tassel. Plants carrying both the *Lfy1* and early flowering traits display an additive phenotype, suggesting that these genes act in different developmental pathways.

Phase change in inbred and exotic lines of maize

--Scott Poethig and Hilli J. Passas

The development of the shoot has traditionally been divided into two discrete phases: a juvenile phase characterized by a variety of vegetative traits and the inability to produce reproductive structures, and an adult phase characterized by a different set of vegetative traits and the ability to produce reproductive structures. This interpretation implies that the reproductive and vegetative development of the shoot are linked in some way, although there is no conclusive evidence for this assumption. To determine if reproductive development in maize is correlated with the change from a juvenile to adult phase of vegetative development, we studied the expression of phase-specific vegetative traits in inbred and exotic lines that varied in total leaf number (i.e., in the "timing" of tassel initiation). Figure 1 is a graph of the last completely waxy leaf, the last leaf with any epicuticular wax (a juvenile trait), the first node with epidermal hairs (an adult trait), the ear node, and the total leaf number in 10 inbreds and exotic varieties of maize ranging in leaf number from 15 (Black Mexican Sweet) to 23 (Sc76). As expected from previous studies, the position of the ear was tightly correlated with leaf number; with the exception of Argentine popcorn, there were 5 or 6 nodes between the ear and tassel. Argentine popcorn was unusual in having only 3 nodes between the topmost ear and tassel. The loss of epicuticular wax was highly correlated with the appearance of epidermal hairs, although in a few inbreds (e.g. Sc76 and A632) hairs appeared on completely waxy leaves. This observation suggests that the juvenile and adult programs are not mutually exclusive.

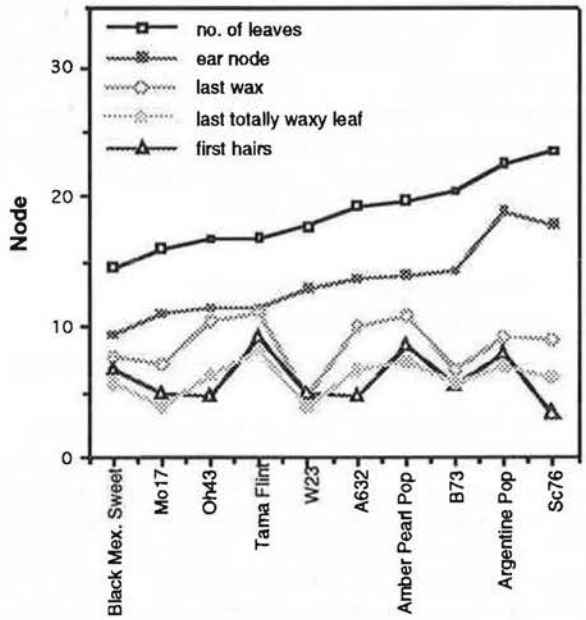

Figure 1: The expression of vegetative and reproductive traits in inbred and exotic lines of maize

With respect to relationship between vegetative and reproductive development, the most interesting feature of these data is that the vegetative maturation of the shoot is unrelated to total leaf number or ear position. Although both the ear and the tassel are only produced by meristems that are in an adult phase, the positions of the tassel and ear do not correlate with the change in the vegetative phase of the shoot. Therefore, while it is still unclear whether the adult phase is a prerequisite for reproductive development, it is likely that the initiation of the ear and tassel is not a direct consequence of the change in the vegetative phase of the shoot.

Phase change along axillary branches

--Matthew M. S. Evans and Scott Poethig

During development maize plants pass through a juvenile vegetative phase and an adult vegetative phase. Juvenile phytomers, or segments, unlike adult phytomers, possess prop roots and leaves with a visible waxy bloom. The primary shoot of a plant first produces juvenile leaves, then produces adult leaves, and finally terminates in a tassel. In some genetic backgrounds maize plants produce axillary branches, called tillers, which appear to have the same structure as the primary axis. We were interested in determining the duration of the juvenile phase of development in tillers originating at different positions. We wanted to determine whether tillers repeated the entire developmental program of the main stem or whether tillers sensed their position and underwent the transition from juvenile to adult development at a position parallel to that of the main stem.

Using the recessive mutation *teosinte branched*, which causes plants to produce a large number of tillers, we scored the duration of the juvenile phase of development on tillers originating at different positions along the primary axis of the plant. Phase change appears to occur in the same manner on tillers as it does on the primary axis of the plant. However, the developmental phase is not reset to a basal level in each tiller, and consequently tillers are not complete reproductions of the main stem. Tillers sense their position on the plant, and the duration of the vegetative phases reflects that position. As shown in Table 1, each tiller has a shorter juvenile phase than the main stem, or tillers from lower positions. The transition from the juvenile to the adult phase appears to occur after approximately the same number of phytomers from the base of the plant, either along the main axis or along a tiller. In the families examined, epicuticular wax is produced on the first five leaves of a plant, whether leaves are located on the main stem and the tiller or on the main stem only. Tillers also produce fewer leaves before tasseling than the main stem; moreover, tillers from higher nodes produce fewer leaves than tillers from lower nodes. However, tassel formation does not occur at the same position from the base of the plant on the tillers as it does on the main stem.

Table 1. Pattern of expression of phase-specific traits on the main stem and tillers of *teosinte branched* plants.

Shoot Position	Last Leaf with Wax	Last Node with Prop Roots	Last Leaf with an Axillary Shoot	Number of Leaves
Main stem	5.1 ± 0.4[c]	8.2 ± 0.5[c]	12.4 ± 0.3[c]	16.9 ± 0.4[c]
Tiller in the axil of leaf 2	3.1 ± 0.4[c]	5.5 ± 0.8	8.9 ± 0.8[b]	12.4 ± 0.8[a]
Tiller in the axil of leaf 3	2.1 ± 0.6[b]	5.3 ± 0.9[b]	8.0 ± 1.1[a]	11.7 ± 1.1[b]
Tiller in the axil of leaf 4	1.0 ± 0.7[a]	4.3 ± 0.6[c]	6.1 ± 0.6	9.9 ± 0.5[a]
Tiller in the axil of leaf 5	0.4 ± 0.6	3.4 ± 0.6	5.3 ± 1.3	9.1 ± 1.0

[a]Mean is significantly different from the mean on the following tiller at the 0.05 level.
[b]Mean is significantly different from the mean on the following tiller at the 0.01 level.
[c]Mean is significantly different from the mean on the following tiller at the 0.001 level.

These results indicate that vegetative phase change occurs globally throughout the plant and is not confined to the shoot apical meristem of the primary axis. The developmental phase of an axillary shoot reflects the developmental phase of the part of the main shoot from which it originated.

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Imprinting of a zein post-transcriptional regulator

--Sumita Chaudhuri and Joachim Messing

The level of expression of methionine-rich 10kD zein in maize endosperm is determined by a post-transcriptional regulator, *Zpr10/22* (TAG 78:761, 1989; MGG 225:331, 1991). In mature endosperm of BSSS53, the level of 10kD zein is several times higher than in W64A, A654, A619 and Mo17 lines. Studies involving crosses of BSSS53 with W64A, A654 and A619 indicate that *Zpr10/22* functions in a dose dependent manner in determining the 10kD zein level. For example, in reciprocal crosses of BSSS53 and W64A (Fig. 1), there is increased accumulation of 10kD zein with increasing doses of the *Zpr10/22* allele from the BSSS53 parent. A similar dosage response is seen in the steady-state level of 10kD zein mRNA in immature endosperms of the same reciprocal crosses. However, in reciprocal crosses of BSSS53 and Mo17 the level of 10kD zein in the hybrids resembles that in the female parent instead of a dosage response (Fig. 2A). The steady-state 10kD zein mRNA level also shows maternal effect throughout endosperm development of the hybrids (Fig. 2B). This maternal effect cannot be due to maternal cytoplasmic or sporophytic factors since the 10kD zein level segregates with *Zpr10/22* in backcross populations.

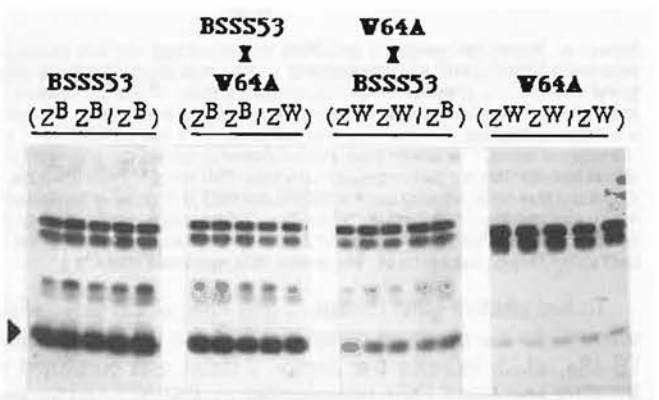


Figure 1. Western blot analysis of SDS-PAGE fractionated total zein from mature endosperms of BSSS53, W64A and their reciprocal crosses using polyclonal antibody raised against 10kD zein. The arrowhead indicates the 10kD zein position. Z^B and Z^W represent the *Zpr10/22* allele from BSSS53 and W64A respectively.

There are two possible explanations for this maternal effect. First, unequal gene dosage from the parents in the endosperm leads the allelic composition of *Zpr10/22* to differ in the reciprocal hybrids (BSSS53 BSSS53/Mo17, Mo17 Mo17/BSSS53). Thus, at least two doses of the Mo17 allele may be necessary to downregulate the 10kD zein level. Ordinarily, two doses of the BSSS53 allele are not required to upregulate 10kD zein level. For instance, one dose is sufficient to upregulate the 10kD zein level in crosses with W64A.

Imprinting, i.e. unequal gene function following passage through male or female parent, is the other possible explanation for this maternal effect. As seen in crosses with W64A, the BSSS53 allele is functional irrespective of which parent transmits it, and is therefore not the allele imprinted. According to this model, therefore, the Mo17 allele is subject to imprinting so that it is a dominant downregulator of 10kD zein expression only when transmitted by the female parent.

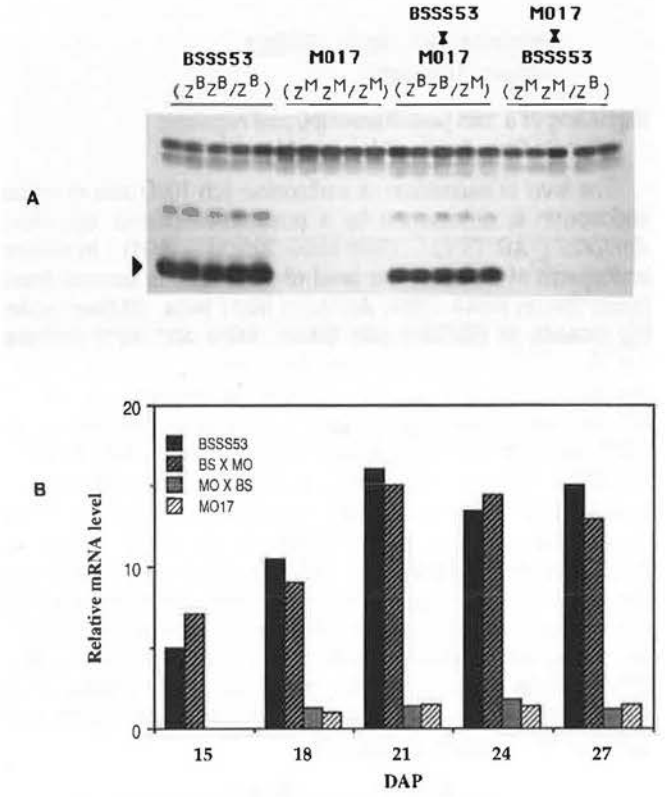


Figure 2. A. Western blot analysis of SDS-PAGE fractionated total zein from mature endosperms of BSSS53, Mo17 and their reciprocal crosses using polyclonal antibody raised against 10kD zein. The arrowhead indicates the 10kD zein position. Z^B and Z^M represent the *Zpr10/22* allele from BSSS53 and Mo17 respectively. B. Relative levels of 10kD zein mRNA in immature endosperms at various days after pollination (DAP) from BSSS53, Mo17 and their reciprocal crosses. The relative levels were determined by successively probing slot-blot filters of RNA with 10kD zein gene probe and A copy of the 27kD zein gene probe (the levels of 27kD A zein gene mRNA are comparable in BSSS53 and Mo17 at all stages of development). Hybridization intensities obtained with the two probes were determined by densitometry and the level of 10kD zein mRNA normalized against 27kD A gene mRNA. The ratio obtained for Mo17 at DAP 18 was given a value of 1 and all other ratios represented relative to it.

To test whether gene dosage or imprinting of the Mo17 allele accounts for the observed maternal effect, the B-A translocation TB-4Sa, which includes the *Zpr10/22* locus, was converted to BSSS53 and Mo17 lines (represented as BSSS53-TB4Sa and Mo17-TB4Sa) and used in various crosses (see Table 1). Frequent non-disjunction of the B centromere during pollen gametogenesis causes variations in the dosage of *Zpr10/22* contributed by the male parent, leading to recovery of hypoploid, hyperploid or euploid endosperms on the same ear. The hyperploid endosperms arising out of the crosses BSSS53 X Mo17-TB-4Sa and Mo17 X BSSS53-TB-4Sa are identical in *Zpr10/22* gene dosage. However, the gametic origin of each allele is different. Comparison of the 10kD zein levels and the 10kD zein mRNA levels between these two groups of hyperploid endosperms is therefore critical in testing whether gene dosage or imprinting accounts for the maternal effect. Since maternal effect is not observed in crosses of BSSS53 and W64A, the cross W64A X BSSS53-TB-4Sa should yield progeny that correlate 10kD zein and its mRNA level with *Zpr10/22* dosage.

Our analyses show that the levels of 10kD zein as well as 10kD zein mRNA are similar to BSSS53 in all progeny arising from the cross, BSSS53 X Mo17-TB-4Sa. Likewise, progeny from Mo17 X BSSS53-TB-4Sa are similar to the maternal parent Mo17 in 10kD zein and its mRNA levels. Thus, the expression of 10kD zein appears not to be determined by the dosage of *Zpr10/22* in these

Table 1. Crosses to test gene dosage vs. imprinting of the Mo17 allele. The B-A translocation TB-4Sa carrying *Zpr10/22* on the translocated chromosome 4 arm was backcrossed five times with inbred lines BSSS53 and Mo17 to generate BSSS53-TB-4Sa and Mo17-TB-4Sa. The expected gene dosage and allelic composition of *Zpr10/22* in the endosperm is indicated next to each cross. Z^B, Z^M and Z^W represent *Zpr10/22* alleles from BSSS53, Mo17 and W64A, respectively.

♀	Crosses	♂	gene dosage in endosperm	
BSSS53	X	M017-TB4Sa	$z^Bz^B/-$	hypoploid
			z^Bz^B/z^Mz^M	hyperploid
			z^Bz^B/z^M	euploid
M017	X	BSSS53-TB4Sa	$z^Mz^M/-$	hypoploid
			z^Mz^M/z^Bz^B	hyperploid
			z^Mz^M/z^B	euploid
W64A	X	BSSS53-TB4Sa	$z^W z^W/-$	hypoploid
			$z^W z^W/z^Bz^B$	hyperploid
			$z^W z^W/z^B$	euploid

crosses but rather by the gametic origin of the Mo17 allele. However, progeny from W64A X BSSS53-TB4-Sa with varying doses of the BSSS53 allele differ in the levels of 10kD zein expression, suggesting a dosage effect.

Taken together, our results indicate that the observed maternal effect in reciprocal crosses of BSSS53 and Mo17 cannot be explained by considering unequal gene dosage contributions of the parents in the hybrids. This indicates imprinting of the Mo17 allele function whereby it is effective as a dominant downregulator of 10kD zein level only when it is transmitted through the female gametophyte. However, this study cannot determine whether the Mo17 allele is directly imprinted or its function modified in response to other imprinted factors in Mo17. For this, it would be necessary to introgress the Mo17 allele in a non-imprinted line such as BSSS53 or W64A and study the effect of its transmission through the male and female gametophytes.

Zeon1, a member of a large retrotransposon family

--Weiming Hu, O. Prem Das and Joachim Messing

Studies on the tandemly duplicated 27kD zein genes had identified a mitotic DNA rearrangement at this locus in particular stocks of A188 (Das et al., PNAS 87:7809, 1990). This appeared to be a two-step process, consisting of a homologous recombination between the repeats of the duplicated *S* allele, and a second rearrangement at the 5' end (Fig. 1). Cloning the rearranged allele has now shown that the second event is the insertion of a 7.3kb element 1.1kb upstream of the A gene (Fig. 1). By using PCR primers near the insertion site, we have shown that this insertion is only found in the *Ra* allele which has only the A copy, and not in the *S* allele which has both the A and B copies of the duplication (Fig. 1). The *Ra* allele is found in many other inbred lines, e.g. W64A, B37 and A619.

We have characterized the 7.3kb insertion element which we designate *Zeon1* (zein retrotransposon) because it has many of the characteristics of a retrotransposon. Its insertion site is flanked by a typical 5bp target site duplication (Fig. 1). In addi-

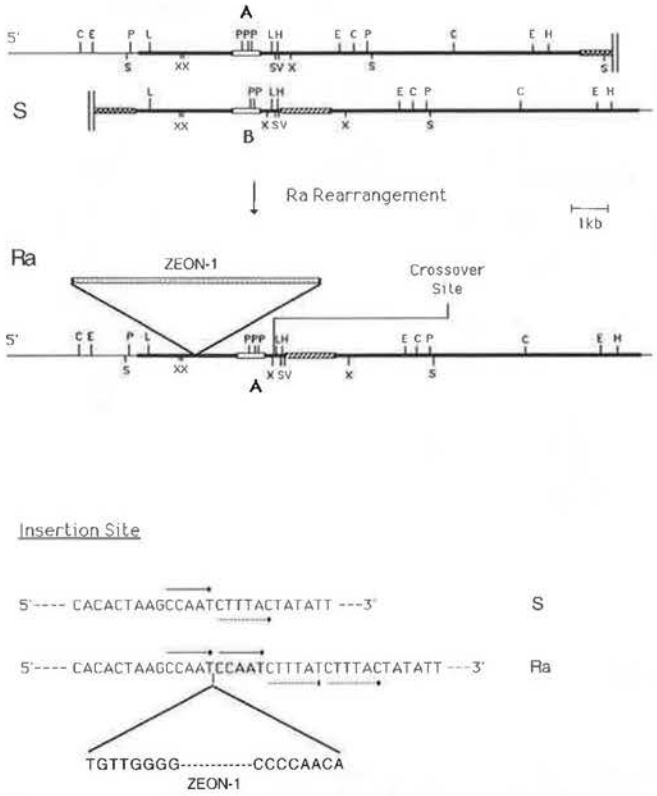


Figure 1. The upper panel shows the restriction maps of *S* and *Ra* alleles. The A and B copies of the duplications of the *S* allele are drawn on separate lines to highlight homology. Homologous regions are represented by the heavy line, open boxes indicate coding sequences and the striped box represents an insertion unique to the B copy. The crosshatched box separates the duplications. The *Ra* allele has a 7.3kb insertion, *Zeon1*, located 1.1kb upstream of the coding sequence. In addition, its formation requires a second homologous crossover between the repeats at the indicated site. Transcription direction is from left to right. Restriction site key: B = *Bgl*I, C = *Sca*I, E = *Eco*RI, H = *Hin*dIII, L = *Sal*I, P = *Pst*I, S = *Sac*I, X = *Xba*I. The lower panel shows nucleotide sequences at the *Zeon1* insertion site in the *S* and *Ra* alleles. Note the 5bp target site duplication, the 8bp terminal inverted repeats flanking *Zeon1*, and the imperfect 6bp duplication immediately adjacent to the insertion.

tion, a second imperfect duplication is present at one end (Fig. 1). Two LTRs (long terminal repeats) of 649bp and 662bp have the typical structure of U3-R-U5, including nucleotide sequences similar to a maize TATA box and a poly-A signal. *Zeon1* also has a PBS (primer binding site) and a purine-rich sequence at the 5' and 3' ends of the internal sequence. Its PBS consists of ten nucleotides complementary to the 3' end of Lys-tRNA; most plant retrotransposons have a Met-tRNA binding site. The internal sequence of *Zeon1* has several open reading frames. The largest one is a gag-related ORF of 375 aa, that includes a putative zinc finger in the nucleic acid binding domain. Comparison of the zinc finger region of *Zeon1* to that of other retrotransposons and retroviruses shows that *Zeon1* has one extra amino acid (Fig. 2).

Zeon-1	1984	tgt	ata	ttc	tgt	ggt	gaa	gac	aag	ggc	cat	acc	acc	agg	atg	tgc	cac
	322	C	I	F	C	G	E	D	K	G	H	T	T	R	M	C	H
Tnt-1	232	C	Y	N	C	N	Q	P	-	G	H	F	K	R	D	C	
Copia	232	C	H	H	C	G	R	E	-	G	H	I	K	K	D	C	
MMULV	504	C	A	Y	C	K	E	K	-	G	H	W	A	K	D	C	
RSV	509	C	Y	T	C	G	S	P	-	G	H	Y	Q	A	D	C	
HIV-1	392	C	F	N	C	G	K	E	-	G	H	T	A	R	N	C	

Figure 2. A comparison of the gag-related ORF of *Zeon1* with the conserved nucleic acid binding domain found in retroviruses and retrotransposons is shown. Amino acid sequences are indicated in capital letters, and the corresponding nucleotide sequence for *Zeon1* is indicated in lower case. *Tnt1* is a retrotransposon from *Nicotiana tabacum*; *Copia* is a retrotransposon from *Drosophila melanogaster*; MMULV is the Moloney murine leukemia virus; RSV is the Rous sarcoma virus; HIV-1 is the human immunodeficiency virus. The conserved cysteine, histidine and glycine residues involved in the zinc-binding domain are marked.

The lengths of the other longest ORFs are 92 aa and 113 aa, but none of them shows significant homology to reverse transcriptases.

Using probes for the LTR and for an internal sequence, we estimate a copy number of around 1000 for LTR-related sequences, and between 300-400 for the internal sequence. Sequence comparisons show two regions of 91bp and 120bp in the LTR of *Zeon1* that are homologous to the maize retroelement *Cin1* (Shepherd et al., Nature 307:185, 1984). Sequences homologous to the LTR are also found at the 3' flanking region of a 19kD zein gene (Kriz et al., MGG 207:90, 1987).

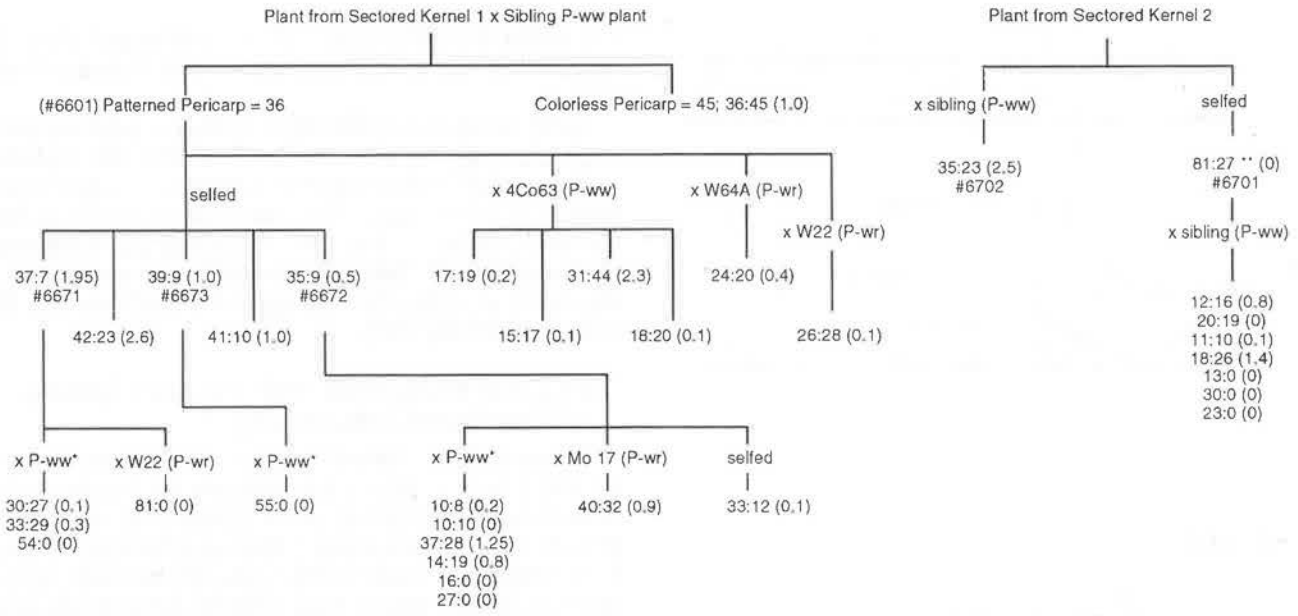
A new allele of the *P* gene that conditions patterned pericarp

--O. Prem Das and Joachim Messing

Earlier studies had identified isolates of the inbred line A188 carrying a heritable activity that increased the frequency of somatic mutations of several maize genes (Das et al., PNAS 87:7809, 1990, Das and Messing, Methods in Molecular Genetics, K. W. Adolph, ed., Academic Press, Orlando, In Press). Somatic mutations of the *P* gene in these A188 isolates and their derivatives were recovered as colorless pericarp sectors on the uniform red background in *P-rr*/*P-ww* plants. Mutations causing a pericarp sector can be transmitted to the underlying kernel at a maximal frequency of 50% if the sector covers the germinal half or more of a kernel (Anderson and Brink, Am. J. Bot. 39:637, 1952). Among progeny ears from eight kernels bearing such sectors, two showed a patterned or variegated phenotype in pericarp. This phenotype was not seen among >6,000 ears screened over the course of these experiments, indicating that transmission of the mutation causing the sector was responsible for the new phenotype in the two cases.

Inheritance of the patterned pericarp phenotype has been studied over five additional generations of one isolate, and two of the other. Genetic analysis of three generations of progeny from one sectored kernel and two generations from the other is presented in Fig. 1. The sectored kernels were from ears obtained by growing *P-rr*/*P-ww* plants and their F2 progeny in an isolation plot. Both kernels evidently received *P-ww* from the male parent, as judged by segregation in the succeeding generation (Fig. 1). Segregation of the patterned phenotype was consistent with the phenotype being conditioned by an allele of *P*, designated *P-pr* for *p*atterned pericarp and *r*ed cob. Pigmentation conditioned by this allele in pericarp, cob and tassel glume edges was highly variable, but variegation could be detected only in pericarp. Pigmentation in pericarp varied from color only at the silk attachment point to almost full color, and cob color varied from almost colorless to red. Homozygotes among sibling progeny displayed less pigmentation in pericarp than heterozygotes. But this was insufficient to account for the extent of variation in pericarp color, and it is not yet clear if the additional variation has a genetic basis.

Molecular analysis using two probes derived from the *P* gene (kindly provided by Dr. Thomas Peterson; Lechelt et al., MGG 219:225, 1989; Grotewold et al., PNAS 88:4587, 1991), and 27 methylation-insensitive restriction enzymes showed no differences between *P-pr* and *P-rr*. This indicated that no major insertion, deletion or rearrangement of the *P* gene was associated with the mutation to *P-pr*. However, eight restriction enzymes sensitive to cytosine methylation showed that the *P* gene in leaf DNA from plants carrying *P-pr* was hypermethylated relative to *P-rr*. Representative data with *Sal*I are shown in Fig. 2. Increased methyla-



LEGEND

= family accession number
* = plants homozygous for P-ww among the progeny of families 6601, 6671, 6672 or 6673.
** = one of the patterned class was fully red; see text for details.

NOTES

1. Each entry gives the segregation data for a family (progeny of one ear) as the number of ears with patterned pericarp vs colorless pericarp.
2. The values in brackets give the uncorrected chi-square values assuming that patterned phenotype was conditioned by an allele of P.
   Except for the first generation, expected segregation ratios were based on molecular identification of the genotype of the parent contributing P-pr.

Figure 1. Genetic analysis of the patterned pericarp phenotype.

tion was detected with probe 15, but not with probe 8B (Fig. 2). The location of the two probes showed that 5' and 3' flanking sequences of the gene were hypermethylated, but not the intervening region covering most of the transcribed region. In particular, the 5' region is located >5kb away from the 5' end of the functional transcript; *Ac* insertions in this region affect phenotype (Moreno et al., Genetics 131:939, 1991), indicating that it carries determinants of gene function.

To test if methylation correlated with phenotype, leaf DNA from 38 plants of #6601, 40 of #6671, 24 of #6672 and #6673, 14 of #6701 and 10 of #6702 was analyzed on Southern blots probed with probe 15 (family numbers are defined in Fig. 1). A methylation-insensitive restriction enzyme was used to identify *P* alleles, and *Sal*I was used to determine their methylation status. Among these 126 samples, 90 carried a *P-rr*-like gene as identified by the first enzyme. All 90 showed at least one of the higher MW *Sal*I bands corresponding to hypermethylation. No methylation changes were detected for *P-ww*. All but one of the 90 had patterned pericarp. The ear from the exceptional plant had fully red pericarp. This was the least hypermethylated sample, with

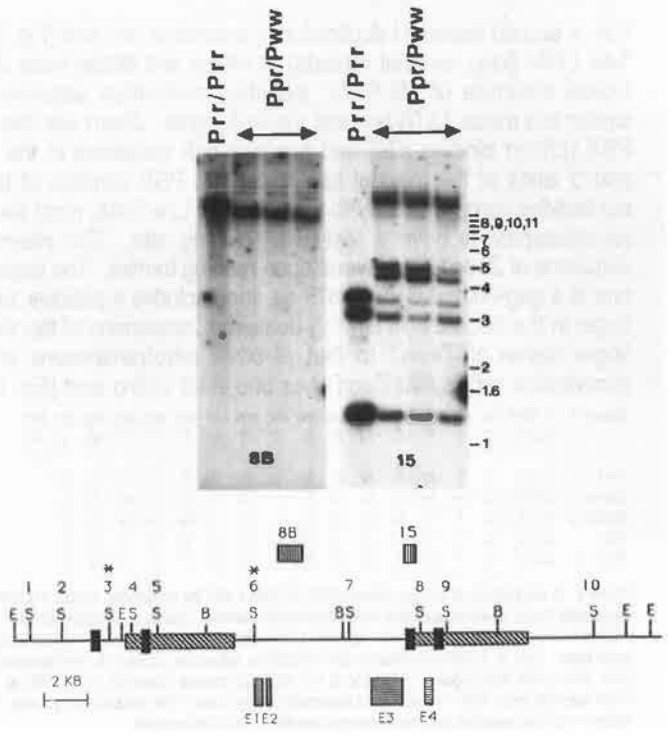


Figure 2. Hypermethylation in *P-pr*: A restriction map of the *P-rr* gene is shown. A large direct repeat at the locus is represented by the cross hatched box. Exons E1, 2 and 3 (horizontally striped boxes) make up the functional transcript, and an alternate transcript arises from E1+2+4. The probes used in the Southern analysis are shown above the map. Probe 15 detects the four repeats specific to the locus represented by the black box, and probe 8B detects the large intron. *Sal*I sites (S) are numbered; E = *Eco*RI, B = *Bam*HI. Leaf DNA from a *P-rr*/*P-rr* plant is compared to leaf DNA from three sibling *P-pr*/*P-ww* progeny after digestion with *Sal*. No differences are seen with 8B, controlling for partial digestion, but probe 15 detects a number of higher MW bands. Three bands are detected for *P-rr*, of 1.2kb (two comigrating bands), 3.0kb and 3.4kb. In *P-pr*, the higher MW bands can be classed into partial hypermethylation (two bands of MW 4.2 and 4.6 from methylation of sites 4 and 8) and full methylation (two unresolved bands of >10kb from methylation of sites 1, 2 and 4, and cutting at 5 and a site to the left that is not shown, or methylation of 8 and 9, and cutting at 7 and 10). Sites 3 and 6 are methylated in *P-rr*, and remain so in *P-pr*. Sites 5 and 7 are unchanged in methylation. With seven other enzymes, including *Hae*III, *Hpa*II, *Msp*I, *Eco*RII and *Pst*I, probe 15 gave higher MW bands, while probe 8B showed no differences These results indicate that the regions around the four filled boxes are hypermethylated at both CpG and CpXpG in *P-pr*. However, the intervening regions comprising much of the transcription unit appear to be unchanged in methylation.

only one of the two higher MW *Sal*I bands representing partial methylation being present. This plant was open pollinated in a greenhouse where no *P-rr* plants were present. Of five progeny plants from this ear, one gave a fully red ear, consistent with the possibility that this was a stable revertant to *P-rr*. Analysis of the second generation of progeny is under way.

In summary, the *P* gene in *P-pr* appears to be hypermethylated, and hypermethylation is linked to its patterned phenotype. In a discussion of the role of DNA methylation in epigenetic phenomena, Holliday defined epimutation as a mitotically heritable change in the methylation of a gene (Holliday, Science 238:163, 1987). Forward or reverse epimutations were defined as an increase or decrease in methylation, respectively. Based on this definition, the origin of *P-pr* may represent a forward epimutation. Furthermore, this example suggests that a change in the methylation of a gene can be heritable through meiosis, and can contribute to allelic diversity.

Developmental changes in the methylation of *P-pr*

--O. Prem Das and Joachim Messing

A new allele of the *P* locus, *P-pr*, has been isolated that is associated with a change in methylation of the *P* gene (preceding note, this issue). Molecular analysis of leaf DNA from 126 progeny segregating for *P-pr* and *P-ww* had identified 90 individuals carrying *P-pr*. These 90 samples displayed a wide range in the relative intensities of the higher MW bands corresponding to full, partial and basal methylation defined by *Sal*I digests. Basal methylation (represented by bands of 1.2, 3 and 3.4kb) is equivalent to *P-rr*, partial methylation (bands of 4.2 and 4.6kb) arises from methylation of two *Sal*I sites in cognate positions in the two pairs of repeats detected by probe 15, and full methylation gives two bands of >10kb (see Fig. 2 of previous note for details). Variation ranged from predominantly full methylation (low levels of partial, and undetectable basal bands) to predominantly basal (low levels of partial, and undetectable full methylation). However, all leaf DNA samples were chimeric, i.e. contained more than one set of bands.

Chimeric methylation in a tissue may result either from an increase or a decrease in methylation during development. Immature embryos and endosperms showed predominantly full methylation, and the bands corresponding to basal and partial methylation showed a modest increase with kernel development. Mature embryos and endosperms showed a further increase. This suggested a decrease rather than an increase in methylation with development. All tissues of mature plants, including brace roots, cob, culm, husks, pericarp, silks, tassel stems and tassel glumes, were chimeric for methylation. Data from sections of leaf and silk tissue were also consistent with a decrease in methylation during development. Silks and brace roots showed more methylation than tissues of earlier origin, suggesting that methylation did not decrease uniformly with time or with ongoing cell division. These changes may resemble the developmental changes in the activity and methylation of *Spm* (e.g. Banks et al., Genes Dev. 2:1364, 1988; Fedoroff and Banks, Genetics 120:559, 1988) and *Mu* (e.g. Chandler and Walbot, PNAS 83:1767, 1986; Martienssen et al., Genes Dev. 4:331, 1990). However, we detect a decrease in methylation, rather than the increase seen with transposons.

Animal and plant genes whose activity correlates with methylation are usually demethylated in expressing tissue. In contrast, demethylation in *P-pr* occurs in all tissues, despite a phenotype confined to floral tissues and pericarp. Demethylation in leaf DNA (ratio of partial and basal methylation to full methylation) correlated qualitatively with pigmentation in pericarp. Furthermore, DNA from heavily pigmented pericarp of mature kernels was less methylated than from lightly pigmented kernels. This was true in comparisons among ears, and even between sectors differing in pigmentation on a single ear. These correlations suggest a role for demethylation in the patterned phenotype of *P-pr*. Preliminary data show reduced P transcripts in pericarps of plants carrying *P-pr* compared to *P-rr*.

The origin and properties of *P-pr* can be accounted for by the following model. *P-pr* may have originated in a somatic event which changed the methylation state of the *P-rr* gene. This state was transmitted through meiosis at least twice, in the two inceptions of this allele. The properties of this allele are such that after fertilization, it is gradually converted to a demethylated state through development. This occurs in both endosperm and plant tissues; in the latter, demethylation appears to be controlled independently in individual organs. Expression in clones of pericarp cells carrying a demethylated gene and not in those carrying a methylated gene may account for the variegated phenotype. Demethylation may occur after the separation of gamete precursors from other cells, enabling normal transmission of the methylated state.

Interactions of *P-pr* with other *P* alleles

--O. Prem Das and Joachim Messing

Since *P-pr* is a hypermethylated derivative of *P-rr*, it was of interest to determine the phenotype of heterozygotes between the two. Therefore, *P-pr*/*P-ww* plants of the family #6601 (Fig. 1, preceding note) were crossed reciprocally to plants homozygous for *P-rr-4026* (the parent of *P-pr*) in the W22 inbred background. Molecular analysis was used to distinguish *P-rr*/*P-pr* from *P-rr*/*P-ww* progeny plants in a small-scale greenhouse planting. All *P-rr*/*P-ww* plants showed full red pigmentation in pericarp, as expected from the dominant *P-rr* allele. Surprisingly, *P-pr*/*P-rr* plants showed variable patterned pigmentation in pericarp. Larger plantings of families from reciprocal crosses of families 6601, 6671, 6672 and 6673 (Fig. 1, preceding note) to *P-rr-4026* gave similar results. When *P-ww* homozygotes from these families were used in such crosses, all progeny displayed uniform red pericarp color. Therefore, effects caused by the genetic background or by the *P-ww* allele were unlikely. In contrast, when plants carrying *P-pr* were crossed to *P-rr*, pericarp pigmentation was highly variable, ranging from fully red to color only at the silk attachment point. Striking patterns, and sectors with differing patterns, both of which are typical of *P-pr*, were also seen. Qualitatively, pigmentation was heavier than in *P-pr*/*P-ww* plants, and in *P-pr*/*P-wr* plants from crosses to W22.

These observations suggest that *P-pr* suppresses the normal expression of *P-rr*. The degree of suppression varies between progeny, and in sectors of an ear. In addition to *P-rr*, *P-pr* also suppresses pericarp pigmentation conditioned by three other *P* alleles, *P-cw* (white-cap pericarp, colorless cob), *P-or* (orange pericarp, red cob) and *P-ow*. For all three, suppression was variable as with *P-rr*, and control crosses to *P-ww* siblings showed no reduction or variation in pigmentation. However, crosses to inbred lines carrying the *P-wr* allele (W22, Mo17, B73 and BSSS53) show the typical variation in pericarp pigmentation expected from *P-pr*, and no reduction or variation in cob pigmentation. These

properties of *P-pr* may resemble the phenomenon termed co-suppression observed for transgenes in tobacco and petunia (Matzke et al., EMBO J. 8:643, 1989; Napoli et al., Plant Cell 2:279, 1990; and van der Krol et al., Plant Cell 2:291, 1991). As in co-suppression, *P-pr* may suppress itself (note the lower pigmentation in *P-pr*/*P-pr* vs. *P-pr*/*P-ww*, preceding note), and related genes. Of particular interest is the possibility of a heritable change in *P-rr* as a result of interaction with *P-pr*. This can be determined either by changes in phenotype or by changes in DNA methylation in outcrossed progeny from a heterozygote.

PUSA, BIHAR, INDIA
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Antiquity of maize in India

--M. Kumar and J. K. S. Sachan

Post-Columbian introduction of maize into India by the Portuguese in the 16th century or later has been accepted by most of the maize workers. However, the peculiar features of maize being grown in remote northeastern Himalayan tracts adjoining Burma and Tibet have stimulated an interesting discussion among maize workers on the possible pre-Columbian introduction of maize in these hilly tracts of the Himalayas. This curiosity has led to extensive work on various aspects of the NEH maize. Some observations on ethno-botany (Stonor and Anderson, 1949; Marszewski, 1968, 1978), plant type (Mukherjee et al., 1971; Singh, 1977, 1989; Sachan and Sarkar, 1982), pachytene analysis (Gupta and Jain, 1971; Dash et al.; 1986, Pande et al., 1988; Kumar and Sachan, 1992), chromosome banding (Mohan and Raut, 1980; Sachan et al., 1982; Pande et al., 1983) and biochemical assays (Pereira et al., 1983) have been reported.

Jeffreys (1965) has suggested that maize had been introduced by the Arabs and not by the Portuguese, in the pre-Columbian era. The Indian names for maize, like Makka jouri (Mecca sorghum), Makka jola (Mecca sorghum), Makkai (grain of Mecca), Mukka Cholam (Mecca sorghum) etc. provide evidence for such a hypothesis. Kuleshov (1928) reported that varieties similar to those described from the Naga tribes are widespread in Central Asia from Persia and Turkestan to Tibet and Siberia. However, Ashraf (1990, personal communication) has discounted such a diffusion of maize in India by the Arabs, and instead cited the mention of maize as "Markataka" in ancient Sanskrit religious texts, 'Vishnu Purana' and 'Apasthamba Saruta Sutra'. Etymology of this terminology and subsequent derivation of the term "Mak" or "Maka" appears to be convincing (Ashraf, 1990). Further depiction of so-called maize "ears" in Indian sculptures in Somnathpur and other Hoysala temples of 12-13 century A.D. as well as some other older Hindu and Buddhist temples has been cited (Johannessen and Parker, 1989) as evidence of pre-Columbian diffusion of maize in India. However, depiction of maize "ears" in Hoysala temples was refuted by Sachan and Payak (Nature, 1989).

Stonor and Anderson's (1949) contention of uniqueness of maize grown by various ethnic groups of erstwhile greater Assam is further supported by the presence of four new knob forming positions at 1Lb, 2Lt, and 9Lb in these NEH strains (Kumar, MNL60, 1992) which are hitherto unknown in maize of the West Hemisphere. It is interesting to note that these knob positions, though absent in maize, are present in Mexican teosinte. Similarly, some new knob positions in two Sikkim Primitive strains, SP1 and SP2, have been identified earlier also (Gupta and Jain, 1971). These knob positions, 7L, 8S, 8L and 10La, were not present in evolved varieties. Hence, it can be concluded that there were two sets of maize introductions in NEH (a) in prehistoric times through a sea/land route much before the discovery of America by Columbus in 1492, and (b) in the post-Columbian era by Christian missionaries, material which essentially resembles Caribbean germplasm. Presence of both low and high knob number groups of maize strains in the NEH region of India further suggests two possible lineages (a) Nal-Tel-Chapalote complex, (b) Confite Morocho and to some extent Palomero Toluqueno.

The pre-Columbian introduction must have taken place through trans-Pacific routes. Otherwise, there would have been traces of this kind of maize along the trade routes during the post-Columbian era. The absence of such traces suggests that pre-Columbian introduction of maize into the Himalayan region might have taken place through routes across southeast Asia and the Pacific islands (Sachan et al., 1978; Ashraf, 1985, 1987).

Deep involvement of maize in the customs, tradition and economy of tribal people in the centre NEH further supports the prehistoric introduction of maize in these areas (Thapa, 1966). Also two written records, namely Tien, non Pen tS'ao (Chinese) and Vamsavali (Nepalese) support the view that maize was cultivated in the Arunanchal, Bhutan, Sikkim and North Burma in the pre-Columbian time (Marszewski, 1978).

RALEIGH, NORTH CAROLINA
North Carolina State University

The effect of *Rf1* on the methomyl sensitivity of cms-T callus

--Mark E. Williams and C. S. Levings, III

The dominant allele of the *Rf1* gene substantially reduces the expression of URF13, the protein responsible for the T-toxin and methomyl sensitivity of cms-T maize. Based on a root-tip growth assay, Kuehnle and Earle (Theor. Appl. Genet. 78:672, 1989) showed that at certain concentrations of methomyl, root-tips of the *Rf1*-containing version of W64Acms-T were significantly less sensitive. We tested for this effect on callus growth using the isogenic background A188cms-T x T204 with and without a single *Rf1* allele. As shown in Figure 1, the growth of both versions is completely inhibited at 1.3mM methomyl. However, at 0.65mM methomyl, while growth of A188cms-T x T204 *rf1 rf1* is still

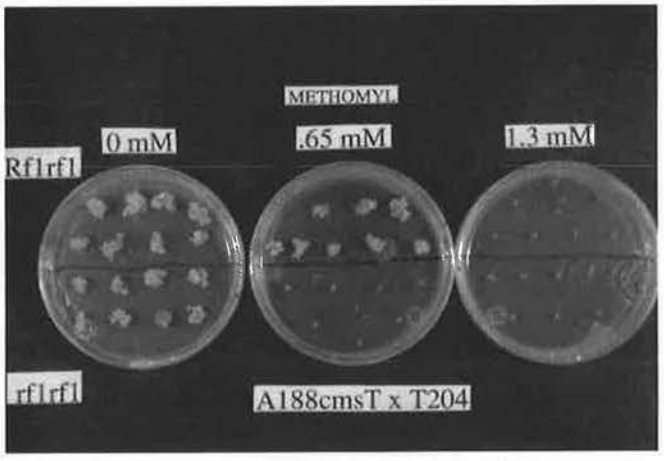

Figure 1. Growth of A188cms-T x T204 +/- *Rf1* on various concentrations of methomyl.

completely inhibited, the growth of A188cms-T x T204 *Rf1 rf1* is substantial (about 75 percent of growth on 0mM methomyl). Normal callus is completely unaffected at either of these methomyl concentrations.

In animal systems, many genes which have a selectable phenotype in cell culture have been cloned by the technique of marker rescue (Watson et al., Recombinant DNA, pp. 180-188, 1983). Since genomic DNA containing a selectable marker has also been shown to transform maize (BMS) protoplasts at a high rate (Antonelli and Stadler, Theor. Appl. Genet. 80:395, 1990), this method could be applied to attempt to clone the *Rf1* locus. The donor line of genomic DNA would be homozygous *Rf1 Rf1* and the recipient protoplasts would be a cms-T line which is *rf1 rf1* followed by selection on 0.65mM methomyl. An ideal recipient line would be BMS; we have backcrossed cms-T into BMS for 4 generations, but have not as yet been able to establish ideal suspension cultures.

RIVERSIDE, CALIFORNIA
University of California

Analysis of synonymous codon usage in maize

--Sheila L. Fennoy, Gita Surti and Julia Bailey-Serres

Synonymous codon usage was previously examined for a small number of maize nuclear genes (Murray et al., Nucl. Acids Res. 17:477-497, 1980; Campbell and Gowri, Plant Physiol. 92:1-11, 1990; Hamilton and Mascarenhas, MNL65:2-3, 1991). We have completed a codon usage analysis of 101 nuclear genes, obtained from GenBank (Release 73, 9/92) and EMBL (Release 32, 9/92) databases and the literature. Codon usage tables were generated with the Genetics Computer Group program CODON FREQUENCY (Devereux et al., Nucl. Acids Res. 12:387-395, 1984). The relative synonymous codon usage (RSCU) of the 101 genes was calculated to show the non-uniformity in synonymous codon usage in maize (Table 1). Codons ending in G and C (GC3) were most frequently used and are indicated in boldface type. The preference for codons ending in G or C reflects the high GC content of the maize genome.

To examine synonymous codon usage among genes, correspondence analysis, a multivariate statistical analysis, was performed on the codon frequency tables of the 101 maize genes (Sharp and Lloyd, Mol. Gen. Genet. 230:288-294, 1991). The analysis produced a two dimensional plot depicting the first and second most influential factors that distinguish the patterns of codon usage of individual genes. The displacement of genes in Dimension 1 reflected the differences among genes in bias for GC3. Genes that plotted above zero, in Dimension 1, had GC3 values ranging from 30 to 70 percent; those below zero had values ranging from 70 to 90 percent. The displacement of genes in Dimension 2 was less extreme. Dimension 2 reflected the differences among genes in use of the set of codons most common to maize (Table 1). In summary, while the genes separated in Dimension 1 based on GC3 content, they separated in Dimension 2 by codon selection.

Genes were grouped by subcellular location, function or condition for induction to discern differences in codon usage that reflected characteristics of expression (Fig. 1). Of the non-zein genes, synonymous codon usage of highly expressed genes encoding structural and photosynthetic proteins was biased towards high GC3 content. The ABA-inducible genes were biased in GC3 and

Table 1. Summary of Codon Usage. Presented are: amino acid, AA; sum of the frequencies of the codons, N; relative synonymous codon usage, RSCU; relative composition of each amino acid in all genes, AC. Bias in codon usage in maize is measured by the RSCU. RSCU mathematically describes the disproportionate use of synonymous codons. The most commonly used synonymous codons are those ending in C and G and are shown in boldface type.

AA	CODON	N	RSCU	AC
Ala	GCU	841	0.92	0.098
	GCC	**1319**	**1.45**	
	GCA	511	0.56	
	GCG	**955**	**1.05**	
Leu	UUA	102	0.19	0.087
	UUG	407	0.75	
	CUU	527	0.97	
	CUC	**993**	**1.83**	
	CUA	220	0.41	
	CUG	**1000**	**1.85**	
Gly	GGU	640	0.83	0.083
	GGC	**1417**	**1.83**	
	GGA	476	0.61	
	GGG	566	0.73	
Val	GUU	512	0.77	0.072
	GUC	**883**	**1.33**	
	GUA	199	0.30	
	GUG	**1062**	**1.60**	
Ser	AGU	182	0.43	0.067
	AGC	**640**	**1.54**	
	UCU	353	0.85	
	UCC	**647**	**1.55**	
	UCA	300	0.72	
	UCG	**376**	**0.90**	
Pro	CCU	459	0.84	0.059
	CCC	**581**	**1.06**	
	CCA	455	0.83	
	CCG	**701**	**1.28**	
Glu	GAA	555	0.49	0.061
	GAG	**1724**	**1.51**	
Arg	CGU	231	0.66	0.056
	CGC	**643**	**1.85**	
	CGA	125	0.36	
	CGG	**314**	**0.90**	
	AGA	207	0.59	
	AGG	**569**	**1.63**	
Thr	ACU	369	0.76	0.052
	ACC	**777**	**1.61**	
	ACA	328	0.68	
	ACG	**459**	**0.95**	
Lys	AAA	370	0.39	0.051
	AAG	**1540**	**1.61**	
Asp	GAU	645	0.68	0.051
	GAC	**1240**	**1.31**	
Ile	AUU	442	0.81	0.044
	AUC	**1013**	**1.86**	
	AUA	181	0.33	
Gln	CAA	465	0.61	0.041
	CAG	**1066**	**1.39**	
Phe	UUU	332	0.50	0.036
	UUC	**1006**	**1.50**	
Asn	AAU	353	0.55	0.034
	AAC	**923**	**1.44**	
Tyr	UAU	236	0.45	0.028
	UAC	**808**	**1.55**	
His	CAU	265	0.63	0.023
	CAC	**572**	**1.37**	
Met	AUG	891	1.00	0.024
Cys	UGU	151	0.49	0.017
	UGC	**470**	**1.51**	
Trp	UGG	420	1.00	0.011
TER	UGA	45	1.35	0.003
	UAA	23	0.69	
	UAG	32	0.96	

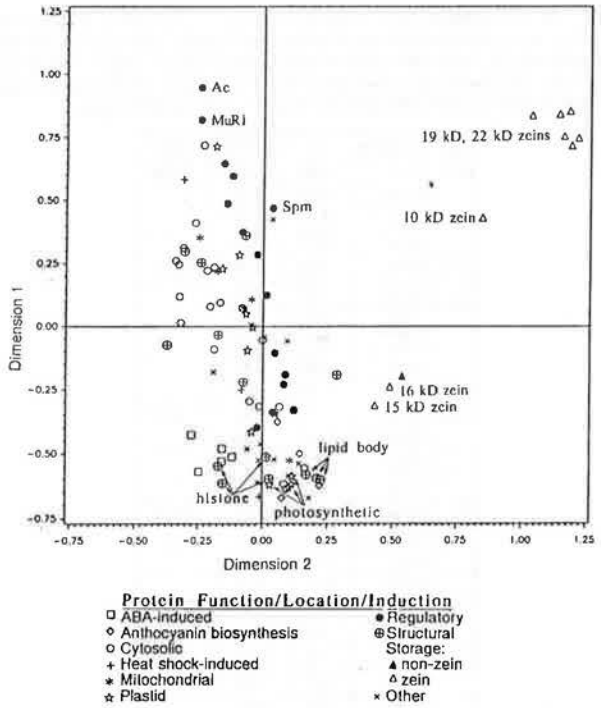

Figure 1. Correspondence Analysis of Codon Usage. This multivariate statistical analysis was done on 101 maize gene sequences to discern differences in codon usage. Coding sequences are identified by subcellular location, function, or induction characteristics of the protein. The two axes, Dimension 1 and 2, depict the first and second most influential factors for dispersion. The origin represents the average codon usage for all genes. The distance between genes on the graph is a measure of their dissimilarity in synonymous codon usage.

codon choice as demonstrated by the displacement in Dimensions 1 and 2 (Fig. 1). The group of regulatory genes included the transcription factors, transposable elements, phosphatases and kinases. These genes were predominantly distinguished from each other by their bias in GC3. Genes encoding transcription factors had higher GC3 values than those encoding kinases and phosphatases. The open reading frames of transposable elements, *MuR1*, *Ac* (ORFa),and *Spm* (TNPa), were GC3 poor and showed an extreme bias in codon usage. Of those genes encoding structural proteins, the histones and lipid-body-associated proteins were most biased in GC3. These genes are most likely highly expressed. The genes encoding cytosolic enzymes had average GC3 content and plotted at negative values in Dimension 2. The storage proteins included zein and non-zein proteins. As noted by others (cf. Hamilton and Mascarenhas, MNL65:2-3, 1991), the genes encoding the 19 and 22kD zeins of endosperm have unusual codon usage. The correspondence analysis clearly demonstrates the relatively low GC3 content and extreme codon usage bias of the 19 and 22kD zeins. In contrast, the 15 and 16kD zeins showed near average GC3 content but distinct codon selection.

Synonymous codon usage reflects the co-adaptation of the population of charged tRNAs and the coding sequence. The degree of non-random codon usage in each gene may reflect its rate of translation and/or the mutational bias of the genome. We plan to test whether codon usage affects the rate of elongation in vivo and in vitro in maize. The analysis may provide important information on translational control mechanisms in plants.

The RSCU and correspondence analyses presented here should prove useful for designing degenerate oligonucleotides for polymerase chain reaction amplification of coding sequences in maize.

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University of Minnesota

Big rings in corn

--Charles R. Burnham

Goal: a multiple interchange stock, which when crossed with normal, gives a ring of 20. The following multiple interchange stocks have been established:

1. 1-5-6-7-8, when crossed with N = a ring of 10.
2. 3-2-4-9-10, when crossed with N = a ring of 10.
3. 5-7-1-9-10, when crossed with N = a ring of 10.
4. 6-3-2-4-8, when crossed with N = a ring of 10.
5. 5-7-1-9-10-8, when crossed with N = a ring of 12.

Stock #1 x #2 = 2 rings of 10. Stock #3 x #4 = 2 rings of 10. Stock #4 x #5 = one ring of 20. I attempted to combine #1 and #2 by crossing #1 x #2 then backcrossing to 1 or 2 and selfing the progeny. Extensive tests were made but no success.

Another approach, using the Mangelsdorf multiple marker stock with one marker in each chromosome from 1-10:*bm2*, *lg1*, *a1*, *su*, *pr*, *Y* or *y*, *gl1*, *j1*, *wx*, *g1*, has not been tried.

Cross 1-5-6-7-8 x multiple marker, backcross to 1-5-6-7-8. Self the fertiles, select those segregating for the markers only in chromosomes 3, 2, 4, 9, 10, the chromosomes in the other ring: 3-2-4-9-10. Select those plants homozygous for those markers.

Cross the 3-2-4-9-10 stock with the multiple marker stock, backcross to 3-2-4-9-10. Self the fertiles, select ones segregating for markers only in 1, 5, 6, 7, and 8, the chromosomes in the other ring, 1-5-6-7-8. Select those plants homozygous for these markers.

Cross the two stocks: 1-5-6-7-8 *a*, *lg*, *su*, *wx*, *g1* with 3-2-4-9-10 *bm1*, *pr*, (*Y* or *y*), *gl1*, *j1*. The F1s have 2 rings of 10, are more than 95% sterile, and do not shed pollen.

The ideal situation would be for several of the markers in the normal chromosomes in one ring to be closely linked with the interchange break points in the interchange chromosomes in the other ring. This is an application of the scheme used by Inman to construct rings with more chromosomes.

The F1 between the two stocks will have two rings of 10 and the normal chromosomes in each ring will have a genetic marker. Use the F1s for colchicine treatment to study what happens to produce the plants that are about semisterile rather than 95% or more sterile. See Helmy Ghobrial's reports in earlier newsletters. Germinating seedlings were watered with a colchicine solution and transplanted to the field. Occasional plants shed pollen. All branches of the plant shed pollen. Also on the few such plants that had tillers all branches also shed pollen. Clues to the explanation should be provided by the genetic markers.

Would the F1 between the two stocks backcrossed to one parent without the markers, followed by selfs, be a method of combining 1-5-6-7-8 with 3-2-4-9-10?

A multiple-use inbred, A188

--Charles R. Burnham

The various chromosomal interchange stocks in my "Chromosome identification" set and in the "All-Arms marker" set were in different backgrounds. They were converted to inbred A188 by successive backcrosses. It is a sturdy plant with dark green leaves, a good ear with a strong tendency to be two-eared. The second ear often develops if the top ear is removed. When ears are bagged but not pollinated, one or two side ears develop from

the shank at the base of the ear on some of the plants. If they are under the ear bag, I have been able to pollinate them many days later. Break off the early unpollinated ear. This is the behavior here at Minnesota.

The inbred also was used successfully by Ed Green to generate plants from tissue culture. It is not susceptible to smut. Merle Jenkins said it is susceptible to many other corn diseases. All plants are *r-g r-g*, some plants are *C1 C1*, some *c1 c1*. It has the dominant white cap factor which dilutes *Y Y* endosperms.

Origin: The A188 inbred came from: (4-29 x 64) $4\text{-}29_4$, i.e., (4-29 x 64) backcrossed to 4-29 four times.

4-29 = Extraction out of Silver King

64(A48) = Extraction out of No. West Red Dent.

Balanced lethals for chromosome 6

--Charles R. Burnham

Several luteus (*l*) and albino (*w*) genes closely linked to *y* are carried in the Coop as *Y +/y l* and *Y +/y w*.

If normal progeny from the yellow seeds of one stock are crossed with normal progeny from the yellow seeds of the other stock, crosses between *Y y* heterozygotes in both will give ears segregating for *Y* and *y*. The *y* seeds will be *y l +/y + w*, a balanced lethal. Progeny from crosses between them and a normal will be heterozygous for either *l* or *w*, with few exceptions.

To establish the crossover *Y + +/y l10 w15*, one such balanced lethal was crossed with a *Y Y* stock. The plants were selfed, the *Y* and *y* seeds on each ear were planted. Most segregated 3 green: 1 yellow or 3 green: 1 white. The desired crossover segregated green:white plus a few yellow seedlings. The combination, *l w*, is a white seedling, but a crossover that separates them is a yellow seedling. To identify these ears, the linkage was so close that 75 or more seeds per ear were tested. If the ancestry of such a stock were not known, the rare yellow seedlings would be difficult to explain.

Pale yellow endosperm color genes

--Charles R. Burnham

Some yellow endosperm cultivars have a pale yellow gene also. This can be tested by crossing with a pure white endosperm stock (*y y*) and selfing the F1s. At one time I had two pale yellow stocks, which, when crossed, gave deep yellow seeds. In F2 the ratio was 9 deep yellow: 6 pale: 1 white. I do not have those stocks. There were reports in the Newsletter on genes for yellow and pale yellow endosperm. If those stocks are in the Coop they might be tested in crosses for the above behavior. One pale yellow was associated with viviparous (*vp*) seeds. A cross on *y y* showed the heterozygous *vp* plants were *Y Y*.

Seedling survival after desiccation

--Charles R. Burnham

When I was at West Virginia University, classification for certain virescents was difficult in the field because temperatures were too high. If started in the greenhouse in sand, separation was easy. Leaves were cut back, seedlings removed from the sand, virescents were separated from the green seedlings, then transplanted to the field. They were watered and a mulch of dry soil was added on top. Without the mulch, the soil dries out and watering every day is needed.

I was transplanting late one evening and I left when it was dark. I went out the next evening, after a hot day, and found that some 20 feet or so of seedlings were still uncovered. I decided to put them in anyway, watered them well and added the dry soil mulch. I was surprised when I took notes later in the summer. There was no gap in the planting. The seedlings had survived even after more than 20 hours of drying.

I finally tested it here at Minnesota in the greenhouse. Old seed samples were being started in sand. Often when the seed ears were old, a few seeds on a few ears of the same stock would germinate in sand and were then transplanted.

One ear culture had a lot of extra plants. I clipped the leaves, removed them from the sand and left them lying on top of the dry sand in another bench. It was a hot day and after various intervals of time, the plants were transplanted in sand and watered. Seedlings recovered after many hours of drying. Dr. Dale Hicks took pictures, the data are on the slides.

How did the dried-up roots recover? I should have dug them up after recovery.

Viability of pollen

--Charles R. Burnham

I remember E. M. East telling me in 1930 that a rainstorm came up before he had made pollinations with all the bags of pollen he had collected. He threw them in a shed. The next day was clear and he decided to use the bags of pollen he had collected the previous day. He was surprised by the success of the pollinations.

A few years ago, I was test-crossing a lot of plants from selfs of cultures from a stock which, although heterozygous for *ms1*, did not segregate male steriles in the self progeny. I ran out of ears on *ms* plants and left the bags in the field. The next day more ears were ready and I used those bags of pollen. Again, there were well-filled ears.

Is it possible that the occasional contaminations are from pollen grains from other plants lodged on the tassel at the time of bagging, even though few and greatly outnumbered?

Notes on pollination, ear and tassel bagging, etc.

--Charles R. Burnham

Ear bagging. First cut off the husks that extend beyond the ear tip. Often the ear bag can be pushed far down over the ear if the seam side of the bag is next to the stalk. Or the flat ear bag can be used to cut the sheath of the ear next to the stalk. Do not use the knife. The ear tissue next to the stalk is very tender.

If the top ear is to be removed, make a short cut across the sheath near the base of the ear, at right angles to the stalk. Break the ear out sideways. This preserves most of the sheath to prevent breakage of the stalk. Make certain that the entire ear is removed.

Tassel bagging. If the tassel is near the end of pollen shedding, check for full florets at the base of the tassel branches. These are likely to still shed pollen. This avoids bagging of plants that have finished shedding pollen.

Pollination. If there is very little pollen, most of it may be held between the outer and inner folds at the bottom of the bag. Open the bag so that the pollen held there is free to flow.

If many pollinations are to be made with the same tassel bag of pollen fold bag over on itself endwise. Press the edges of the fold tightly together. Pour the pollen plus anthers into the open end. Tap the bag and the pollen will pass into the bottom of the bag.

Tear off the top part with the anthers.

Pollen from tassels that are shedding pollen but broken off and lying on the ground, with or without the tassel bag can be used for full seed sets. The stalk can be pushed into the wet ground or placed in a jar of water. Some pollen can be shaken out of the tassel every few hours after the peak shedding. Another peak is late in the day.

If the ear bag has been on for some time there may be a large mass of silks inside the ear bag. At one time, the procedure was to go through and cut the silks back on ears to be pollinated the next day. This is not necessary. At the time of pollination the ear bag can be pinched and twisted to break off the silks well above the tip of the ear. Or the ear bag can be pulled up part way and the pollen poured in at the tip of the ear. The ear bag is then pushed back over the ear and covered with the tassel bag. If grasshoppers are a problem, eating holes in the tassel bag, the ear is still protected by the ear bag.

Labeling. A manila tag, 1 1/2" x 2 1/2", is stapled on the inner fold of the tassel bag some 3 or 4 inches from the bottom end of the bag. These are prepared during the winter months. At the time of pollination, the pollen source is written on the lower half of the tag plus the date. The male plant and culture number can be recorded plus the traits of the female and male parent. My own practice was to stamp on the female culture number when the pollinations were complete.

At harvest, the pollinated ear is husked with a husking pin, and the pollination tag is nailed on the ear with a thin lath nail which has a larger head. I used a small, smooth stone to pound in the nails. This is preferable to rubber bands which disintegrate within a year. The pollinated ears from one year were not shelled until the results from the next year's plantings were obtained. Separations for aleurone color, yellow vs. pale yellow and white endosperms are easier when the kernels are still on the ear. Remove one by one.

Many of the above ideas were the result of suggestions from my students.

Preliminary mapping and biochemical analysis of *opaque8*

--G. I. Graham, J. Suresh and R. L. Phillips

The opaque endosperm mutations in maize have been valuable for elucidating the control of kernel storage proteins, as well as providing sources of improved nutritional quality. Here we describe another opaque mutant named *opaque8*. Seed for this study was provided by Dr. O. E. Nelson, Jr. in an unknown inbred background. The phenotype is controlled by a single recessive gene; segregation on a B73 x *o8* F2 ear was 174 normal:57 opaque. RFLP mapping based on 20 F2 opaque individuals indicates the gene is 2.5 map units away from the sequence detected by *umc134* on chromosome 2. *Opaque8* appears to be between *umc131* (12.5 map units) and *bnl12.09* (7.5 map units). The order of the 3 marker loci is:

bnl12.09----------*umc134*----------*umc131*
5 10

The exact location of *o8* in reference to *umc134* has not yet been determined. This mutation, like other opaques, appears to increase lysine. Because the background was unknown, we analyzed F2 kernels of crosses of *o8* and B73, A619, and A188. Kernels were separated based on phenotype (opaque vs. normal), pooled, and ground together. Preliminary evidence indicates that the *o8* sample had higher lysine levels by 10.8, 11.7, and 13.0% for the B73, A619, and A188 backgrounds, respectively. Zein protein levels did not appear to be different for either the B73 or A619 backgrounds. However, in the A188 background, the *o8* zein-2 protein fraction was significantly lower than the normal counterpart. Isoelectric focusing (IEF) and SDS-PAGE showed no particular zein proteins to be absent.

The effect of benzyladenine and thidiazuron on endosperm development and DNA endoreduplication

--L. Schweizer, G. L. Yerk, R.L. Phillips, R. L. Jones and F. Srienc

Cytokinins have been associated with mitotic cell division. Cytokinin levels increase greatly after fertilization and during the periods when there is rapid seed and fruit growth (Bohner and Bangerth, Physiol. Plant. 72:316-320, 1988; Carnes and Wright, Plant Sci. 57:195-203, 1988; Schrieber et al., Plant Physiol., in press, 1993). In maize kernels, the decrease in mitotic activity coincides with the decrease in cytokinin levels (Kowles and Phillips, Int. Rev. Cytol. 112:97-136, 1988; Schrieber et al., Plant Physiol., in press, 1993). Following the reduction in mitotic activity, there is an increase in DNA content per nucleus. This increased DNA content per nucleus has been identified in all inbreds and hybrids examined (Kowles and Phillips, PNAS USA 82:7010-7014, 1985). Endoreduplication of the nuclear DNA is responsible for the observed increases in DNA content per nucleus. These data indicate that there may be a tightly coupled developmental regulation between cell division, DNA endoreduplication, and cytokinin levels in developing kernels.

The purpose of this study was to determine the relationship between cytokinin levels and mitosis and DNA endoreduplication during kernel development. Benzyladenine, an adenine derivative, and thidiazuron (N-phenyl-N'-1,2,3-thiodiazol-5-ylurea), a urea derivative with cytokinin activity were exogenously applied to maize inbred line W64a. This line was chosen for its small seed size, thus alterations in cell number would be more easily detected.

W64a was planted in 1990 and 1991 at St. Paul. All ears were bulk pollinated with W64a pollen on the same day. Each year, four replicates in a randomized complete block, containing eight treatments of exogenously applied cytokinins, were evaluated. The eight treatments were control (not sprayed), control spray (sprayed with dimethylsulfonate (DMSO), a solvent used to solubilize the benzyladenine and thidiazuron), three concentrations of benyzladenine: 10^{-3}M, 10^{-4}M, and 10^{-5}M and three concentrations of thidiazuron: 10^{-3}M, 10^{-4}M, 10^{-5}M. Spray treatments were applied on the leaf subtending the ear until run-off occurred each day from 6 to 10 days after pollination (DAP).

Kernel samples were collected from the middle third of the ear each day from five to twenty-two DAP. Samples were fixed in 3:1 (95% ethanol: glacial acetic acid) overnight. The following day, the samples were transferred to 70% ethanol and stored at -20 C. Squashes of endosperm tissue from the control were made with propionic carmine. At least 1000 cells per endosperm were scored for mitotic activity for each day from 6 to 22 DAP. Mitotic index was calculated as the percentage of mitotically active nuclei among the total nuclei.

Nuclei preparations were made according to the method of Kowles et al. (The Maize Handbook, M. Freeling and V. Walbot, eds., 1993). These preparations were stained with Mithramycin A and analyzed by flow cytometry to determine the DNA content per

nucleus and the total cell number per endosperm. In 1990, four replicates per treatment were measured, and in 1991 three replicates per treatment were analyzed. In both years treatments were evaluated each day from 8 to 18 DAP. The following parameters were measured on a per endosperm basis: average DNA content per nucleus, total nuclei number, number of 3C, 6C, 12C, 24C, 48C, and 96C nuclei.

Analysis of variance was performed on the data using a split-split plot design with the treatments being the whole plot factor and heat units being the sub-plot factor. Heat units were utilized rather than days after pollination to standardize the stage of kernel development across environments. The data for the two years were analyzed separately due to heterogeneity of error variances.

Mitotic index peaks at 8 DAP and then declines rapidly. The change in total nuclei number per endosperm over cumulative heat units followed the same pattern in 1990 and 1991, however, the same data taken as a function of days after pollination plateaus at different time points, indicating that the use of heat units to remove environmental effects is appropriate. No significant variation was observed among the treatments for average DNA content per nucleus and 3C nuclei number per endosperm. Significant variation was seen for total nuclei number, and the number of 6C, 12C, 24C, 48C, and 96C nuclei per endosperm. The lowest concentration of thidiazuron increased total cell number as did the benzyladenine treatments. Conversely, the highest concentration of thidiazuron reduced total cell number. These results indicate that cytokinins have an effect on both mitosis, as measured by total cell number per endosperm, and DNA endoreduplication, as measured by the number of 12C, 24C, 48C, and 96C nuclei. The mechanism by which the observed changes in DNA content and cell number occur is unknown. Benzyladenine has been implicated in transcriptional control of gene expression. Although a number of mechanisms have been proposed for the action of thidiazuron on mitotic tissues, a clear model is still unavailable.

Based on these findings, measurement of endogenous cytokinin levels in kernels treated with benzyladenine and thidiazuron at different developmental times is warranted. In particular, measurement of endogenous levels of purine cytokinins and ribonucleoside and ribonucleotide levels which are altered during metabolism of cytokinins, might aid in elucidating the mechanisms by which these compounds act.

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Allele-specific degradation in endosperm development

--G. L. Yerk, R. L. Phillips and R. V. Kowles

Endosperm tissue is a product of the union of one sperm nucleus with two polar nuclei, forming a triploid tissue. Following a period of free nuclear division, during which the endosperm is a syncitial tissue, cell walls are laid down giving rise to a cellularized, uninucleate tissue. Thereafter, endosperm growth proceeds by two processes--increases in cell number and increases in cell size. At 8 to 10 days after pollination (DAP), there is a peak in mitotic index defined as the percentage of mitotic cells from the total endosperm cell population. After this period, mitotic activity declines rapidly, to almost zero. At the same time, DNA content per nucleus begins to increase dramatically reaching peak levels between 16 and 18 DAP (Kowles and Phillips, PNAS USA 82:7010-7014, 1985). The increase in DNA content per nucleus is the result of polytenization of the chromosomes (Kowles et al., Dev. Genet. 11:125-132, 1990). Subsequent to the peak in DNA content per nucleus, a reduction in DNA content per nucleus is observed (Kowles and Phillips, 1985). In the case of inbred A188, the reduction in DNA content per nucleus is approximately one third of the maximum DNA content per nucleus.

The purpose of this study was to further characterize the reduction in DNA content per endosperm nucleus observed in endosperm nuclei sampled after 18 DAP. Furthermore, because the reduction in DNA content per nucleus for A188 was approximately one third, experiments were constructed to test whether preferential degradation of the paternal genome had occurred.

Paired-plant reciprocal crosses were made for B73 x Mo17 and A188 x B73 in the summers of 1990 and 1991. Several individuals within each inbred were also self-pollinated. All pollinations for comparison were made on the same day. Endosperm tissue was removed from kernels and frozen for DNA extraction at a time period estimated to correspond to the peak in DNA content (early) and one estimated to correspond to the time period after which degradation had begun using the CTAB method. Endosperm DNA was digested with *Hin*dIII or *Eco*RI. Digested DNA was electrophoresed in 0.8% agarose and transferred to Immobilon N. Blots were probed using clones from a *Pst*I genomic library from the University of Missouri-Columbia (umc) and a 9kb fragment containing the ribosomal genes. For situations where neither allele was absent, autoradiograms were scanned using Apple scan and ratios of band intensity within a lane were determined using Image 1.44 (NIH) to determine whether degradation of one allele was occurring.

In 1990, the maternal allele was consistently and completely lost in the B73 x Mo17 hybrid at the late sampling date but was present in the reciprocal hybrid, Mo17 x B73, at the late sampling date and in both reciprocal hybrids at the early date. This was true for all six of the maize chromosomes tested. The other four chromosomes were not evaluated due to a limited amount of endosperm DNA. Multiple probing of two of the chromosomes demonstrated that the pattern of allele loss is the same for both arms of the chromosome, as well as multiple sequences on the same arm of a chromosome. Probing with pZMR1, the 9kb ribosomal DNA repeat, demonstrated the same pattern of allele loss as seen with the UMC clones. This repeat unit contains the 18S, 5.8S, and 26S ribosomal DNA sequences.

In 1991, allele loss was not as complete. Some probes showed reduced banding intensity while others were completely absent. In addition, the allele being degraded came from B73 in some cases and from Mo17 in other cases. For example, hybridization with *umc15* and *umc131*, both of which demonstrated complete allele loss in 1990, indicated that the restriction fragment from the Mo17 parent was reduced in intensity. In the case of *umc131*, the reduction was 50%, while for *umc15* the reduction was 42%. The results with *umc4* showed a different pattern of allele loss. In this material a reduction in banding intensity for both the Mo17 and B73 restriction fragments mapping to chromosome 2 were observed in the B73 x Mo17 hybrid at the early sampling date. At the later sampling date, the same bands were absent in both hybrids. Additional restriction fragments located on chromosome 8 were present in both hybrids at both the early and late sampling times. Probing of the same material with *umc39*, located adjacent

to *umc4*, indicated that the restriction fragment from Mo17 was reduced in intensity compared to the B73 restriction fragment.

The observed pattern of allele loss was more complex for the A188 x B73 materials in 1990 compared to the Mo17 x B73 materials. Alleles were either present in both reciprocals at both the early and late sampling dates or they were completely absent in all cases. The missing allele came from A188 in some cases and from B73 in other cases. Once again, six of the ten chromosomes were evaluated. In addition, the pattern of allele loss varied within a chromosome. Thus, alleles from different parents were lost at different locations on the same chromosome.

In 1991, both reductions in intensity and complete absence of alleles were observed in the A188 x B73 materials. Again, the alleles involved came from A188 in some cases and B73 in others. If an allele was absent, it was missing in both reciprocals at both sampling dates.

Several conclusions have been drawn from these experiments. First, allele specific degradation is occurring in both sets of material examined. In 1990, the degradation was always complete. In 1991, degradation was partial in some cases and complete in others. This difference may be due to different environmental conditions in the two growing seasons. The rate of endosperm development as measured by DNA content and cell number per endosperm is extremely dependent on heat units (Schweizer et al., PNAS submitted). Second, the pattern of allele loss was not necessarily the same from one season to the next. In some instances, a probe which was completely absent in one season showed only partial reduction in the other season. In other instances, probes showing either complete or partial degradation in one season demonstrated no degradation in the other season. Third, the pattern of allele specific degradation is not universal from one pair of lines to another. The number of patterns that may exist cannot be estimated due to the small number of combinations examined. Fourth, the patterns observed within a pair of lines for a given probe are consistent from one plant to another. Finally, the loss of a particular allele cannot be attributed to artifacts from self-pollination as the maternal allele is often lost.

A number of possibilities exist which might explain the observed patterns of allele loss. These include sequence specific recognition at these sites, protein marking of these sites, conformational differences at these sites, and the possibility that these may be active transcriptional sites. The overall purpose for the degradation of these DNA sequences may be to provide nucleotides which can be stored and used by the developing and germinating embryo. Experiments are in progress to test a number of these hypotheses.

Characterization of defective kernel mutants (deks) in four genetic backgrounds by flow cytometry

--G. L. Yerk, R. V. Kowles and R. L. Phillips

Endoreduplication of the nuclear DNA is a prominent feature of maize endosperm development. Following the peak mitotic period, at 8 to 10 days after pollination (DAP), an increase in DNA content per nucleus is observed. This increase is due to polytenization of the chromosomes caused by endoreduplication of the DNA. During the endoreduplication process, two different cell cycles are occurring in the endosperm simultaneously, the mitotic cell cycle and the endoreduplicating cell cycle. The mitotic cell cycle consists of G_1, S, G_2, and M-phases. The endoreduplicating cell cycle consists only of alternating S and G-phases; thus, DNA synthesis occurs but there is no chromosome condensation, strand separation, karyokinesis nor cytokineses. The endosperm may be divided into three regions on the basis of these cycles. In the central region of the endosperm at 10 to 12 DAP, the cells begin to undergo the endoreduplicating cell cycle. These cells are not synchronized in their position in the cell cycle. Once a nucleus has undergone the endoreduplication process, it does not become mitotic again. Thus cells which are 48C can be found near to other cells which are 12C or 96C. The second region occurs around the periphery of the endosperm. Here the conventional mitotic cell cycle is the rule. Although the mitotic index peaks between 8 and 10 DAP, cells in this peripheral region continue to be mitotic once endoreduplication has begun in the cells of the central region of the endosperm. The third region is a transitional region located between the first two regions. It contains both mitotic and endoreduplicating cells.

Using flow cytometry, it is possible to monitor both the mitotic and endoreduplicating cell cycles at the same time. Mitotic cells are represented by nuclei with 3C to 6C DNA content where C is the haploid DNA content per nucleus. Endoreduplicating cells comprise the nuclei with DNA content greater than 6C. Among the endoreduplicating nuclei, cells in the G-phase appear as peaks at the 12C, 24C, 48C, 96C, ..., DNA contents, while those in the S-phase appear between these peaks on output from the flow cytometer. By analysis of nuclear preparations from maize endosperm at different DAP, it is possible to follow the progression of the mitotic and endoreduplicating cell cycles. Different inbred lines display characteristic patterns of endoreduplication. These differences may be the percentage of cells in the S- vs. G-phase, as well as in the number of rounds of endoreduplication that they undergo. These differences in pattern of endoreduplication are controlled by a maternal effect, that is, a protein or transcript encoded by the nuclear genome of the female parent which controls a developmental function in the endosperm.

The purpose of this research was to identify a number of mutants in various inbred backgrounds which represent different types of alterations in the endoreduplicating cell cycle in maize endosperm.

Normal seed of seventy-two lines segregating for defective kernel phenotype were planted in the field in 1991. Among the 72 lines, seventeen lines were produced by ethylmethylsulfonate (EMS) mutagenesis of inbred W23, twenty-four lines were derived by EMS mutagenesis of inbred A188, fourteen lines were generated by EMS mutagenesis of a hybrid, and seventeen lines were induced by transposition of *Mu*. Seed of the EMS mutagenized hybrid was provided by Dr. G. Neuffer, University of Missouri, Columbia. Seed of the *Mu*-induced lines was provided by Dr. Martha James, Iowa State University. Other lines were available from previous EMS experiments by Dr. R. L. Phillips. In all of the mutant lines used, either one or two loci were responsible for the defective kernel phenotype.

Several plants within a line were self-pollinated. Kernel samples for W23 lines and *Mu* lines were collected at 18 DAP. Those for the A188 lines and the hybrid lines were collected at 16 DAP. Both defective and normal kernels from a segregating ear were removed using a scalpel and placed in 3:1 (95% ethanol: glacial acetic acid). The following day, the samples were placed in 70% ethanol and stored at -20 C. Pairs of nuclear preparations of normal and defective kernels from a segregating ear were made according to the method of Kowles and Phillips (Dev. Genet.

11:125-132, 1990). Nuclear preparations of normal kernels were made from a single endosperm. Those of defective kernels were made using one to three endosperms due to the reduced size of the material. Endosperm nuclei from all pairs of normal and defective kernels from one genetic background were prepared and stained the same day. Nuclear preparations of W23, *Mu* and hybrid lines were analyzed using an Ortho Diagnostics flow cytometer. A Becton Dickinson flow cytometer was utilized to evaluate the A188 lines. Mean DNA content per nucleus was determined based on the fluorescence values obtained from analysis of the Mithramycin stained nuclei. Cell counts were made using a hemocytometer. Three to five counts were made from a nuclear preparation. DNA content and cell number data within a pair of normal and defective kernels from the same segregating ear were compared using a standard t-test. Comparisons between lines are invalid due to differences in pollination date.

Ten classes of mutants were identified among the materials studied. They are lines with: 1) lower DNA content per nucleus due to no or almost no endoreduplication and lower cell number in the *dek* vs. the normal endosperm, 2) lower DNA content per nucleus due to fewer rounds of endoreduplication and lower cell number in the *dek* vs. the normal endosperm, 3) lower DNA content having the same number of rounds of endoreduplication but fewer nuclei in the endoreduplicating peaks and lower cell number in the defective endosperm when compared to normal, 4) lower average DNA content per nucleus and equivalent cell number in the *dek* vs. the normal endosperm, 5) decreased DNA content per nucleus and increased cell number in the *dek* compared to the normal endosperm, 6) equivalent DNA content per nucleus and reduced cell number in the *dek* compared to the normal, 7) higher average DNA content per nucleus and higher cell number in defective endosperm compared to normal, 8) equivalent DNA content per nucleus and cell number in the *dek* compared to the normal, 9) increased DNA content per nucleus and reduced cell number in *dek* compared to normal lines, and 10) higher average DNA content per nucleus and equivalent cell number in the *dek* and the normal endosperm. Table 1 lists the number of mutants for each class identified within each background.

Table 1. Frequency of ten types of mutants in DNA content and cell number in four genetic backgrounds.

Class	W23 EMS lines	A188 EMS lines	Hybrid EMS lines	Mutator lines
1	1	4	2	0
2	7	12	3	7
3	0	3	0	2
4	8	1	5	5
5	0	0	0	2
6	1	0	0	0
7	0	1	0	0
8	0	1	0	0
9	0	0	0	1
10	0	0	2	0

These mutants should provide the necessary materials for use in further elucidating the biochemistry, molecular, and cell biology of the endoreduplication process.

SALINAS, CALIFORNIA
Comnuts Inc.

Distribution of perennial maize populations

--D. L. Shaver

We will distribute our perennial maize populations to anyone interested in them. Cornnuts has 3 basic perennial populations, two of them tetraploid, one diploid.

Population I. Originally synthesized at Brookhaven in 1962. Has since been maintained by mass selection for perennialism in isolated plantings. It has periodically been serially backcrossed to 4N maize, the last such backcross was to D. E. Alexander's "4N Syn. B". Present theoretical proportion of maize germplasm is 15/16. The other 1/16 comes from *Zea perennis*, the clone from Randolph he designated "E16515". This population derives from 28 generations of mass selection.

Population II. This second 4N population has the same origins as Population I, except that 5 generations ago, it was outcrossed to a 2N stock having the genes, *gt/gt*, and *id/id*. The resultant triploids were sibbed with the larger pollen grains, separated out by mechanical screening, so as to restore the population to tetraploidy. Since then, it has also been maintained in isolation with mass selection.

Population III. This 2N population was derived by crossing *Zea diploperennis* to maize stocks having *gt/gt* and *id/id*, followed by mass selection for perennialism.

We will be happy to respond to letter requests for any of the above.

SOFIA, BULGARIA
Bulgarian Academy of Science

An attempt to use embryo-endosperm marker in obtaining CMS maize analogues

--Toma Dankov, Miglena Kruleva and Zlatka Bojilova

The method of Chase for in vivo androgenic obtaining of maize analogues is known (J. Hered. 54:152-158, 1963), and the endosperm marker created by him for this purpose, known as the marker of Chase. Because of the double fertilization in maize, the endosperm marker is not a sure guarantee for distinguishing androgenetic haploids from the hybrid seeds.

We have at our disposal an embryo-endosperm marker (Compt. Rend. Bulg. Acad. Sci., in press) which is dominantly inherited, and we created sterile analogues to it with T, S and C cytoplasm.

In 1991 in an isolation plot we pollinated the above sterile analogues (in backcross five) with Mo17, which is a universal maintainer of all three types of sterility. 165 ears were obtained in T-type, 41,028 seeds, from which 195 were without embryo and endosperm marker; in S-type, 143, 37,222 and 170 respectively and in C-type 100, 31,155 and 50. The greater part of the haploid seeds were not weak and wrinkled.

In 1992 102 plants of T-type from the seeds without marker, 91 plants of S-type and 33 of C-type were grown directly in the field. From them 38, 37 and 21 respectively were androgenic haploids and entirely female and male sterile. The 3 plants which showed female sterility from T-type, 2 from M-type and 2 from C-type were pollinated by Mo17. Only two plants of T-type and one of C-type had signs of dihaploids (of Mo17) and were also pollinated by this line. The remaining greater part of the plants were hybrid, maybe because then our sterile analogues were in BC6.

We consider that in our embryo-endosperm marker and its sterile analogues of T, S and C-type the formation of androgenic haploid seeds is heritable and can be used for accelerated creation of sterile analogues. The investigations continue.

A new source with a high percentage of haploid seeds

--Toma Dankov, Miglena Kruleva and Zlatka Bojilova

In 1991 in a 0.5ha growing area from the hybrid of the two lines (PX-32 = B-432 x 155) we found about 10% haploid plants--low growing, weak and usually female and male sterile and 6% self pollinated or dihaploid--weaker than those of B-432 line. With great difficulties we managed to obtain 10 haploid plants with 3-8 seeds each, or 38 seeds total and a comparatively easier greater number of normally seeded ears than the supposed dihaploids. On one of the ears there were 256 seeds, of which 63 were haploid seeds, which equals 20.7%.

In 1992 nearly all the haploid seeds and normal seeds from the ear with a high percentage of haploid seeds were sown in separate numbers. We obtained very few plants because of the drought, and almost no seeds from the haploid progenies. From the normal seeds of the ear with 20.7% haploids we grew 123 plants, which were entirely uniform. Thirty self pollinated ears with 7941 seeds were obtained from them, where 1102 or 13.88% were haploid. The study of the new genotype continues, concerning its similarity with B-432 line and the haploid type.

STANFORD, CALIFORNIA
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Unexpectedly extended pollen viability in California

--Greg Barnes and Virginia Walbot

Anecdotal reports suggest that maize pollen remains functional for 30 minutes or less under a variety of climatic conditions. Heat and humidity are both thought to contribute to pollen aging. Our field site has particularly low humidity (20-40% RH) and mild temperatures (mid-day highs of 25-28 C are typical) through the pollinating season. Even though pollen is rarely shed prior to 9 am, we have routinely bagged tassels the preceding night to avoid contamination on the assumption that all pollen viability would be lost overnight.

To determine viability, pollen was collected from a *bz2* tester in a W23/K55 hybrid background at approximately 9:30 am, fractionated in a glassine envelope to remove anthers and other debris, and the "purified" pollen was kept in a glassine bag in our field lab under ambient conditions of light and temperature. Generous aliquots of this pollen were delivered to cut-back ears of the same *bz2* tester in a set of detasseled rows flanked on three sides by *Bz2* material. Most contaminating pollen should yield purple kernels, but no purple contaminants were detected in the 15 ears examined:

Pollen storage (hours)	Kernels per ear	Average K/ear
0	306, 174	240
1	415, 351, 288	351
2	274, 117, 104	165
4	232, 68, 55	118
8	101, 79	90
23	88, 6	47

Averaging the yield of the 0 and 1 hour time samples we set the expected yield at 310 kernels. Seed set decreases about 50% by the 2 hour point and is progressively lower at later time points. It is striking, however, that yield is still substantial (29%) at 8 hours and 23 hours (15%) after pollen collection. We cannot completely rule out contaminating pollen from a neighboring *bz2* tester on one side of the detasseled area, however, the absence of purple kernels means that contamination is probably low.

To confirm the result a second experiment involved recipient *bz2 R-g* W23/K55 ears crossed by stored *Bz2 r-g* W23 pollen; the recipient ears were in a large *bz2* tester plot; none of the recipient plants were detasseled, allowing self-pollination to occur. In this test purple kernels are from the stored pollen and bronze kernels represent contaminants:

Pollen storage (hours)	Kernels per ear	Average K/ear	# *bz2* K
0	353, 316	335	0
1	278, two smutty ears	278	1
2	75, 24	50	1
4	36, 8, 0	15	0
8	0, 0, 0	0	2
24	21, 8, 0	10	0
32	4, 0, 1	2	0
48	0, 0, 0	0	0

A low level of contamination occurred in this experiment, however, it is clear that considerable functional pollen persists for two hours, and that small amounts of viable pollen are present in samples taken at 4-32 hours. That any pollen survives for 24 hr or more is surprising.

The phenomenon of unexpectedly long-lived pollen will be pursued to determine the contributions of pollen genotype, prevailing environment and storage conditions to pollen viability. This summer we will repeat these experiments with additional genotypes, concentrating on time points between 2 and 24 hours. At the initial time points, there is a considerable difference in the viability of *bz2* pollen vs. the *Bz2 r-g* pollen. We will also titrate pollen amounts, obtain pollen samples from tassels of different ages and vary the storage conditions.

Impact of cold stress on fractionation through cytochrome oxidase versus alternative oxidase respiration

--Carol Thornber, Abdol-Ali Soltani and Virginia Walbot

Maize is a chilling-sensitive plant: temperatures below 15 C suppress growth and prolonged exposure to temperatures of 10 C or less will kill seedlings. We previously reported that maize seedlings subjected to a chilling stress at 15 C for up to a few days accumulate *Adh1* mRNA and ADH1 protein and enzymatic activity (Christie et al., Plant Physiol. 95:699-705, 1991); within 2 days of return to normal growing conditions, *Adh1* mRNA and protein levels return to pre-stress levels. Because ADH1 can partially compensate the cell for a loss of respiration during hypoxia, we hypothesized that the rapid appearance and strong induction of ADH1 activity indicated suppression of mitochondrial respiratory activity during chilling treatment. We also reasoned that plants lacking ADH1 or doubly null (ADH1- ADH2-) would be more severely affected by cold treatment than normal seedlings.

To compare the chilling sensitivity of B37N and ADH1- or ADH1- ADH2- plants, five seedling growth experiments were conducted under slightly different conditions. Plants were germinated for 7 days in growth chambers at 25 C-30 C with 12-16 hours of light/24 hours. For chilling treatment, trays of seedlings were switched to a growth chamber kept at 10-15 C with the same light regime as the control seedlings remaining at 25-27 C. Visual inspection of B37N seedlings after 1-7 days at 15 C indicated that anthocyanin pigmentation was strongly induced in the leaf sheaths compared to the control seedlings, growth was almost completely suppressed, and the seedling leaf tips were desiccated. The seedlings survived upon return to control conditions.

Up to 50% mortality of B37N seedlings, measured as a failure to grow when returned to control conditions, was achieved after 14-18 days of chilling stress. In contrast, the ADH1- and ADH1-ADH2- seedlings were more sensitive to chilling stress, with up to 50% mortality after 4 days of cool temperature treatment. The seedlings exhibited water-logging followed by desiccation of the leaf tips to a greater extent than did chilled B37N seedlings. The control ADH-deficient plants grew more slowly than did control B37N seedlings, so direct comparisons of these genotypes is not possible, however, wet and dry weight accumulation was decreased more in the chilled ADH-deficient lines compared to their control than was the case for B37N. These growth experiments substantiate the hypothesis that ADH expression is likely to be important for long-term survival during chilling stress.

The hypothesis that mitochondrial respiration would be affected by chilling stress was tested by measuring oxygen consumption. The measurements were made in 0.1M phosphate buffer, pH 6.3, or in this buffer with addition of either 10mM SHAM or 0.5mM KCN to selectively inhibit alternative oxidase or cytochrome oxidase activity, respectively. Seedlings were grown for 10 days at 31-32 C with 12 hr 85uE m^{-2}/12 hr dark; control seedlings remained in these conditions. For chilling treatment, trays of seedlings were transferred to 15 C day/10 C night with the same light/dark regime. In each experiment, four replicate samples were examined for each condition. Each experiment was repeated two or three times; the results from one experiment are reported here.

Each sample consisted of root tissue from one seedling; 0.1g of root tissue was sliced into 0.5cm sections in buffer just prior to each measurement. Oxidase activity and oxidase capacity were determined using standard methods (Moëller et al., Physiol. Plant. 72:642-649, 1988) in a Hansatech oxygen electrode at 35 C; an A/D board in an IBM-PC transformed the electrode output into a data file. Oxidase capacity is defined as the maximum level of pathway engagement measured when a competing pathway that uses substrate for the same function is fully inhibited. Oxidase activity is a measure of the degree of pathway engagement when competing pathways are both active. With B37N (see Figure 1), about twice as much oxygen consumption occurs through the cytochrome oxidase pathway as in the alternative oxidase pathway in roots of 10 day-old seedlings. Note that the 4 control samples exhibited virtually identical respiratory properties in this (and other) experiments. During cold treatment, however, root-to-root variation in respiration can be very large. This is particularly striking for cytochrome oxidase activity after 4 days of cold treatment. We interpret this variation as reflecting true physiological differences among the seedlings; that is, in some seedlings cytochrome oxidase activity is greatly depressed by cold treatment. We do not understand the origin of these differences between seedlings; only seedlings of similar size (developmental age) were selected for measurement.

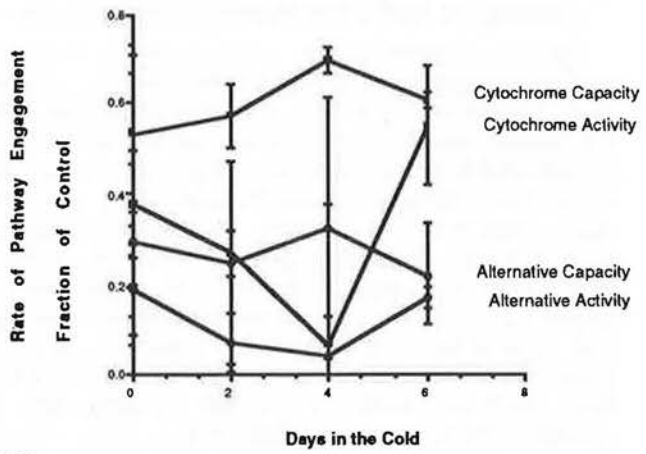


Figure 1.

Compared to B37N seedling roots, the 10 day-old ADH1-ADH2- double null roots had much lower cytochrome oxidase activity (Figure 2). Both the activities and capacities of the two oxidases were similar in the double nulls. The ADH- material is in a different nuclear background than B37N, consequently either this difference or the absence of ADH could impact oxidase content. Similar to the B37N seedlings, the ADH double null seedlings also showed greater root-to-root variation in cold-stressed materials than in the ambient control populations (Figure 2).

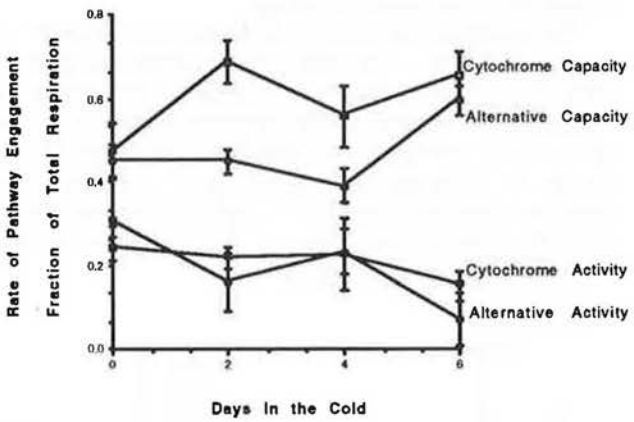


Figure 2.

Contradictory to our initial hypothesis neither oxidase capacity nor activity was significantly affected by chilling stress. In the B37N seedlings with normal levels of ADH, cytochrome oxidase and alternative oxidase capacities and activities remained relatively constant through 6 days of cold treatment. Increases in alternative oxidase have been measured after chilling treatment of other tissues (such as sliced potato tubers), however, we find no evidence for preferential use of alternative oxidase during chilling treatment of B37N seedlings. In the ADH double nulls, the two oxidases also remain relatively constant in both activity and capacity. There is, however, a significant increase in alternative oxidase capacity from after 6 days in the cold. A substantial change in capacity probably reflects increased accumulation of alternative oxidase protein after prolonged chilling treatment.

In summary we have found that respiratory activity and capacity are relatively insensitive to chilling stress in two genotypes of maize and are certainly not rapidly modulated by chilling. Both alternative oxidase and cytochrome oxidase activities and capacities remain relatively constant through 6 days of chilling treatment. Thus, it is unlikely that the rapid induction of ADH activity in normal seedlings is in direct response to a lack of mitochondrial respi-

ration. ADH activity may be required to modulate cytoplasmic pools of key metabolites during chilling stress. This is a different role than observed during oxygen deprivation where maintenance of glycolysis and ATP production by production of catalytic amounts of NAD+ is the key contribution of ADH.

Anthocyanins protect DNA from ultraviolet radiation-induced damage

--Ann E. Stapleton and Virginia Walbot

Anthocyanins extracted from plants absorb ultraviolet radiation. Measurements in situ demonstrate that the epidermis of plants absorbs 95%-99% of incoming UV-B radiation; flavonoid compounds (such as anthocyanins) and cuticular waxes are most likely the agents of UV absorption (Stapleton, Plant Cell 4:1353, 1992). Because anthocyanins and other flavonoids absorb radiation in the UV range, it is commonly suggested that these compounds could shield plant DNA from UV damage. This hypothesis has not previously been tested, however, nor has the extent of protection been quantified. In maize there are at least five anthocyanins present in purple tissues (Harborne and Gavazzi, Phytochem. 8:999, 1969; Styles and Ceska, Phytochem. 11:3019, 1972). We have investigated the ability of these compounds to protect DNA in situ from UV damage.

We developed a sensitive assay for two major types of UV-induced DNA damage, the cyclobutane pyrimidine dimer and the pyrimidine(6,4)pyrimidone, in DNA extracted from maize plants exposed to UV. We used chemiluminescent detection of antibodies specific to the two types of damage (Mori, Photochem. Photobiol. 54:225, 1991); we can detect as few as 10^7 cyclobutane pyrimidine dimers with this assay. The amount of UV-induced DNA damage in *B Pl* (purple) maize plants was less than the amount in isogenic *b pl* (green) plants (Fig. 1).

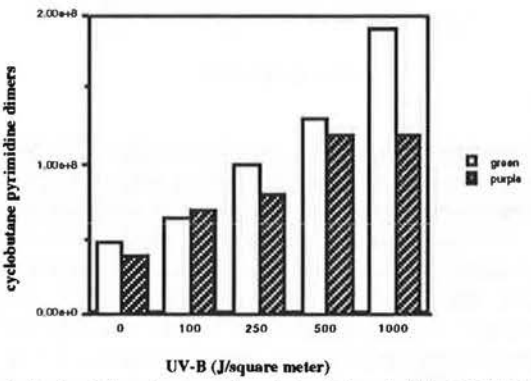


Figure 1. Number of dimers in green and purple sheath tissue irradiated with UV-B. Isogenic *B Pl* and *b pl* maize plants were grown for 6 weeks in the greenhouse, sheath tissue was excised, irradiated with UV-B, and DNA prepared using urea/SDS lysis buffer and phenol extraction (P. Bedinger, personal communication). The DNA was slot-blotted in quadruplicate onto Nytran, baked, blocked with 5% dried milk in TBS with 0.2% Tween-20, then washed and reacted with the monoclonal antibody to cyclobutane pyrimidine dimers at a 1:2000 dilution. Secondary antibody conjugated to horseradish peroxidase was used at a 1:3000 dilution. Enhanced chemiluminescence detection of specifically bound secondary antibody was performed according to Amersham's protocol. The luminescence was detected on X-ray film, which was densitometer traced. The signal from a plasmid with a known number of dimers (assayed by T4 endonuclease V protocol; Mellon, PNAS USA 83:8878, 1986) was compared with the DNA samples to determine the number of dimers in each sample. The means of the four quadruplicate samples are presented above. A permutation test was used to compare the four measurements from the green plants to the four measurements from the purple plants at each UV-B dose; at $P<0.1$ the difference between the green and purple samples was significant in the 250 and 1000 samples.

We conclude that anthocyanins protect maize DNA from UV-induced DNA damage. This suggests that anthocyanins, which are located in the epidermal vacuole, must be between the DNA and the source of UV. We used confocal laser microscopy to examine the location of the nucleus in leaf and sheath samples. In leaf epidermal cells the nucleus is in the interior third of the cell. Thus in leaves the epidermal nuclei and the mesophyll nuclei are shielded from UV by the anthocyanin in the vacuole. However, in sheath tissue the epidermal nuclei are in the outer portion of the cell; the nuclear DNA is thus not protected by anthocyanins present in the vacuole. We suggest that, in sheath, either the epidermal nuclei are more tolerant of UV damage or underlying DNA molecules (either in the mesophyll or in organelles in the epidermis that are localized below the vacuole) are protected by anthocyanin.

We are now measuring the relative levels of UV-induced DNA damage in chloroplasts, mitochondria and nuclei.

TAEJON, KOREA
Chungnam National University

Tillering depends on temperature

--Heebong Lee, Wonkoo Lee, Heechung Ji, Manki Baek and Bongho Choe

In order to determine the effects of temperature during seed germination period on tillering formation, inbreds which have been developed for hybrids with tillers were grown in temperature controlled growth cabinets. Constant temperatures of 15, 20, 25 and 30 C were applied from germination to 30 days after germination. The number of tillers per plant was observed weekly during seedling growth. It was found that the initiation of tiller formation was greatly accelerated at the temperature of 20 C. Temperature above 25 C apparently was not effective in inducing tiller formation. Effects of other factors such as fertilizers or soil water content on the tillering should be studied. The effects of temperature on maize tiller formation were also indirectly estimated when maize hybrids or inbreds which had two to three tillers without failure in Korea, where temperature is below 25 C during germination-seedling growth, were planted in the Philippines, where daily temperature is above 25 C, and did not show the usual number of tillers.

Inbreds whose tiller height is taller than the main stem

--Heebong Lee, Wonkoo Lee, Heechung Ji and Bongho Choe

We have attempted to develop hybrids with two tillers which bear harvestable ears. In order to develop such hybrids we developed a few inbreds from our indigenous local maize lines. Among the inbreds developed for tillering hybrids, one line was characterized by having higher tiller length than the main stem, while ordinary tillering inbreds usually have lower or lagging tiller height compared with the main stem. The height of the main stem of the inbred was about 70cm, while the heights of the first and second tillers were about 77cm and 73cm, respectively. We assume that one of the reasons for the lower main stem and higher tiller height of the inbred may be due to the fact that the root activities of the main stem may stop earlier than those of the tillers, which were developed later than the main stem. The agronomic characters of the inbred when crossed with other inbreds were not yet confirmed. But we assume that the inbred may be useful for developing tillering hybrids for silage

production (inbred seed is available upon request).

Super thin pericarp, is it possible?

--Manki Baek, Wonkoo Lee, Heebong Lee, Heechung Ji and Bongho Choe

As one measure of developing hybrids with thin pericarp for human consumption, we have used Korean local glutinous maize lines which have been traditionally used for human consumption. A few of the lines collected and observed for thickness of pericarp shows pericarp the thickness of which ranges from 25 to 40 millimicrons (most of the inbreds from the U.S. showed over 60 millimicrons). The heritability estimated was also very high (over 70%). The thickness of pericarp after being peeled off and dried was measured by micrometer using a microscope without any chemical treatment.

TUCSON, ARIZONA
University of Arizona

The use of bulk segregant analysis to map dwarf mutants

--Tim Helentjaris, Ivone Torres-Jerez and Tom McCreery

In our efforts to study various dwarf mutations in maize, we have found it necessary to establish better linkages between their genomic locations and the maize molecular marker set. We have found it convenient to use bulk segregant analysis as described by Michelmore et al. (PNAS 88:9828-9832, 1991). Usually 100+ F2 seeds were planted either in the greenhouse or field. All of the mutant plants were identified and leaves collected from them as one pool (usually 10+ plants) and multiple normal plants as a second pool. The material from each pool was lyophilized, ground, and DNA was extracted. Genomic DNA for each pool was digested with *Hin*dIII, *Eco*RI, and *Eco*RV, electrophoresed in an agarose gel, and blotted to a nylon membrane. A collection of RFLP probes were selected for the chromosome arm where each mutant was known to be located. Close linkage of the mutant to a RFLP locus was scored from the hybridization results based upon a polymorphism being evident in the F2 population and that two allele fragments could be detected in the "normal" pool and only one in the mutant pool.

Four mutants have been evaluated so far. *br2* was found to be most closely located to a region containing *npi272* and *bnl5.59* near 1C. The location for *ct1* on chromosome 8S falls in a region containing *npi110* and *umc32b*. *ct2* is located on chromosome 1S and lies within a region containing *umc157* and *npi97b*. *na2* is located in a region on 5S containing *umc27a* and *umc166*.

Use of random cDNA probes for RFLP mapping

--Tom McCreery, Tim Helentjaris and Ivone Torres-Jerez

In our undertaking to enlarge the maize molecular marker map, we have begun initial efforts to both map and sequence randomly chosen cDNA clones. Two different libraries have been investigated. The first was constructed in LambdaZapII with *Not*I-*Eco*RI linkers ligated to ds cDNAs prepared from 14 DAP sweet corn endosperms. No size selection was incorporated prior to ligation to the *Eco*RI site of the phage arms. Bluescript phagemids were excised from the library using R408 helper phage, transfected into *E. coli* XL1Blue cells, and plated out on selective media. 672 colonies were picked into microtiter dishes and screened. Cultures of each colony were used as template in PCR labelling reactions containing a digoxigenin-modified nucleotide. The probes were hybridized to Southern blots and detected using AMPPD chemiluminesence activated by an Ab-conjugate of alkaline phosphatase. The Brookhaven recombinant inbred collection was used for assigning map positions for all polymorphic loci detected by these clones.

The results of this initial screen are summarized in Figure 1. For a number of reasons, many clones from this library proved unsuitable at different steps in the process and had to be discarded. The resulting informative clones (both complex and simple) were sent to Linkage Genetics (Salt Lake City, Utah) for

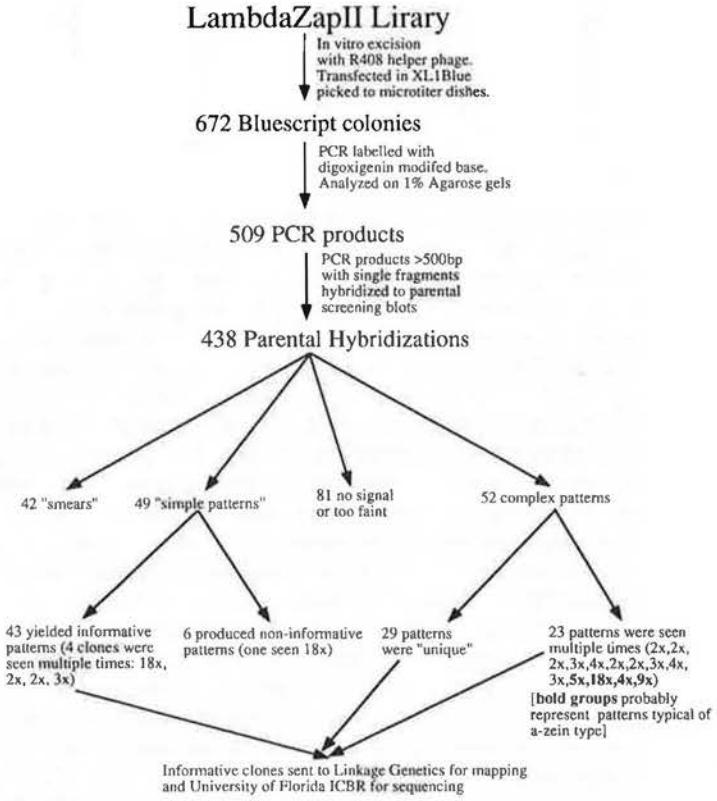


Figure 1.

mapping with the RI progeny. The mapping data yielded 99 probes that mapped to 161 loci. These probes are found throughout the genome (see Figure 2) with an asymmetric distribution. Particularly noteworthy is the large number of markers mapped to the short arm of chromosome 4, many of which we have determined to represent various zein genes. This should not be surprising as zeins comprise a large percentage of expressed genes in developing seeds. Interestingly most of these zein clones are found in a large "inverted" array comprising most of 4S. Prior to this study there were very few RFLP markers on this chromosome arm, which suggests several interesting possibilities for consideration. Most RFLP markers in the past have utilized genomic clones from *Pst*I libraries as a selection for low-copy-number sequences. That some areas of the genome are underrepresented by this general pool could suggest that methylation occurs non-randomly in maize. Alternatively different areas of the genome may contain different levels of unique sequences. In any case, the use of randomly chosen cDNAs prepared from different tissues may facilitate closing

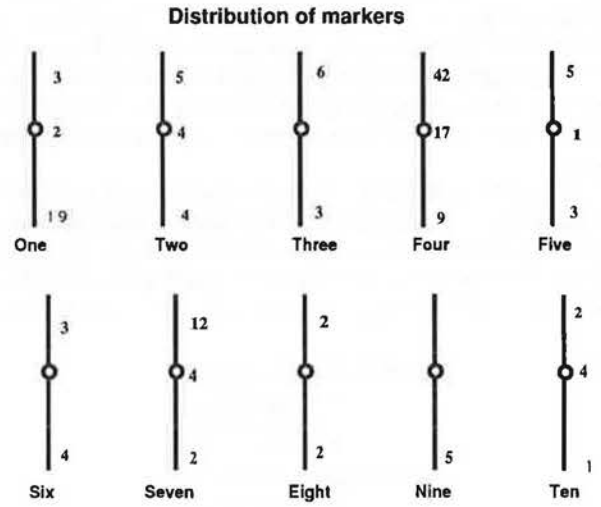


Figure 2.

some of the "gaps" in the current maize molecular marker map.

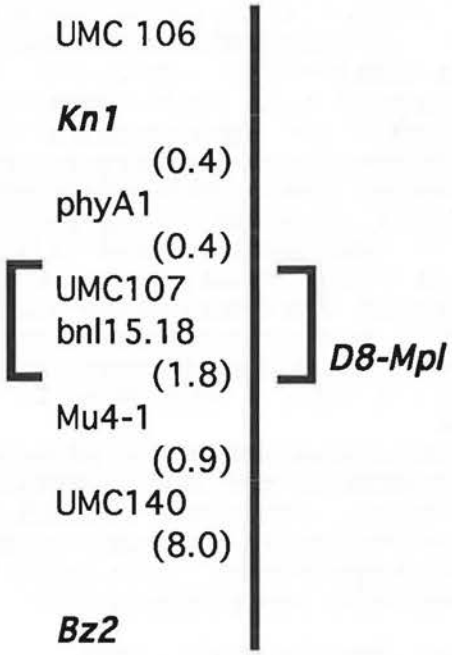
Many of these clones were also sent to the University of Florida (R. Ferl) for sequencing. Very few have been sequenced to date but it is our intention to both map and sequence all of these clones in order to attempt to assign both functions and genome locations to these cDNAs. One clone was found that possessed a short but significant homology to *bt1* and mapped to at least five different chromosomal locations, suggesting that this strategy should produce some interesting insights at the same time we are enlarging the maize marker map. While our success rate from start to finish with this library has been lower than we would like, we have been much more successful with a library prepared by Stratagene of maize leaves, and many of these clones previously have been sequenced by C. Baysdorfer at California State University. >90% of the clones picked from this library amplify and we have been able to detect genomic polymorphisms for almost all of these. The difference we believe lies in the construction of the libraries. The first library was constructed without imposing any size selection upon the inserts and many have been in the range of only 150-200bp, which is too small to produce an effective hybridization signal or meaningful sequence comparison to existing databases. The latter utilized an oligo-dT-primer-linker and a size selection which resulted in most of the randomly-chosen clones being in the +500bp range. We believe this fact explains most of the differences we have observed in our success and would urge others to utilize both steps in constructing their own cDNA libraries, no matter what their desired purpose.

For future use we are constructing cDNA libraries using RNA isolated from both etiolated seedlings and developing seeds. However three principal differences in the construction will be used: 1) LambdaZipLox (Gibco BRL) will be used as a cloning vector which possesses the site-specific Cre-*lox* recombinational machinery to provide automatic in vivo subcloning, 2) directional libraries will be constructed utilizing *Not*I-oligo-dT primers, *Sal*I adapters and *Sal*I-*Not*I digested arms, and 3) the cDNA will be size-selected using a sephadex column before ligating it to the vector arms. Once this library is prepared clones will be isolated from it in three classes as either "abundant and common between tissues", "abundant and specific to the tissue of origin", and as "rare" using subtraction and screening strategies. We will then proceed with sequencing and mapping of this set of clones to determine how much more efficient this process is with this strategy.

Dominant dwarfs

--Rodney G. Winkler and Tim Helentjaris

D8-1 is a dwarf mutant proposed to be involved in gibberellin (GA) reception. In contrast to dwarfs deficient in the biosynthesis of GA, *D8-1* is dominant and GA-nonresponsive. To facilitate the identification of putative *D8* clones isolated by transposon tagging, 224 backcross progeny were analyzed to resolve the map location of *D8-Mpl*. The following cross was utilized: *zb7 kn1-wt d8-wt bz2* X (*zb7 Kn1-0 D8-Mpl Bz2 /zb7 kn1-wt d8-wt bz2*). 121 *bz2* kernels and 103 *Bz2* kernels from this cross were planted and seedlings were treated with GA_3 at 7 and 14 and 21 days. The *zb7* phenotype did not express well under the summer greenhouse growth conditions used (temperature sensitive?), however *Mpl* and *Kn* were easily scored. Recombinants were selected (11 *kn1-wt d8-wt Bz2*; 14 *Kn1-0 D8-Mpl bz2*; 2 *Kn1-0 d8-wt bz2*) and analyzed with molecular markers (*umc106, phyA1, umc107, bnl15.18, Mu4, umc140*) in the region to define a precise location for the *D8* locus. The suggested map is shown below [ed. note - the map should be read from bottom to top]:



Estimated map distances (cM) are shown in parentheses. *D8-Mpl* was not resolved from *umc107* and *bnl15.18* in this population but was approximately 1.8cM distal from an endogenous *Mu4* element linked to *D8-Mpl*. Two results are slightly different than expectations. The map position of *D8* suggested by this study is more proximal than in the "working" maize genetic map (MNL, 1992). Secondly the region between *Kn1* and *Bz2* is more condensed than expected from previous reports (MNL63:2, BNL and UMC maps 1992). However, the predicted map order of the RFLP markers in this region is the same as that observed in the BNL and UMC maize RFLP maps. A larger population has been planted in the greenhouse to more precisely define the position of *D8-Mpl* rela-

tive to the closest markers and *zb7*.

A second dominant anther-eared dwarf, *D9* (*D*-2319* = *D9*, isolated by G. Neuffer and mapped by him to chromosome 5S (MNL65:51), was RFLP mapped by the "bulked segregant" method (PNAS 88:9828, 1991). *D9* mapped to an interval of 5S that contains duplicate loci with chromosome 1L (region of 1L containing *D8*). Thus *D9* is probably a duplicate of *D8*. Similar to the six known dominant alleles of *D8* (MNL66:21), *D9* is dominant and GA-nonresponsive (does not grow in response to GA). *D9* and *D8* alleles set a maximum limit on plant growth by uncoupling the normal relationship between GA and growth.

_Mu_ tagging of dwarfs

--Rodney G. Winkler and Tim Helentjaris

Two dwarfs were isolated from selfed ears from active Robertson's Mutator lines. Maize *d*-3010* is andromonoecious, GA_3 responsive, and was mapped to an interval between *bnl3.06* and *umc81*, suggesting that it may be an allele of *d3*. Seed from an allelism cross is drying in the greenhouse. Analysis of "bulked segregant" pools (PNAS 88:9828, 1991) has identified a linked *Mu1* element. A higher resolution mapping experiment is in progress. Maize *d*-3685* is a semidominant dwarf that is GA_3 responsive and appears to be extremely sensitive to environmental conditions, expressing well in the greenhouse, but expressing weakly in the Tucson fall field season. Dwarfs in this family were in repulsion to *B*, suggesting that *d*-3685* maps to chromosome 2 (an allele of *d5* ?). RFLP mapping confirmed the chromosome 2 map position by showing linkage to *umc6*.

VICTORIA, BRITISH COLUMBIA
University of Victoria

The color of music - pigment notes

--E. Derek Styles

The American Dream. Although not everyone can be born a queen, king, prince or princess (who would want to be, nowadays?), in theory any citizen should be able to rise (?) to become a president. True, a certain amount of native ability is required, but is insufficient if the more basic necessities of 'resources, friends and helpers' are lacking. Thus it is for presidential aspirants, and thus it is for their genes, and of course thus it is also for the pigmenting genes of maize. Presidential hopefuls and pigmenting genes remain untested potentials until all the basic requirements for their expression are present. In theory, inherent differences in potential can be evaluated by offering all the same opportunity, for example, different *R* or *P* alleles can be compared against the same genetic background. This is at best a limited evaluation, and might be equated to finding differences in presidential potential by having everyone live in the same suburban environment, and limiting all to an adequate but minimal income. A different approach might be to offer everyone unlimited 'resources, friends and helpers' and evaluate differences in presidential potential in the 'best of all possible worlds'. For maize pigment genes, this would mean comparing pigment potentials of different alleles when every other genetic and environmental factor favors maximum pigmentation. Ideally, there should be no duplicate factors present (only one candidate at a time per party being evaluated), and it should be recognized that evaluation of current worth is at best temporal, as it has been achieved and can be further altered by 'experience'.

The above wandering waffle is by way of introducing and justifying my current approach to the study of the flavonoid pigment genes in maize. Clearly it will soon be possible to recognize differences at known loci in precise molecular detail, and at that time there may be a terminology problem in using the very word 'allele', especially if a multitude of differences are recorded at a single locus. It is quite realistic to expect reports of differences in large sequences of bases; differences in single base pairs and in numbers of triplet repeats, and differences in methylation patterns, etc., in the near future. At that point the limiting factor will be in our ability to recognize the effects of such differences, if any, at the phenotypic level. Restrictions on the expression of an allele's potential will limit our ability to recognize the effect of a specific change at the base level. It will be an advantage, therefore, to arrange for the 'best of all possible worlds' when attempting to evaluate the potential of a specific allele, meaning that all genetic and environmental factors should favor maximum pigmentation, so that the only limiting factor is the particular allele being evaluated.

***P*-locus phenotypes.** Allelic symbols for the *P* locus offer only broad categorizations, and the collection of *P* locus factors incorporated into the inbred strain 4County63 by R. A. Brink and sent to the National Seed Storage Laboratory in Fort Collins, Colorado (see descriptions in MNL40:149-160, 1966) demonstrate that *P* alleles actually form a continuous series in terms of cob and pericarp pigment when compared in that particular background. The unstable factor for orange pigment (*Ufo1*) found by Dr. Charles Burnham (see MNL61:100) appears to 'release' the tissue specific controls of *P-WR* alleles so that pigments normally restricted to the cob are produced not just in the cob, but also in the pericarp, culms, leaves, and other plant parts. By itself, *Ufo1* has no expression, so that *P-WW* alleles remain unpigmented even when *Ufo1* is present. The expression of an intermediate *P*-allele, determining a grainy type of pericarp pigment (from the 4Co63 collection, specific designation unfortunately lost, but probably 65-CFS-364), is not altered by the presence of *Ufo1*. This 'grainy' allele occasionally mutates to a sectored form, with irregular sectors in the cob, husk and pericarp. This sectored form yields *P-RR* alleles at high (~10%) frequency, and these *P-RR* plants are very darkly pigmented in husks, cob, pericarp and even culms, in fact they are among the strongest *P-RR* alleles in my stocks. The sectored form is distinct from *P-VV* or *P-MO* alleles, not only in the type of sectoring, but also in the very dark and clear sectors consistently seen in the husks and cobs. Homozygous sectored forms breed true except for mutation to *P-RR*. Heterozygous *P-WR/'P-sect'* plants yield *P-WR*, *'P-grainy'*, *'P-sect'* and some *P-RR* progeny. If, as this evidence seems to suggest, the grainy phenotype results from a controlling element at an otherwise very strong *P-RR* allele, this would explain why, in its controlled state, the allele appears not to respond to *Ufo1*, and it is like *P-MO* and *P-VV* in this respect.

Pith pigment. In line with attempts to get the maximum expression of *R* and *P* alleles, I have been particularly interested in those alleles or combinations of enhancing factors that have strongly pigmented piths. Interestingly enough, the 'grainy' *P* allele described above is easily distinguished from other *P* alleles by its orange pith pigment. The pith of the sectored form is white with dark red sectors, and the secondarily derived *P-RR* allele has a solid dark red pith to go with the other darkly pigmented tissues.

R alleles that determine anthocyanins in the pith (with other 'appropriate' non-allelic friendly helpers) include the Group-D *R* alleles, and *R-nj-6*, a compound allele derived from *R-st:nj* (stippled crown). *Pl* is a required but not sufficient factor for pith anthocyanin, and the enhancing factor *a3* definitely favors pith pigmentation by the above listed alleles, although it is not an absolute requirement. Many of the lines with strong pith anthocyanin also carry the recessive aleurone enhancer *in*, even though selection was made on the basis of pith pigment rather than for aleurone enhancement.

The aleurone intensifier *in*. If *in* can enhance anthocyanin production in the pith, does it have an enhancing role in other tissues also? Some miscellaneous observations on possible effects of the *in* factor may be helpful or misleading, depending on whether the observations hold up with further testing: 1) Red pollen is typically produced by some (but not all) *Pl r-ch* (or *R-ch*) strains, but some strains of non-cherry red anthered *R* alleles (with *Pl*) can also produce red pollen. Some (but not all?) of our red pollen producing non-cherry strains also carry *in*. 2) Some *c2 R-r Pl* have purple anthers indistinguishable from sib *C2 R-r Pl* plants. I am not sure how typical this is, but so far I have noted this only in *c2 Whp* strains that also carry *in*. 3) Some *r-g* colorless aleurone strains, and even some *c2 whp* colorless aleurone strains, have a 'dirty' pericarp, that if it were covering a colored aleurone, might well be called a 'sheen'. There seems to be a more than accidental correlation between at least one type of 'dirty pericarp' and the presence of the *in* factor. Unfortunately the correlation is not one hundred percent. Perhaps another factor such as *Pl* may be required? 4) The 'pericarp sheen' typical of *in* appears not to contain flavonoids, at least we have not been able to isolate flavonoids when attempting to extract the 'sheen' from the pericarp.

Although the above observations should be treated with caution until they have been tested properly, I offer them in the hope that they may add or arrange a few pieces in the larger puzzle. To my mind, they would make the most sense if *in* determined a membrane characteristic and affects, among others, the semi-permeable membrane that separates the pericarp and the endosperm tissues.

The salmon silk factor *sm*. According to Anderson (NY Cornell Univ. Exp. Sta. Mem. 48:539-554, 1921), the intensity of the salmon silk color is related to that of the pericarp: *'A sm P'* have salmon silks and *'A sm p* [possibly *P-WR?*]' having brown silks. There was a report in the 1960 Newsletter (Kramer, MNL34:111) of a salmon silk character appearing in progeny from UV treated pollen that showed good expression in the absence of red pericarp, but it was not stated whether the *P* allele was *P-WW* or *P-WR*. In my experience, *sm* expression is more variable with *P-WR*, but is usually good to adequate with only the occasional line showing 'brown' rather than 'salmon' silks. Occasional families show a dominance of the *sm* factor, but I have never been able to isolate the factor or factors that allow the dominant expression. Until a few years ago, I had never found an exception to the rule that *P* locus activity in the form of a *P-RR* or *P-WR* allele was required for *sm* expression. I now have *P-WW sm* stocks that consistently give good salmon silks. The *sm* allele is one that I obtained from the Coop stocks in 1973, and it has never shown any deviation in its expression to this point, and I have no reason to suspect that it has changed. I have derived *r-g P-WW sm* and *bz2 P-WW sm* lines from the original *P-WW sm* family, to confirm that the silk color is not derived from 3-hydroxy anthocyanins controlled by the *R* locus. Phenotypically, the *P* allele is unremarkable, with no evidence of any cob or pericarp pigment, but I have yet to test it with other sources of *sm*. It is possible that this particular *P* allele is unique in being active only in the silks, and that will be my next test in trying to track down the reasons for this apparent anomaly in *sm* expression.

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The role of transposons in the origin and morphogenesis of maize
--Walton C. Galinat

The first direct evidence of host regulation over the transposons that control gene expression during the origin and morphogenesis of maize comes from connecting-link stocks developed to analyze floral differences between teosinte and maize. Previous studies by others of host regulated transposon excision and incision involved color producing genes expressed as spots in the aleurone or stripes in the pericarp. By extrapolation to genes in general, McClintock predicted that this transposon process controlled evolution and morphogenesis.

In the connecting-link stocks the teosinte gene *Pd-Ac* (*pd*) on chromosome 3 that represses development of the pedicellate female spikelet had a variegated expression along the ear (Fig. 1). Normally in teosinte, the female spikelets are solitary in adapting for the protective function of grain enclosure within a fruitcase while the male spikelets on the same plant are paired showing host regulation by sex over *Ac* transposition and *Pd* expression. In maize, both male and female spikelets are paired. Paired female spikelets in maize are adaptive for the domestic function of increased productivity per ear and easier shelling. Stabilizing selection in certain of my stocks restored absolute sexual control in producing female spikes that were stable for either two-ranks of single spikelets as in teosinte or else two-ranks of paired spikelets as in maize.

Apparently during the origin of maize, the *Ac* transposon of *Pd* became banished to exile, perhaps into some distant intron of a different chromosome. The pairing of spikelets, by a reactivation of the second spikelet, then had a freedom of expression without sexual discrimination. Humankind's greatest plant breeding achievement was well underway.

Presumably the different transposons can be identified molecularly by different known probes, and in due course someone will do that for the *Pd* transposon that I have perhaps incorrectly indicated here as being the *Ac* transposable element.

Bifurcation at different levels during the floral phase
--Walton C. Galinat

Bifurcation of the spike (ear), the rachis (*mr*) and the spikelet (*pd*) are shown as different target levels of expression in Figure 2. The rachis and spikelet levels of expression are for genes (*mr*, *pd*) involved in the origin of corn from teosinte. The *pd* gene has been described here in the item on the role of transposons.

The *mr* gene (multiranking on short arm chromosome 2) controls another key domestic trait with symptoms of transposon involvement. Normally in teosinte, two-ranking occurs in both the vegetative and floral phases, while maize retains this primitive condition in only the vegetative phase. In the floral phase of maize, the central spike of both tassel and ear are usually multiranked,

Variegated expression of paired female spikelets in a connecting link stock between teosinte and maize. Normally in teosinte, pairing is confined to the male spikelets. Plants with variegated female pairing may also have variegated male pairing as if the pd gene had lost its normal sexual recognition and regulation.

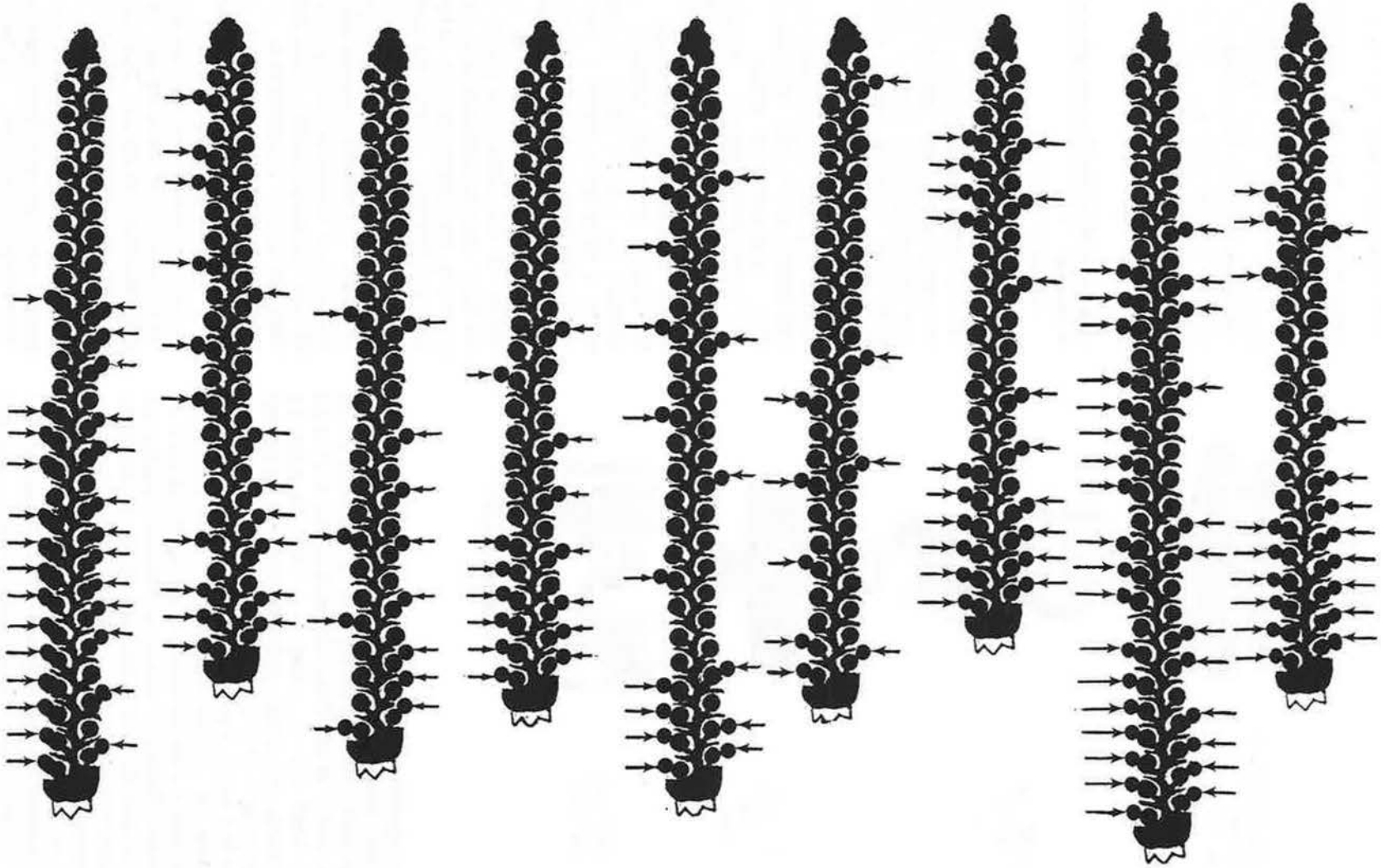

Fig. 1

Target in Developmental Time for Bifurcation Expression

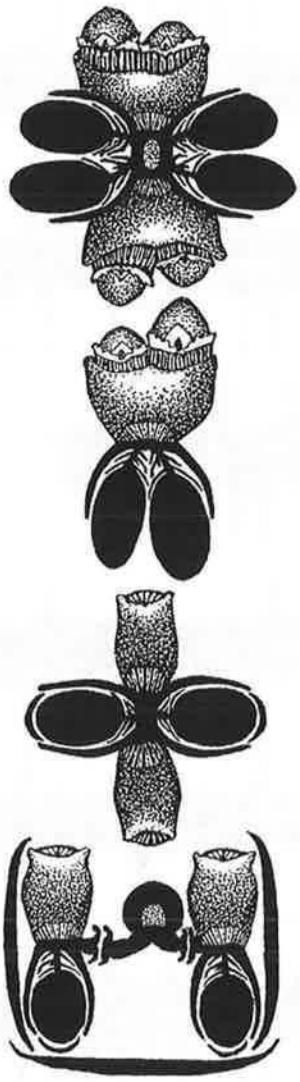

Figure 2.

while the branches, or secondary ears, remain at the primitive two-ranked level. Energy-deprived inflorescences that develop under a vascular system that is genetically reduced or diverted on one hand, or environmentally stunted on the other, may revert to the primitive condition of two-ranking. In contrast, the higher the level of energy and vascular supply endowment to the terminal spike, the higher its floral ranking and kernel row number. Mutations are well known to corn breeders that result in vegetative multiranking that is usually unstable and confined to the upper nodes between the ear and tassel. At very high kernel row numbers, the whole plant may have many-ranked leaves, and every plant in a homozygous family (*mr*-v, *mr*-v) will express the phenotype. All of these changes in ranking, including the environmentally induced phenocopies, are based on a certain genetic condition at the *mr* locus on chromosome 2S. The possibility cannot be ignored that the movement of transposons involves the consumption of energy. A role for transposons in both spikelet bifurcation and non-divergent rachis bifurcation during the origin of maize seems possible. The various levels, or movements, of bifurcation in floral morphogenesis are illustrated in Figure 2.

Maize origin from two kinds of four-rowed teosinte

--Walton C. Galinat

In their effort to collect, grow, and isolate more productive mutant forms of teosinte, the first corn breeders of 8000 years ago included two kinds of four-rowed teosinte. One had two ranks of paired spikelets due to the *pd* mutation. The other had four ranks of single spikelets due to the *mr* mutation. They hybridized in their isolated gene pool. The double mutant from recombination was the first eight-rowed corn (Fig. 3).

On the color of the New England Flints and their sweet corn derivatives

--Walton C. Galinat

In attempting to develop an authentic type of multi-colored northern flint corn for use in the reconstructed farming of the 1620's at Plimoth Plantation, I chose the Rhode Island flint for the background as the closest and purest, having been maintained since Colonial Days by the University of Rhode Island and members of the Society for the Preservation of the Jonnycake Tradition in Rhode Island. But there was a breeding problem with R.I. flint. It was homozygous for both the *C-I* dominant color inhibitor and for dingy pericarp color. When a *C* allele was substituted for the *C-I* color inhibitor, any aleurone color would barely show through the dingy or straw color of the pericarp.

The early descriptions of the corn grown in New England before the English started corn farming and selection state that it was eight-rowed and of many distinct colors. The best and most detailed description comes from a letter of John Winthrop, Jr. to the Royal Society (1662) reprinted in full in the New England Quarterly Vol. X, No. 1 (1937). According to this report, the white and yellow that is between a straw color and a pale yellow are the most common. It goes on "there are also (ears) of very many other colours, as red, yellow, blew, olive colours, and greenish and some very black and some of intermediate degrees of such colours, also many sorts of mixt colours and speckled or striped, and these various coloured eares often in the same field and some graines that are of divers colours in the same eare." (The original spelling of Winthrop is retained in the quote.)

The separate listing of yellow from straw color and white suggests to me that the straw color is dingy pericarp on a snow white endosperm. The absence of aleurone color in the present R.I. flint may be due to selection by the English farmers for a corn cereal that was more similar in color to the European small grain cereals which they were more accustomed to eating. Some of the early sweet corn inbreds such as C3 and C13 have the same dingy pericarp as Rhode Island flint while others of older divergence such as Luther Hill have colorless pericarp and snow white endosperm. The only color retained in some strains of Rhode Island flint is that 1% red pericarp under the variety name of King Philip - the Indian Chief involved in a bloody war. The mixture of 1% red ears had excitement value at harvesting time because the tradition was the husking of a red ear gave license to kiss the one of choice.

In any case, the necessary genetic changes have been made by breeding manipulations in Rhode Island flint so as to restore the original beauty to this corn with a great variety of gorgeous colors. Perhaps the spirits of the Wampanoag and Naragansett Indians will smile on this.

The origin of maize in the F_2 from a hybrid between two kinds of four-rowed teosinte.

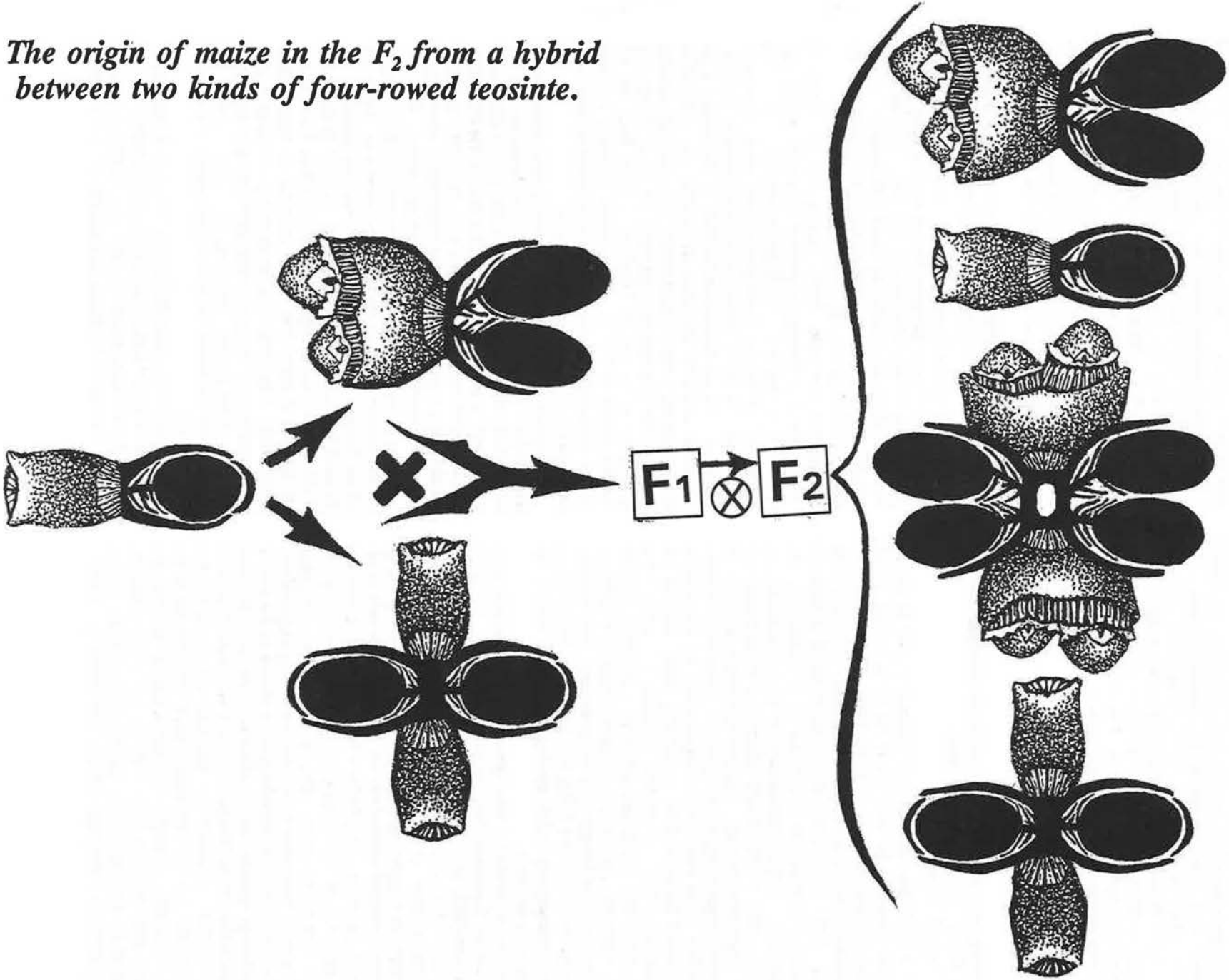

Fig. 3

Pollen production by *Vg* tassels

--Walton C. Galinat

Pollen production by vestigial glume (*Vg*) sweet corn has always presented a breeding problem. In a normal background, the *Vg* gene has a stronger effect on the tassel while the reverse with the stronger effect on the ear is the desired objective. The naked flowered tassels sun-blast and, thereby, become effectively male-sterile. Modifying genes that restore some degree of pollen shedding to *Vg* plants include intermediate tunicate alleles, intrusive tassels and anther color. To these we now add small anthers bred to fit the reduced tassel glumes. The small anther gene (*sa*) was found to be linked to *Vg* on chromosome 1.

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Screening large populations for recessive *bx1* genotypes; a variation of the $FeCl_3$ root-tip squash assay

--Kevin D. Simcox

The qualitative methods most commonly used to identify *bx1/bx1* genotypes are variations of the $FeCl_3$ root-tip squash method of Hamilton (Weeds 12:27-30, 1964). This procedure involves squashing root-tips of germinated kernels in a drop of 0.1M $FeCl_3$. Formation of a dark blue-purple coloration indicates the presence of DIMBOA and other related 1,4-benzoxazinones. Homozygous recessive *bx1* genotypes either lack or accumulate very small quantities of DIMBOA and subsequently do not produce the dark blue-purple coloration. This procedure was used on several thousand kernels to map the *bx1* gene to the short arm of chromosome four using monosomic analysis and B-A translocations (Simcox and Weber, Crop Sci. 25:827-830, 1985). Although this procedure identifies very few false negatives, application of this procedure to screen large populations for mutations in the DIMBOA biosynthetic pathway (i.e., transposon tagging or EMS mutagenesis) would be laborious and time consuming. I have used the following variation of the $FeCl_3$ root-tip squash procedure to screen approximately 30,000 seedlings for Mutator-induced mutations at the *bx1* locus.

Kernels are planted in either greenhouse seedling beds or in dark-colored Tupperware tubs containing vermiculite. To obtain even germination, use a shallow planting depth and heat beds or tubs above 72 C. Cover the beds with a black plastic sheet; I use landscaping plastic, or cover the tubs with aluminum foil. After four days, the etiolated coleoptiles (etiolated coleoptiles are used because chlorophyll will react with $FeCl_3$) are sprayed with an alcoholic $FeCl_3$ solution (50g $FeCl_3 \cdot 6H_2O$ dissolved in 500ml of 95% ethanol, 0.1N HCl; Tipton et al., Biochemistry 6:2866-2870, 1967), using a plant mister. After 15 min, rinse the coleoptiles with tap water. Coleoptiles heterozygous or homozygous dominant *Bx1* will form dark blue-purple longitudinal streaks on the side of the coleoptile opposite from the direction of the spray (droplets of the solution form on the opposite side of the coleoptile, soaking into the tissue). Recessive *bx1* genotypes will not react to the $FeCl_3$ solution, but may form slight brown discoloration due to the HCl in the solution. To confirm the negative reaction, seedlings are sprayed again with the $FeCl_3$ solution, and if still negative, seedlings are removed from the planting media and the root-tips are squashed on a filter paper saturated with the $FeCl_3$ solution.

Using this procedure I have identified 4 putative Mutator-induced *bx1* mutants out of 30,000 F1 seedlings. Approximately 45 min is needed to screen 2,000 seedlings and seedling beds or tubs can be replanted very 5 days. Care should be taken in selecting what dominant *Bx1* source is used as a parent. There is a great deal of variation between inbred lines in the amount of DIMBOA that accumulates in seedlings and adult plants (Klun and Brindley, J. Econ. Entomol. 59:711-718, 1966; Dunn et al., Can. J. Plant Sci. 61:583-593, 1981). It is also important to screen seedlings prior to the emergence of the initial leaf from the coleoptile. Once the leaf emerges, the fully expanded coleoptile becomes unresponsive to treatment and variable results are obtained scoring the emerging leaf blade. Since cyclic hydroxamates are primarily localized in meristematic regions and vascular tissue, it is important not to use adult tissue in screening procedures.

The use of the "modified dry blot" procedure for RFLP analysis

--Kevin D. Simcox and Michael D. McMullen

Our lab is constantly searching for the elusive combination of Southern hybridization protocols that will result in rapid turnover, good signals, and repetitive use of membranes. We would like to describe our latest attempt to reach hybridization nirvana, adapting the "modified dry blot" protocol of Kempter and coworkers (TIG 7:109-110, 1991) for RFLP analysis.

Digested genomic DNA (approximately 10µg of DNA/40µl/lane) is electrophoresed through a 0.8% agarose gel, in 1 X RBE running buffer (40mM Tris-Acetate, pH 7.3, 2mM Na_2 EDTA, 0.02mM NaOAc, 32mM glacial acetic acid). After electrophoresis, the gel is stained in ethidium bromide and photographed. The gel is prepared for DNA transfer using 2 X gel volumes of solution for each step: 1) depurinate in 0.25N HCl for 20 min; 2) denature twice in 0.4M NaOH, 15 min each; 3) neutralize once in 0.25M Tris-Acetate, 0.1M NaCl, pH 8.0 for 15 min; and 4) incubate in the 0.025M Tris-Acetate, 0.1M NaCl, pH 8.0 transfer solution for 15 min. Slowly agitate gel during treatment using a rotary platform, and rinse the gel in ddH_2O between each treatment (except after transfer solution).

Cut 5 pieces of Whatman 3MM chromatography paper and one piece of Gene Screen Plus hybridization membrane (DuPont) the same size as the gel. Soak one piece of 3MM paper in the transfer solution and lay on plastic wrap. Lay the gel onto the 3MM paper (doesn't matter which side of the gel is up) and gently roll out air bubbles using a pipet. Pre-wet the hybridization membrane in ddH_2O and equilibrate for 15 min in transfer solution. Place the membrane on top of the gel, soak a second piece of 3MM paper in transfer solution and stack on top of the membrane. Gently roll out air bubbles with a pipet and place the four remaining dry pieces of 3MM paper on top of the stack. Cut a 2" (2.5cm) stack of paper towels slightly larger than the gel. Place towels on top and add 1kg of weight (one Sigma Chemical Co. catalog does nicely) on top of the stack. The minimum time for transfer is 1 hr, but we routinely go 2 hrs or overnight depending upon the time of day. After 2 hrs transfer, DNA is not detected in the gel. The membrane can be dried either at room temperature or in vacuo at 80 C for 2 hrs.

The membrane is pre-hybridized (5 X SSC, 2 X Denhardt's, 50mM Tris-HCl, pH 8.0, 5mM Na_2 EDTA, 0.5% sarcosine, 1.0ml of 10mg/ml boiled salmon sperm) in a 65 C water bath for a minimum of 3 hrs. We use Dazey Micro-Seal pouches (8" X 12", 2MILS

thick), sealing the pouches with a ClampCo sealer (Fisher Sci.). We normally use 30 to 40ml of solution per pouch, with 1 to 8 membrane(s)/pouch. The pre-hybridization solution is replaced with hybridization solution (5 X SSC, 2 X Denhardt's, 50mM Tris-HCl, pH 8.0, 5mM Na_2 EDTA, 0.5% sarcosine, 10% dextran sulphate, 1.0ml of 10mg/ml boiled salmon sperm). A G-50 purified random-primed ^{32}P-dCTP probe (100ng isolated insert or linearized plasmid, 20µCi ^{32}P-dCTP; Feinberg and Vogelstein, Anal. Biochem. 132:6-13, 1983) is boiled for 5 min and added to the hybridization pouch. Air bubbles are removed and the sealed pouch is incubated 24 to 48 hr in a 65 C water bath. The membranes are washed five times for 5 min each in 2 X SSC, 0.1% SDS at 65 C (400ml/wash), then an additional five times in 0.2 X SSC, 0.1% SDS at 65 C. The membranes are wrapped in Saran wrap and autoradiography is performed as described in Maniatis et al. (Molecular Cloning, CSH Laboratory Press, 1982) for 24 to 48 hrs. Membranes are stripped in 0.4M NaOH for 30 min and neutralized in 0.4M Tris-HCl, 0.1 X SSC, and 0.1% SDS for 30 min. The membranes are returned to pre-hybridization solution or wrapped in Saran Wrap to prevent drying.

Starting from DNA digestion, we have reduced the amount of time to detect single-copy maize sequences with ^{32}P-dCTP nick translocated probes to a minimum of four days. The Gene Screen Plus membrane was found to be superior to Zeta-Probe and Hybond-N, measured by signal intensity and background remaining after stripping the membranes. We have stripped and re-hybridized Gene Screen Plus membranes eight or more times maintaining signal intensity without increasing background. The hybridization solution is re-used to probe additional membranes. After use, the hybridization solution is stored in a 50ml Falcon tube at -20 C. The used hybridization solution is added directly to the pouch, along with 1.0ml of 10mg/ml boiled salmon sperm, and incubated for 2 days at 65 C. The resulting hybridization signal is comparable to the original hybridization up to 2 weeks after initial hybridization. This technique was developed with considerable input from Mark Jones and Brenda Schult (USDA, ARS, Wooster, OH 44691).

Mapping of multiple disease resistance genes on the short arm of chromosome six

--Kevin D. Simcox, Michael D. McMullen and Raymond Louie

The satellite region distal to the NOR on the short arm of chromosome six contains resistance genes to two potyviruses, Maize Dwarf Mosaic Virus (*Mdm1*) and Wheat Streak Mosaic Virus (*Wsm1*), and a recessive source of resistance to the fungal pathogen *Cochliobolus heterostrophus* race O (*rhm1*).

Maize Dwarf Mosaic Virus. Several unlinked loci are involved in resistance in maize to MDMV. Presently, all characterized sources of resistance to MDMV have involved the *Mdm1* allele on the short arm of chromosome 6. The short arm of chromosome 6 is also responsible for resistance to all known strains of MDMV. McMullen and Louie (MPMI 2:309, 1989) initially placed *mdm1* in the interval between the distal marker, *umc85*, and the proximal marker, *bnl6.29*. A F2 population was generated using the MDMV resistant inbred Pa405 and a susceptible B73 inbred homozygous recessive for *polymitotic1* (*po1 mdm1*) to further characterize the map position of *mdm1*. F2 progenies were planted in the summer of 1991 disease nursery and inoculated with MDMV strain A (MDMV-A). DNA was prepared according to McMullen and Louie (1989) from whorl tissue of each F2 individual and genotype using RFLP markers on the short arm of chromosome 6. Plants were rated for resistance to MDMV-A three times throughout the growing season and scored for fertility. During the winter of 1991, field scores for resistance of some individuals were also confirmed by screening F3 progeny in the greenhouse.

The F2 mapping data cleared up several ambiguities in the reported map order of RFLP markers on chromosome 6S. The position of the NOR was found to map within the *umc85-bnl6.29* interval (Figure 1). The NOR probe, pZMS1, containing the 3.0kb spacer region, detects an *Eco*RI polymorphism in about 10% of the repeat units in Pa405. Because of the difficulty in scoring the heterozygotes (5% versus 10%) the NOR was scored as a dominant marker. Data from a Pa405/*y* M14 x *y* M14 backcross and analysis of F2 recombinants placed the NOR proximal to *umc85* and distal to *bnl6.29a* (Table 1). The *po1* locus was found to be tightly linked to *umc85* and we have yet to identify a recombinant between these two loci. Mapping of *mdm1* in our F2 population agreed with the initial placement of *mdm1* within the *umc85-bnl6.29* interval distal to the NOR (Figure 1). Our best estimate would place *mdm1* 0.5cM proximal to *umc85*. One difference between other 6S RFLP maps and our mapping data is that we have less recombination across 6S. Our F2 recombination data agree with the map distances produced from a Pa405/*y* M14 x *y* M14 backcross.

Table 1. Number of recombinant chromosomes between *po1*, *mdm1*, NOR, and RFLP markers on the short arm of chromosome 6.

Interval	# Recomb.	Interval	# Recomb.
A) Pa405 *Po1 Mdm1 Y1/po1 mdm1 y1* F2, n=185			
npi245--umc85/po1	4	*bnl6.29--npi235*	2
umc85/po1--mdm1	2	*npi235--npi101*	3
mdm1--NOR	1	*npi101--umc59*	13
B) Pa405 *Mdm1 Y1*/M14 *mdm1 y1* x M14 *y* BC1, n=952			
umc85--mdm1	2	*bnl6.29--npi101*	3
mdm1--NOR	1	*npi101--y1*	16
NOR--*bnl6.29*	3		

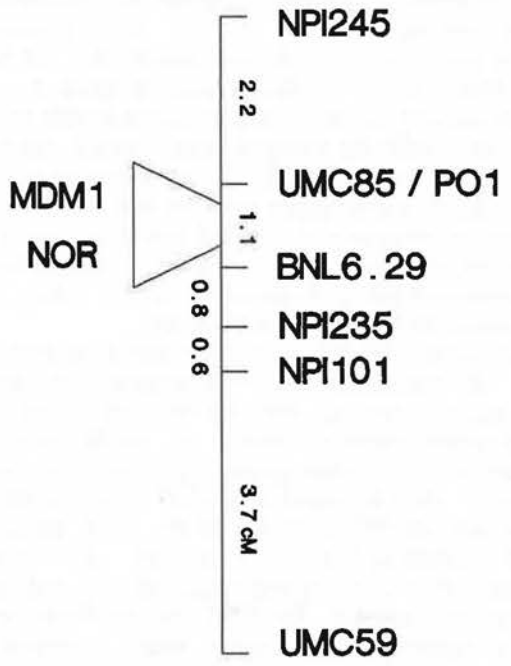

Figure 1. RFLP linkage map of the short arm of chromosome 6.

Wheat Streak Mosaic Virus. Although WSMV is an economically important pathogen of wheat, maize acts as an oversummering host. Only a few maize genotypes are susceptible to WSMV, and these lines are also susceptible to MDMV. These two viruses share approximately 38% protein coat similarity, but WSMV and MDMV differ in both symptoms produced in susceptible host and vector relationships. Resistance in maize to WSMV was first genetically defined by M. D. McMullen and R. Louie (Phytopath. 81:624, 1991) and appears to be conferred by at least two dominant genes, *Wsm1* and *Wsm2*. In crosses between Pa405 and the WSMV susceptible inbred Oh28, either gene is sufficient to confer resistance to WSMV. *wsm1* has been mapped to the short arm of chromosome 6 and is tightly linked to *po1/umc85* (data not shown). The mapping of *wsm1* near *mdm1* shows that these genes are either allelic or are tightly linked.

Cochliobolus heterostrophus. A recessive chlorotic-lesion type resistance to *C. heterostrophus*, causal agent of the southern corn leaf blight, was first identified in a Nigerian inbred in 1968 (J. Craig and J. M. Fajemisin, Plant Dis. Reptr. 53:742, 1969). D. Smith and A. Hooker proposed the symbol *rhm1* for the recessive chlorotic-lesion resistance and placed this locus on chromosome 6 using a series of *y1*-marked reciprocal translocations (Crop Sci. 13:330, 1973). Zaitlin and co-workers (Genome, in press, 1993) have mapped *rhm1* to the short arm of chromosome 6, near *umc85*. We are interested in determining the location of *rhm1* in relation to *mdm1* and the NOR. Two approaches were used to localize *rhm1*, B-A translocations which are used to identify loci distal to the A-chromosome breakpoint and near-isogenic line analysis (NIL's), which defines the chromosome segment retained during introgression of *rhm1*. The B-A translocation TB-6Sa was crossed as a male onto the inbred RB37 *rhm1* and the F1 progeny were screened for resistance to *C. heterostrophus*. If *rhm1* is distal to the NOR, resistant F1 progenies could result from fertilization of the egg (6, *rhm1*) by a sperm nucleus hypoploid (6B) for the region of chromosome six distal to the reported breakpoint of TB-6Sa in the NOR. Resistant progenies were recovered in the F1 and subjected to RFLP analysis. The markers proximal to the TB-6Sa breakpoint (*bnl6.29a* and *npi235*) detected both the RB37 *rhm1* and the TB-6Sa alleles in the resistant hypoploid progeny, verifying that the TB-6Sa breakpoint and the *rhm1* locus were distal to *bnl6.29a* (Figure 2, panel D and E, respectively). RFLP markers distal to the TB-6Sa breakpoint (*npi245*, *umc85*, and the *Eco*RI polymorphism detected with the rDNA spacer probe) were hemizygous for the RB37 *rhm1* allele in all of the hypoploid progeny (Figure 2, panels A, B, and C, respectively). The B-A translocation data indicate that the *rhm1* locus maps distal to the breakpoint in the NOR heterochromatin.

Near-isogenic line analysis of three *rhm1* conversions (RB37 *rhm1*, RH95 *rhm1*, and RVa35 *rhm1*) suggests that the chromosome segment retained from the Nigerian inbred 024-2-4, through at least 6 backcross generations, extends from the *Eco*RI polymorphism in the rDNA spacer region through *npi245*. The same RFLP allele is present in all *rhm1* converted lines when probed with *npi245*, *umc85*, and the rDNA spacer probe (Figure 2, panels A, B, and C, respectively). All recurrent parents were polymorphic with their respective converted *rhm1* line for these RFLP markers. The RB37 *rhm1* and RH95 *rhm1* conversions matched their respective recurrent parents at the *bnl6.29* and *npi235* loci, indicating that the introgressed chromosome segment from the Nigerian inbred 024-2-4 was entirely

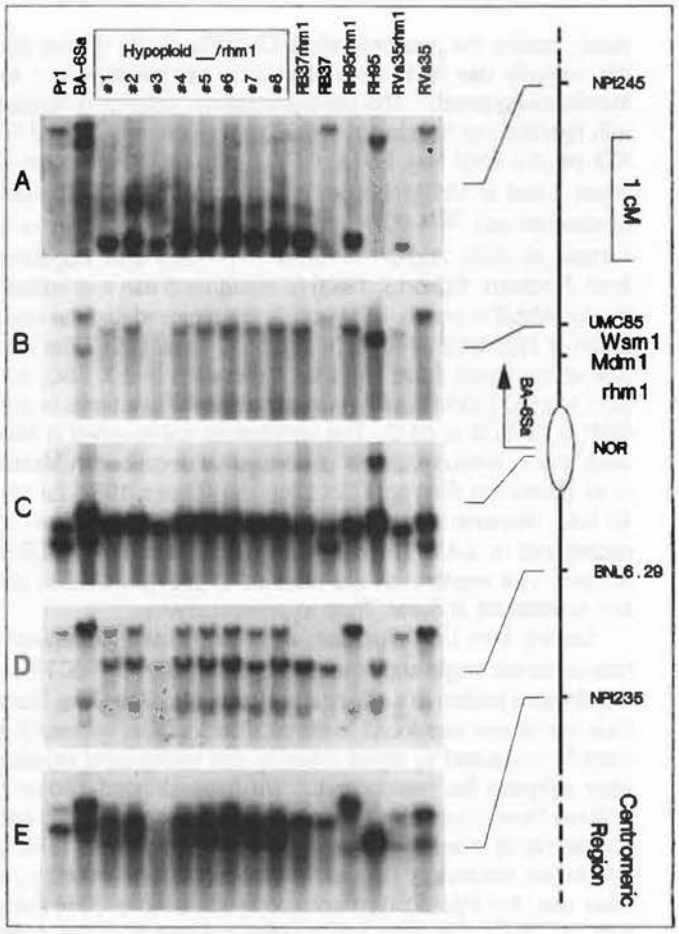

Figure 2. B-A translocation and near isogenic line analysis of *rhm1* conversions. DNA's represented in lanes 1 through 16 are: Pr1 and TB-6Sa/Pr1 (lanes 1 and 2), hypoploid F1 progenies of RB37 *rhm1*/TB-6Sa) lanes 3 through 10), and the *rhm1* conversion lines with the corresponding recurrent parents (lanes 11 through 16). Panels A through E represent the same hybridization membrane probed with *npi245* (panel A), *umc85* (panel B), pZMS1 (panel C), *bnl6.29a* (panel D), and *npi235* (panel E).

distal to *bnl6.29* (Figure 2, panels D and E, respectively). However, the introgressed segment in RH95 *rhm1* may extend through *bnl6.29* and *npi235*. Inoculation of the three *rhm1* conversion lines with MDMV-A, -B, and WSMV indicates that the introgressed segment from the original Nigerian source of *rhm1* carried both *mdm1* and *wsm1* as well as *rhm1*. We are currently crossing Pa405 and RH95 *rhm1* to map *mdm1* and *wsm1* in relation to *rhm1*.

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Mapping the *HtN* resistance gene to the long arm of chromosome 8
--Kevin D. Simcox and Jeffrey L. Bennetzen

Single gene resistance to *Setosphaeria turcica*, causal agent of the northern corn leaf blight in maize, is conditioned by four dominants, *Ht1*, *Ht2*, *Ht3*, and *HtN*. *Ht1* and *Ht2* have been previously mapped to the long arm of chromosome 2 (Hoisington and Coe, in Development and Application of Molecular Markers to Problems in Plant Genetics, pp. 19-24, 1989) and the long arm of chromosome 8 (Zaitlin et al., MNL66:69-70, 1992), respectively. The *ht3* gene has not been mapped, but was shown by Hooker (MNL55:87-88,

1981) to segregate independently of both *ht1* and *ht2*. No information is yet available on the map location of the *HtN* gene.

We are interested in studying interactions between genes that specify resistance to *S. turcica*. The identification of flanking RFLP markers would be useful in confirming allele status in segregating populations. To determine the map position of *HtN*, race 1 of *S. turcica* (avirulent on *HtN* and virulent on *Ht1* genotypes) was used to identify resistant and susceptible backcross individuals (W22 *HtN*/A619 *Ht1* x A619 *Ht1*) by greenhouse screening. Assignment of *HtN* to a linkage group involved selecting RFLP markers separated by approximately 50cM from each of the ten homologs and hybridizing RFLP clones to membranes containing DNA's from equal numbers of resistant and susceptible backcross progeny. Linkage to *HtN* was initially detected with *umc16*, located on the long arm of chromosome 3. Two duplicate loci were detected, *umc16a* and *umc16b*. Hybridization of chromosome 3 markers *bnl15.20* and *bnl10.24a* indicated that *HtN* was linked to *umc16b* (4cm, SE=3, n=45), but not linked to *umc16a* (48cM, SE=9, n=33) on chromosome 3. Helentjaris and co-workers (Genetics 118:296-299, 1988) mapped duplicate regions of the maize genome and found that the region around *umc16* on the long arm of chromosome 3 was duplicated on chromosome 8, near *idh1*. Linkage of *HtN* with *umc30a* (5cM, SE=3, n=60) and *umc117* (7cM, SE=5, n=27) placed *HtN* on the long arm of chromosome 8 in the region of *idh1*. An additional 99 backcross progeny were used to generate the following map order; *umc48* - 9cM - *umc30/umc117* - 1.1cM - *HtN*. The backcross (W22 *HtN/gl18 htN v16 j1* x *gl18 htN v16 j1* was used to map *HtN* with respect to morphological markers on the long arm of chromosome 8. No linkage was observed between *HtN* and *gl18*, however *HtN1* was found to be 13cM (n=336) proximal to *virescent16* and 30cM (n=112) proximal to *japonica1*.

When W22 *HtN* was crossed with A619 *Ht1* the expected 15:1 segregation was observed in the F2 (Table 1). An absence of independent segregation between *HtN* and *Ht2* was observed (Table 1), as expected, since Zaitlin and co-workers (1992) placed *Ht2* within the *umc48-umc89* interval, near *HtN*. The recovery of susceptible F2 progeny in the W22 *HtN*/A619 *Ht2* cross indicates that these genes are not allelic. The absence of linkage between *HtN* and A619 *Ht3* confirms the findings of Hooker (1981) that *Ht3* is not linked to the region on the long arm of chromosome 8 containing *HtN* and *Ht2* (Table 1). The markers flanking *Ht2* will be useful in confirming whether an inbred contains either *Ht2* or *Ht3*. Problems in identification arise since both of these genes have very similar phenotypes when inoculated with an avirulent race of *S. turcica*, and differential races have not been reported. Upon attempting to map *Ht3* out of RB37 *Ht3* using interval mapping we discovered that resistance in RB37 *"Ht3"* mapped near *Ht2* (K. D. Simcox and M. D. McMullen, unpublished data). Since both *HtN* and *Ht2* segregate independently of *Ht3*, we can only assume that our RB37 *"Ht3"* source contained *Ht2* and not *Ht3*. The contamination of *Ht3* material by *Ht2* has been seen by others (Dave Zaitlin, personal communication) and represents a serious problem, especially when inappropriate genotypes are used in disease surveys.

Table 1. F2 segregation data between *HtN* and the *Ht1*, *Ht2*, and *Ht3* genes.

Crosses	Observed R	S	Expected ratio	χ^2 value	P value
W22 *HtN*/A619 *Ht1* F2	148	11	15:1	0.12	0.50-0.75
W22 *HtN*/A619 *Ht2* F2	176	4	15:1	8.4	>0.005
	390	15	15:1	4.5	0.05-0.025
W22 *HtN*/A619 *Ht3* F2	188	12	15:1	0.02	0.90-0.95
	151	11	15:1	0.08	0.75-0.90
	152	3	15:1	4.64	0.05-0.025

This is a summary of selected genetic research information (new factors; mapping; cloning; trait inheritance) reported in recent literature and in this News Letter (numbers preceded by "r" refer 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.

aa = amino acid; BS = Base Sequence; BSH = Broad Sense Heritability; GCA = General Combining Ability; GxE = Genotype by Environment interactions; HR = Horizontal Resistance; NSH = Narrow Sense Heritability; PC = Personal Communication; QTL = Quantitative Trait Loci; R = Resistance or Reaction; RFLP = Restriction Fragment Length Polymorphisms; RI = Recombinant Inbreds; RM = Restriction Map; RS = Recurrent Selection; SCA = Specific Combining Ability; unc. = uncovered; VR = Vertical Resistance.
* with symbols identifies loci needing allelism tests, documentation, gene-specific clarification of the functional role, or standardization of the symbol.

CHROMOSOME 1

adh1 -10- *bnl7.25* -18- *npi238* -3- *bnl8.29* -12- *bm2* -3- *umc84* --67:33

adh1: BS, promoter, Anaerobic Response Element (ARE) binding protein (GCBP-1) --r504

adh1: BS, Z-DNA in promoter --r210

adh1: RM, BS, promoter dissection --r413

adh1-3F1124::Mu3, *adh1-3F1124r17*, genomic, RM, BS --r354

agrr22 - *bnl5.62* - *agrr152* - *bnl8.05* - *p1* - *bnl12.06* - *agrc587* - *agrc512* - *agrx1176* - *agrr34* - *bnl8.29* - *agrc707* - *umc84* - *bnl6.32*, QTL for regenerability near *bnl8.05, umc84* --r20

an1 probe vs. plant height QTL; ECB R QTL, 40% of height variation, Mo17/H99 F2; 16% of ECB R --67:6

anther culture and callus QTLs near *umc11, umc 167, umc128*, RFLP maps QTL --r723

asg59 -4.9- *asg31* -9- *umc94* -7.5- *bnl5.62* -12- *php20603* -10.8- *php20537* -9.2- *php20689* -16.4- *php20640* -16.6- *umc76* -7- *umc137* -6.1- *umc11* -13.1 - *umc13* -9.1- *bnl12.06* -5, 2- *php1122* -8- *bnl7.21* -13.1 - *umc133* -6.4- *umc67* -5.2- *php20682* -3.7- *php20654* -4.3- *php20674* -0.8- *php20575* -9- *bnl5.59* -6.3- *php20644* -9.5- *php20855* -9.9- *umc23* -2.2- *umc33* -1.1- *php20527* -4.7- *php20668* -1.8- *php20661* -4.7- *umc128* -4.9- *umc50* -3.4- *php20870* -10.5- *bnl8.10* -12.6- *umc107* -6.9- *php20518* -12.5- *bnl7.25* -12.4- *bnl8.29* -14.2- *umc84* -5.3- *php15058* -3.6- *php20557* -8.8- *bnl6.32*; *php20689* - *umc157* - *php20640*; *umc11* - *php20006*; *php20575* - *php20044* - *php20644*; *umc33* - *amp1* - *umc128*; *php20870* - *umc83* - *bnl8.10* - *npi120* - *umc107*; *umc76* - *(umc6, asg26, asg57, asg35b, asg45, asg69, asg75, asg3, asg78, asg11)*; *bnl5.59* - *asg62* - *umc128* - *(umc83, umc37, php2722)* - *php20870*; *umc107* - *(asg54b, asg68)* - *bnl8.29*; *umc50* - *npi238* - *bnl6.32* --67:56

bnl(tas1h) and *bnl(tas1c)*: end 1S, first report, telomere-associated sequence, subtelomeric; telomere-length QTL near *ynh20* --r90

bnl5.62 - *rpa(umc94a)* - *umc157* - *umc76* - *umc8a* - *umc67* - *bnl5.59* - *umc58* - *umc128* - *umc106* - *umc84* - *bnl6.32*, F2 vs. anther culture doubled haploids;disturbed segregations, recombination variations --r55

bnl5.62 - *umc94* - *php20537* - *php20603* - *php20689* - *umc76* - *php20640* - *umc11* - *umc13* - *bnl12.06* - *bnl17.21a* - *umc133* - *php20654* - *bnl5.59* - *php20682* - *php20674* - *php20575* - *php20644* - *php20044* - *php20855* - *umc37* - *umc23a* - *php20661* - *php20668* - *umc128* - *amp1* - *umc83* - *php20870* - *umc50b* - *bnl8.10* - *umc107a* - *php20518* - *bnl17.25* - *bnl18.29* - *php20557* - *umc84* - *bnl6.32*, plant height QTLs near *bnl12.06* and near *amp1* --r46

bnl5.62 -5- *umc164* -26- *umc115* -3- *sr1* -13- *zb4* -17- *umc76* -8- *umc162* -8- *umc11* --67:31

br2 near *npi272*, *bnl5.59* --67:109

Bz2 promoter, transient expression --r68

Bz2: genomic, RM, BS, promoter, transcriptanalysis, sense/antisense; stress-response (heat shock, auxin, PR) homology --r608

bz2::Mu1, *bz2::Mu9*, *Mu* reactivation by UV exposure --r717

cat2, *sod4* --response to cercosporin toxin --r752

ct2 near *umc157 npi97b* --67:109

d-3*, by RFLPs, on 1S? --67:82

D8: *bz2* -8.0- *umc140* -0.9- *Mu4-1* -1.8- (*umc107, bnl15.18, D8*) -0.4- *phyA1* -0.4- *Kn1* - *umc106* --67:110

f1, *bm2* linkage with QTLs *pd**, *tr** --67:22

hcf7: first report, uncovered by TB-1La, high chlorophyll fluorescence: defective processing of 16S rRNA --67:42

hm1: centr. - *php20644* -5- *hm1* -5- *php20044*; *br2* -0.1- *hm1* (P. Sisco, PC), *656, *1369, *1062, *Hm1-B73*, genomic, cDNA, RM, BS: L02540, HCTR, H. carbonum toxin reductase ; homology to NADPH-dependent dihydroflavonol-4-reductase (*a1*) --r325

Hm1: HC-toxin reductase, detoxifies HC-toxin --r453

hm1-656::Mu1, *hm1-1369::Mu3*, *hm1-1062*, *hm1-1040::Spm*, *Hm1*(B79), genomic, RM --r83

Kn1: RM --r271

Kn1: RM, *Mu* methylation, recombination --r409

Kn1-174, *Kn1-174a*, alteration of *Kn1-O* by insertion mutagenesis, excision and rearrangement --67:2

kn1-del: pollen lethal; hypoploid with TB-1La embryonic lethal --67:3

mdh4 with leaf number, plant height, yield QTLs, coded RFLPs --r183

npi234, *npi238*, yield, grain moisture, plant/ear height QTLs --r780

npi262 - *npi272* - *bnl5.59*, QTL for androgenesis near *npi262* --r132

npi411 -22- *npi354* -17- *npi428* -30- *npi453* -26- *npi429* -28- *amp1* -12- *npi447* -7- *npi255* -26- *bnl7.25* -9- *npi238* -3- *gdh1*, QTLs, heterosis, GxE --r660

npi447, *adh1* genomic/YAC --r181

P1: RM, *Ac* transposition and replication --r116

P1-pr: RM, allele: patterned pericarp and red cob; hypermethylated relative to *P1-rr* --67:95

P1-rr, RM, BS: Z11879, *Ac* insertion sites --r22

P1-VV reconstitution, RM, *Ac* transposition sites --r478

p1: *Ac* inserts, BS --r263

pgm1 - *adh1* - phi1, recombination variation with genotypes and environments --r692

php20537 -19.6- *php20640* -13- *umc11* -30.1- *bnl12.06* -31.9- *umc105b* -10.5- *php20654* -3.4- *bnl5.59* -11.4- *php2044* -8.3- *php20855* -12.3- *umc23* -8.6- *php20661* -30.9- *umc107* -13- *ope13.19* -20.4- *umc86* -3- *php20557* --67:80

QTLs vs. teosinte for ear disarticulation, lateral branch length, pedicellate spikelets near *umc83*, *umc107* --r156

stalk-rot (*Fusarium graminearum*, *Gibberella zeae*) R QTL, near *umc23* --67:81

T1-3(5597): T -20- *zb4* -4- *p1* --67:46

T1-3(5982): T -15- *zb4* -6- *p1* --67:46

T1-3k: *zb4* -4- *p1* -10- T --67:47

T1-4b: *zb4* -2- T -2- p1 --67:47

T1-5(6899): *zb4* -4- *p1* -3- T --67:47

tls1: first report, uncovered by TB-1Lc; near *bnl8.29*, *php15058*, *php20557*, *bnl6.32* (conclusion: in distal third of 1L), *tasselless1*: plants generally lack tassels, have ear shoots but no ear, variable; in some backgrounds, pubescent, leathery at 4-8 leaf stage (similar to *bs1* of Woodworth, stocks apparently lost; relationship to *bs1* of Micu unclear) --67:51

tub1 (=*tua1*), *tub2* (=*tua2*) (in tandem), PCR genomic sequence analysis indicates at least 7 genes in tubulin family --r476, r711

zb4 -9- *ts2* -2 - *p1* -11- *npi401* -4 - *npi214* -1- *umc167*; *php20575* -9- *php20644* -16- *php20855* -11- *br1* -9- *f1*; *zb4* -29- *umc76* -15- *npi286* -0 - *p1* -10- *npi262* -14- *umc67* -6- *bnl5.59*; *php20644* -1-

umc58 -7 - *php20855* -11- *br1* -6 - *f1* -12- *umc128* -3- *umc83*; *umc128* -7- *umc83* -3- *an1* -5 - *bnl8.10* -4- *umc184a(glb1)* -0- *umc140* -8- *umc106* -4- *npi255*; *umc140* -8- *umc106* -4 - *npi155* -9 - *gs1* -13- *npi238* -9 - *bm2* -9- *umc84*; *bnl12.06* -14- *npi262* -5 - *as1* -3 - *umc167* -6- *bnl5.59* -14- *php20644*; *php20644* -16- *php20855* -3 - *br1* -0- *umc33* -6 - *f1* -16- *bnl8.10* -11- *umc140* -5 - *umc107* -8- *adh1* --67:32

CHROMOSOME 2

(*npi417*, *npi254*) -34- *npi287* -21- *npi(b1)* -15- *npi297* -5- *npi456* -27- *bnl8.21* -10- (*npi465*, *npi59*) -6- *npi392*, QTLs, heterosis, GxE --r660

agrc938 - *agrc538* - *umc6* - *agrc593* - *agrr239* - *agrp62* - *agrc479*, QTL for regenerability near *agrr239*, *agrp62* --r20

anther culture and callus QTLs, near *umc6*, *umc34*, *umc131*, *umc5*, *umc49*, *umc36* --r723

B1, promoter dissection --r248

B1-Peru, *B1-I*, genomic, RM, BS, promoter, tissue specificity --r549

bnl(tas1a) end 2S, *bnl(tas1p)* and *bnl(tas1g)* end 2L, first report, telomere-associated sequence, subtelomeric --r90

bnl8.45 - *umc53* - *rpa(bnl7.49b)* - *umc6* - *umc34* - *umc8b* - *umc55* - *umc98* - *rpa(umc22)* - *rpa(umc125a)* - *rpa(bnl8.44a)* - *umc36* --r55

bnl8.45 -21.2- *umc53* - *opb01.4* -16.6- *umc34* -8.9- *php10033b* -2- *php10012* -1.8- *umc131* -9- *php20005* -13.7- *umc135* -33.5- *php20668* -45.8 - *umc36* -1.8- *umc93* --67:80

d-3685* associated with *b1* and *umc6*, prospective *Mu1*- associated allele of *d5* --67:111

et2 (was *et*-2352*): first report, uncovered by TB-2Sb, etched endosperm, off-white albino seedlings with occasional greening of leaf tips; revertant sectors occasionally --67:9

H4C14 subfamily: monosomic analysis, members of H4 histone subfamilies are located on at least 7 chromosomes --r114

hrg1 expression: ethylene, wounding; promoter dissection --r670

Hrg1-W22, *Hrg1-Ac1503*, *Hrg1-Zd*, genomic, BS: X63134, M36635, X64173, hydroxyproline-rich glycoprotein; sequence evolution, incl. *Z. diploperennis* --r557

lg1, *gl2*, *b1*, *v4* linkage with QTLs *pd**, *tr** --67:22

npi297, *npi239*, stalk/root lodging QTLs --r780

npi298, genomic/YAC --r181

NRase: 2 near centromere (D. Grant, PC), cDNA, NADH:nitrate reductase, EC1.6.6.1 --r95

o8: first report, *bnl12.09* -5- (*o8*, *umc134*) -10- *umc131*, opaque: increased lysine levels --67:102

php20568 -10.9- *umc53* -30- *umc6* -7 - *umc44* -10.2 - *umc61* -11.5 - *bnl10.42* -4.1 - *umc34* -1.9- *bnl8.04* -6- *umc134* -8.1- *php10012* -4.2- *umc131* -11.6- *umc139* -10.5 - *php20005* -11 - *php20017* -4.5- *umc4* -7.6 - *umc122* -7.7 - *bnl6.20* -19.7 - *umc12* -15.2 - *umc36* -7.5- *php20581* -7 - *php20622* -9.6- *bnl17.14*; (*php20568*, *bnl8.45*) - *umc53*; (*php20568*, *npi583*) - *php20005*; *bnl8.04* - *asg25* - *php10012*; *umc131* - (*asg28*, *asg29B*, *asg65*, *asg83*, *umc22* - *umc4* - (*asg23*, *asg77a*, *asg56*) --67:57

php20568b - *bnl8.45* - *umc53* - *umc61* - *umc34* - *php10012* - *umc131* - *umc139* - *php20005* - *php20017* - *umc4* - *bnl6.20* - *umc36* - *php20581a*, plant height QTLs near *umc61* and *umc131* --r46

plant height QTL, coded RFLPs --r183

QTLs vs. teosinte for ear ranking, glume induration near *umc53*, *umc34* --r156

RFLPs, 2L synteny with Sorghum, map order: *umc53 umc78 umc34 bnl7.25 php10012 npi565 jc0767a jc1410 agp2 jc0233 umc131 dek*-1047 umc98 jc0954 umc4 bnl8.44b* --67:7

stalk-rot (*Fusarium graminearum*, *Gibberella zeae*) R QTL, near *php10033b* --67:81

Wrp1 linked with *wx1* T2-9c(2S.33) (23%), T2-9b(2L.33) (18%), T2-9d(2L.83) (19%) --67:33

CHROMOSOME 3

anther culture and callus QTLs, near *bnl5.37*, *umc60*, *umc16*, *umc96* --r723

bnl(tas1b) end 3S, first report, telomere-associated sequence, subtelomeric --r90

bnl5.37 - *npi108* - *bnl15.20* - *umc98*, QTL for androgenesis between *bnl5.37* and *npi108* --r132

bnl8.15 - *agrr116a* - *agrr206* - *agrr184b* - *agrp40* - *bnl15.20* - *agrr144a* - *a1* - *agrc638*, QTL for regenerability near *agrp40* --r20

bnl8.15 - *e8* - *php20042* - *php12006* - *bnl8.35* - *umc10* - *php20576* - *php20511* - *php20558* - *php20509* - *php20802* - *bnl13.05* - *php20508* - *umc26* - *bnl5.37* - *bnl10.24a* - *umc82* - *umc3* - *bnl6.16* - *php15033* - *php20521* - *umc7* - *umc16* - *php1a10a* - *php10080* - *php20726*, plant height QTLs near *php12006* and near *bnl5.37* --r46

bnl8.15 -50.9- *bnl8.35* -11.6- *bnl6.06* -12.1- *php20558* -3.9- *umc42* -17.3- *php20802* -37.9- *opb8.13* -10.6- *ope13.7* -7.8- *bnl6.16* -4.1- *php15033* -15.4 - *php20521* -23.9- *php10080* --67:80

bnl8.15 -8.2- *php20905* -3.2- *e8* -5.8- *umc121* -19.7- *php20042* -13- *php12006* -7.5- *bnl8.35* -10.5- *php20576* -4.6- *php20509* -2.7- *php20558* -4.2- *php20511* -2.7- *umc10* -5.7- *php20508* -6.2- *umc26* -9.4- *bnl5.37* -3.6- *bnl10.24* -10.9 - *umc60* -8.4- *umc82* -8.2- *bnl6.16* -2.8- *php15033* -5.9- *umc3* -8.6- *bnl1.297* -4.5- *php20521* -4.2- *umc17* -4.1- *php1106* -9.6- *php10080* -7.5- *umc63* -9.4- *umc96* -3.2- *php20726* -6.2- *npi457*; *bnl8.35* - (*php20017*, *umc42*); *php20508* - *umc51* - *umc82* - *bnl15.20* - *php15033*; *bnl8.35* - *bnl13.05* - *php20558*; *php20511* - (*php20903*, *umc18*) - *bnl10.24*; *php20576* - (*php20802*, *php20797*) - *umc26*; *php20508* - *gst-III* - *bnl10.24*; (*bnl8.15*, *asg64*) -*umc121* - (*asg16*, *asg24*, *asg48*, *php1544*, *asg46b*, *umc92*) -*umc10* - (*umc102*, *asg67a*, *asg1a*, *asg39*, *asg15*, *asg10*, *asg4*) - *umc63* --67:57

Cg2 -39 - *ra2*; -46- *lg2* --67:65

e8 -24 - *npi249* -19- *npi446* -20- *npi296* -15- *npi52* -23- *npi212* -26- *a1* -11- *npi457* -2- *npi425*, QTLs, heterosis, GxE --r660

lg2, *a1* linkage with QTLs *pd**, *tr** --67:22

Lg3 encodes homeodomain protein --r637

pgd2-null, *pgd2-125null*, *pgd2-86null*, EC1.1.1.44, expression and distribution of 6-PGD --r34

plant height QTL, coded RFLPs --r183

pollen tube growth rate QTLs near *bnl6.06*, *pgd2*; pollen germinability QTLs near *bnl8.35*, *bnl6.06* --r599

QTLs vs. teosinte for pedicellate spikelets, lateral branching near *umc92*, *umc42b*, *umc18a* --r156

Sh2-BMS, genomic, BS: M81603 --r621

sh2-m1::Ds, BS, revertant sequences --67:44

stalk-rot (*Fusarium graminearum*, *Gibberella zeae*) R QTL, near *php20802* --67:81

T1-3(5597): *ts4* -16 - *lg2* -2- T -24- *a1* --67:47

T1-3(5982): *ts4* -31 - *lg2* -8- T -11- *a1* --67:47

T1-3(8995): T -3- *ts4* -21- *lg2* -33- *a1* --67:47

T1-3k: *ts4* -18 - (T, *lg2*) -25- *a1* --67:47

umc16, *umc92*, *umc96*, yield, grain moisture, ear height QTLs --r780

umc32 - *umc121* - *rpa(umc161)* - *umc10* - *bnl6.06* - *umc60* - *umc3* - *umc63* - *umc96* - *rpa2* --r55

Vp1, activation of *C1* promoter --r279

y10 13.4 units distal to *lg2* --67:18

CHROMOSOME 4

agrc94 - *agrp67* - *agrr109* - *agrr27* - *agrc300* - *bnl15.07* --r20

Als1-QJ22, *Als1-XS40*, first report, on 4 near centromere by *wx1* translocations, acetohydroxy acid synthase (AHAS), imidazolinone R selected in vitro; assume same as *dup(als1)* RFLP locus --r496

anther culture and callus QTLs, near *umc42* --r723

bnl(tas1e) end 4S, *bnl(tas1o)* end 4L, first report, telomere-associated sequence, subtelomeric; telomere-length QTL near *bnl8.23* --r90

bnl15.07, yield QTLs --r780

bnl15.45 -4- *npi267* -36- *npi292* -7 - *npi208* -10- *npi444* -3- *npi104* -16- *npi317*, QTLs, heterosis, GxE --r660

bnl5.46 -9.6- *umc49* -8.9- *bnl15.45* -7.5- *ope10.1* -10.5- *ope10.45* -17.5- *umc66* -20.9 - *php20071* -5 - *php20562* -15.7 - *php10025* -27.5 - *bnl15.07* -16.9- *php20563* --67:80

cAc-11, near *aco1*, *pcAc-11*, genomic, RM, BS, cryptic *Ac* --r388

cat3, response to cercosporin toxin --r752

d-3*, by RFLPs, on 4L? --67:82

ECB R QTL, near *bx1*, 17% of variation, Mo17/H99 F2 --67:6

gl3-15, new allele designation for 6 sources; non-complementing with *gl15* --r647

o12 uncovered by TB-4Sa, potential allelism to *dek7* to be determined --67:10

orp1 (probe TSB1), *bnl15.45* - TSB1 - *bnl7.20L*, pZmTSB1, cDNA, BS: M76684, tryptophan synthase beta --r763

pep3, H1r22, pMR15, PEP carboxylase, root-form --r344

pollen germinability QTLs near *zpl1a*, *zpl1d*, *zpl2a* --r599

prh1, first report, ZmPP1, cDNA, M60215, Type 1 protein phosphatase (serine/threonine phosphatase), *E. coli* expression, similarity to corresponding animal enzyme; Southerns indicate multigene family --r639

QTLs vs. teosinte for glume induration near *bnl5.46*, *umc42a* --r156

stalk-rot (*Fusarium graminearum*, *Gibberella zeae*) R QTL, near *bnl15.45* --67:81

su1, *ts5* linkage with QTLs *pd**, *tr** --67:22

ubi1: plambdaUbi-1, genomic, RM, BS, polyubiquitin(7mer), *S1* mapping, transgenic expression, heat shock --r120

umc123 -14 - *php20725* -16.6 - *umc31* -11.5 - *bnl5.46* -20.2 - *umc42* -5.6- *bnl15.45* -6.1- *php20597* -12.3 - *umc66* -4.1 - *umc19* -7.5- *php20595* -10.7- *bnl7.65* -4- *umc158* -7.4- *php20071* -4.7- *umc15* -4.4- *php20562* -6.3- *umc52* -8- *php10025* -18- *php20608* -9.4- *bnl15.07* -7.8- *umc111* -2.2- *bnl8.23* -8.7- *php20563* -6.7- *umc10*; *php20725* - *umc87* - *bnl5.46* - (*umc47*, *umc23*, *umc156*) - *umc66*; *php20595* - *bnl10.05*- *umc158*; *bnl5.46* - *php1106* -*bnl15.45*; *bnl5.46* - *mals* - *umc19*; *bnl5.46* - (*asg76*, *asg38*) -*umc66*; *umc19* - (*bnl5.67*, *asg33*, *asg9a*, *asg27a*, *asg84*, *asg21*, *asg22*) - *asg41A* -*bnl8.23* --67:58

umc31 - *bnl5.46* - *umc156* - *umc19* - *rpa(umc127)* - *rpa(bnl7.65)* - *rpa9b(ssu)* --r55

umc87 - *php20725* - *php20713* - *umc31* - *bnl5.46* - *php1a10b* - *php20597* - *umc47* - *umc23b* - *bnl15.45* - *umc42* - *umc66* - *umc19* - *bnl7.65* - *php20071* - *php20562* - *umc15* - *umc52* - *php10025* - *php20608* - *bnl8.23* - *php20563*, plant height QTLs near *umc42* --r46

Zpr10/(22), maternal/dosage effect --r459

CHROMOSOME 5

ae1 -7.4- *pr1* -0.64 - *lw2* -0.32- *gl8* --67:9

agrc595 - *agrc669* - *agrx43* - *agrr199* - *agrr127* - *agrr215* - *agrx701* - *agrr252* - *agrr211* --r20

Als2-XA17, *Als2-XI12*, first report, on 5L by *wx1* translocations, acetohydroxy acid synthase (AHAS), imidazolinone R selected in vitro, XA17 is R to sulfonylurea but XI12 not; assume same as *dup(als2)* RFLP locus --r496

am1-pra allele of *am1*, was *pra1*; dominance series *Am1* > *am1-pra* > *am1-ref* --r249

anther culture and callus QTLs, near *umc43*, *umc126* --r723

bnl(tas1n) and *bnl(tas2b)* end 5S, first report, telomere-associated sequence, subtelomeric --r90

bnl6.25 - *umc147* - *umc27* - *bnl5.02* - *bnl7.71* - *umc126* - *rpa(umc39b)* - *umc51* - *umc68* - *umc104* --r55

Bt1, *bt1-m*, pBt1cDNA1.7, cDNA, genomic, BS: M79333, M79334, northerns; possible adenylate translocator in amyloplasts --r665

cat1, response to cercosporin toxin --r752

d-6*, by RFLPs, on 5S --67:82

D9 = *D*-2319*, associated with RFLPs on 5S in region that is duplicated on 1L --67:111

ms42 near *bnl6.25*, *umc50*, male sterile: dominant *Ms42-2082* plants male sterile; penetrance affected by background --67:64

na2 near *umc27a umc166* --67:109

npi288-Mo17, *npi288-B73*, BS, allele-specific PCR --r620

npi409 -23- *npi282* -12- *pgm2* -29- *npi256* -12- *amp3* -5 - *npi449* -39- *npi458* -27- *npi288*, QTLs, heterosis, GxE --r660

pep2, H1r22, pMR15, PEP carboxylase, root-form --r344

pollen tube growth rate QTLs near *bnl10.06*, *bnl7.43*; pollen germinability QTLs near *bnl7.43*, *bnl4.36* --r599

pr1 linkage with QTLs *pd**, *tr** --67:22

QTLs vs. teosinte for ear disarticulation near *bnl5.02* --r156

stalk-rot (*Fusarium graminearum*, *Gibberella zeae*) R QTL, near *bnl7.71* --67:81

ubi2, plambdaUbi-2, genomic, RM, BS, polyubiquitin(7mer), *S1* mapping, transgenic expression, heat shock --r120

umc27, genomic/YAC --r181

umc86 -19.7- *bnl8.33* -11.3 - *bnl6.25* -25.6 - *umc72a* -24.3- *umc50* -5.1- *bnl5.02* -11.2 - *bnl7.56* -3.1 - *php20872* -8.1 - *umc43* -3.2- *umc1* -1.3- *php06012* -2.9- *php15024* -3.2- *php15018* -6.1 - *umc40* -5.6- *php20589* -9.7 - *php10014* -4.8- *bnl5.71* -6.7- *bnl15.27* -8.5- *umc126* -6.1 - *umc54* -5.7 - *umc51* -4.3- *php20531* -7.7 - *php20566* -8.4- *umc108* -13.9 - *umc68* -13.4 - *bnl5.24* -9.2- *php20909* -2.2- *php20523* -8.9 - *umc104* -8.4 - *php10017*; (*umc86*, *umc59*, *php20045*) - *bnl6.25* - (*php1163*, *umc90*) - *umc50*; *php20872* - *bnl6.10* - *php06012*; *umc126* - *umc26* -*php20531*; *umc108* - *php1544*- *bnl5.24*; (*umc86*, *npi409*) -*umc72a* - *umc107* - *bnl5.02*; *um40* - (*php20715*, *bnl7.71*, *umc138*) -*php10014*; *umc72a* - *php20898* - *bnl7.56* - *php1477* - *umc43* - *bnl6.22 php20589* - *MALS* - *bnl5.71* - *bnl5.40*; *umc68*; (*umc86*, *umc147*, *asg54a*, *asg73*, *asg55*) - *umc1* - (*php1550*, *asg43*, *asg66*, *asg71*, *asg29a*) -*umc108* - *asg27b* - (*asg85*, *asg9B*, *asg74*, *php10017*) --67:58

umc86b - *bnl8.33* - *bnl6.25* - *umc90* - *umc72a* - *umc107b* - *pgm2* - *php20898* - *bnl7.56* - *php20872* - *php20622* - *umc43* - *php06012* - *php15024* - *php15018* - *php20715* - *bnl7.71* - *php20589* - *php10014* - *amp3* - *bnl5.71* - *bnl5.40* - *umc54* - *php20531* - *php20566* - *php12026* - *umc108* - *umc68* - *bnl5.24* - *php20909* - *php20523* - *php10017* - *umc104*, plant height QTLs near *bnl7.56* and near *bnl5.72* --r46

umc90 -16.3- *umc27* -10.8 - *bnl5.02* -11.4 - *bnl6.10* -14.6 - *bnl7.71* -16- *bnl5.71* -15.1 - *umc49m* -31.5- *umc49u* -33- *umc104* --67:80

CHROMOSOME 6

agrp144 - *agrr87* - *agrr47* - *agrr189* - *agrr37* - *agrr118a* - *bnl15.37* --r20

anther culture and callus QTLs, near *umc59*, *umc65*, *umc38*, *umc132*, *umc134* --r723

bnl6.29 - *php20527* - *php20528* - *umc59* - *php20854* - *php20045* - *php06007* - *umc65* - *umc113a* - *umc21* - *m3-4* - *umc46* - *php10016* - *bnl15.37* - *umc138* - *php20904* - *umc62* - *mdh2* - *php20595* - *php20569* - *php20599*-, plant height QTLs near *php06007* --r46

bnl6.29 -6.6- *php20527* -2.9- *php20854* -4.3- *umc59* -7.4- *ph20045* -18.7- *umc65* -11.7- *umc21* -7- *bnl3.03* -13.5- *bnl15.37* -4.5- *umc46* -10.7- *bnl5.47* -14.4- *umc138* -14.2 - *php20904* -11.1 - *umc132* -23.2- *umc62* -5.1- *php20595* -3.5- *php20599* -11.7 - *npi280*; *umc85* - *bnl6.29* - (*php06007*, *php20719*) -*php20045*; *bnl5.47* - *php10016* - *php20904*; (*bnl6.29*, *php20528*) -*php20854*; *php20045* - *umc113* - *umc21*; (*asg79*, *asg40*) - *bnl6.29*; *umc21* - (*asg51*, *asg50*, *asg6a*) -*umc132* - (*asg18*, *asg36b*, *asg14b*, *asg47*, *umc28*, *npi280*) --67:59

bnl6.29, *umc85*, genomic/YAC --r181

cps2 (was *hcf133*): first report, uncovered by TB-6Lc, chloroplast protein synthesis: pale green/yellow green, nonviable, decreased levels of thylakoid membrane complexes (2-fold) and RUBISCO (20-fold), and decreased assocation of all chloroplast mRNAs with polysomes; allele *cps1-2* more severe than *cps1-1*; from *Mu* screening --67:42

d-9*: TB-6S uncovers, complements *d1, d2, d3, d5, d*-3, d*-4, d*-6, d*-8* --67:82

enp1 with plant height QTL, coded RFLPs --r183

idh2 - mdh2, recombination variation with genotypes and environments --r692

l12 uncovered by *Df* between T6-9e/T6-9(043-1), T6-9(6019)/T6-9(043-1), T4-6(8428)/T4-6(6623), conclude *l12* is most likely between 6L 0.28 and 0.36 --67:65

l10, ms1, si1 uncovered by *Df* between T6-9e/T6-9(043-1), conclude each is most likely between 6L 0.28 and 0.36 --67:65

Mdm1: *npi245* -2.2- (*umc85, po1*) -1.1- *bnl6.29* -0.8- *npi235* -0.6- *npi101* -3.7- *umc59; umc85 - wsm1 - mdm1 - rhm1* - (TB-6Sa, NOR) - *bnl6.29a* --67:117

Ms21: stocks lost --67:64

npi377 -17- *npi393* -12- *npi223* -25- *npi252* -11- *bnl5.47 - mdh2*, QTLs, heterosis, GxE --r660

pgd1-null, EC1.1.1.44, expression and distribution of 6-PGD --r34

php06007 -27.7- *bnl6.29* -12.6- *umc59* -19.5- *umc65* -11.6- *opg05.8* -17.8-*php20608* -22 - *umc38* -27- *umc62*

Pl1'-mah, paramutagenic variant; alters *Pl1-rh* but not *pl1* --67:42

pollen tube growth rate QTLs near *npi223* --r599

rhm1: TB-6Sa uncovers *rhm1, npi245, umc85* but not *bnl6.29a* or *npi235*; *rhm1* distal to NOR --67:118

rpa(umc159c) - umc85 - umc65 - umc21 - umc38 - umc132 - umc62 - --r55

T6-9(5454) + B-6Ld, tertiary trisomics --r351

umc21, umc38, yield QTLs --r780

Wsm1 tightly linked to *po1* and *umc85*, wheat streak mosaic virus resistance: one of at least two dominant genes (see *wsm2*) conferring resistance; allelic to *mdm1* or tightly linked (McMullen & Louie, Phytopath. 81:624, 1991) --67:118

y1 uncovered by *Df* between T6-9e/T6-9(043-1), T6-9(6270)/T6-9(043-1), T6-9(6019)/T6-9(043-1), T4-6(055-8)/T4-6(6623), T4-6(8428)/T4-6(6623, T6-10b/T6-10d, but not T6-9(6270)/T6-9(6019) or T4-6(055-8)/T4-6(8428), conclude *y1* is most likely between 6L0.28 and 0.31, though possibly between 6L 0.12 and 0.16 --67:65

y1, pg11 linkage with K6 --67:25

y1, py1 linkage with QTLs *pd*, tr** --67:22

CHROMOSOME 7

agrc261 - agrr128 - agrr49 - agrr73 - agrc701 - agrr186 - agrr202 - agrc525 --r20

anther culture and callus QTLs, near *bnl16.06* --r723

gl1-m8, reversions, cell lineage shoot apex --r75

hcf103-114, blocked in plastoquinone-9 --r128

ij1: genomic, RM, BS: S15063, Southerns, northerns; revertant modifications; no protein homology recognized --r275

in1 mutables from *En/Spm* plot --67:4

nbp1, first report, on 7L, G1a, cDNA, RM, BS: Z11488, M74566, nuclear-encoded chloroplast nucleic acid-binding protein, light-dependent, organ-specific induction; one band in Southerns and northerns --r129

npi277 -7- *npi216* -13- *npi394* -15- *npi283*, QTLs, heterosis, GxE --r660

npi391, ear diameter QTLs --r780

O2 regulation of lysine-ketoglutarate reductase (LSK; EC1.5.1.8) --r84

o5-3038, uncovered by TB-7Lb, pale yellow/sugary, embryo lethal viviparous from *Mu* population; *o5-ref/o5-3038* kernels pale yellow, seedlings yellow-green --67:9

opm082000, 28 cM distal to *php20581* --67:16

pep4: *npi30* -2.6- *pep4 - npi112*, pMR32, cDNA, RM, BS: X61489, C3-PEP carboxylase, EC4.1.1.31 --r344

php20581 -23.8- *bnl15.40* -8.8- *umc98* -4.7- *umc116* -6.9- *bnl15.21* -7- *php15037* -6- *php20708* -1.6- *php20684* -4.7- *umc110* -4.2- *php20746* -6.6- *umc56* -10.3- *bnl8.32* -10.9- *bnl8.39* -15.9- *php20909* -4.9- *php20690* -10.9- *bnl8.44* -13.4- *php20020* -17.2- *php20728* -32.1- *umc35; umc116 - php20569 - php15037; umc56 - (tyk30, bnl14.07, umc125) - bnl8.39 - (php20593, bnl16.06) - php20690 - php20890 - php20020; (php20581, asg8, asg34, asg70) -umc116 - asg72 - - bnl15.21 - (bnl4.24, asg8*, 0 *asg5, asg32, bnl7.61, asg14a, asg36a, umc91, umc35)* --67:59

php20581 -55.9- *bnl8.39* -7.8- *bnl8.32* -14.2- *umc111* -7.7- *umc110* -6.3- *php15037* -6.7- *php20569* -13.9- *php20684* -31.6- *php20690* -3.9- *php20593* -17.3- *php20020* -19.7- *umc35* --67:80

php20581b - php20569 - umc116 - php15037 - php20708 - php20684 - php20746 - umc110 - bnl8.32 - umc125b - bnl8.39 - php20593 - php20690 - bnl8.44 - php20020 - php20728, plant height QTLs near *php20569* --r46

pollen tube growth rate QTLs near *npi400, o2, bnl15.40, zp50, zpb36* --r599

RFLPs, synteny with Sorghum, map order: *jc0678 jc1362 jc0767b bnl15.40 bnl15.21 jc0185 umc110 jc0446 bnl7.61 bnl8.37 bnl14.07 dek*-326 bnl8.39 jc0943 jc0878 bnl8.44a umc35* --67:7

rpa(umc39c) - rpa(umc112a) - bnl15-40 - umc116 - umc110 - rpa(umc111) umc149 - umc125b - bnl7.61 - umc80 - umc151 - bnl8.44b - umc35 --r55

sh6-8601 allelic to *sh6-1295*, not to *cp2*, shrunken, opaque kernel like *sh1*; seedlings pale green, poor viability to lethal; *sh6- 8601* less severely expressed --67:10

sod2, response to cercosporin toxin --r752

Tp1: *gl1* -8.7- *umc116* -3.9- *php20569* -0.8- *tp1* -1.6- *bnl15.21* -6.3- *php15037* -0.4- *sl1* --67:91

CHROMOSOME 8

agrr169 - agrr116b - agrr222 - agrr144b - agrr184a - agrr269 - agrr262 - agrr21 --r20

anther culture and callus QTLs, near *umc120, umc117, umc93, umc7* --r723

*bif1-**, probable recessive *Bif1* allele --67:2

bnl(tas1m) end 8L, first report, telomere-associated sequence, subtelomeric; *npi201b - bnl17.17*(CCCTAAA) - *umc71*, telomere-like sequence, internal --r90

bnl13.05 -14 - *php10040* -3 - *bnl9.11* -12.3 - *umc103* -15.4- *bnl10.39* -11.9 - *bnl9.08* -9.2- *php20714* -10- *bnl2.369* -6.2- *umc89* -6.6- *umc12* -7.2- *bnl12.30* -7- *umc93* -6.8- *umc30* -6.8- *umc117* -8.2- *bnl10.24* -21.9 - *php20793* -3.8- *umc7* -13.4 - *php892; bnl9.11 - umc120 - bnl10.39; umc12 - umc48 - umc93 - umc82 -php892; umc103 - php20727 - bnl9.08; umc7 - (umc3, php892); bnl13.05 - asg53 - umc103 - asg6b umc89 - asg13 - umc93 - (asg67b, asg52b, asg1b, asg17, php892)* --67:60

bnl13.05a - php10040 - bnl9.11 - umc103 - bnl9.08 - bnl10.39 - umc120 - php20714 - umc12 - bnl12.30 - bnl10.24b - php20793 - umc7, plant height QTLs near *bnl9.08* --r46

bnl6.25 -11.1- *php10040* -20.4 - *umc120* -18.1- *opg05.2* -2 - *umc89* -11.7 - *bnl5.62* -50.2- *umc7* --67:80

ct1 near *npi110, umc32b* --67:109

gl23 allelic to *gl18* --67:10

hox1, first report, 8L near *umc89*, RB11, cDNA, BS, Zmhox1a polypeptide, homeobox, DNA binding transcriptional regulator with affinity for *sh1* feedback control element; Southerns show one band --r51

htn1, first report, *umc16b* -4±3- *HtN; umc48* -9±3- (*umc30, umc117*) -1.1±1- *HtN; HtN* -13- *v16; HtN* -30- *j1*, northern corn leaf blight resistance (HtN): *Setosphaeria turcica* resistance --67:119

idh1 with plant height, internode length, node number, yield QTLs, coded RFLPs --r183

npi114 -34- *npi110* -25- *bnl1.45* -21- *npi426* -36- *tpi5* -32- *npi107*, QTLs, heterosis, GxE --r660

pet1 (was *hcf121*), first report, uncovered by TB-8Lc, cytochrome f/b6: pale green/yellow green, nonviable, lack protein components of

cytochrome f/b6 complex; from *Mu* screening --67:42

pollen tube growth rate QTLs near *bnl7.08*, *act1*; pollen germinability QTLs near *npi220*, *bnl13.05*, *bnl9.11*, *niu1*, *bnl9.44L*, *bnl9.08*, *bnl1.45*, *bnl7.08*, *act1*, *bnl2.369*, *bnl10.24b*, *npi268* --r599

psb1 (was *hcf134*), renamed, photosystem II: pale green, nonviable, PS II complex absent; from *Mu* screening, mutable --67:42

rip1, first report, ZmcRIP-3, ZmcRIP-9, cDNA, BS: M83926, M83927, *b-32*, ribosomal-inactivating protein, aa sequence --r44

rip1: *o6* -4- *rip1* (= *b-32* locus; was *alb1*) --r569

rip1? (possible repeat clone; no linkage data), first report, cDNA, BS: M77122, ribosome inactivating protein --r720

rpa(bnl13.05), *bnl9.11 - umc103 - bnl9.44 - rpa(bnl12.30) - rpa(umc30) - rpa(umc150a)* --r55

*ubf**, *uwo1(ubf)* near *npi438*, *umc7*, scMubG7-J, genomic, ubiquitin fusion protein --67:75

umc48, genomic/YAC --r181

CHROMOSOME 9

acp1 with plant height, yield QTLs, coded RFLPs --r183

agrr118b - bz1 - agrr205 - bnl5.04 - bnl8.17 - css1 - agrr90 - agrc595 - bnl5.09, QTL for regenerability near *agrc595* --r20

bz1-m13, pcs64SB, pcs3XB, genomic, RM, BS, alternative splicing --r503

bz1-m13::dSpm, change of state alleles CS-A, CS-B, CS-C, CS-E --67:44

C1, dissection of promoter activation by *Vp1* product --r279

C1-bk ('breaker') from *Dt* plot --67:5

d-3010* between *bnl3.06* and *umc81*, prospective *Mu1*- associated allele of *d3* --67:111

H3C3 subfamily, monosomic analysis, members of H3 histone subfamilies are located on at least 7 chromosomes --r114

npi253 -25- *bz1* -25- *wx1* -6- *bnl5.10* -4- *npi25* -19- *npi427*, QTLs, heterosis, GxE --r660

npi416 - umc81 - bnl7.13 - bnl14.28, QTL for androgenesis near *umc81* and *bnl7.13* --r132

pep1, C4-PEP carboxylase --r344

php10005 - sh1 - umc113b - wx1 - php20052 - umc81 - umc114 - css1 - php20554 - bnl8.17 - umc95 - bnl7.21b - bnl4.28 , plant height QTLs near *wx1* --r46

pollen tube growth rate QTLs near *sh1*, *bnl5.04*, *bnl7.12*; pollen germinability QTLs near *npi253*, *c1*, *sh1*, *bz1*, *acp1*, *bnl5.04*, *bnl7.13* --r599

Rld1: centr. - *npi209* -12- *npi97* -14- *Rld1* --67:33

Trn1-1597, first report, linkage with all *wx1* 9-translocations; *wx1 - Trn1* 16+/-2.7 cM, *torn1*: dominant *Trn1* plants have chlorotic and adherent leaf tissue, which becomes green and healthy after some sunlight exposure but smaller plants with torn leaves result --67:33

umc105a -23.6- *bnl5.10* -1.3- *umc114* -8.1- (*umc95*, *bnl8.17*) -3- *php20554* -10.6- *bnl5.09* -8.3- *opg05.6* --67:80

umc109 -8.8- *php10005* -13.7- *umc113* -7.6- *bz1* -22.1- *wx1* -4.9- *php1460* -6.6- *bnl5.10 php20052* -2.2- *umc81* -3.6- *umc114* -1.4- *bnl5.04* -7.3- *css1* -2.2- *php20554* -4.3- *bnl8.17* -2.7- *umc95* -8.3- *umc140* -27.5- *bnl5.09* -3.3- *bnl14.28; (umc109, umc148) -php10005 - sh1 - bz1; php10005 - bnl3.06 - umc81; php10005 - umc20 - umc114 - bnl7.13 - bnl14.28; (umc109, asg19, asg82, asg63) - bnl5.04 - (asg30a, asg35A, asg44, asg12, umc94, bnl14.28)* --67:60

umc81 - umc153 - rpa(umc112b) - umc114 - umc95 - bnl5.09 - rpa(umc94b) --r55

wd1-Mu, 11 isolates all allelic inter se and with *wd1-ref* and *pyd1-ref* --67:8

wx1, T linkage with QTLs *krn**, *fas** --67:25

wx1-B2, BS --r89

wx1-B3, *wx1-B3-S1*, RM, BS, *Ac* excision --r40

wx1-m1, alternative splicing --r740

Wx1-m5: genomic , RM, BS, intragenic *Ds* transposition --r737

wx1-m9::Ds-cy, *wx-m9::Ac*, methylation --r81

wx1-Mo17, *Wx1-Mo17*, *Wx1-A619*, *Wx1-B73*, *Wx1-C103*, *wx1-st*, *wx1-B*, *wx1-B73*, BS, allele-specific PCR --r620

wx1-Stonor, *wx1-g*, *wx1*-B5, genomic, cDNA(PCR), RM, BS, alternative RNA splicing retrotransposon --r699

CHROMOSOME 10

bnl10.17 - agrc561 - agrr57 - agrc459 --r20

bnl10.17 -18.7- *php20626* -5- *php20753* -2.3- *bnl3.04* -27.9- *umc130* -4.7- *php20646* -1.4- *umc18* -2.9- *php06005* -5.3- *umc155* -5.7- *php15013* -4 - *php10033* -7.9- *php20719* -17.4- *umc44* -2.7- *umc57* -4.1- *bnl10.13* -12.2- *bnl7.49* -12.8- *php20568; php20753 - php20075 - umc130; umc155 - umc146 - php20719; bnl3.04 - glu1 - php20646; umc155 - (asg2, asg42) -umc44 - asg81 -bnl7.49* --67:61

bnl10.17 -31.9- *php20626* -84.4- *php06005* -19- *php20646* -11.8- *php10033a* -2- *php15013* -11.9- *bnl10.13* -19.1- *bnl7.49* --67:80

bnl3.04 - umc130 - umc155 - umc44 - bnl7.49a --r55

glu1, beta-glucosidase, EC3.2.1.21, aa sequence --r192

glu1 with plant height, node number, late season growth, kernel depth, cob diameter QTLs, coded RFLPs --r183

mgs1, Zm13, RM, pollen-specific promoter --r274

Mini10 chromosome carries *Oy1-yg*, unstable --67:25

npi264 - npi563, QTL for androgenesis near *npi563* --r132

npi264, *npi437*, genomic/YAC --r181

npi366 - npi422 - sad1 -10- *glu1* -10- *npi264* -25- *npi461* -4 - *npi436* -12- *npi306* -16- *npi350*, QTLs, heterosis, GxE --r660

orp2 (probe TSB2), (*php06005*, TSB2) - *glu1 - npi105*, pZmTSB2, cDNA, BS: M76685, tryptophan synthase beta --r763

php20075 - php20626 - php06005 - php20646 - glu1 - php15033 - php10013 - php20719 - umc44 - umc57 - bnl10.13 - bnl7.49 - php20568a, plant height QTLs near *php15033* --r46

Rp1-J/Rp1-J, *Rp1-G/Rp1-G*, *Rp1-G/Rp5*, unequal crossing over --r662

Sn1-bol3, cDNA, BS: X60706, transcriptional activator? --r127

stalk-rot (*Fusarium graminearum*, *Gibberella zeae*) R QTL, near *php15013* --67:82

TB-10La, breakpoint between *npi264* and *umc155*, therefore TB is proximal to *Tp2* --67:91

telomere-length QTL near *gln1* --r90

Tp2: *php15013* -11.8- *umc155* -5.7- *npi264* -1.9- *Ds* -1.4- (*Tp2*, *umc163*) -0.8 - *php20719* -1.1- *g1* -11- *umc44a* -1.4- *r1*, *Tp2-x2* and *Tp2-x16*, x-ray induced revertants, not transmitted through pollen, reduced frequency through egg: both lack *php20719* but not *npi264* or *umc155*. --67:91

umc44a, stalk lodging QTLs --r780

UNPLACED

Acc1-s1, *Acc1-s2*, *Acc1-s3*, *Acc1-h1*, *Acc1-h2*, ACCase, ALS, EC 6.4.1.2, alleles: sethoxydim and haloxyfop tolerant --r442

*acp**, EC3.1.3.2, endosperm culture --r464

ad2-2356A, first report, adherent2: fused tassel and upper leaf parts; seedling and juvenile stages normal --67:33

alt1?, *alt2*, *alt3*?, first report, L-alanine:2-oxoglutarate aminotransferase, EC2.6.1.2, *alt1* and *alt3* are postulated loci; *alt2* has electrophoretic mobility variants and forms interlocus hybrid bands with *alt1* --r732

aspartate kinase, aspartate kinase, EC2.7.2.4, isolation of three isoenzymes; two lysine sensitive; one threonine inhibited --r25

cgx1, *cgx2*, first report, chloroplast gene expression: pale green/yellow green, nonviable, decreased levels of thylakoid membrane complexes and RUBISCO but normal chloroplast mRNAs and rRNAs and polysome associations; from *Mu* screening --67:42

cif1, *cim1*, *cim2*, first report, cross-incompatibility --r554, r555

cpi1, first report, lambda ZC7, cDNA, RM, D90549, corn cystatin I, cysteine proteinase inhibitor --r1

cps1, first report, chloroplast protein synthesis: pale green/yellow green, nonviable, decreased levels of thylakoid membrane complexes and RUBISCO, and decreased assocation of all chloroplast mRNAs with polysomes; allele *cps1-2* more severe than *cps1-1*; from *Mu* screening --67:42

crp1 (was *hcf136*), first report, chloroplast RNA processing: pale green/yellow green, nonviable, fails to accumulate monocistronic petB and petD mRNAs and cytochrome f/b6 proteins; from Mu screening --67:42

crp2 (was *hcf143*), first report, chloroplast RNA processing: pale green/yellow green, nonviable, fails to degrade excised group II introns; from *Mu* screening --67:42

cta1, first report, ChitA, ChitB, cDNA, BS, chitinase, EC3.2.1.14, antifungal --r305

d-4* complements *d1*, *d2*, *d3*, *d5*, *d*-3*, *d*-4*, *d*-6*, *d*-8*, *d*-9* --67:82

d-8* complements *d1*, *d2*, *d3*, *d5*, *d*-3*, *d*-4*, *d*-9*; dies at about 15 days --67:82

dks8, defective kernel shootless; from *Mu* screening --67:34

dsy1, non-homologous synapsis from multiple associations of SC --r680

hyp1, first report, mIP9, genomic, BS: X60432, hybrid proline-rich protein, single band in Southerns and northerns; embryo-specific expression --r334

Les-Ua957*, dominant lesion mimic with small (clonal) necrotic lesions to large necrotic sectors; from *Mu* screening --67:33

NADP malate deHase, aa sequence, mol. weight native protein --r7

pep5?, pM500, C4-PEP carboxylase, no polymorphism found --r344

pet2, *pet3*, *pet4*, *pet5*, first report, cytochrome f/b6: pale green/yellow green, nonviable, lack protein components of cytochrome f/b6 complex; from *Mu* screening --67:42

PRms*, induction of pathogenesis-related protein and mRNA --r101

psa1, first report, photosystem I: pale green, nonviable, PS I complex absent; from Mu screening, mutable, expression normalizes with methylation of *Mu* --67:41

psa2, *psa3*, *psa4*, first report, photosystem I: pale green/yellow green, nonviable, PS I complex absent; from *Mu* screening --67:42

psb2, first report, photosystem II: pale green/yellow green, nonviable, PS II complex absent; from *Mu* screening --67:42

rab28, cDNA, genomic, BS: X59138, RAB28, one band in Southerns and northerns --r532

Rs1, encodes homeodomain protein --r637

sod1, *sod3*, response to cercosporin toxin --r752

sod3, complementation of MnSOD deficiency in yeast; transit peptide dissection --r788

Tsc1, tar spot complex (*Phyllacora maydis*) R, first report, dominant R, GCA, SCA --r102

tha1, first report, thylakoid membrane assembly: pale green/yellow green, nonviable, normal levels of RUBISCO, 10-fold reduction in PSII proteins, 5-fold in PSI and cytochrome f/b6 proteins, ATP synthase unaffected; from *Mu* screening --67:42

tha2, first report, thylakoid membrane assembly: pale green/yellow green, nonviable, normal levels of RUBISCO, 4-fold reduction in PSI and PSII proteins, 10-fold in cytochrome f/b6 proteins, 3-fold in ATP synthase; from *Mu* screening --67:42

tua4, *tua5*, *tua6*, *tua3*, bZMPalpha1.5, bZMPalpha9.5, bZMPalpha4.1, bZMPalpha2.6, bZMPalpha2.11, cDNA, BS: X63179, X63180, X63177, X63178, X63176, alpha tubulin, 81 tubulin clones sequenced --r335, r476, r711

Wsm2, wheat streak mosaic virus resistance: one of at least two dominant genes (see *wsm1*) conferring resistance --67:118

B CHROMOSOME

B-chr nondisjunctional regions 2 (prox. euchr.), 3 (prox. heterochr.), 4 (centr., short arm), deletion analysis of meiotic loss of univalents --r99

TRANSPOSABLE ELEMENTS

Ac, *Uq*, non-reciprocal interactions --r91

Ac, ORFa protein, putative *Ac* transposase: expression in *E. coli*; aa sequence; binding domain dissection --r206

Ac, RM, reactivation following regeneration: altered methylation patterns inherited sexually --r81

Ac, transposition and replication at *p1* --r116

dHbr, defective Heartbreaker --r325

Ds1, BS, alternative splicing --r740

Ds1, genomic, BS: Xa54710, X54711, *Z. perennis*, *Z. luxurians* --r426

Hbr, heartbreaker: insertion with imperfect terminal inverted repeat sequence similar to Tourist, same target site specificity (TAA) and same 3bp duplication at target site --67:53

Mu1, *Mu1.7*, RM, loss and restoration of activity; methylation --r87

Mu9, genomic, RM, BS: M76978, candidate for autonomous *Mu* --r286

Mu9, reactivation by UV exposure --r717

MuA, *MuA2*, *MuR1*, mapping autonomous elementswith *wx1* translocations --r571

ruq-st, *ruq31*, *ruq66*, genomic, BS: Xa59774, X59775, Xa59776, sequences *Ds1*-like --r531

Tourist element family, first report, BS, copies 1k-50k; 133bp average size, conserved terminal IR, 3-bp flanking direct repeat, target-site specificity --r89

Uq2, 3, 4, 5, 6, 5 newly activated germinal *Uq*'s, linkages independent of *Uq1*; *Uq2* 'allelic' to *Uq4*, *Uq3* to *Uq5* --r511

Zeon1, RM, BS, zein retrotransposon: 1000 copies of LTR-related sequences, 300-400 of internal sequence --67:94

PLASTID/CHLOROPLAST

cp-clpP, *cp-rps12*, *cp-rpl20*, first reports, RM, BS: X60548, proteolytic sub-unit of ATP-dependent protease, ribosomal proteins S12, L20, translational analysis --r736

cp-ndhA, first report, BS, NADH dehydrogenase subunit A --r435

cp-rps16, first report, genomic, RM, BS: X60823, ribosomal protein --r342

cp-psaI, first report, BS: X61188, PSI-I, identification of photosystem 1 gene --r574

trnG(GCC), *trnG(UCC)*, *trnfM*, *trnG(psi)*, first report, BS: L02941 --r573

MITOCHONDRIA

atp1, *atp6*, *cox3*, RM, BS, promoter dissection --r553

atp6-C, *atp9*, *coxII* in cms-C, BS --r152

cms-T, methomyl R selection in culture --r372

mt-nad5, transcriptional analysis, common mechanism for *nad5* transcription in higher plants --r525

nad5, cDNA, RM, BS: M74160 --r149

ORF1 of 2.3kb linear, product localization --r386

orf25, in TURF 2H3 region, pB10N pB10NR, genomic, RM, variant expression --r727

orf25, RM, transcript modification, nuclear factors --r268

S1 S2, linear plasmids; phylogenetic relationships --r577

T-urf13, *Iorf221*, T-a106, genomic, *Rf1 Rf2* control of transcription --r572

PROBES, CLONES

ABP1, cDNA, beta-D-glucohydrolase, EC3.2.1.21, auxin-binding proteins --r477

*als**, pSOG108, pSOG109, genomic, BS: X63553, X63554, acetohydroxyacid synthase, EC4.1.3.1.8, likely clones of *als1* and *als2* --r198

BE-I*, MB9, MB10, cDNA, RM, BS, starch branching enzyme I, potential clone of *su1*; northerns show one band --r29

cab*, cab-m7, genomic, BS, LHCPII, mesophyll cell specific --r48
cab*, gCAB48, genomic, BS: X63205, chlorophyll a/b binding protein type I --r355
cab*, photoregulated promoter dissection --r416
cab-m1 (=*lhcb1* locus) BS:M87020, photoregulated promoter dissection --r38
casein kinase 2 (CK-2), ck2, cDNA, BS: X61387, sequence identities with human CK-2 --r155
chfi* corn (activated) Hageman factor inhibitor(CHFI), first report, 8-1, cDNA, BS: X54064, 12kDa trypsin inhibitor --r739
cyp* cyclophilin family, CHEM7, cDNA, BS, cyclophilin (CyP), stress-induced; Southerns show 6-7 bands; northerns one band --r437
emb5, first report, cDNA, BS: M90554, ABA-responsive, embryo specific transcripts by northerns; Southerns not described --r749
fm1, fm2, first report, FM1A, FM1B, FM2, cDNA, RM, BS: X61391, X61392, ferritin; northerns --r402
fungus resistance factor?; maize basic protein-1, MBP-1, aa sequence --r177
gbf* family, first report, pGF14-12, cDNA, BS: M96856, G-box binding factor (GF14), G-box element protein factor; bZIP domain; Southerns show several bands, northerns one --r150
glutamine synthase family, pGS107, pGS112, pGS117, pGS122, pGS202, cDNA, RM , BS, glutamine synthetase GS1, GS2, pGS202 plastidic, others cytosolic; tisssue-specific abundances --r592
glutelin-1, -2, -3, cDNA, glutelins G1-204, G1-164, G2, G3, G1, 5-10 gene copies; G2, 1-2 copies; G3, 2-3 copies --67:78
*grp** gene family, CHEM1, CHEM2, cDNA, BS:X 61121, 157 aa glycine-rich protein, stress induced; identity to ABA induced cDNA; Southerns indicate 5 members --r153
h2b* histone family, first report, cDNA, BS: X57312, X57313, H2B, Southerns show multiple bands --r323
HSP18 family, g/cMHSP18-1, cDNA, genomic --67:70
HSP18 family, HSP26, HSP70, pHSP18, pHSP26, pHSP70, cDNA, Southerns with pHSP18 show multiple bands; pHSP26 single; pHSP70 1-3 bands --r333
HSP60, cDNA, BS: Z11546, Z11547, clone identification; mRNA induction --r541
HSP80, scMHSP80-6-4, cDNA --67:71
ldh1, genomic, RM, BS: Z11754, LDH1, (s)lactate:NADH oxidoreductase candidate, EC1.1.1.27, hypoxia, anaerobic regulatory element (ARE) --r250
NIA, pZmnr1, pZmnr1S, cDNA, BS: M77792, NADH:nitrate reductase, EC1.6.6.1, transgenic expression in *E. coli* --r95
nitrate reductase, NAD(P)H, p1501, cDNA, BS: X64446, EC1.6.6.1 --r404
omt1, pMC1, pMG18/32 , cDNA, genomic, RM, BS: M73235, lignin O-methyltransferase, EC2.1.1.6, expression in *E. coli*, one band in Southerns, one mRNA --r124
*pep** (ppc) family (4 members), *nmdh** family (2-3 members), *me** family (2 members), *ssu** (rbcS) family (3 members), *pdk** (ppdk) family (3 members) --r493
*pep**, C4 PEP carboxylase, EC4.1.1.31, Southerns: methylation vs tissue expression --r772
*pep**, pepcZm1, pepcZm1', pepcZm2A, pepcZm3A, genomic, RM, BS: X63869, X63870, X63871, C4-PEP carboxylase, promoter dissection --r605
*pgam** phosphoglycerate mutase family, first report, PGAM1, PGAM2, PGAM3, PGAMg, cDNA, RM, BS: M80912, PGAM-i: 2, 3-bisphosphoglycerate-independent phosphoglycerate mutase, E.C.5.4.2.1., several bands in Southerns, one in northerns --r257
polygalacturonase gene family, W2265, W2247, B7317; 3C12, genomic; cDNA, RM, BS: X62383, X62384, X62385, polygalacturonase? --r11
*risp** (=*isp1* locus), MR2.4, TR12, cDNA, BS: M772224, M772225, Rieske iron-sulfur protein, EC1.10.2.2 --r298
stress-induced protein, mRNA, CHEM4, cDNA, BS, thaumatin-like protein?, Southerns show multiple bands --r224
synthetic tandem repeats (STR), DNA polymorphism --r436
transcription factor IID, ZMTFIID-1; ZMTFIID-2, cDNA, RM, BS, Southerns show two or more bands --r270
tua3, *tua4*, *tua5* may be in tandem with *tua*-7*, *tua*-8*, PCR genomic sequence analysis indicates at least 7 genes in α tubulin family --r476
*ubf**, MubG7, MubG10, ubiquitin fusion protein --67:72
ubf9 gene family , first report, pUBF9, genomic, RM, BS: M68937, ubiquitin fusion protein, multiple bands in Southerns; copy number, transcript levels --r117
*ubi** family, g/cMub1, cDNA, genomic, polyubiquitin --67:72
YAC library; ordered array --r181
*yptm** family, yes, yptm1, yptm2, cDNA, BS: X63277, X63278, ras-related proteins; organ-specific transcripts in northerns --r510
zein -300 element, pMS1, pMS2, RM, promoter dissection, binding proteins --r548
zein-22kD, 22Z-4, BS, O2 protein binding site in promoters; prolamin box --r607
zein-27kd, RM, BS: X56117, X56118, X53514, 27kd zeins; duplication variations --r140
zeinE19 (zE19), RM, BS, 19kDa zein, promoter dissection --r245
zp-gZ22.8, zp-psi-gZ22.8, contiguous tandem sequences, pgZ22.8H3, p-psi-gZ22.8, genomic, BS: X61085, gene candidate and pseudogene --r677
*zrp3** Zea root preferential mRNA family, pZRP3, cDNA, RM, BS-Z12103, ZRP3 polypeptide, root-specific mRNA, cortical; Southerns show multiple bands --r326
*zsf4** alpha-zein family cluster, first report, zsf4c1, zsf4c2, zsf4c3, zsf4c4, zsf4c5, genomic, RM, BS: X55722, X55723, X55726, X55724, X55661, alpha zein --r396
ZSF4C1 - *ZSF4C2* - ZSF4C3 - *ZSF4C4* - ZSF4C5, cosmid contig, RM, BS: X55726, five SF4 a-zein genes --r395

OTHER INHERITANCE

Ed. note:
Systematic treatment of continuous-data information and representation of genetic factors controlling traits that are subject to environmentally sensitive measurable variations (i.e., QTLs), are in the planning and development phase as part of the maize genome database prototype. This is a challenging effort, either for the "Zealand" paradigm or for database representation. Pending coherent protocols for representing this category of information, and data standards for experimental design and analysis, summaries are given simply in narrative form in the following. Among the many studies that must await suitable representation in the database are the expanding variety of QTL experiments, and the complex allometric treatments of Miranda and colleagues at Campinas.

Al tolerance: selection --r394
Al tolerance; acid phosphatase --r232
alkali R, yield, GCA, SCA --r632
allele frequencies of tropicals during random mating of temperate x tropical sweet corn composite --r583
androgenesis, QTLs on 1, 3, 9, 10 --r132
antagonists S-(2-aminoethyl)L-cysteine and 5-methyl tryptophane R: tissue culture seleciton --r242
anther culture and callus QTLs, associations with 1, 2, 3, 4, 5, 6, 7, 8 --r723
aphid (*Rhophalosiphum maidis*) R, GCA, SCA --r64
bromoxynil tolerance, association with environmental stress R --r688
clomazone tolerance, recessive R to herbicide; BSH --r689
cms-C, cms-S, cms-T, genotype x cytoplasm effects on chiasma frequency, univalent frequency --r370
cold tolerance, C-bands, guard cells, chloroplast number --r451
cold-shock proteins --r712

corn earworm (*Helicoverpa zea*) (CEW), maize weevil (*Sitophilus zeamais*) (MW), fall armyworm (*Spodoptera frugiperda*) (FAW) R: GCA; SCA; maternal & reciprocal effects --r744
drought and heat R and heat shock proteins --r567
drought and heat R, chloroplast structure, and ABA --r566
drought and heat R, membrane damage, and ABA --r568
drought R, ABA, and leaf epidermal characteristics --r565
drought resistance vs. ethephon --r415
drought resistance, glutathione reductase --r516
drought-adaptive traits: selection --r265
ear ('cob') characteristics as discriminators of productivity --r649
ear morphology, evolution --r56
early generation testing strategies; heritability --r60
earworm (*Helicoverpa zea*) growth vs. silk cuticular lipids --r773
earworm (*Helicoverpa zea*) R multigenic --r759
earworm (*Helicoverpa zea*) R screening, antibiosis --r761
earworm resistance --r762
fall armyworm (*Spodoptera frugiperda*) growth vs. resistant corn --r750
fall armyworm (*Spodoptera frugiperda*)(FAW) R: RS, BSH --r745
floret bisexuality, evolution --r57
forage traits; BSH; RFLP; genetic distance --r72
forage yield, RFLPs and genetic diversity, heterotic groups; diallel; GD --r457
Fusarium ear rot R and silk phenolics --r560
Fusarium ear rot R SCA GCA --r561
Fusarium R and degradation of silk --r562
GCA, SCA relationships --r634
genetic similarity; RFLPs vs pedigrees; GD --r9
genetic variance components in nested mating designs --r254
glyphosate-tolerant isoform selected in tissue culture BMS-R1, 5-enol-pyruvyl-shikimate-3-phosphate synthase, EC2.5.1.19 --r217
grain quality trait correlations: hardness, protein, test weight, kernel density, oil, starch content --r164
GxE selection, sweet corn --r30
head smut (*Sphacelotheca reiliana*) R --r61
head smut (*Sphacelotheca reiliana*) R vs. su1 --r32
histone H3 and H4 subfamilies, members of these histone subfamilies are located on at least 7 chromosomes --r114
husk number, shank length, yield: SCA; GCA --r552
hybrid diversity evaluated by RFLP patterns; GD --r635
IHP vs. ILP: associations with glutamate dehydrogenase and glutamine synthetase activities in the kernel --r145
iron deficiency chlorosis --r499
larger grain borer (*Prostephanus truncatus*) R, maize weevil (*Sitophilus zeamais*) R, phenolics vs grain insect resistance; negative correlation --r21
leaf freckles and wilt/Goss's wilt (*Clavibacter michiganense*) R, GCA, SCA, BSH, NSH; gene number 1-5 --r497
lodging correlates, yield, BSH, NSH --r582
lysine-threonine R: selection in culture --r463
maize dwarf mosaic virus (MDMV) R: BSH, gene number --r315
maize streak virus R --r42
maize x tripsacum crossability --r350
maize x *Z. m. mexicana*, tillers, green mass, *Fusarium* R --r517
maternal effect on DNA endoreduplication, 369
metalochlor tolerance and metabolism --r131
microprojectile bombardment, inheritance of transgenic hpt --r721
molecular marker data: identification of useful parental lines --r174
nematode (*Meloidogyne incognita*) R diallel, GCA, SCA --r751
nicosulfuron response in sweet corn --r651
nitrogen-response vs prolificacy --r4
Northern leaf blight (*Exserohilum turcicum*) R and components, associations with *wx1* translocations on 3, 4, 6; year-dependent --r82
oil and protein: long-term selection, methods --r173
oil content, NSH --r392
osmotic potential: selection --r70
PEP carboxylase, nitrate and ammonium vs. mRNA expression --r663
pericarp thickness, NSH, BSH --r313
phosphorus accumulation: GCA, SCA, multifactorial --r551
photosynthetic efficiency, chlorophyll content, leaf area, yield, diallel --r456
plant height, QTLs on 1 centr, 1L, 2S, 2 centr, 3S, 3L, 4 centr, 5S, 5 centr, 6L, 7L, 8 centr, 9 centr, 10 centr --r46
plant height QTLs, with coded RFLPs, associated with markers in 1S, 1 centr, 1L, 2, 3 centr, 3L, 4L, 5S, 6L, 7L, 8S 8 centr, 8L, 9 centr, 10L --r183
plant height, with coded RFLPs, associated with markers in 1S, 1 centr, 1L, 2 S, 3 centr, 3L, 4L, 5S, 6L, 7L, 8(S), 8 centr, 8L, 9 centr, 10L --r183
plant height, yield QTLs with RFLPs --r283
pollen tube growth rate (3 centr., 5 near ga2 and ga10, 6L, 7 centr., 8 centr., 9), germinability (3 centr., 4 near ga1, 5 centr., 8, 9) QTLs --r599
potyvirus R screening --r230
QTLs, heterosis, GxE --r660
QTLs, maize vs. teosinte (spikelet pairing, 1L, 3, 4S, 2S; ear ranking, 2S, 5S, 9, 3L, 4 centr, 8L, 10L; glume induration, 4S, 2S, 3L, 1L, 5S; ear disarticulation, 1L, 5S, 2S, 4 centr) --r156
regenerability QTLs on 1S, 1L, 2L, 3L, 9L --r20
relationship of Mexican races of maize based on numerical taxonomy and GxE interactions --r593
RFLPs vs. pedigree, heterotic groups; GD --r460
RFLPs, QTLs for yield, lodging, plant/ear height, maturity, yield components --r780
RLFPs, isozymes, pedigree, inbreeding depression: GD --r638
root characters in seedling vs. root lodging --r653
root morphology vs. lodging screening of hybrids --r652
root rot (*Fusarium solani*) R --r168
RS for yield in o2 population --r400
salinity response --r134
salt R: selection in culture --r421
seed growth characters, leaf senescence --r133
seed heterosis --r770
seed quality, GCA, SCA --r171
stalk strength, in vitro digestibility: RS --r629
Stiff Stalk Synthetic: RS, selection methods --r376
SW corn borer (*Diatraea grandiosella*) R, fall armyworm (*Spodoptera frugiperda*) R and leaf polypeptides --r92
telomere length variation 25-fold among inbreds, 3 loci, on 10S near gln1, on 4L near bnl8.23, on 1L near ynh20, account for 50% of variation --r90
transcript from cp-orf221 (1100-nt), one or two dominant factors for presence vs. absence --r572
transgenics: segregation of bar, uidA --r646
yield, RFLP marker selection during inbreeding --r779
yield selection, GCA, SCA for traits --r661
yield, with coded RFLPs, associated with markers in 1 centr, 1L, 2 centr, 3 centr, 4 centr, 4L, 6L, 8 centr, 8L, 9 centr --r183
yield, rubisco; drought stress --r444
yield: RS --r196
zein diversity --r755

--Assembled unrestricted by a Prof. Ligate Committee: Georgia, Doug, Mary, Pat, and Ed
(It is our intention to develop database protocols by which these data will be structured on an ongoing basis).

IV. MAIZE GENETICS COOPERATION STOCK CENTER

Maize Genetics Cooperation ◦ Stock Center

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&
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During the 1992 calendar year, 2,606 seed samples were supplied in response to 229 requests. Of these, a total of 54 requests were received from 17 foreign countries. The numbers of requests and seed samples distributed are similar to figures for calendar year 1991.

As a result of favorable rainfall distribution, the past growing season was the most successful of recent years. About three acres of nursery were grown. Good increases were obtained of numerous stocks that were in low supply. There was an increase of the basic set of *wx1*-marked translocations, together with supplementary stocks from other sources, particularly those derived by Don Robertson. Increases were made of a basic set of B-A translocations with breakpoints distributed over the genome and of about fifty additional ones located in the long arms of chromosomes 3, 4, or 10.

Special plantings were made of several categories of stocks, including goldens, yellow stripes, necrotics, viviparous kernels, nuclear male sterile and numerous additional endosperm or seedling mutants. Some tests for allelism were made within groups.

Greenhouse sandbench plantings were made to determine or confirm genotypes relative to seedling traits. Field plantings were also grown to develop pedigree information with regard to mature plant traits. Such information is used to determine or verify genetic constitutions of sources used to perpetuate stocks and supply seed requests.

During the year a reel-type irrigation system was obtained that should provide much greater assurance of perpetuating field-grown stocks that are weak or ill-adapted to heat or drought stress conditions. In addition, a second coldroom essentially doubling current seed storage capacity has been completed and will be fully operational as soon as seed drawers are available. This additional capacity will find immediate use for storage of seed samples currently held at room temperature pending availability of supplementary long-term storage. It will also be needed in the near future for stocks anticipated to be received from other established collections.

Catalog items change from year to year, so requests should be based on the latest listing. A revised listing of stocks available upon request will continue to appear each year in the *Maize Genetics Cooperation • News Letter*. This listing, which is widely available throughout the maize community, has served as a basis for seed requests. Information on genetic stock availability will also be accessible electronically over the internet (see below).

In making requests, you should indicate both the code number and the genotype of each stock. This information allows us to recognize typographical errors in some cases, or to seek verification of intent when these two types of information are in conflict. In addition to mail, phone and FAX, we can now also accept stock requests by e-mail. Please note our new FAX number and e-mail address.

Each year, additional stocks are received that include mutant alleles of known genes, or gene combinations, or unidentified and untested mutant traits. During the past year numerous stocks were received from the collection developed by Marcus M. Rhoades. Over the course of the next few years the Stock Center will need to assemble numerous additional unique genetic stocks from collections maintained by several individuals who are approaching, or who have reached, retirement from active research. In some cases, stocks will need to be grown to obtain increases of good quality seed for continued storage. In other cases, seedstocks may be suitable for immediate storage in the National Seed Storage Laboratory at Fort Collins, with working samples held at the Stock Center.

We wish to re-emphasize that if you submit genetic stocks to our collection that involve traits requiring special techniques, facilities or skills for classification (e.g., allozyme variants, disease resistance traits, etc.), the stocks should be homozygous for the designated alleles. In that way, the samples may be propagated by selfing, sibbing or intercrossing without the necessity for classifying segregating progenies. We anticipate that greater diagnostic capabilities will become available to us in the future.

It is sometimes necessary to discontinue supplying samples of particular listed items because of insufficient seed supply or because of detected pedigree errors. In these cases, we will attempt to substitute stocks with closely similar genotypes.

We expect that during the next several months a support scientist will be employed by the USDA-ARS who will serve as the Curator of the Maize Genetics Cooperation • Stock Center. We hope to select this individual soon.

We recently purchased a Macintosh Quadra 950 computer and plan to use the relational database program, 4th Dimension, to store all information on our genetics stocks. Entering data on the stocks will begin soon. It is anticipated that computerizing the Stock Center's data will enable us to serve you better.

We have been collaborating with Ed Coe's efforts in creating a Maize Genome database. This is part of the Plant Genome database effort being sponsored by the National Agriculture Library. We have plans to tie our stock center database in with the Maize-DB (and therefore also with the Plant Genome-DB and GRIN at NAL) to allow users access to information about available maize genetic stocks. Our hopes are that a user will be able to find a stock of interest using an on-line database and directly request stocks from within the database program. The request will be transmitted electronically through the internet to us.

Marty Sachs
Director

Earl Patterson
Co-director

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 *br2 hm1*
115B *br2 Vg1 bm2*
115C *v22*
115D *bz2-m; A1 A2 C1 no Ac Pr1 R1*
115E *br2 Vg1*
116A *bz2-m; A1 A2 Ac C1 Pr1* R1
116C *an1 bm2*
116D *an1-bz2-6923*
116I *bz2 gs1 Ts6 bm2*
117A *br2*
117B *br2 bm2*
117D *tb1*
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
124A *v*-5688*
124B *j*-5828*
124C *w*-8345*
124D *v*-5588*
124E *w*-018-3*
124F *w*-4791*
124G *w*-6577*
124H *w*-8054*
124I *v*-032-3*
124J *v*-8943*
124K *yg*-8574*
125A *Les2*
127A *bz2 zb7 bm2*
127B *dek1*
127C *dek2*
127D *dek22*
127E *f1*
127F *Msc1*
127G *Tlr1*
128A *ij2*
128B *l16*
128C *l17*
128D *pg15*
128E *pg16*
128F *v25*
129A *w18*
129B *wlu5*
130A *o10*

CHROMOSOME 2

201F *ws3 lg1 gl2 b1*
203B *al1*
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 A and B)
218E *ba2*
218F *B1 ba2*
219B *B1-Peru; A1 A2 C1 r1-g*
219C *Ch1*
220A *Les1*
220B *ws3 lg1 gl2*/T2 2T *Tripsacum*
220F *os1*
221B *B1 gs2*
222A TB-1Sb-2L4464
222B TB-3La-2S6270
223A Trisomic 2
224A *w*-4670*
224B *v*-5537*
224F *w*-062-3*
224G *yel*-8630*
224H *whp1; A1 A2 C1 c2 R1*
224J *ijmos*-7335*
224K *glnec*-8495*
227A *dek3*
227B *dek4*
227C *dek16*
227D *dek23*
227E *Les4*
228A *l18*
228B *spt1*
228C *v26*
229A *Ch1 rf3*
229B *v24*

CHROMOSOME 3

301A *cr1*
302A *d1-6016*
302E *d1-tall*
303A *d1 rt1 Lg3*
303B *d1 Rf1 lg2*
303F *g2*
303G *g2 d1*
304A *d1 ys3*
304B *d1 Rg1 ys3*
304G *Lg3 Rg1*
305A *d1 Lg3*
305D *d1 Rg1*
305K *d1 cl1; Clm1-4*
307C *pm1*
308A *d1 ts4 lg2 a1-m; A2 C1 Dt1 R1*
308B *d1 ts4*
308C *d1 lg2 a1-m; A2 C1 Dt1 R1*
308E *ra2*
308G *d1 ts4 a1-m; A2 C1 Dt1 R1*
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; Clm1-3*
311D *cl1-p; Clm1-4*
311E *rt1*
311F *ys3*
311G *Lg3 ys3*
312C *ys3 ts4 lg2*
312D *Lg3*
313A *gl6*
313C *Lg3 Rg1 gl6*
313E *Lg3 gl6*
314F *Rg1 gl6 lg2*
314G *gl6 lg2*
315B *Rg1 gl6*
315D *A1-b(P415)*
316A *ts4*
318A *ig1*
318B *ba1*
318C *y10-7748*
319C *lg2 a1-m et1; A2 C1 dt1 R1*
319D *lg2 a1-m et1; A2 C1 Dt1 R1*
319F *lg2 a1-st et1; A2 C1 C2 Dt1 R1*
320A *lg2*
320D *A1 sh2; A2 B1 C1 dt1 Pl1 R1*
320F *A1 sh2; A2 b1 C1 pl1 R1*
320I *A1 sh2; A2 C1 R1*
321A *A1-d31; A2 C1 R1*
322A *A1-d31 sh2; A2 C1 dt1 R1*
322B *A1-d31 sh2; A2 C1 Dt1 R1*
322D *a1; A2 B1 C1 Pl1 R1*
322E *a1-m; A2 B1 C1 dt1 Pl1 R1*
322F *a1-m; A2 b1 C1 dt1 pl1 R1*
322G *a1; A2 C1 C2 R1*
323A *a1-m; A2 C1 Dt1 R1*
323B *a1-m; A2 B1 C1 Dt1 Pl1 R1*
323C *a1-m sh2; A2 B1 C1 dt1 Pl1 R1*
324A *a1-st; A2 C1 Dt1 R1*
324E *a1-st et1; A2 C1 Dt1 R1*
324G *a1-st; A2 C1 dt1 R1*
325A *a1-p et1; A2 C1 dt1 R1*
325B *a1-p et1; A2 B1 C1 Dt1 Pl1 R1*
325C *a1-x1*
325D *a1-x3*
325G *a3*
325J *a1-p; A2 C1 Pr1 R1*
326A *sh2*
326B *vp1*
326C *Rp3*
327A TB-3La
327B TB-3Sb
327C TB-3Lc
327D TB-3Ld
328A Trisomic 3
329A *v*-9003*
329B *v*-8623*
329C *w*-022-15*
329D *yd2*
329E *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
332B *dek5*
332C *dek24*
332D *Wrk1*
332E *gl9*
332F *gl19*
332G *dek6*
332H *dek17*
332I *Lxm1*
332J *ms23*

CHROMOSOME 4

401A *Rp4*
401D *Ga1-S*
401J *Ga1-M*
402A *st1*
402C *fl2 st1*
402D *Ts5*
403A *fl2 Ts5*
404A *Ts5 su1 zb6*
405B *la1*
405D *la1 su1 gl3*
405G *la1 gl4 su1*
406C *fl2*
406D *fl2 su1*
407B *fl2 bm3 su1*
407D *su1*
407E *su1-am*
408B *bm3 su1*
408E *bm3*
408K *su1 se1*

409A *su1 zb6 Tu1*
410D *su1 zb6 gl3*
412C *su1 gl3*
412E *su1 j2 gl3*
413B *gl4 su1*
414A *bt2*
414B *gl4*
414C *gl4 o1*
414E *de*-414E*
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*
418E *dp1*
418F *o1*
418G *v17*
419B *su1 gl3 ra3*
419F *gl3 Dt6; a1-m A2 C1 R1*
420A *su1 Dt4; 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 Trisomic 4
423A TB-4Lb
423B TB-4Lc
423C TB-4Ld
423D TB-4Le
423E TB-4Lf
427A *dek7*
427B *dek25*
427C *Ysk1*
427D *orp1; orp2*
427E *dek8*
427F *dek10*
427G *Ms41*
427H *dek31*
428A *gl5; gl20*
428B *lw4; lw3*
428C *nec5*
428D *spt2*
428E *wt2*
428F *lw4; Lw3*
428G *bx1*

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 ps1-vp7 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 pr1 br3; A1 C1 R1*
507A *a2; 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 *ps1-vp7*
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 *v2 sh4*
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*
520F *A2 Dap1; A1 C1 C2 R1*
520G *A2 pr1 Dap1; A1 C1 C2 R1*
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 Trisomic 5
527A *dek18*
527B *dek9*
527C *dek26*
527D *dek27*
527E *grt1*
527F *nec7*
527G *sh5 pr1*

CHROMOSOME 6

601D *rgd1 Y1*
601E *po1-ms6*
601F *po1-ms6 pl1; y1*
601G *po1-ms6 Pl1; y1*
602A *po1-ms6 wi1; y1*
602K *y1-gbl*
603A *l10; y1*
603C *l12; y1*
603D *w15; y1*
604A *pb4 pl1; y1*
604B *pb4 Pl1; y1*
604F *ms1-si; y1*
604H *ms1; y1*
604I *Y1 ms1*
605A *wi1 Pl1; y1*
605F *Y1 wi1 pl1*
606A *Y1 pg11; pg12 Wx1*
606B *pg11; pg12 wx1 y1*
606C *Y1 pg11; pg12 wx1*
606D *pg11; pg12 Wx1 y1*
606E *pl1; y1*
606F *Pl1; y1*
607A *"Pl1" y1 Pl1-Bh1; A1 A2 c1 R1 sh1 wx1*
607B *"pl1" y1 Pl1-Bh1; A1 A2 c1 R1 sh1 wx1*
607C *su2; y1*
607D *pl1 su2; y1*
607F *Pl1 su2; y1*
608G *Y1 l11*
609B *Y1 wi1 pl1*
609C *Y1 wi1 Pl1*
609D *Y1 su2*
610B *Pl1 Dt2; a1-m A2 C1 R1*
610C *pl1 sm1; P1-RR*
610H *Y1 pl1 Dl2; a1-m A2 C1 R1*
611A *Pl1 sm1; P1-RR*
611D *Pt1*
611E *w1*
611H *py1*
612A *w14*
612B *po1-ms6*
612C *l*-4923*
612D *oro1*
613A 2NOR; *A1 a2 bm1 C1 pr1 R1 v2*
613F *whs*-8613*
613L *w*-8954*
613M *yel*-039-13*
613R *wh*-8889*
613T *pg*-6656*
613U *wh*-8624*
614A TB-6Lb
614B TB-6Sa
614C TB-6Lc
615A Trisomic 6
627A *dek28*
627B *dek19*
627C *vp*-5111*

CHROMOSOME 7

701B *In1-D*
701D *o2*
703A *o2 v5 gl1*
703J *Rs1-o*
705A *o2 gl1*
705B *o2 gl1 sl1*
705C *o2 ij1-ref::Ds*
705D *o2 bd1*
707A *y8 v5 gl1*
707B *in1; A1 A2 C1 pr1 R1*
707D *v5*
707E *vp9*
707F *y8 gl1*
708A *ra1*
708G *y8*
709A *gl1*
710H *ms7 gl1 Tp1*
711B *ij1-ref::Ds*
711G *ts*-br*
712A *ms7*
713A *Bn1*
713B *bd1*
714B *o5*
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 Trisomic 7
727A *dek11*
727B *wlu2*

CHROMOSOME 8

801A *gl18*
801B *v16*
801C *v16 j1*
801D *v16 ms8 j1*
801G *gl18 v16*
803A *ms8*
803B *nec1*
803D *gl18 ms8*
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
827A *dek20*
827B *dek29*
827C *Bif1*
827D *Sdw1*
827E *Clt1*

CHROMOSOME 9

901D *yg2 C1-l 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 *yg2 c1 sh1 wx1 gl15; A1 A2 K9S-s 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-l 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 *C1 sh1 wx1; A1 A2 K9S-l* R1
905B *C1 sh1 ms2; A1 A2 R1*
905C *C1 bz1 Wx1; A1 A2 R1*
905D *C1 sh1 wx1; A1 A2 K10 K9S-l R1*
905E *C1 sh1 wx1 v1; A1 A2 C2 R1*
906A *C1 wx1; A1 A2 Ds Pr1 R1 y1*
906B *C1 wx1; A1 A2 Ds pr1 R1 Y1*
906C *C1-l Wx1; A1 A2 Ds R1*
906D *C1-l; A1 A2 R1*
906G *C1-l Wx1; A1 A2 Ds R1*
907A *C1 wx1; A1 A2 R1*
907D *C1 wx1; A1 A2 B1 pl1 R1*
907E *C1-l wx1; A1 A2 R1 y1*
907G *C1-l(p); A1 A2 B1-b pl1 R1*
907H *C1-l(m); A1 A2 b1 pl1 R1*
908B *C1 wx1 v1; A1 A2 R1*
908D *C1 wx1 gl15; A1 A2 R1*
908E *C1 wx1 gl15; A1 A2 pr1 R1*
908F *C1 wx1 da1; A1 A2 R1*
908H *C1 wx1; A1 A2 R1 y1*
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 *C1 sh1-bz1-x2 Wx1; A1 A2 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; pg11 y1*
914F *wx1 pg12; pg11 y1*
914G *Wx1 pg12; pg11 Y1*
914H *wx1 pg12; pg11 Y1*
915A *wx1* (other alleles from O. Nelson available)
915B *wx1*-a
915C *w11*
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 *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 *Df3; Tp3-9*
920L *ygzb*-5588*
920M *wnl*-034-5*
921A TB-9La
921B TB-9Sb
921C TB-9Lc
921D TB-9Sd
922A Trisomic 9
924A *wd1 C1 + Ring9 Wd1 C1-I; A1 A2 R1*
927A *dek12*
927B *dek13*
927C *dek30*
927D *Les8*
927E *Zb8*
927F *C1; a1-r A2 Dt7 R1*
928A *v28*
928B *wlu4*
928C *C1 wx1 Bf1; A1 A2 r1*
930C *wx1 ms2 Bf1; A1 A2 r1*

CHROMOSOME 10

X01A *oy1*
X01B *oy1 R1; A1 A2 C1*
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 *bf2 ms11*
X04D *bf2*
X05A *zn1 bf2*
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; A1 A2 C1 wx1 y1*
X13I *r1-g; A1 A2 C1 Wx1 y1*
X14A *lsr1 r1-r; A1 A2 C1*
X14F *r1 v18; A1 A2 C1*
X14G *r1 sr2 v18; A1 A2 C1*
X15C *R1-g; A1 A2 C1*
X15D *r1-ch; A1 A2 C1 Pl1*
X16B *r1; A1 A2* abnormal-10 *C1*
X16C *R1-ch; A1 A2 B1 C1 pl1*
X16D *r1 sr2; A1 A2 C1*
X16E *r1 K10-ll; A1 A2 C1 C2*
X16F *R1 K10-ll; A1 A2 C1 C2*
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; 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; A1 A2 bz2 C1 C2*
X18H *R1-nj* (purple embryo Chase); *A1 A2 bz2 C1*
X19A *Lc1*
X19B *w2*
X19C *l1 w2*
X19D *o7*
X20B *l1*
X20C *v18*
X20F *yel*-8721*
X21A TB-10La
X21B TB-10L19
X21C TB-10Lb
X22A TB-10Sc
X23A Trisomic 10
X24A *cm1*
X24B *nec*-4889*
X24C *nec*-5876*
X24D *wh*-7165*
X24E *yel-gr*-8631*
X24F *wh*-8129*
X25A *R1-scm2; a1-st A2 C1 C2*
X25B *R1-scm2; A1 A2 C1 c2*
X25C *R1-scm122; A1 A2 C1 C2 pr1*
X25D *R1-scm2; A1 a2 C1 C2*
X25E *R1-scm2; A1 A2 c1 C2*
X26A *r1-x1; A1 A2 C1*
X26B *R1-scm2; A1 A2 C1 C2*
X26C *R1-sc122; A1 A2 C1 C2*
X27A *dek14*
X27B *dek15*
X27C *w2-dek21*
X27D *Les6*
X27E *gl21*
X27F *Vsr1*
X27G *Oy1-700*
X27H *orp2; orp1*
X27I *l19*

UNPLACED GENES

U140C *l4*
U141A *ms22*
U141B *ms24*
U141C *o9*
U141D *o11*
U142A *o12*
U142B *o13*
U142C *rd3*
U142D *ub1*
U142E *y11*
U142F *y12*
U240A *Les7*

MULTIPLE GENE STOCKS

M141A *A1 A2 B1 C1 C2 Pl1 Pr1 R1-g*
M141B *A1 A2 B1 C1 C2 pl1 Pr1 R1-g*
M141C *A1 A2 b1 C1 C2 Pl1 R1-g*
M141D *A1 A2 b1 C1 C2 pl1 R1-g*
M241A *A1 A2 B1 C1 C2 Pl1 Pr1 r1-g*
M241B *A1 A2 B1 C1 C2 pl1 Pr1 r1-g*
M241C *A1 A2 B1 C1 C2 Pl1 Pr1 R1-r*
M340A *A1 A2 B1 c1 C2 pl1 Pr1 R1-g*
M341B *A1 A2 B1 C1 C2 pl1 Pr1 R1-r*
M341C *A1 A2 b1 C1 C2 Pl1 Pr1 R1-r*
M341D *A1 A2 B1 c1 C2 Pl1 Pr1 R1-r*
M341E *A1 A2 b1 c1 C2 pl1 Pr1 R1-g*
M341F *A1 A2 b1 C1 C2 pl1 Pr1 R1-r*
M441A *A1 A2 B1 C1 C2 Pl1 Pr1 R1-r wx1*
M441B *A1 A2 B1 C1 C2 pl1 Pr1 R1-r wx1*
M441D *A1 A2 B1 C1 C2 Pl1 Pr1 r1-r*
M441E *A1 A2 B1 c1 C2 Pl1 Pr1 r1-r*
M441F *A1 A2 b1 C1 C2 pl1 Pr1 R1-g wx1*
M541F *A1 A2 C1 C2 Pr1 R1*
M641B *A1 A2 C1 C2 Pr1 R1 wx1*
M641D *A1 A2 C1 C2 Pr1 r1 wx1 y1*
M741C Stock 6 high haploid *A1 A2 B1 C1 C2 Pl1 R1-r*
M741F Stock 6 high haploid *A1 A2 C1 C2 pl1 R1-g* scutellum colored *y1*
M741G Stock 6 high haploid *A1 A2 C1-I C2 pl1 R1-g wx1 y1*
M841A *A1 A2 C1 C2 pr1 R1 su1*
M841B *a1 A2 C1 C2 R1 su1*
M841C colored scutellum *A1 A2 C1 C2 Pr1 R1*
M841E colored scutellum *A1 A2 C1 C2 pr1 R1*
M941A *A1 A2 c1 C2 Pr1 R1 wx1 y1*
MX17A *A1 A2 b1 C1 C2 pl1 Pr1 r1-g*
MX40A Mangelsdorf's tester *a1 bm2 g1 gl1 j1 lg1 pr1 su1 wx1 y1*
MX41A *A1 A2 C1 C2 gl1 pr1 R1 wx1 y1*
MX41B *A1 A2 C1 C2 gl1 pr1 R1 su1 wx1 y1*
MX41C *a1 a2 bz1 bz2 c1 c2 pr1 r1 wx1 Y1/y1*
MX41D *a1 A2 C1 C2 gl1 pr1 R1 su1 wx1 y1*

POPCORNS

P142A Amber Pearl Popcorn
P142B Argentine Popcorn
P142C Black Beauty Popcorn
P242A Hulless Popcorn
P242B Ladyfinger Popcorn
P242C Ohio Yellow Popcorn
P342A Red Popcorn
P342B Strawberry Popcorn
P342C Supergold Popcorn
P342D South American Popcorn
P442B White Rice Popcorn

EXOTICS AND VARIETIES

E542A Black Mexican Sweet Corn *A1 A2* B chromosomes present *Bz1 Bz2 C1 C2 Pr1 R1*
E542B Black Mexican Sweet Corn B chromosomes absent
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* Tetraploid
N103D *P1-WR* Tetraploid
N104B *A1 A2 C1 pr1 R1* Tetraploid
N104C *su1 wx1* Tetraploid
N106D *sh1 Wx1 Y1* Tetraploid
N106E *sh1 wx1 y1* Tetraploid
N107B W23 Tetraploid
N107C Synthetic B Tetraploid

CYTOPLASMIC TRAITS

C337A NCS2
C337B NCS3

CYTOPLASMIC STERILES AND RESTORERS

C736A R213; *Rf1 rf2*
C736B Ky21; *Rf1 Rf2*
C736C B37; *rf1 Rf2*
C736D N6; *rf1 Rf2*
C836A Wf9; cms-T, *rf1 rf2*
C836B Wf9; N cytoplasm, *rf1 rf2*

WAXY RECIPROCAL TRANSLOCATIONS

wx01A T1-9c (1S.48 9L.22) *wx1*
wx01B T1-9(5622) (1L.1 9L.12) *wx1*
wx03A T1-9(8389) (1L.74 9L.13) *wx1*
wx04A T2-9c (2S.49 9S.33) *wx1*
wx05A T2-9b (2S.18 9L.22) *wx1*
wx06A T2-9d (2L.83 9L.27) *wx1*
wx07A T3-9(8447) (3S.44 9L.14) *wx1*
wx08A T3-9c (3L.09 9L.12) *wx1*
wx10A T4-9e (4S.53 9L.26) *wx1*
wx11A T4-9g (4S.27 9L.27) *wx1*
wx12A T4-9(5657) (4L.33 9S.25) *wx1*
wx13A T4-9b (4L.9 9L.29) *wx1*
wx15A T5-9(4817) (5L.06 9S.07) *wx1*
wx16A T5-9d (5L.14 9L.1) *wx1*
wx17A T5-9a (5L.69 9S.17) *wx1*
wx18A T6-9(4778) (6S.8 9L.3) *wx1*
wx20A T6-9b (6L.1 9S.37) *wx1 y1*
wx21A T6-9(4505) (6L.13 9) *wx1*
wx22A T7-9(4363) (7 9) *wx1*
wx23A T7-9a (7L.63 9S.07) *wx1*
wx24A T8-9d (8L.09 9S.16) *wx1*
wx25A T8-9(6673) (8L.35 9S.31) *wx1*
wx26A T9-10(8630) (10L.37 9S.28) *wx1*
wx27A T9-10b (10S.4 9S.13) *wx1*
wx28A T5-9(8386) (5L.87 9S.13) *wx1*

NON-WAXY RECIPROCAL TRANSLOCATIONS

Wx30A T1-9c (1S.48 9L.22) *Wx1*
Wx30B T1-9(4995) (1L.19 9S.2) *Wx1*
Wx30C T1-9(8389) (1L.74 9L.13) *Wx1*
Wx31A T2-9c (2S.49 9S.33) *Wx1*
Wx31B T2-9b (2S.18 9L.22) *Wx1*
Wx32A T3-9(8447) (3S.44 9L.14) *Wx1*
Wx32B T3-9(8562) (3L.65 9L.22) *Wx1*
Wx32C T3-9c (3L.09 9L.12) *Wx1*
Wx33A T4-9e (4S.53 9L.26) *Wx1*
Wx33B T4-9(5657) (4L.33 9S.25) *Wx1*
Wx33C T4-9g (4S.27 9L.27) *Wx1*
Wx34A T5-9c (5S.07 9L.1) *Wx1*
Wx34B T5-9(4817) (5L.06 9S.07) *Wx1*
Wx34C T4-9b (4L.9 9L.29) *Wx1*
Wx35A T5-9(8386) (5L.87 9S.13) *Wx1*
Wx35B T5-9a (5L.69 9S.17) *Wx1*
Wx35C T5-9d (5L.14 9L.1) *Wx1*
Wx36A T6-9(4778) (6S.8 9L.3) *Wx1*

Wx37A T6-9(8768) (6L.89 9S.61)
Wx1
Wx37B T7-9(4363) (7 9) *Wx1*
Wx37C T6-9(4505) (6L.13 9) *Wx1*
Wx38A T7-9a (7L.63 9S.07) *Wx1*
Wx38B T8-9d (8L.09 9S.16) *Wx1*
Wx38C T8-9(6673) (8L.35 9S.31)
Wx1
Wx39A T9-10(8630) (10L.37 9S.28)
Wx1
Wx39B T9-10b (10S.4 9S.13) *Wx1*

INVERSIONS

I143B Inv1c (1S.35-1L.01)
I143C Inv1d (1L.55-1L.92)
I143D Inv1(5131-10) (1L.46-1L.82)
I243A Inv2(8865) (2S.06-2L.05)
I243B Inv2(5392-4) (2L.13-2L.51)
I343A Inv3a (3L.38-3L.95)
I343B Inv3L (3L.19-3L.72)
I343C Inv3(3716) (3L.09-3L.81)
I344A Inv9a (9S.7-9L.9)
I443A Inv4b (4L.4-4L.96)
I443B Inv4c (4S.86-4L.62)
I444A Inv2a (2S.7-2L.8)
I543A Inv4e (4L.16-4L.81)
I743A Inv5(8623) (5S.67-5L.69)
I743B Inv6(8452) (6S.77-6L.33)
I743C Inv6(3712) (6S.76-6L.63)
I843A Inv6(8604) (6S.85-6L.32)
I943A Inv7(5803) (7L.17-7L.61)
I943B Inv7(8540) (7L.12-7L.92)
I943C Inv7(3717) (7S.32-7L.3)
IX43A Inv8a (8S.15-8S.38)
IX43B Inv9b (9S.05-9L.87)

V. GENE LIST AND WORKING MAPS

GENELIST: A table of the defined and designated gene loci of maize, derived by extraction from the Maize Genome Database, follows. Included are the symbol for the locus; the chromosome (L=long arm, S=short arm) and map location ("+-" denotes a map location near the position listed); the name and a brief description of the phenotype; and references to original descriptions. Stocks of variants may be obtained from the Maize Genetics Stock Center (preceding section); many variations (e.g., isozymes and RFLPs) occur naturally among generally available strains. The gene list was compiled by Ed Coe and Mary Polacco. Georgia Davis and Pat Byrne participated in collection and refining of the data and descriptions; Stan Letovsky and Denis Hancock aided in deriving the output.

NOMENCLATURE: New definitions of standards and criteria are presented in the following section. Oliver Nelson chaired the committee that has carefully developed this standard.

MAPS: Working maps follow the table. The traditional linkage map is in the center, showing recombination distances in centimorgans. 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. To the right 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) or to one arm (vertical line from near the centromere to the end of the arm). Substantive changes in the maps this year include a general revision for chromosome 8, and local revisions in 6S and in 1L near *adh1*.

The cytological map of each chromosome, immediately to the left of the linkage map, shows the arms in proportion and locations of selected aberrations. B-A translocations, which generate hemizygous segments, are shown as TB-..., and A-A translocations as T with chromosome numbers and identifiers (see MNL 55:140ff.); placements on the linkage map are in relation to cytogenetic mapping data (see MNL 52:129ff., 59:159ff., 60:149ff., et seq.). 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; genes spanned by the line may or may not). 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 line are uncovered.

To the left are RFLP and isozyme loci on the Core Map derived at the University of Missouri. Maximum likelihood treatment of multipoint data (Mapmaker) has until now only been suitable for F2 data, providing a map on which statistical qualifiers can be stated. Loci for which the order is uncertain (LOD scores do not differ by 3) are marked with a dotted line. Highlighted with boxes are Core Markers, a spaced set of widely used, informative markers (Gardiner et al., Genetics, in press) available from the laboratory of Shiaoman Chao. Loci new to the Core Map include ones probed by sequenced cDNAs (table below). Accompanying the Core Map is a parallel list of markers mapped at Brookhaven National Laboratory, in map order and matched to the Core Map; cooperation of Ben Burr in providing the large and complex current data set for this parallel representation is greatly appreciated. Tools for analysis of these data by maximum likelihood have just become available and are being applied to the data at Brookhaven. These and the several other available RFLP maps are largely consistent with each other, and plans for merging of the accumulated data to produce a consolidated map are in progress. Dashed cross-lines show interrelated locations; these are mutually interdependent (i.e., derived from information from each source by circular logic).

Construction of a map that integrates the locations of genes, cytogenetic variants, and molecular markers requires systematic compilations of data (which are in progress under the Maizedb program), but further requires new mapping engines under development.

The maps were compiled and prepared by Ed Coe with participation by Gerry Neuffer, Jack Gardiner, and Shiaoman Chao; technical help of Susan Melia-Hancock, Oscar Heredia-Diaz, Theresa Musket, and Guilin Xu is appreciated.

The current Plastid Chromosome Genetic Map, prepared by Steve Rodermel, follows the nuclear working maps. For the Mitochondrial Map, see MNL 64:165.

MAP IT: The value of mapping with probes of known function cannot be overstressed. This gives functional significance to particular places in the genome, important as additional studies (particularly in quantitative genetics) progress. 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). The Brookhaven set of recombinant inbreds can be probed and the data sent to Ben Burr for inclusion in the data resource. The probe can be sent to Missouri for mapping in the Immortal F2 population and inclusion in the Core Map resource. We would also use the probe in correlation to physical and conventional markers. Included in this Newsletter is a sample form with 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.

QUALITY of these resources is enhanced each year by corrections, clarifications, and suggestions provided by Cooperators; your input is welcome and needed.

Ed Coe

Mapped loci probed by cDNA clones (Keith et al., 1992): identification number and sequence similarity.

umc No.	Chrom.	cDNA No.	Sequence similarity	umc No.	Chrom.	cDNA No.	Sequence similarity
303	2	4		344	2	64	brain specific 14-3-3 protein, tau chain
304	7	5	thiol protease	346	6	68	
312	7	13		354	10	86	
313	3	16	NADP malic enzyme	357	1	92	
314	2	17	tobacco 31kd ribonucleoprotein	360	9	95	
316	1	21	chlorophyll a/b binding protein	364	10	103	
317	3	25		365	5	108	GTP-binding protein
322	3	30	vacuolar ATPase, proteolipid subunit	369	6	116	elongation factor 1 alpha
323	8	31		371	2	133	
324		32		379	6	146	cell cycle protein CDC48p
329	3	38		382	5	149	
337	9	54		384	2	154	
338		56		386	9	158	enolase
340	9	59		392	5	173	
342	1	61					

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
a1	3L-149	anthocyaninless1, colorless aleurone, green or brown plant, brown pericarp with *P1-RR*, for alleles and interactions, see Coe et al., 1988, encodes dihydroflavonol reductase	116
a2	5S-35	anthocyaninless2, like *a1*, but red pericarp with *P1-RR*, encodes flavanone dehydrase/oxidase?, naringenin, 2-oxoglutarate 3-diooxygenase?	204
a3	3L-132	anthocyanin, recessive intensifier of expression of *R1* and *B1* in plant tissue	257
abp1	3L-67+-	auxin binding protein1, putative auxin receptor, single band in Southerns (Lobler 1990), encodes auxin binding protein	456
Ac		activator: autonomous transposable element, regulates *Ds* transposition and dissociation, *Ac9* is element isolated from *wx1-m9*	281
Ac2		activator: similar to *Ac*	95
acc1		acetyl-coenzyme A carboxylase1, tissue-culture selected resistance to cyclohexanedione (e.g., sethoxydim) and aryloxy phenoxypropionate (e.g., haloxyfop) herbicides, encodes acetyl-coenzyme A carboxylase	339
aco1	4S-39+-	aconitase1, electrophoretic mobility, monomeric, encodes aconitate hydratase	51, 474
aco2		aconitase2, electrophoretic mobility, encodes aconitate hydratase	474
aco3		aconitase3, electrophoretic mobility, encodes aconitate hydratase	474
aco4		aconitase4, electrophoretic mobility, monomeric, encodes aconitate hydratase	474
acp1	9L-56+-	acid phosphatase1, electrophoretic mobility, cytosolic, dimeric, encodes acid phosphatase	111, 165
acp2		acid phosphatase2, electrophoretic mobility, dimeric, encodes acid phosphatase	111, 165, 219
acp4	1L-176	acid phosphatase4, electrophoretic mobility, monomeric, encodes acid phosphatase	219
acpt1		acyl carrier protein1, cDNA sequence, deduced transit peptide, encodes acyl carrier protein	425
ad1	1L-108	adherent1, seedling leaves, tassel branches, and occasionally top leaves adhere	226
adh1	1L-134	alcohol dehydrogenase1, electrophoretic mobility, null alleles are known, dimeric, intra/interlocus hybrid bands occur, encodes alcohol dehydrogenase	404
adh2	4S-27	alcohol dehydrogenase2, electrophoretic mobility, null alleles are known, dimeric, intra/interlocus hybrid bands occur, encodes alcohol dehydrogenase	402
adk1	6S-0	adenylate kinase1, electrophoretic mobility, plastidial, encodes adenylate kinase	471
adr1		alcohol dehydrogenase regulator1	238
ae1	5L-57	amylose extender1, glassy, tarnished endosperm, high amylose content, encodes 1,4-alpha-glucan branching enzyme	466
afd1	6L	absence of first division1, male and female sterility, anaphase I equatorial	161
agt1		agravitropic1, primary root unresponsive to gravity	106
al1	2S-4	albescent plant1, variably cross-banded to white leaves, pale yellow endosperm, some alleles viviparous	349
ald1	8L-80+-	aldolase1, cytosolic aldolase, cDNA and genomic clones, Southern blots give single or double band, promoter functional in transient expression assay (Dennis et al. 1988), encodes aldolase	174, 224
alh1	1L-161+-	a-subfraction lysine-rich histones1 (was H1a), electrophoretic mobility, encodes histone la	446
alpha		*a1* locus component (see beta), determines reduced aleurone and plant color, brown pericarp	245
als1	4-55+-	acetolactate synthase1, tolerance to imidazolinone herbicides, acetohydroxyacid synthase has altered herbicide inhibition kinetics, encodes acetohydroxyacid synthase	9, 54, 327
als2	5L-50+-	acetolactate synthase2, tolerance to imidazolinone herbicides, acetohydroxyacid synthase has altered herbicide inhibition kinetics, encodes acetohydroxyacid synthase	9, 54, 327
alt1		L-alanine:2-oxoglutarate aminotransferase1, electrophoretic mobility, *alt1* and *alt2* interact to form heterodimers, encodes L-alanine:2-oxoglutarate aminotransferase	469
alt2		L-alanine:2-oxoglutarate aminotransferase2, electrophoretic mobility, *alt1* and *alt2* interact to form heterodimers, encodes L-alanine:2-oxoglutarate aminotransferase	469
alt3		L-alanine:2-oxoglutarate aminotransferase3, electrophoretic mobility, encodes L-alanine:2-oxoglutarate aminotransferase	469
am1	5S-20	ameiotic1, male and female sterility, anaphase I equatorial	336, 367
amp1	1L-85+-	aminopeptidase1, electrophoretic mobility, cytosolic, monomeric, encodes aminopeptidase	334
amp2	1-64+-	aminopeptidase2, electrophoretic mobility, monomeric, encodes aminopeptidase	334
amp3	5S-35+-	aminopeptidase3, electrophoretic mobility, monomeric, encodes aminopeptidase	334
amp4		aminopeptidase4, electrophoretic mobility, monomeric, encodes aminopeptidase	334
amy1		amylase1, electrophoretic mobility, monomeric, encodes alpha amylase	64
amy2	5S-17+-	amylase2, electrophoretic mobility, monomeric, encodes beta amylase	63
an1	1L-104	anther ear1, andromonoecious dwarf, intermediate stature, few tassel branches, responds to gibberellins, *an1-6923* includes deletion of *Bz2+*	113, 123
anl1	5S-29+-	anthocyaninless lethal1, colorless aleurone, small kernels, embryo lethal	74
ant1	5L	adenine nucleotide translocator1, open reading frame encodes a polypeptide of 40,519 Da (Baker, 1985), encodes adenine nucleotide translocator, mitochondrial, candidate	15
ant2		adenine nucleotide translocator2, cDNA sequence corresponds to genomic sequence (Winning et al., 1991), actively transcribed in basal meristem, not in green leaves (Bathgate et al., 1989), encodes adenine nucleotide translocator, mitochondrial, candidate	17
aph1		aphid resistance1	61
ar1	9L-62	argentia1, virescent seedling, greens rapidly, husk leaf tips striped	127

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
ars1		autonomous replicating sequence1, autonomously replicates in yeast, 11,000 copies in maize	30
ars2		autonomous replicating sequence2, autonomously replicates in yeast, 10,000 copies in maize	30
ars3		autonomous replicating sequence3, autonomously replicates in yeast, 28,000 copies in maize	30
as1	1-56	asynaptic1, synaptic failure in male and female	24
ask1	7S-16+-	aspartate kinase1, lysine-threonine resistance in cultures and seedlings, increased threonine in kernels, encodes aspartate kinase	97
ask2		aspartate kinase2, lysine-threonine resistance, encodes aspartate kinase	97
atn1		anaerobic tolerant null1, enhances survival of ADH-null under anoxia	246
atp1	3-62+-	ATPase1, partial cDNA sequence identical to proteolipid of *Avena sativa* vacuolar ATPase, single copy, gene-specific cDNA probe, encodes proteolipid, vacuolar ATPase	62, 222
ats1	8	atrazine susceptible1, lacks glutathione S-transferase	172
b1	2S-49	colored plant, dominant *B1* plants have anthocyanin in major plant tissues, some alleles affect aleurone and embryo color (for alleles, see Coe et al., 1988), regulates flavonoid enzymes	119
ba1	3L-102	barren stalk1, ear shoots and most tassel branches and spikelets absent	184
ba2	2-74+-	barren stalk2, like *ba1*, but tassel more normal	184
ba3		barren stalk3	338
baf1	9S-54+-	barren stalk fastigiate1, ear shoots often absent, tassel branches erect	75
bd1	7L-109	branched silkless1, ear silkless, branched at base, tassel proliferated, bushy	229
beta		*a1* locus component (see alpha), determines aleurone and plant color, red pericarp	245
bf1	9L-137	blue fluorescent1, homozygous *bf1* seedlings, homozygous or heterozygous anthers, fluoresce blue under ultraviolet light, anthranilic acid accumulates, anthranilate synthase has altered inhibition kinetics	455
bf2	10L-33	blue fluorescent2, similar to *Bf1* in expression, shows earlier, stronger seedling fluorescence	5
Bg		transposable element, Bergamo, regulatory element mediating *o2-mr*	386
bif1	8-0	barren inflorescence1, dominant *Bif1* plants have ear and tassel with many fewer spikelets, bare rachis appendages	321
bk2	9L-82	brittle stalk2, brittle plant parts after 4-leaf stage	242
blh1	1S	bleached1, dominant *Blh1* plants have pale green midveins and base in upper leaves	313
bm1	5S-41	brown midrib1, brown pigment over vascular bundles of leaf sheath, midrib, and blade, especially evident on the midribs of healthy leaves at flowering. Lignin content at maturity 86% of normal	126
bm2	1L-161	brown midrib2, like *bm1*	50
bm3	4-55+-	brown midrib3, like *bm1*, has lowered activity of catechol O-methyl transferase. Silage corn with *bm3*, having improved digestibility, is in production.	124, 235
bm4	9L-141	brown midrib4, like *bm1*	47
bn1	7L-71	brown aleurone1, yellowish brown aleurone color	237
br1	1L-81	brachytic1, short internodes, short plant, no response to gibberellins	225, 228
br2	1L-64+-5	brachytic2, like *br1*	248
br3	5	brachytic3, like *br1*	418
bre1		branching enzyme1, maize kernel cDNA homologous to starch branching enzyme I of bacteria, deduced 64-amino acid transit peptide, highly expressed in early stages of kernel development, encodes starch branching enzyme?	12
brn1	3S-19	brown aleurone1, brown kernel, brown embryo, seedling lethal	379
Bs-1		barley stripe1, transposable element, retrovirus-like, 1-5 copies in genome	210
bs1		barren sterile1, plant weak, with little or no tassel and usually with only a vestige of pistillate inflorescence, shank, husks	286
bt1	5L-42	brittle endosperm1, mature kernel collapsed, angular, often translucent and brittle, encodes phospho-oligosaccharide synthase	268, 475
bt2	4S-48	brittle endosperm2, like *bt1*, endosperm ADPG pyrophosphorylase subunit (compare *sh2*), encodes ADP glucose pyrophosphorylase	124, 454
btn1		brittle node1, tassel breakage in B73 inbred line	220
bu1		leaf burn1, leaves show burning, sometimes horizontal bands, accentuated by high temperature	146
bv1	5L-47	brevis plant1, short internodes, short plant	250
bv2		brevis plant2, plant height 50-70% of normal, possible allelism with *rd1*	350
bx1	4S	benzoxazinless1, cyclic hydroxamates (blue color in crushed root tip with FeCl3), which inhibit *Ostrinia nubilalis* and *Helminthosporium turcicum*, present in *Bx1* roots, absent in *bx1*	87
bz1	9S-31	bronze1, modifies purple aleurone and plant color to pale or reddish brown, anthers yellow-fluorescent, allele *bz1-m4* = *sh1-bz1-m4*, encodes flavonol (O)3-glucosyl transferase	366
bz2	1L-106	bronze2, like *bz1*, anthers not fluorescent, *an1-6923* mutation includes deletion for *Bz2*, potential function flavonoid acylation, glycosylation, transport, or deposition	331
c1	9S-26	colored aleurone1, *C1* colored, *c1* colorless, *C1-I* dominant colorless, *c1-p* pigment inducible by light (see Coe et al., 1988), encodes C1 protein	108
c2	4L-98	colorless2, 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, encodes naringenin-chalcone synthase	171

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
car1	1S	catalase regulator1, dominant *Car1* determines increased enzyme activity level, encodes CAR1 product	392
cat1	5S-17+-	catalase1, electrophoretic mobility, cytosolic/glyoxysomal, tetrameric, intra/interlocus hybrid bands occur, encodes catalase	27
cat2	1S	catalase2, electrophoretic mobility, null allele is known, cytosolic/glyoxysomal, tetrameric, intra/interlocus hybrid bands occur, encodes catalase	389
cat3	4L-118+-	catalase3, electrophoretic mobility, null allele is known, mitochondrial, tetrameric, intralocus hybrid bands occur, expressed largely in leaf mesophyll cell of young seedlings, encodes catalase	391
cdh1		cinnamyl alcohol dehydrogenase1, electrophoretic mobility, encodes cinnamyl alcohol dehydrogenase	138
ce1		curled entangled1, dominant *Ce1* plants have rolled leaves that tend to be entangled	68
cfl2		complementary to *fl2*	335
cfr1	1S	coupling factor reduction1, chloroplast ATP synthase affected, seedlings pale green and greatly reduced in vigor	109
cg1	3S-35	corngrass1, dominant *Cg1* plants have narrow leaves, extreme tillering	417
cg2	3	corngrass2, dominant *Cg2* plants have narrow leaves, high tillering, mutable	262
cgl1		Colletotrichum graminicola resistance1, dominant *Cgl1* plants are resistant	14
cgx1		chloroplast gene expression1, reduced RUBISCO, thylakoid polypeptides, chloroplast rRNA, mRNA's appear normal and mostly associated with polysomes	16
cgx2		chloroplast gene expression2, reduced RUBISCO and thylakoid polypeptides, plastid mRNA's, rRNA's normal and mostly associated with polysomes	16
ch1	2L-155	chocolate pericarp1, dominant *Ch1* ears have tan to dark brown pericarp and cob	8
cif1		cross-incompatibility in female1, reduced seed set when homozygous *cif1* female is crossed with male homozygous recessive for *cim1* and *cim2*	360
cim1		cross-incompatibility in the male1 (with *cif1*, *cim2*)	360
cim2		cross-incompatibility in the male2 (with *cif1*, *cim1*)	360
Cin		Cinteotl corn insert: repetitive sequences dispersed in the genome	173
cl1	3S-60	chlorophyll1, white to green seedlings, depending upon alleles of modifier *clm1*, pale yellow endosperm	125
clh1		c-subfaction lysine-rich histones1, electrophoretic mobility, encodes histone lc	446
clm1	8	modifier of *cl1*, dominant *Clm1* alleles confer greening in *cl1* seedlings, does not restore endosperm carotenoids	125
clt1	8L-41	clumped tassel1, dominant *Clt1* plants have variable dwarfing, developmental anomalies	149, 319
cm1	10L-64+-	chloroplast modifier1, white or yellow stripes on leaves (compare *ij1*), conditions chloroplast modifications that are maternally inherited	448
cms-C		female-transmitted male sterility, C type, restored by *Rf4*	25
cms-S		cytoplasmic male sterility, female-transmitted male sterility, S type, restored by *Rf3*	213, 216
cms-T		cytoplasmic male sterility, female-transmitted male sterility, Texas type, restored by *Rf1 Rf2*	213, 216
cp1	7S-25+-	collapsed1, endosperm collapsed and partially defective	260
cp2	7S-25+-	collapsed2, endosperm rough, collapsed, partially defective, seedling very light green with darker streaks, lethal	326
cps1		chloroplast protein synthesis1, reduced levels of RUBISCO and all thylakoid membrane complexes, unaltered chloroplast mRNA, decreased chloroplast polysomes	16
cps2	6L	chloroplast protein synthesis2, 20-fold reduced RUBISCO, 2-fold reduced thylakoid polypeptides, decreased chloroplast polysomes	16
cr1	3S-26	crinkly leaves1, plant short, leaves broad, crinkled, foreshortened	120
cr4	10	crinkly leaves4, plants short with rough, extremely crinkly leaves and club tassel, aleurone mosaic	442
crp1		chloroplast RNA processing1, fails to accumulate monocistronic petB and petD mRNA's, lacks cytochrome b6/f	16
crp2		chloroplast RNA processing2, fails to degrade group II introns in chloroplast	16
css1	9L-66+-	sucrose synthase1, enzyme of embryo and other tissues, (compare *sh1*), encodes sucrose synthase	278
ct1	8-22+-	compact plant1, semi-dwarf plant, ear furcated	306
ct2	1S-10+-	compact plant2, semi-dwarf plant with club tassel	153
cta1		chitinase A1, cDNA sequence, peptide sequence of maize 28kDa chitinase A, encodes chitinase	198
ctb1		chitinase B1, cDNA sequence, protein sequence of maize chitinase B, encodes chitinase	198
cto1		cob turned out1, ear inverted to a sheet or tube, kernels internally placed, variable expression	463
cx1	10L-33+-	catechol oxidase1, electrophoretic mobility, null allele is known, monomeric, no hybrid bands, encodes catechol oxidase	357
Cy	5L	cycler: regulatory element mediating *bz1-rcy*	396
d1	3S-44	dwarf plant1, andromonoecious, short, compact plants, responds to gibberellins, *d1-t* intermediate in height	113
d2	3	dwarf plant2, like *d1*	451

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
d3	9S-59	dwarf plant3, like *d1*	92
d5	2S-34	dwarf plant5, like *d1*	451
d8	1L-132	dwarf plant8, dominant *D8* plants resemble *d1*, not responsive to gibberellins, (compare *Mpl1*, probable allele)	347
d9	5S-6	dwarf plant9, dominant *D9* plants semidwarf with broad, dark green leaves, not andromonoecious, not responsive to gibberellins	314, 315
da1	9	dilute aleurone1, aleurone color diluted	131
dap1	5L-96+-	dappled aleurone1, dominant *Dap1* kernels show patches of normal and abnormal aleurone cells, effect with colored aleurone is conspicuous	443
dek1	1S-27	defective kernel1, germless, floury endosperm, anthocyanins and carotenoids absent, cultured embryos not obtained	324, 325
dek2	1L	defective kernel2, discolored, scarred endosperm, lethal, cultured embryos green	324, 325
dek3	2S	defective kernel3, germless, cultured embryos white with green stripe	324, 325
dek4	2L	defective kernel4, germless, floury endosperm, cultured embryos green, narrow leaved	324, 325
dek5	3S	defective kernel5, shrunken endosperm, white seedling with green stripes	324, 325
dek6	3L	defective kernel6, shrunken endosperm, lethal, cultured embryos normal	324, 325
dek7	4S-36+-	defective kernel7, shrunken sugary endosperm, white seedling with green stripes	324, 325
dek8	4L	defective kernel8, shrunken endosperm, lethal, cultured embryos green, small	324, 325
dek9	5L	defective kernel9, crumpled endosperm, lethal, anthocyanins and carotenoids reduced, cultured embryos not obtained	324, 325
dek10	4L	defective kernel10, collapsed endosperm, lethal, cultured embryos green, curled, stubby	324, 325
dek11	4S	defective kernel11, etched endosperm, lethal, cultured embryos white with green stripes	324, 325
dek12	9S	defective kernel12, collapsed endosperm, lethal, cultured embryos green, narrow-leaved, curled	324, 325
dek13	9L	defective kernel13, defective opaque endosperm, lethal, cultured embryos pale green with green stripes	324, 325
dek14	10S	defective kernel14, collapsed endosperm, lethal, cultured embryos yellow-green	324, 325
dek15	10L	defective kernel15, collapsed floury endosperm, lethal, cultured embryos green	324, 325
dek16	2L	defective kernel16, floury endosperm, lethal, cultured embryos normal	410
dek17	3L	defective kernel17, collapsed endosperm, lethal, cultured embryos not obtained	410
dek18	5S	defective kernel18, collapsed endosperm, lethal, cultured embryos green, narrow-leaved	410
dek19	6L	defective kernel19, collapsed opaque endosperm, lethal, cultured embryos green	410
dek20	8L-41+-	defective kernel20, collapsed endosperm, lethal, cultured embryos green	410
dek22	1L	defective kernel22, collapsed endosperm, lethal, cultured embryos not obtained	71, 409
dek23	2L	defective kernel23, defective crown, lethal, cultured embryos not obtained	71, 409
dek24	3S	defective kernel24, collapsed endosperm, lethal, cultured embryos normal	409
dek25	4S-13+-	defective kernel25, shrunken endosperm, lethal, cultured embryos normal	409
dek26	5L	defective kernel26, collapsed endosperm, lethal, cultured embryos normal	409
dek27	5L	defective kernel27, collapsed endosperm, lethal, cultured embryos green	409
dek28	6S	defective kernel28, opaque endosperm	409
dek29	8L-52+-	defective kernel29, collapsed endosperm, viable, cultured embryos green, narrow-leaved	409
dek30	9L	defective kernel30, floury endosperm, lethal, cultured embryos green, narrow-leaved	409
dek31	4L-82+-	defective kernel31, pitted endosperm, lethal	408
dek33	5L-55+-	defective kernel33, opaque, floury, dented, wrinkled kernel with floury endosperm, occasionally viviparous	316
dep1	6	defective pistils1	288
des17	8L	defective seedling17, reduced height, partial suppression of primary root growth, contorted leaves, lethal	140
dia1	2-83+-	diaphorase1, electrophoretic mobility, cytosolic, monomeric, encodes dihydrolipoamide dehydrogenase	51, 474
dia2	1L-161+-	diaphorase2, electrophoretic mobility, cytosolic, dimeric, encodes dihydrolipoamide dehydrogenase	474
dib1		dichotomously branched1, main axis branches into two normal tops, most often at node 4-8 but variable, associated with aneuploidy	286, 287, 289
dp1	4L-118	distal pale1 seedling leaf tip virescent (E.G. Anderson, unpublished)	
Ds		designator for transposable factors regulated by *Ac*, modifies gene function and/or chromosome breakage (termed "*Ds-2*"), *Ds2* designates element isolated from *Adh1-2F11*	281
dsc1	4S	discolored kernel1, crumpled, discolored, germless lethal	199, 393
dSpm		defective *Spm*: designator for transposable factors regulated by *Spm*	395
dsy1		desynaptic1, male and female sterility, synaptic failure	157
dsy2		desynaptic2, like *dsy1*	160
dsy3		desynaptic3, like *dsy1*	155
dsy4		desynaptic4, like *dsy1*	155
Dt1	9S	Dotted1, regulates controlling element at *A1*, responding *a1-m* alleles express colored dots on colorless kernels and purple sectors on brown plants, encodes transposase	362

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
Dt2	6L	Dotted2, like *Dt1*	332
Dt3	7L	Dotted3, like *Dt1*, but expression variable	332
Dt4	4	Dotted4, like *Dt1*, but dots chiefly on crown of kernel	99
Dt5	9S	Dotted5, like *Dt1*	99
Dt6	4	Dotted6, like *Dt1*	428
du1	10L-31	dull endosperm1, glassy, tarnished endosperm, affects soluble starch synthase and branching enzyme IIa	124, 269
dv1		divergent spindle1, chromosomes unoriented at metaphase I, partial male and female sterility	70
dy1		desynaptic1, chromosomes unpaired in microsporocytes, partial male and female sterility, possibly defect in the synaptonemal complex, expressed later as sporadic loss of chiasma maintenance	308
e1	7L-59+-	esterase1, electrophoretic mobility, null allele is known, dimeric, intralocus hybrid bands occur, encodes esterase	399
e2		esterase2, presence-absence only, encodes esterase	401
e3	3	esterase3, electrophoretic mobility, dimeric, intralocus hybrid bands occur, encodes esterase	400
e4	3S-69+-	esterase4, electrophoretic mobility, null allele is known, monomeric, encodes esterase	179
e5(I)		esterase5(I), electrophoretic mobility, duplicate factor with *E5(II)*, encodes esterase	264
e5(II)		esterase(II), electrophoretic mobility, duplicate factor with *E5-(I)*, encodes esterase	264
e6		esterase6, presence-absence only, encodes esterase	264
e7		esterase7, presence-absence only, encodes esterase	264
e8	3S-14	esterase8, electrophoretic mobility, null allele is known, dimeric, intralocus hybrid bands occur, encodes esterase	264
e9		esterase9, electrophoretic mobility, null allele is known, encodes esterase	264
e10		esterase10, electrophoretic mobility, encodes esterase	264
eg1	5L	expanded glumes1, glumes open at right angle	48
el1	8L	elongate1, chromosomes uncoiled during meiotic metaphase and anaphase in male and female, frequent unreduced gametes	367
emp1	1S	empty pericarp1, germless, unfilled kernel	393
emp2	2L-74+-	empty pericarp2, germless, unfilled kernel	393
emp3	8L-89	empty pericarp3, small, extremely collapsed, defective, poorly viable kernel.	
En		enhancer: transposable element (equivalent to *Spm*), autonomous, regulates *I* (=*dSpm*) transposition (e.g. at *g2-m* = *pg-m* = *pg14-m*)	345
eno1	9S-56+-	enolase1, cDNA clone pZm245 complements enolase mutant in E. *coli*, encodes enolase	239
enp1	6L-13+-	endopeptidase1, electrophoretic mobility, null allele is known, monomeric, encodes endopeptidase	285
et1	3L-161	etched1, pitted, scarred endosperm, virescent seedling, plastid membranes altered	434
et2	2S	etched2, endosperm etched, seedlings off-white albino, with occasional greening of leaf tips	444
f1	1L-86	fine stripe1, virescent seedling, fine white stripes on base and margin of older leaves	254
fae1		fasciated ear1, small, rounded ears branched at their tips	408
fbr1		few-branched1, dominant *Fbr1* plants have tassel reduced to 0-3 branches, bract replaces next-to-bottom branch	313
Fcu		factor Cuna: controlling element of *r1-cu*	163
fl1	2S-68	floury1, endosperm opaque, soft, dosage effect with *fl1-ref* allele, but *o4* allele is recessive	181
fl2	4S-39	floury2, endosperm opaque, soft, dosage effect	124, 310
fl3	8L-24	floury3, endosperm opaque, soft, dosage effect	304
g1	10L-50	golden plant1, seedling and plant with distinctive golden yellow cast, stub of cut seedling displays golden vs. green	113, 115
g2	3S-0	golden plant2, golden pale-green, weak plants, sheaths whitish yellow-green, *pg-m* of Peterson (1960) is mutable allele carrying *En*	201
g6	9S-42+-	golden plant6, dominant *G6* plants golden, lighter yellowish sheaths	320
ga1	4S-13	gametophyte factor1, *Ga1* pollen grains are competitively superior to *ga1* on *Ga1* silks, *Ga1-S* super-gametophyte	214
ga10	5	gametophyte factor10	162
ga2	5L-55	gametophyte factor2, *Ga2* pollen grains are competitively superior to *ga2*	46
ga7	3L-167	gametophyte factor7, *ga7* pollen from heterozygotes is only 10-15% functional regardless of silk genotype	364
ga8	9S-50+-	gametophyte factor8, *Ga8* pollen grains are competitively superior to *ga8* on *Ga8* silks	398
gcb1		GC binding protein1, binds to anaerobic responsive element (ARE) of *Adh1* promoter	333
gdh1	1L-154+-	glutamic dehydrogenase1, electrophoretic mobility, null allele is known (cold sensitivity), intra/interlocus hybrid bands occur, encodes glutamic dehydrogenase	355
gdh2	10	glutamic dehydrogenase2, electrophoretic mobility, intralocus hybrid bands occur, encodes glutamic dehydrogenase	164
gl1	7L-36	glossy1, cuticle wax altered, leaf surface bright, water adheres	237
gl2	2S-30	glossy2, like *gl1*	182
gl3	4L-93	glossy3, like *gl1*	182

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
gl4	4L-62	glossy4, like *gl1*	432
gl5	4S-36+-	glossy5, like *gl1*, duplicate factor with *gl20*	124, 429, 432
gl6	3L-69	glossy6, like *gl1*	124
gl7		glossy7, like *gl1*	124
gl8	5L-68	glossy8, like *gl1*	124
gl9	3L	glossy9, expression poor	124
gl11	2S-49+-	glossy11, like *gl1*, abnormal seedling morphology	426a
gl13	4L	glossy 13, glossy leaf	433
gl14	2	glossy14, like *gl1*, duplicate factor with *gl24*	6
gl15	9L-66	glossy15, glossy leaf surface expressed after 3rd leaf	6
gl17	5S-34	glossy17, like *gl1*, but semi-dwarf with necrotic crossbands on leaves	368
gl18	8-0+-	glossy18, like *gl1*, expression poor	6
gl19	3S	glossy19, like *gl1*, barren plant with no ear or tassel	320
gl20		glossy20, like *gl1*, duplicate factor with *gl5*	429
gl21	10S	glossy21, like *gl1*, duplicate factor with *gl22*	320
gl22		glossy22, like *gl1*, duplicate factor with *gl21*	313
gl23		glossy23, like *gl1*	430
gl24		glossy24, like *gl1*, duplicate factor with *gl14*	430
glb1	1L-121	globulin1, electrophoretic mobility, null allele is known, embryo protein, encodes globulin, 63,000 kDa	234, 403
glb2		globulin2, presence-absence, encodes globulin, 45,000 kDa	234
glu1	10L-27+-	beta glucosidase1, electrophoretic mobility, cytosolic, dimeric, intralocus hybrid bands occur, plastid localization, encodes beta glucosidase	356
got1	3L-120	glutamate-oxaloacetate transaminase1 (possibly = *Ta1*), electrophoretic mobility, null allele is known, glyoxysomal, dimeric, intralocus hybrid bands occur, encodes aspartate aminotransferase	390
got2	5L-96	glutamate-oxaloacetate transaminase2, electrophoretic mobility, null allele is known, plastidial, dimeric, intralocus hybrid bands occur, encodes aspartate aminotransferase	166
got3	5S-35+-	glutamate-oxaloacetic transaminase3, electrophoretic mobility, null allele is known, mitochondrial, dimeric, intralocus hybrid bands occur, encodes aspartate aminotransferase	166
gpa1	10L-50+-	glyceraldehyde-3-phosphate dehydrogenase A1, chloroplastic, A subunit, encodes glyceraldehyde-3-phosphate dehydrogenase (NADP+) (phosphorylating)	41
gpc1	4-47+-	glyceraldehyde-3-phosphate dehydrogenase C1, cytosolic, C subunit, type 3 gene, coding region has sequence homology to *gpc2*, unique 3' untranslated region, constitutive expression, encodes glyceraldehyde-3-phosphate dehydrogenase C, cytosolic	41, 272, 382
gpc2	6S-0+-	glyceraldehyde-3-phosphate dehydrogenase C2, cytosolic, C subunit, cDNA clone isolated, coding region has homology to *gpc1*, unique 3' untranslated region, constitutive expression, encodes glyceraldehyde-3-phosphate dehydrogenase C, cytosolic	382
gpc3	4-47+-	glyceraldehyde-3-phosphate dehydrogenase C3, cytosolic, C subunit 3, coding sequence homology to *gpc4*, unique 3' untranslated region, encodes glyceraldehyde-3-phosphate dehydrogenase C, cytosolic	382
gpc4	5L-57+-	glyceraldehyde-3-phosphate dehydrogenase C4, C subunit, electrophoretic mobility, coding sequence homology to *gpc3*, unique 3' untranslated region., encodes glyceraldehyde-3-phosphate dehydrogenase C, cytosolic	383
grp1		glycine-rich protein1, protein with high glycine content and repetitive glycine stretches, putative cell wall components, encodes glycine-rich protein	96
grt1	5L	green tip1, pale yellow seedling with green first leaf tip, lethal	320
gs1	1L-135	green stripe1, grayish green stripes between vascular bundles on leaves, tissue wilts	118, 295
gs2	2S-54	green stripe2, like *gs1*, but pale green stripes, no wilting	124
gs3	6L	green stripe3, like *gs2*	320
gs4	10	green stripe4, dominant *Gs4* plants are like *gs1*	314
gt1	1	grassy tillers1, numerous basal branches, vegetatively totipotent in combination with *id1* and factors for perennialism	406
h1	3	soft starch1, endosperm soft, opaque	303
Hbr		Heartbreaker: element similar to Tourist	209
hcf1	2L	high chlorophyll fluorescence1, affects NADP+ oxidoreductase, green seedling	291
hcf2	1L	high chlorophyll fluorescence2, missing cytochrome f/b6 complex, yellow-green seedling	291
hcf3	1S-26+-	high chlorophyll fluorescence3, missing PSII thylakoid membrane core complex, green seedling	291
hcf4	1L	high chlorophyll fluorescence4, affects CO2 fixation, green seedling	292
hcf5	6S	high chlorophyll fluorescence5, affects PSII reaction, green seedling	294
hcf6	1S	high chlorophyll fluorescence6, missing cytochrome f/b6 complex, green seedling	249
hcf7	1L	high chlorophyll fluorescence7, defective processing of 16S rRNA	16
hcf12	1L	high chlorophyll fluorescence12, green seedling	249
hcf13	1L	high chlorophyll fluorescence13, affects CO2 fixation, green seedling	249

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
hcf15	2L	high chlorophyll fluorescence15, affects photophosphorylation, yellow-green seedling, may survive	249
hcf18	5L-67+-	high chlorophyll fluorescence18, major loss of PSI, other thylakoid complexes reduced, yellow-green seedling	294
hcf19	3L	high chlorophyll fluorescence19, affects PSII thylakoid membrane core complex, green/yellow-green seedling	249
hcf21	5L	high chlorophyll fluorescence21, affects CO2 fixation, Rubisco, green seedling	292
hcf23	4S	high chlorophyll fluorescence23, affects photophosphorylation, yellow-green seedling, may survive	249
hcf26	6S	high chlorophyll fluorescence26, affects electron transport, yellow-green, viable seedling	249
hcf28	10L	high chlorophyll fluorescence28, affects CO2 fixation, green seedling	294
hcf31	1S	high chlorophyll fluorescence31, missing chlorophyll a/b binding protein, yellow-green seedling	294
hcf34	6L	high chlorophyll fluorescence34, affects photophosphorylation, yellow-green seedling	249
hcf36	6L	high chlorophyll fluorescence36, affects electron transport, green seedling	294
hcf38	5L	high chlorophyll fluorescence38, affects cytochrome f/b6 complex, alpha and beta components of CF1, green seedling	249
hcf41	1L	high chlorophyll fluorescence41, affects PSII thylakoid membrane core complex, green seedling	249
hcf42	9L	high chlorophyll fluorescence42, affects Rubisco, green/yellow-green seedling	292
hcf44	1L	high chlorophyll fluorescence44, affects PSI membrane core complex, pale-green seedling, lethal	292
hcf46	3L	high chlorophyll fluorescence46	249
hcf47	10S	high chlorophyll fluorescence47, affects cytochromes, yellow-green seedling	294
hcf48	6L	high chlorophyll fluorescence48, affects electron transport, yellow-green seedling	294
hcf50	1L	high chlorophyll fluorescence50, missing PSI thylakoid membrane core complex, seedling slightly pale green	292
hcf101	7L	high chlorophyll fluorescence101, affects PSI thylakoid membrane core complex	294
hcf102	8L	high chlorophyll fluorescence102, affects cytochrome f/b6 complex	293
hcf103	7L	high chlorophyll fluorescence103, reduced photosystem II activity due to absence of plastoquinone (PQ-9)	83
hcf104	7L	high chlorophyll fluorescence104, photosystem I-deficient	83
hcf106	2-74+-	high chlorophyll fluorescence106, affects PSI, PSII, cytochrome f/b6	271
hcf108	5	high chlorophyll fluorescence108, ATPase-deficient	83
hcf111	7L	high chlorophyll fluorescence111, cytochrome b6/f-deficient	83
hcf113	9S	high chlorophyll fluorescence113, multiple effects, yellow-green seedlings	82
hcf316	10S	high chlorophyll fluorescence316, affects chlorophyll a/b binding protein, yellow-green seedling	294
hcf323	6S	high chlorophyll fluorescence323, affects photophosphorylation, coupling factor, green seedling	294
hcf408	6L	high chlorophyll fluorescence408, affects chlorophyll a/b binding protein, yellow-green seedling	294
hex1	3S-35+-	hexokinase1, electrophoretic mobility, null allele is known, cytosolic, monomeric, encodes hexokinase	472
hex2	6L-60+-	hexokinase2, electrophoretic mobility, null allele is known, cytosolic, monomeric, encodes hexokinase	472
hfi1		Hageman factor inhibitor1, cDNA clone, partial amino acid sequence,yeast-expressed product inhibits trypsin, encodes corn(activated) Hageman factor inhibitor	470
his1		histone H1 family, cDNA (see *alh1*), encodes histone I	361
his3		histone H3 family, (see *clh1*) 60-80 copies/diploid genome, encodes histone 3	66
his4		histone H4 family, 100-120 copies/diploid genome (Chaubet et al., 1986), encodes histone 4	67, 68
hm1	1L-64	*Helminthosporium carbonum* susceptibility1, disease lesions vs. yellowish flecks (resistant) on leaves with race 1, encodes NADPH HC-toxin reductase	462
hm2	9L-82+-	*Helminthosporium carbonum* susceptibility2, like *hm1*, masked by *Hm1*	307
hox1	8-52+-	homeobox1, protein product binds to *sh1* promoter (feedback control element), is found in nuclei, transcript found in many tissues, protein not in root, encodes HOX1 protein	28
hox2		homeobox2, similar to *hox1*, but sequence predicts not allelic to *hox1* (Bellmann and Werr 1992), encodes HOX2 protein	28
hpt1		hygromycin phosphotransferase1, transgenic chimeric gene, single dominant locus, coding region origin *E. coli* introduced by particle bombardment, encodes hygromycin-B kinase	468
hrg1	2S	hydroxyproline rich glycoprotein1, cDNA, genomic clones, single site (Southern analysis), accumulates preferentially in provascular cells, encodes hydroxyproline-rich glycoprotein	439
hs1	7S-0	hairy sheath1, dominant *Hs1* plants have abundant hairs on leaf sheath	453
hsf1	5	hairy sheath frayed1, dominant *Hsf1* plants have pubescent sheaths and leaf margins, liguled enations at leaf margins	33
hsp1		heat shock protein1, genomic clones, single copy (Southern blots), transcribed (Northern blots), transgenic (petunia) expression, encodes hsp 70	380
ht1	2L-121	*Helminthosporium turcicum* resistance1, dominant *Ht1* plants resistant	188

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
ht2	8L-66+-	*Helminthosporium turcicum* resistance2, dominant *Ht2* plants resistant	189
ht3		*Helminthosporium turcicum* resistance3, (from *Tripsacum floridanum*), dominant *Ht3* plants resistant	190
htn1	8L-80+-	northern corn leaf blight resistance1, (was *HtN*) resistance to *Setosphaeria turcica* (*Helminthosporium turcicum*)	414
hyp1		hybrid proline-rich protein1, genomic sequence, mRNA accumulates in immature zygotic embryos, in ovary, encodes hybrid proline-cysteine rich protein (HyPRP)	215
id1	1L-104+-	indeterminate growth1, requires extended growth and short days for flowering, vegetatively totipotent with *gt1* and factors for perennialism	416
idh1	8L-66+-	isocitrate dehydrogenase1, electrophoretic mobility, null allele is known, cytosolic, dimeric, intra/interlocus hybrid bands occur, encodes isocitrate dehydrogenase	166
idh2	6L-78+-	isocitrate dehydrogenase2, electrophoretic mobility, null allele is known, cytosolic, dimeric, intra/interlocus hybrid bands occur, encodes isocitrate dehydrogenase	166
ig1	3L-90	indeterminate gametophyte1, low male fertility, polyembryony, heterofertilization, polyploidy, androgenesis (male and female affected)	230
ij1	7L-52	iojap striping1, many variable white stripes and margin patterns on leaves (compare *cm1*), conditions chloroplast defects that are cytoplasmically inherited	200
ij2	1L-161+-	iojap striping2, like *ij1*, chloroplast inheritance unknown	320
in1	7S-20	intensifier1, intensifies aleurone anthocyanin pigments, *In1-D* dominant dilute	135
Ins1	9S	insertion1: located upstream of *bz1-R*, up to 50 copies in genome (Southerns)	359
Ins2	9S	insertion2: 447 bp element upstream of *bz1-R*	359
is1		cupulate interspace1	143
isp1		iron-sulfur protein1, cDNA, functional in yeast, encodes Rieske iron-sulfur protein, mitochondrial	193
isr1	10L-64+-	inhibitor of striate1, dominant *Isr1* plants have reduced expression of *sr2* and other leaf-striping factors	231
j1	8L-80	japonica striping1, white stripes on leaf and sheath, not often expressed in seedling	115
j2	4L-87	japonica striping2, extreme white striping of leaves, etc.	124
K		knob, general symbol for heterochromatic structures (knobs) that are heritably polymorphic in size and are found at characteristic positions on the chromosomes, homology with 185bp probe	340
K3L	3L-115	knob, heterochromatic structure	94
K10	10L-98+-	knob, heterochromatic appendage on long arm of chromosome 10, neocentric activity distorts segregation of linked genes	259
kn1	1L-133	knotted1, dominant *Kn1* plants have localized proliferation of tissue at vascular bundles on leaf	43
kn2		knotted2, dominant *Kn2* plants have finger-like projections of leaf at the ligule	137
l1	10L-64+-	luteus1, yellow pigment in white tissue of specific chlorophyll mutants *w1*, *w2*, *j1*, *ij1*, others	252, 253
l3		luteus3, lethal yellow seedling	207
l4		luteus4, lethal yellow seedling	207
l6	9S-31+-	luteus6, like *l4* (W.H. Eyster, 1935, unpublished data)	124
l7	9S-42	luteus7, yellow seedling and plant, lethal	131
l10	6L-19	luteus10, like *l4*, fails to convert protochlorophyllide to chlorophyllide	374
l11	6S	luteus11, yellow seedling with green leaf tips, lethal	13
l12	6L-16	luteus12, like *l11*	88
l13	10L-94	luteus13, dark yellow, lethal seedling, fails to convert protoporphyrin IX to Mg-protoporphyrin	273, 320
l15	6L-30	luteus15, like *l4*	378
l16	1S	luteus16, like *l4*, leaves bleach to paler yellow in patches	320
l17	1L	luteus17, like *l4*, leaves with lighter yellow crossbands	320
l18	2L	luteus18, like *l4*	320
l19	10S	luteus19, like *l4*	320
la1	4S-36	lazy plant1, prostrate growth habit	206
lbl1		leaf bladeless1, leaf blade reduced to absent, low temperature enhances expression	290
lc1	10L-68	red leaf color1, dominant *Lc1*, anthocyanin in coleoptile, nodes, auricle, leaf blade, etc., (compare *Sn1*)	104
lcs1		last cluster-second1, thylakoid membrane polypeptide, electrophoretic mobility	299
lct1		last cluster-third1, thylakoid membrane polypeptide, electrophoretic mobility	299
lct2		last cluster-third2, thylakoid membrane polypeptide, presence-absence	299
les1	2S-58	lesion1, dominant *Les1*, large necrotic lesions resembling disease lesions formed by fungal infections on susceptible lines	323
les2	1S-0+-	lesion2, dominant *Les2*, small white lesions resembling disease lesions formed by fungal infections on resistant lines	323
les3	10	lesion3, like *les1*	11
les4	2L	lesion4, dominant *Les4*, late expression of large necrotic lesions on leaf blade and sheath	185
les5	1S	lesion5, like *les2*	185
les6	10S	lesion6, like *les4*	185
les7		lesion7, dominant *Les7*, late expression of small chlorotic lesions	185
les8	9S-50+-	lesion8, dominant *Les8*, late expression of small, pale green lesions	185

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
les9	7L-32+-	lesion9, dominant *Les9*, late expression of small necrotic lesions	185
les10	2-83+-	lesion10, like *Les1*	186
les11	2	lesion11, like *les1*	316
les12	10	lesion12, dominant *Les12*, many small to medium, chlorotic to necrotic lesions form in clusters on the leaf blade beginning at 5 leaf stage and rapidly coalesce to form large senescent areas that may spread over the whole leaf and cause early death	316
les13	6L	lesion13, dominant *Les13*, frequent small to medium necrotic spots on leaf blade, sheath and culm, appearing at the 5 leaf stage	316
les14	3L	lesion14, dominant *Les14*, many small brown necrotic spots with light centers, some with anthocyanin halos on leaf blade beginning at the 6 leaf stage, no reduction in height or vigor	316
les15	2	lesion15, dominant *Les15*, tiny yellowish green plants with many small chlorotic and necrotic lesions on speckled yellow green leaf blade background that looks like iron deficiency symptoms	316
les16	10	lesion16, dominant *Les16*, pale green plant develops small chlorotic lesions on the leaf blade just before flowering	316
les17	3L	lesion17, dominant *Les17*, profuse small to medium chlorotic and necrotic lesions expressed at 8-10 leaf stage causing plants to have a light green color, occasional normal green non-lesion sectors appear on leaves	316
lfy1		leafy1, dominant *Lfy1*, increased number of leaves	405
lg1	2S-11	liguleless1, ligule and auricle missing, leaves upright, enveloping	113, 114
lg2	3L-101	liguleless2, like *lg1*, less extreme	37
lg3	3-65	liguleless3, dominant *Lg3*, no ligule, leaves upright, broad, often concave and pleated	341
lg4	8L-24+-	liguleless4, dominant *Lg4*, no ligule or auricle but vestiges sporadically in blade	134
lhcb1	3	light harvesting chlorophyll a/b binding protein1, gene-specific cDNA probe,low expression in bundle sheath cells, encodes chlorophyll a/b binding protein	407, 482
lhcb2	7	light harvesting chlorophyll a/b binding protein2, gene specific cDNA probe, expressed in dark	407, 450, 482
li1	10L-33+-	lineate leaves1, fine, white striations on basal half of mature leaves	81
lls1	1S	lethal leaf spot1, chlorotic-necrotic lesions resembling *Helminthosporium carbonum* infection	461
ln1	6	linoleic acid1, lower ratio of oleate to linoleate in kernel	89
lo2	9S-50	lethal ovule1, ovules containing *lo2* gametophyte abort	308
loc1		low oil content1, low oil content in kernel, associated with albino seedlings	352
lp1	4	lethal pollen1, *lp1* pollen fails in competition with *Lp1*	305
lty1		light yellow endosperm1	101
lty2		light yellow endosperm1	101
lu1	5S-29	lutescent1, pale yellow green leaves	412
lw1	1L-134+-	lemon white1, white seedling, pale yellow endosperm	459
lw2	5L-67.6	lemon white2, like *lw1*	459
lw3	5L-107+-	lemon white3, like *lw1*, duplicate factor with *lw4*	459
lw4	4-55+-	lemon white4, like *lw1*, duplicate factor with *lw3*	459
lxm1	3	lax midrib1, dominant *Lxm1*, leaves with wide, flat, flexible midrib	312
mc1		mucronate1, dominant *Mc1*, opaque endosperm	387
mch1		maize CRY1 homolog1, ribosomal protein gene family (cDNA probe)	243
mch2		maize CRY1 homolog2 ribosomal protein gene family (cDNA probe)	243
mct1		modifier of *cox2* transcripts1, changes transcripts of mitochondrial gene	85
mdh1	8-10+-	malate dehydrogenase1, electrophoretic mobility, null allele is known, mitochondrial, dimeric, intra/interlocus hybrid bands occur, encodes malate dehydrogenase	328
mdh2	6L-78+-	malate dehydrogenase2, electrophoretic mobility, null allele is known, mitochondrial, dimeric, intra/interlocus hybrid bands occur, encodes malate dehydrogenase	328
mdh3	3L-146	malate dehydrogenase3, electrophoretic mobility, null allele is known, mitochondrial, dimeric, intra/interlocus hybrid bands occur, encodes malate dehydrogenase	328
mdh4	1L-104+-	malate dehydrogenase4, cDNA sequence, electrophoretic mobility, null allele is known, cytosolic, dimeric, intra/interlocus hybrid bands occur, encodes malate dehydrogenase	62, 328
mdh5	5S-17	malate dehydrogenase5, electrophoretic mobility, null allele is known, cytosolic, dimeric, intra/interlocus hybrid bands occur, encodes malate dehydrogenase	328
mdm1	6S-4.4	maize dwarf mosaic virus resistance1, dominant *Mdm1*	284
me1	3L-125	NADP malic enzyme1, electrophoretic mobility, null allele is known, tetrameric, encodes malate dehydrogenase (oxaloacetate decarboxylating) (NADP+)	166
me3	3S-26+-	NADP malic enzyme3, cDNA sequence, deduced plastid transit peptide, single copy, encodes malate dehydrogenase (oxaloacetate decarboxylating) (NADP+)	62, 222, 381
mei1		meiosis1, dominant *Mei1*, chromosomes sticky in metaphase I, male sterile	156, 159
mep1	5L	modifier of embryo protein1, affects quantities of *Glb1* protein forms	403
mg1		miniature germ1, germ 1/4 to 1/3 of normal, viable	240
mgs1	10-50+-	male-gametophyte specific1, mRNA in cytoplasm of both vegetative cell (pollen grain) and pollen tube, not expressed in shoot, root, kernel, ovule, silk, encodes MGS1 protein	176, 445
mgs2	4L-98+-	male gametophyte-specific2, cDNA with pectate lyase homology	54

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
mi1	1	midget plant1, small plant (H.S. Perry, 1935, unpublished data)	124
mmm1	1L-104+-	modifier of mitochondrial malate dehydrogenases1, mobilities altered	328
mn1	2-68+-	miniature seed1, small, somewhat defective kernel, fully viable, invertase reduced	261
mn2	7	miniature seed2, small kernel, loose pericarp, extremely defective but will germinate (R.J. Lambert, unpublished)	
mn3	6-8+-	miniature seed3, small kernel, etched/pitted endosperm, viable	442
Mod		modifier: inactive Spm element, enhances excisions elicited by active *Spm*	283
Mp		modulator of pericarp: transposable factor affecting *P1* locus, parallel to *Ac-Ds*	40
Mpi1		transposable element, 10-15 copies in the genome	477
mpl1	1L-134+-	miniplant1 dominant *Mpl1*, andromonoecious, intermediate dwarf (compare *D8*, possible allele), not responsive to gibberellins	177
Mr	9S	mutator of *R1-m*: transposable factor	60
Mrh	5	mutator: controlling element of *a1-m-rh*	369
ms1	6L-20	male sterile1, anthers shriveled, not usually exserted, affected at microspore vacuolation	419
ms2	9L-64	male sterile2, like *ms1*, affected between vacuolation and pore formation	129, 131
ms3	3	male sterile3, anthers shrivelled, not usually exserted	129, 131
ms5	5-42+-	male sterile5, anthers not exserted, affected at microspore mitosis	22
ms7	7L-32+-	male sterile7, like *ms2*	22
ms8	8L-66	male sterile8, like *ms5*, affected in meiosis	22
ms9	1S-26+-	male sterile9, like *ms5*, affected in meiosis	22
ms10	10L-45+-	male sterile10, like *ms5*, affected at microspore vacuolation	22
ms11	10	male sterile11, like *ms5*, affected at microspore mitosis	22
ms12	1	male sterile12, like *ms1*, affected at microspore vacuolation	22
ms13	5S	male sterile13, like *ms5*, affected at microspore vacuolation	22
ms14	1-56+-	male sterile14, like *ms5*, affected at microspore mitosis	22
ms17	1S-23	male sterile17, like *ms1*, affected variably in meiosis	121
ms20		male sterile20	131
ms21	6	male sterile21, pollen grains developing in presence of *Ms21* are defective and nonfunctional if *sks1*, normal if *Sks1*	247, 397
ms22		male sterile22, affected in meiosis	476
ms23	3L	male sterile23, affected in meiosis	476
ms24		male sterile24, like *ms1*, affected in microspore mitosis	476
ms28	1S-19+-	male sterile28, anaphase I disturbed, spindle persists	159
ms41	4L	male sterile41, *Ms41*	320
ms42	5S	Ms42 plants male sterile, penetrance varies	3
ms43	8L	male sterile43, anaphase I impaired	156, 159
ms44	4L-98+-	male sterile44, *Ms44*	2
msc1	1L	mosaic1, *Msc1* aleurone mosaic for anthocyanin color	320
msc2	5S	mosaic2, *Msc2* aleurone mosaic for anthocyanin color	320
mst1	10L-70	modifier of *R-st*, *Mst1* affects expression of *R1-st*	10
Mu		mutator: freely transposable element, *Mu1* designates element isolated from *Adh1-S3034*	377
Mu4		mutator4: elements with terminal inverted repeats similar to *Mu1*	452
Mu5		Mutator5: element with inverted terminal repeats similar to *Mu1*	452
Mu8		Mutator8: 1.4 kbp element within *wx1-mum5*, terminal inverted repeats similar to *Mu1*	133
Mut	2S	mutator: controlling element for *bz1-m-rh*	369
mv1	3	mosaic virus resistance1, resistance to "corn stripe"	35
na1	3L-113	nana plant1, short, erect dwarf, no response to gibberellins	197, 251
na2	5S-17+-	nana plant2, like *na1* (H.S. Perry, unpublished)	
nabp1	7S-25+-	nucleic acid binding protein1	54
nbp1	7L	nuclear encoded chloroplast nucleic acid binding protein, genomic and cDNA clones, product is imported in vitro into chloroplasts, expressed only in leaf, encodes nucleic acid-binding protein	84
NCS1		nonchromosomal stripe1, maternally inherited light green leaf striping	413
NCS2		nonchromosomal stripe2, maternally inherited pale green and depressed striping, mitochondrial	73
NCS3		nonchromosomal stripe3, maternally inherited striations, distorted plants, mitochondrial	73
NCS5		nonchromosomal stripe5, maternally inherited stunted growth, yellow stripes, aborted kernels, mitochondrial cytochrome oxidase subunit 2 (*cox2*) alteration	329
NCS6		nonchromosomal stripe6, maternally inherited stunted growth, yellow stripes, aborted kernels, mitochondrial cytochrome oxidase subunit 2 (*cox2*) alteration	244
nec1	8-22+-	necrotic1, chlorotic seedling that stays rolled, wilts and dies	274
nec2	1S-34	necrotic2, green seedling develops necrotic lesions at 2-3 leaf stage, lethal (E.G. Anderson, 1952, unpublished data)	
nec3	5-42+-	necrotic3, seedling emerge with tightly rolled leaves that turn brown and die without unrolling, manually unrolled leaves tan with dark brown crossbands	311
nec4	2S-34+-	necrotic4, seedling yellow, leaf tips necrotic, lethal	187
nec5	4L	necrotic5, pale green seedling becoming necrotic, dark brown exudate, lethal	320

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
nec6	5S-35+-	necrotic6, like *nec3*	320
nec7	5L	necrotic7, seedling becoming necrotic in crossbands, lethal	320
nl1	10L-33+-	narrow leaf1, leaf blade narrow, some white streaks	124
nl2	5S-25	narrow leaf2, dominant Nl2, leaves narrow and distorted, tillering	321
nnr1		nitrate reductase(NADH)1, leaf, scutellum cDNA's, flavin and cyt b domains functional in *E.coli*, may be allelic to nnr3, encodes nitrate reductase (NADH)	167
nnr3		nitrate reductase(NADH)2, scutellum cDNA, cyt b domain functional in *E.coli*, encodes nitrate reductase (NADH)	55
NOR	6S	nucleolus organizer: encodes ribosomal RNA's	279
o1	4L-98	opaque endosperm1, endosperm starch soft, opaque	124, 310
o2	7S-16	opaque endosperm2, like *o1*, high lysine content, regulates b-32 protein (see *pro1*), reduced lysine degradation (lysine-ketoglutaric reductase)	124, 310
o5	7L-32+-	opaque endosperm5, like *o1*, virescent to yellow or white seedlings	373
o7	10L-90	opaque endosperm7, like *o1*, high lysine content	296
o8	2-74+-	opaque endosperm8, higher in lysine	169
o9		opaque endosperm9, crown opaque and light in color, frequently with a cavity, base or abgerminal side of kernel often corneous	305
o10	1L	opaque endosperm10, like *o1*	305
o11		opaque endosperm11, thin, opaque, somewhat shrunken kernels with greyish cast	305
o12	4S	opaque endosperm12, thin, etched or scarred kernels, variable in size, plants chlorophyll deficient and small, with pollen but few ears	305
o13		opaque endosperm13, opaque, etched kernels with rim of corneous starch on abgerminal side	305
o14	6	opaque endosperm14, large opaque kernel with mostly floury starch except for a small amount of corneous starch near the base of the abgerminalside, normal green seedling develops yellow striped appearance and is slow in growth	316
obf1	1-56+-	octopine synthase binding factor1, encodes protein with bZIP motif that binds to transcriptional enhancer sequences (ocs-elements)	415
obf2	9-56+-	octopine synthase binding factor2, encodes protein with bZIP motif that binds to transcriptional enhancer sequences (ocs-elements)	415
og1	10S-19	old gold stripe1, dominant *Og1*, variable bright yellow stripes on leaf blade	257
omt1		O-methylthransferase1, genomic and cDNA clones, sequence, single site (Southern analysis), transgenic expression in *E. coli*, encodes O-methyltransferase	77
ora2		orange endosperm2	100
ora3		orange endosperm3	101
oro1	6S	orobanche1, 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	273
oro2		orobanche2, like *oro1*	273
orom1		orobanche modifier1, dominant *Orom1* partially corrects chlorophyll loss in *oro1*	273
orp1	4S-47+-	orange pericarp1, duplicate factor with *orp2*, pericarp orange over *orp1 orp2* kernels, lethal, tryptophan auxotroph, encodes tryptophan synthase	322
orp2	10S-33+-	orange pericarp2, duplicate factor with *orp1*, encodes tryptophan synthase	322
os1	2S	opaque endosperm-small germ1, opaque crown, kernel larger, lighter color, viable, reduced oil content	424
oy1	10S-15	oil yellow1, 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	130
P		plant color component at *R1*, anthocyanin pigmentation in seedling leaf tip, coleoptile, anthers	103, 435, 436
p1	1S-26	pericarp color1, dominant *P1*, red pigment in cob and pericarp (for alleles, see Coe et al., 1988)	112, 258
pam1		plural abnormalities of meiosis1, desynchronized meiotic divisions and premeiotic mitosis, male sterile, incompletely female sterile	158
pam2		plural abnormalities of meiosis2, like *pam1*	160
pb1	6L-20+-	piebald leaves1, very light, irregular green bands on leaf	93
pb4	6L-17+-	piebald leaves4, like *pb1*	93
pd1	3	paired rows1, single vs. paired pistillate spikelets, quantitative, one of a family of loci differentiating maize vs. teosinte	241
pdc1	8L-24+-	pyruvate decarboxylase1, cDNA and genomic sequences, encodes pyruvate decarboxylase	223
pdc2	8-0+-	pyruvate decarboxylase2, cDNA sequence, single copy, encodes pyruvate decarboxylase	343
pdc3	1S	pyruvate decarboxylase3, cDNA sequence, single copy, encodes pyruvate decarboxylase	343
pdf1		pink doublet-faster band1, thylakoid membrane polypeptide, dominant increase in electrophoretic mobility	298
pdk1	6L	pyruvate, orthophospate dikinase1, products (mRNA, protein) most abundant in mesophyll chloroplasts, transcripts vary, depending on tissue, by inclusion/exclusion of exon encoding chloroplast transit peptide, encodes pyruvate, orthophosphate dikinase	152
pe1		perennialism1, vegetatively totipotent in combinations with *gt1* and *id1*	406

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
pep4	7	phosphoenolpyruvate carboxylase4, cDNA for presumptive anaplerotic C3 isozyme based on sequence homology, gene specific probe maps to new site, expression in green leaves>etiolated leaves>roots, encodes phosphoenolpyruvate carboxylase	221
pet1	8L	photosynthetic electron transport1, high leaf chlorophyll fluorescence, palegreen, lacks cytochrome b6/f, reduced PSII at higher intensity light	16
pet2		photosynthetic electron transport2, lacks cytochrome b6/f polypeptides	16
pet3		photosynthetic electron transport3, lacks cytochrome b6/f polypeptides	16
pet4		photosynthetic electron transport4, lacks cytochrome b6/f polypeptides	16
pet5		photosynthetic electron transport5, lacks cytochrome b6/f polypeptides	16
pg11	6L-38	pale green11, duplicate factor with *pg12*, seedling light yellowish green, mature plant pale and vigorous	365
pg12	9-61	pale green12, duplicate factor with *pg11*	365
pg13		pale green13, seedling light yellowish green, stunted growth	411
pg15	1S	pale green15, seedling light yellowish green, bleaches to near white in patches, lethal	320
pg16	1L	pale green16, seedling light yellowish green	320
pgd1	6-8+-	6-phosphogluconate dehydrogenase1, electrophoretic mobility, null allele is known, cytosolic, dimeric, intra/interlocus hybrid bands occur, encodes 6-phosphogluconate dehydrogenase	166
pgd2	3L-67+-	6-phosphogluconate dehydrogenase2, electrophoretic mobility, null allele is known, cytosolic, dimeric, intra/interlocus hybrid bands occur, encodes 6-phosphogluconate dehydrogenase	166
pgm1	1L-121+-	phosphoglucomutase1, electrophoretic mobility, null allele is known, cytosolic, monomeric, encodes phosphoglucomutase (glucose-cofactor)	166
pgm2	5S-0	phosphoglucomutase2, electrophoretic mobility, null allele is known, cytosolic, monomeric, encodes phosphoglucomutase (glucose-cofactor)	166
ph1	4S-0+-	pith abscission1, cob disarticulation, quantitative, one of a family of loci differentiating maize vs. teosinte	144
phi1	1L-149	phosphohexose isomerase1, electrophoretic mobility, null allele is known, cytosolic, dimeric, intralocus hybrid bands occur, encodes glucose-6-phosphate isomerase	166
php1	10S-27+-	chloroplast phosphoprotein1, isozyme, encodes chloroplast phosphoprotein	54
pi1		pistillate florets1, duplicate factor with *pi2*, secondary florets develop ("Country Gentlemen" or "Shoe Peg" expression) in *pi1 pi2* ears, quantitative character	194
pi2		pistillate florets2, duplicate factor with *pi1*	194
pl1	6L-49	purple plant1, *Pl1* plants have sunlight-independent pigment in plant, light-dependent in *pl1*, *Pl1-Bh1* allele shows colored patches in aleurone tissue of *c1* (colorless) kernels and in plant, regulates flavonoid enzymes, encodes *Pl1* protein	119
pm1	3L-73+-	pale midrib1, midrib and adjacent tissue lighter green, reduced plant vigor	38
pmg1		phosphoglycerate mutase1, cDNA sequence corresponds to sequence of purified protein, also partial genomic sequence, amino acid sequence similar to alkaline phosphatases (yeast, *E. coli*, human), encodes phosphoglycerate mutase, cofactor independent	170
pn1	7L-112	papyrescent glumes1, dominant *Pn1*, long, thin papery glumes on ear and tassel	145
po1	6S-4	polymitotic1, repeats 2nd meiotic division in male and female	20
ppg1	5L	pale pale green1, white seedling with faint green, white necrotic crossbands, lethal	320
pr1	5L-67	red aleurone1, changes purple aleurone to red, flavonoid 3'-hydroxylase	108
prh1	4L-62+-	ser/thr protein phosphatase1, PCR clone from root mRNA, expressed in *E. coli* as active kinase, 4-8 copies by Southern analyses, highest expression in roots, seedling shoots, encodes serine/threonine specific protein phosphatase	422
pro1	8L-22	proline requiring1, crumpled opaque kernel, b32 protein isoforms and null, green-striped lethal seedling	147
ps1	5S-39	pink scutellum1, some alleles viviparous, endosperm and scutellum pink, seedling white with pink flush	426
psa1		photosystemI1, lacks photosystem I core complex polypeptides	16
psa2		photosystemI2, lacks photosystem I core complex polypeptides	16
psa3		photosystemI3, lacks photosystem I core complex polypeptides	16
psa4		photosystemI4, lacks photosystem I core complex polypeptides	16
psb1	8L	photosystemII1, lacks protein components of photosystem II core complex, pale green	16
psb2		photosystemII2, lacks photosystem II core complex polypeptides	16
psei1		cystatin1, cDNA clone encodes 135 amino acid protein, includes a signal peptide fragment and has papain-inhibitory activity, expressed in endosperm maximally 2 weeks after flowering, encodes cysteine proteinase inhibitor	1
pt1	6L-60	polytypic ear1, dominant *Pt1*, proliferation produces irregular growth on ear and tassel	309
ptd1	1L-116+-2	pitted endosperm1, small seed with pitted, scarred endosperm and small germ, usually lethal, seed with larger embryos will germinate to produce small, non-flowering plants with large, necrotic, mottled sectors on leaves	393
ptd2	7L	pitted endosperm2, pitted, cracked endosperm, small germ, generally lethal, approximately 10% of seed produce plants with narrow, frayed leaves with necrotic margins and with sterile, rudimentary ear and tassel	393

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
px1	2L	peroxidase1, electrophoretic mobility, null allele is known, monomeric, encodes peroxidase	175
px2		peroxidase2, electrophoretic mobility, monomeric, encodes peroxidase	263
px3	7L-112+-	peroxidase3, electrophoretic mobility, monomeric, encodes peroxidase	263
px4		peroxidase4, electrophoretic mobility, null allele is known, monomeric, encodes peroxidase	263
px5		peroxidase5, presence-absence, encodes peroxidase	263
px6		peroxidase6, presence-absence, encodes peroxidase	263
px7		peroxidase7, electrophoretic mobility, null allele is known, monomeric, encodes peroxidase	263
px8		peroxidase8, electrophoretic mobility, monomeric, encodes peroxidase	36
px9		peroxidase9, electrophoretic mobility, null allele is known, monomeric, encodes peroxidase	36
py1	6L-69	pigmy plant1, leaves short, pointed, fine white streaks	451
py2	1L-161+-	pigmy plant2, like *py1*	320
pyd1	9S	pale yellow seedling1, deficiency for short terminal segment of chromosome arm, lethal, (for alleles, see Coe et al., 1988)	280
r1	10L-64	colored, dominant *R1*, red or purple color in aleurone and/or anthers, leaf tip, brace roots, etc. (for alleles, see Coe et al., 1988), regulates flavonoid enzymes, encodes *R1* protein	108
ra1	7L-32	ramosa1, ear and tassel many-branched, tassel branches taper to tip	22, 150
ra2	3S-49	ramosa2, irregular kernel placement, tassel many-branched, upright (R.A. Brink, 1935, unpublished data)	124, 330
ra3	4	ramosa3, (H.S. Perry, 1954, unpublished data)	
rab17	6L-69+-	responsive to abscisic acid17, dehydration-induced protein expressed in roots and shoots of seedlings, cDNA and peptide sequence, encodes glycine-rich protein(RAB17)	72
rab28		responsive to abscisic acid28, cDNA and genomic clones, inducible by ABA in embryos and young leaves, induced by water-stress in leaves, homologous to cotton LeaD-34	351
rab30	1S-0+-	responsive to abscisic acid30, cDNA	54
rBg		receptor of *Bg*	386
rcm1	7-25+-	rectifier1, dominant *Rcm1* restores miniature seed of teosinte cytoplasm to normal	4
rcm2		rectifier2, dominant *Rcm2* weakly restores miniature seed of teosinte cytoplasm to normal	4
rcm3		rectifier3, dominant *Rcm3* restores miniature seed of teosinte cytoplasm to normal, from *Z diploperennis*	4
rcu		receptor of *Fcu*	163
rcy		receptor of *Cy*	396
rd1	1L-149+-	reduced plant1, semi-dwarf plant, possible allelism with *bv2*	306
rd2	6L	reduced plant2, like *rd1*, but not as extreme	154
rd3	3L-77+-	reduced plant3, like *rd1*, anthocyanin interactions	277
rDNA18S	6S	NOR (nucleolus organizer) component, encodes rRNA18S	218
rDNA25S	6S	NOR (nucleolar organizer) component, encodes rRNA25S	218
rDNA5.8S	6S	NOR (nucleolus organizer) component, encodes rRNA5.8S	218
rDNA5S	2L	cluster consisting of several thousand repeated genes, encodes rRNA5S	438
rDt		receptor of Dotted	102
ren1	5L-67+-	reduced endosperm1, small seed with reduced, opaque endosperm, usually lethal, seed with larger embryos produce fertile plants	393, 394
ren2	7L-59+-10	reduced endosperm2, endosperm variably reduced in size, often with loose pericarp and small germ, usually lethal, larger seed may produce small plants with rudimentary sterile tassel	199, 393
ren3	10L-65+-30	reduced endosperm3, reduced endosperm, partially filled to empty pericarp, small germ or germless, larger seed produce fertile plants	394
rf1	3S-62+-	fertility restorer1, dominant *Rf1* restores fertility to cms-T, complementary to *Rf2*	212
rf2	9-56+-	fertility restorer2, see *rf1*	107
rf3	2L-155+-	fertility restorer3, *Rf3* restores fertility to cms-S	44
rf4	8-0+-	fertility restorer4, dominant *Rf4* restores fertility to cms-C, complementary with *rf5* and *rf6*	168
rf5		fertility restorer5, dominant *Rf5* restores fertility to cms-C, complementary with *rf4* and *rf6*	217, 464
rf6		fertility restorer6, dominant *Rf6* restores fertility to cms-C, complementary with *rf4* and *rf5*	217, 464
rf7		fertility restorer7, dominant *Rf7* partially restores fertility to cms-Y	358
rg1	3-67	ragged leaves1, dominant *Rg1*, defective tissue between veins of older leaves, causing holes and tearing	39
rgd1	6-8	ragged seedling1, seedling leaves narrow, thread-like, have difficulty in emerging	233
rgh1	8L-111	rough kernel1, small floury kernel with rough and pitted surface and nonviable embryos	318
rgo1		reversed germ orientation1, embryo faces base of ear, variable frequency, maternal trait	384
rhm1	6-4+-	resistance to *Helminthosporium maydis*1, chlorotic-lesion reaction with race O	420
ri1	4S-27	rind abscission1, dominant *Ri1*, cob disarticulation, quantitative, one of a family of loci differentiating maize vs. teosinte	144
rip1	8L-24+-	ribosome-inactivating protein1, electrophoretic mobility, encodes abundant 32kD endosperm protein, cytosolic	147, 270, 467
rld1	9-137+-	rolled leaf1, dominant *Rld1* plants have leaves tightly rolled and tend to be entangled, ligular flaps on abaxial surface of leaf, resembles *Ce1*	33
rMrh		receptor of *Mrh*	369

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
rMut		receptor of *Mut*	369
rp1	10S-3	resistance to *Puccinia sorghi*1, dominant *Rp1* resistant	265, 266
rp3	3-67+-	resistance to *Puccinia sorghi*3, dominant *Rp3* resistant	478
rp4	4S-24	resistance to *Puccinia sorghi*4, dominant *Rp4* resistant	478
rp5	10S-0	resistance to *Puccinia sorghi*5, dominant *Rp5* resistant	388
rp6	10S-3+-	resistance to *Puccinia sorghi*6, dominant *Rp6* resistant	478
rpp9	10S-3+-	resistance to *Puccinia polysora* and *Puccinia sorghi*9, dominant *Rpp9*	460
rs1	7S-0+-	rough sheath1, dominant *Rs1* has extreme ligule disorganization	232
rs2	1-56+-	rough sheath2	232
rs4	7-30+-	rough sheath4, dominant *Rs4* has rough leaf sheaths, vascular bundles enlarged	314
rt1	3S-60+-	rootless1, secondary roots few or absent	203
ruq		receptor of *Uq*	139
S		seed color component at *R1*, anthocyanin pigmentation in aleurone, (see also cms-S)	103, 216, 436
sad1	10L-33+-	shikimate dehydrogenase1, electrophoretic mobility, plastidial, monomeric, encodes shikimate dehydrogenase	474
sbd1	6L	sunburned1, sun-exposed leaves greyish-waxy	314
sdw1	8L-22+-	semi-dwarf1, dominant *Sdw1* plants have shortened internodes, erect leaves	32
sdw2	3	semi-dwarf2, short plant, 1/3-1/2 normal height, with normal green erect leaves, does not respond to gibberellins, no anthers in ear	316
se1	4L-118+-	sugary-enhancer1, high sugar content with *su1*, light yellow endosperm, freely wrinkled in III677a	132
sen1	3	soft endosperm1, duplicate factor with *sen2*, endosperm soft, opaque	440
sen2	7	soft endosperm2, duplicate factor with *sen1*	440
sen3	1	soft endosperm3, duplicate factor with *sen4*, like *sen1*	440
sen4		soft endosperm4, duplicate factor with *sen3*	440
sen5	2	soft endosperm5, duplicate factor with *sen6*, like *sen1*	440
sen6	5	soft endosperm6, duplicate factor with *sen5*	440
sft1		small flint type1, ears on *sft1* plants produce only small flint endosperms, +/*sft1* ears are normal	102
sg1		string cob1, dominant *Sg1* reduced pedicels	142
sh1	9S-29	shrunken1, inflated endosperm collapses on drying, forming smoothly indented kernels, sucrose synthase-1 of endosperm (compare *css1*), homotetramer, encodes sucrose synthase	196
sh2	3L-149.2	shrunken2, inflated, transparent, sweet kernels collapse on drying, becoming angular and brittle, endosperm ADPG pyrophosphorylase subunit (compare *bt2*), encodes ADP glucose pyrophosphorylase	267
sh4	5L-67+-	shrunken4, collapsed, chalky endosperm	458
sh5	5-29+-	shrunken5, sides of kernel collapsed	430
sk1	2S-56	silkless ears1, pistils abort, no silks	211
sks1	2L-83+-	suppressor of sterility1, pollen grains developing in presence of *Ms21* are defective and nonfunctional if *sks1*, normal if *Sks1*	397
sl1	7L-50	slashed leaves1, leaves slit longitudinally by necrotic streaks	182
sm1	6L-59	salmon silks1, silks salmon color with *P1-RR*, brown in *P1-WW*	7
sn1	10L-64+-	scutellar node color1, anthocyanin in coleoptile, nodes, auricle, leaf blade, etc. (compare *Lc1*)	148
sod1		superoxide dismutase1, electrophoretic mobility, plastidial, Cu-Zn dimeric, intralocus hybrid bands occur, encodes superoxide dismutase	18
sod2	7L-71+-	superoxide dismutase2, electrophoretic mobility, cytolosic, Cu-Zn dimeric, encodes superoxide dismutase	57, 58
sod3		superoxide dismutase3, electrophoretic mobility, mitochondrial, Mn tetrameric, intralocus hybrid bands occur, cDNA complements yeast mutant, encodes superoxide dismutase	18
sod4	1S-26+-	superoxide dismutase4, electrophoretic mobility, cytosolic, Cu-Zn dimeric, intralocus hybrid bands occur, encodes superoxide dismutase	18
spc1	3L-90+-	speckled1, dominant *Spc1*, brown speckling on leaves and sheath at flowering, supporting tissues weak	321
spc2	1L	speckled2, green seedling with light green speckles	320
spc3	3L	speckled3, green seedling with dark and light green speckles	320
Spm		suppressor-mutator: autonomous transposable element (equivalent to *En*), regulates *dSpm* (=*I*) transposition and function at *a1-m1*, *a1-m2*, *bz1-m13*, etc.	282
sps1		sucrose phosphate synthase1, cDNA encodes a 1068 amino acid leaf protein, transgenic (*E. coli*) directs sucrose phosphate synthesis, encodes sucrose-phosphate synthase	480
spt1	2L	spotted1, pale green, weak seedlings with dark green spots	320
spt2	4S	spotted2, like *spt1*	320
sr1	1S-0	striate leaves1, many white striations or stripes on leaves (A.M. Brunson, 1935, unpublished data)	124
sr2	10L-98	striate leaves2, white stripes on blade and sheath of upper leaves	208
sr3	10S	striate leaves3, virescent and striate to striped	153
sr4	6L	striate leaves4, seedlings pale luteus, later leaves white-striped	313

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
srp1		signal recognition particle RNA1, gene family, encodes 7SL RNA	56
ssu1	4L-98+-	ribulose bisphosphate carboxylase small subunit1, encodes ribulose bisphosphate carboxylase, small subunit	53
st1	4S-62	sticky chromosome1, small plant, striate leaves, pitted kernels resulting from sticky chromosomes, *st1-e* heightened by high temperature	23
stAc	10-33+-	stabilized Activator, RFLP locus (P. Chomet, unpublished)	51
su1	4S-66	sugary1, 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), encodes starch debranching enzyme I	86
su2	6L-58	sugary2, endosperm glassy, translucent, sometimes wrinkled, encodes starch branching enzyme?	131
su3		sugary3, endosperm glassy, smoother than *su1*	441
sup1		suppressor1, dominant *Sup1* modifies *o2* kernels to semi-transparent	275
sy1		yellow scutellum1	426
ta1		transaminase1, electrophoretic mobility, dimeric, intralocus hybrid bands occur,possibly = *Got1*	263
tb1	1L-130+-	teosinte branched1, many tillers, ear branches tassel-like	49
td1	5-42+-	thick tassel dwarf1 (E.G. Anderson, unpublished)	
te1	3L	terminal ear1, stalked ear appendages at tip, varying to infolded ears	276
tga1	4-47+-	teosinte glume architecture1, glumes indurated, erect, long, boat-shaped, factor transferred from teosinte	98
tha1		thylakoid assembly1, reduced polypeptides of photosystem II, photosystem I, cytochrome b6/f, normal coupling factor, normal RUBISCO, missing polypeptides appear to be synthesized normally	16
tha2		thylakoid assembly2, reduced polypeptides of cytochrome b6/f, photosystems I and II, coupling factor, missing polypeptides appear to be synthesized normally	16
thc1		thiocarbamate sensitive1, sensitive to Eradicane	346
tl		tasselless, trait was previously symbolized as a gene, *tl1*, but inheritance is complex and irregular, associated with aneuploidy	286
tlr1	1L	tillered1, dominant *Tlr1*, extreme tillering	320
tls1	1L-158+-	tasselless1, plants generally lack tassels, have ear shoots but no ear, variable, in some backgrounds, pubescent, leathery at 4-8 leaf stage, similar to *bs1* of Woodworth and Micu	457
Tourist		family of transposable elements, 1-50k copies in genome, average length 133 bp	45
tp1	7L-46	teopod1, dominant *Tp1*, many tillers, narrow leaves, many small partially podded ears, tassel simple	256
tp2	10L-48	teopod2, like *tp1*	344
tpe1		thin pericarp1, reduced cell number in pericarp (from *Coroica*)	141
tpi1	7L-59	triose phosphate isomerase1, electrophoretic mobility, plastidial, dimeric, intra/interlocus hybrids occur with *Tpi2*, encodes triose phosphate isomerase (plastidial)	473
tpi2	2L-100	triose phosphate isomerase2, electrophoretic mobility, plastidial, dimeric, intra/interlocus hybrids occur with *Tpi1*, encodes triose phosphate isomerase (plastidial)	473
tpi3	8-0+-	triose phosphate isomerase3, electrophoretic mobility, cytosolic, dimeric, intra/interlocus hybrids occur with *Tpi4* and *Tpi5*, encodes triose phosphate isomerase (cytosolic)	473
tpi4	3L-60+-	triose phosphate isomerase4, electrophoretic mobility, cytosolic, dimeric, intra/interlocus hybrids occur with *Tpi3* and *Tpi5*, encodes triose phosphate isomerase (cytosolic)	473
tpi5	8L-111+-	triose phosphate isomerase5, electrophoretic mobility, cytosolic, dimeric intra/interlocus hybrids occur with *Tpi3* and *Tpi4*, encodes triose phosphate isomerase (cytosolic)	473
tpm1		thylakoid peptide modifier1, dominant decrease in electrophoretic mobility	297
tr1	2S	two-ranked ear1, distichous vs. decussate phyllotaxy in ear axis, quantitative, one of a family of loci differentiating maize vs. teosinte	241
trAc	1S-1+-	transposed Activator sequence	54
trn1	9	torn1, dominant *Trn1* plants have chlorotic and adherent leaf tissues on later leaves, which become green and healthy after sunlight exposure but are torn	317
tru1		tassels replace upper ears1, upper ear branches tassel-like, tillers bear ears	408
ts1	2S-74	tassel seed1, tassel pistillate and pendant, if removed, small ear with irregular kernel placement develops	117
ts2	1S-24	tassel seed2, like *ts1*, but branches variably pistillate and staminate	117
ts4	3L-73	tassel seed4, tassel compact silky mass, upright, with pistillate and staminate florets, ear silky and proliferated	348
ts5	4S-53	tassel seed5, dominant *Ts5*, tassel upright with scattered, short silks, branches mostly pistillate toward the base	122
ts6	1L-158	tassel seed6, dominant *Ts6*, tassel pistillate to mixed, compact, ear with irregular kernel placement	330
tsc1		tar spot complex1, *Tsc1*, resistance to tar spot complex	59
tu1	4L-101	tunicate1, dominant *Tu1*, kernels enclosed in long glumes, tassel glumes large, coarse	79, 80

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
tua1	1L-130+-	alpha tubulin1, mRNA expressed primarily in roots, member of tandem repeat (see *tua2*), encodes alpha tubulin	53, 300
tua2	1L-130+-	alpha tubulin2, member of tandem repeat (see *tua1*), separated by 1.5 kbp, preferentially expressed in radicles, root tips and coleoptiles (Montoliu et al., 1989), 6 alpha tubulin genes identified (Villemur et al., 1992), encodes alpha tubulin	300, 465
tua3		alpha tubulin3, alpha tubulin family, mRNA expressed in all dividing cells examined, encodes alpha tubulin	301
tua4		alpha tubulin4, belongs to alpha tubulin subfamily I, with *tua1* and *tua2*, gene specific cDNA probe, encodes alpha tubulin	465
tua5		alpha tubulin5, alpha tublin subfamily II with *tua6*, gene specific cDNA probe, encodes alpha tubulin	465
tua6		alpha tubulin6, alpha tubulin subfamily II, gene specific cDNA probe, encodes alpha tubulin	465
tub1		beta tubulin1, genomic clones sequenced, gene-specific probe (by Southern blot) hybridizes to a single transcript size, encodes beta tubulin	195
tub2		beta tubulin2, cDNA sequenced, single copy (Southern blots), encodes beta tubulin	195
ub1		unbranched1, tassel with one spike	326
ubf9		ubiquitin fusion protein9, genomic sequence, hybridizing mRNA expressed during cell division and/or cell growth, multiple copies in genome, encodes ubiquitin fusion protein	
ubi1		ubiquitin1, genomic sequence, 7 contiguous, direct ubiquitin repeats, transcript specific probe, promoter active in monocots, not in tobacco, encodes polyubiquitin	69
ubi2		ubiquitin2, genomic sequence encodes 7 contiguous ubiquitin monomers, transcript specific probe, encodes polyubiquitin	69
ufo1	10S-3+-	unstable factor for orange1, dominant *Ufo1*, anthers, silks, and most other plant parts orange with *P1-WR* or *P1-RR*, growth retarded	449
Uq1	2	ubiquitous: controlling element mediating *a1-ruq*, *ruq-st* (receptor element), *ruq31*, *ruq66* (receptor elements)	139
v1	9L-63	virescent1, yellowish white seedling, greens rapidly, low temperature accentuates	91
v2	5L-107	virescent2, like *v1*, but greens slowly, low temperature accentuates	115
v3	5L-45	virescent3, light yellow seedling, greens rapidly, low temperature accentuates	91
v4	2L-83	virescent4, like *v2*	91
v5	7S-24	virescent5, like *v1*, but older leaves have white stripes	91
v8	4L-82+-	virescent8, like *v2*, lethal	92
v12	5L-75+-	virescent12, like *v3*	349
v13		virescent13, first leaf with green tip, greens slowly	349
v16	8L-52	virescent16, like *v2*	349
v17	4	virescent17, like *v1*, but greening from base to tip	349
v18	10	virescent18, like *v1*	349
v21	8L-66+-	virescent21, grainy virescent, greening from tips and margins inward	26
v22	1L-104+-	virescent22, like *v1* (E.G. Anderson, unpublished)	
v23	4-43+-	virescent23, like *v1* (E.G. Anderson, unpublished)	
v24	2L	virescent24, like *v1*, except not cold-sensitive and developmentally conditional high chlorophyll fluorescence attibutable to premature assembly of the light harvesting complexes	320
v25	1S	virescent25, greenish white seedling, greens from base upward	320
v26	2S	virescent26, yellowish white seedling with green leaf tip and midrib	320
v27	7L	virescent27, like *v1*	320
v28	9S-7+-	virescent28, like *v1*	320
v29	10L	virescent29, grainy virescent	320
v30	9L-87	virescent30, like *v1*	74
v31	9S-7+-	virescent31, grainy, light green seedling, small green plant with longitudinal white stripes	178
va1	7L-52+-	variable sterile1, variable male and female fertility, cytokinesis fails in anaphase I	21
vg1	1L-85	vestigial glume1, dominant *Vg1* glumes very small, cob and anthers exposed, upper leaves have scant ligules	427
vp1	3L-73+-	viviparous1, embryo fails to become dormant, viable if transplanted, some alleles dormant, chlorophyll and carotenoids unaffected, anthocyanins in aleurone suppressed	128
vp2	5S-38	viviparous2, embryo fails to become dormant, white endosperm, white seedling, anthocyanins unaffected	128
vp5	1S-1	viviparous5, like *vp2*	370
vp8	1L-154	viviparous8, embryo fails to become dormant, chlorophyll and carotenoids unaffected, small, pointed-leaf seedlings	371
vp9	7S-25	viviparous9, like *vp2*, *vp9-4889* dormant, pale aleurone, pale green seedling	371
vp10		viviparous10, yellow endosperm, colored aleurone, green seedlings, adherent	421
vsi1		variable short internodes1, clusters of 2-4 short internodes, predominantly at base of plant but varies in location, temperature sensitive	105
vsr1	10L	virescent striped1, dominant *Vsr1* virescent seedling, greens to white and yellow striped plant	320
w1	6L-78+-	white1, white seedling (yellow with *l1*), plastid transcripts variously aberrant	113, 114, 253

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
w2	10L-80	white2, white seedling (yellow with *l1*), endosperm pitted and spotted (allele *dek21*), plastid DNA content decreased	255
w3	2L-111	white3, like *vp2*, *w3-8686* pale endosperm, pale green seedling in dim light	255
w11	9S-54	white11, like *w1*	92
w14	6L-78	white14, like *w1*	88
w15	6L-13	white15, like *w1*, fails to convert protochlorophyllide to chlorophyllide	88
w16	7S-25+-	white16, like *w1*	302
w17	7S-0+-	white17, like *w1*	302
w18	1L	white18, like *w1*	313
w19	3L-149+-	white19, white plant tissue, identified in plants carrying the a1-x1 deficiency, forming albino chimeras on loss of ring3 carrying *A1-b Sh2*	318, 437
wc1	9L-107	white cap1, dominant *Wc1* kernel with pale yellow endosperm (pearly white with *y1*), emphasized in soft-starch crowns	236
wd1	9S	white seedling1, deficiency for distal half of first chromomere of short arm (for alleles, see Coe et al., 1988)	280
wgs1	5L	white green sectors1, white seedling with green sectors	320
whp1	2L-155+-	white pollen1, duplicate factor with *c2* for pollen color and for anthocyanins, chalcone synthase, encodes naringenin-chalcone synthase	76
wi1	6L-17+-	wilted1, chronic wilting, leaves not as cool as normal, delayed differentiation of metaxylem vessels	354
wi2	3	wilted2, dominant *Wi2*, top leaves wilt under moisture/temperature stress	313
wi3		wilted3, like *wi2*	314
wlu1	3L	white luteus1, pale yellow seedling, lethal	320
wlu2	7L	white luteus2, like *wlu1*	320
wlu3	8L-66+-	white luteus3, like *wlu1*	320
wlu4	9L	white luteus4, like *wlu1*	320
wlu5	1L-81+-	white luteus5, like *wlu1*	313
wrk1	3S-62	wrinkled kernel1, dominant *Wrk1* kernels small and wrinkled	320
wrp1	2	wrinkled plant1, dominant *Wrp1* dwarf, leaves and culm longitudinally corrugated, dosage effect	34, 313
ws1		white sheath1, light yellow leaf sheaths, duplicate factor with *ws2*	227
ws2		white sheath2, see *ws1*	227
ws3	2S-0	white sheath, white leaf sheath, culm, husks	363
ws4	1S	white sheath, dominant *Ws4* seedlings and plants lighter green in sheaths	313
wsm1	6S-4+-	wheat streak mosaic virus resistance1	
wsp		weak streaked plant1, maternally inherited reduced plants	42
wt1	2S-60	white tip1, tip of first leaf white and blunt	431
wt2	4S	white tip2, seedling with white leaf tip and crossbands on first 2 leaves	320
wx1	9S-56	waxy1, amylopectin (stained red by iodine) replaces amylose (blue staining) in endosperm and pollen, (for alleles, see Coe et al., 1988), encodes NDP-glucose-starch glucosyltransferase, starch granule-bound	78
wyg1	7L-32+-	white yellow green seedling1	302
y1	6L-17	pale yellow1, reduced carotenoid pigments in endosperm, some alleles affect chlorophyll in seedlings (e.g. *y1-8549*, see Coe et al., 1988)	86
y3	2S-4+-	pale yellow3, compare *al1*	342
y8	7S-18	pale yellow8, pale endosperm	205
y9	10S-27	pale yellow9, pale endosperm, slightly viviparous, green to pale green seedlings and plants	376
y10	3L-115+-	pale yellow10, pale endosperm, white seedling, lethal	372
y11		pale yellow11, pale endosperm, green seedling	429
y12		pale yellow12, like *y11*	429
yd2	3L-101+-	yellow dwarf2	375
yg1	5L-107+-	yellow-green1, yellow-green seedling and plant	126
yg2	9S-7	yellow-green2, like *yg1* (for alleles, see Coe et al., 1988)	202
ys1	5L-75	yellow stripe1, yellow tissue between leaf veins, reflects iron deficiency symptoms	19
ys2	1S	yellow stripe2, yellow tissue between leaf veins	353
ys3	3L-69+-	yellow stripe3, like *ys1*	481
ysk1	4-43+-	yellow streaked1, dominant *Ysk1*, longitudinal yellow streaks in top 3rd of mature leaves	321
zb1		zebra crossbands1, yellowish crossbands on older leaves	90
zb2		zebra crossbands2, crossbands on seedling leaves	447
zb3	5L-107+-	zebra crossbands3, yellowish crossbands on older leaves (M. Demerec, 1935, unpublished data)	124
zb4	1S-19	zebra crossbands4, regularly spaced crossbands on earlier leaves, enhanced by cool temperatures	180
zb6	4-79	zebra crossbands6, regularly spaced crossbands on earlier leaves, enhanced by cool temperatures	183

SYMBOL	LOCATION	NAME, PHENOTYPE	REF
zb7	1L-130+-	zebra crossbands7, lighter green crossbands on seedlings, glossy	320
zb8	9-42+-	zebra crossbands8, yellow-green crossbands on older leaves, strong anthocyanin expression in leaf tip and blade	320, 321
Zeon1		zein retrotransposon1, 1k copies of LTR-related sequences, 3-400 copies of internal sequence	192
zn1	10L-29	zebra necrotic1, necrotic tissue appears between veins in transverse leaf bands on half-grown or older plants	191
zn2		zebra necrotic2, like *zn1*	151
zp		zein polypeptide1, designator for loci determining zein polypeptides, encodes zein	423, 424
zpb36	7-32+-	zein familyB36, encodes zein	52
zpg1		zebra-stripe pale green1	101
zpl1	4S-13+-	zein polypeptidesL1, *Zp1La - Zp1Lf* complex, encodes zein	479
zpl2a	4S-39+-	zein polypeptidesL2a, encodes zein	479
zpl2b	7S-16+-	zein polypeptidesL2b, encodes zein	479
zpl3a	4L-47+-	zein polypeptidesL3a, encodes zein	479
zpr10/(22)	4-39	zein-protein regulator, elevation of 10kD zein	29
zps10/(22)	9-56+-	zein-protein structural gene10/(22), 10kD zein, RFLP (probe 10kZ-1), encodes zein	29

1. Abe, M., et al. 1992. Eur. J. Biochem. 209:933-937. (*psei1*)
2. Albertsen, M.C. and M.G. Neuffer. 1990. MNL 64:52. (*ms44*)
3. Albertsen, M.C., et al. 1993. MNL 67:64. (*ms42*)
4. Allen, J.O., et al. 1989. Maydica 34:277-290. (*rcm1, rcm2, rcm3*)
5. Anderson, E.G. 1953. MNL 27:5. (*bf2*)
6. Anderson, E.G. 1955. MNL 29:5-6. (*gl14, gl15, gl18*)
7. Anderson, E.G. 1921. Cornell Univ. Agric. Exp. Stn. Memoir 48:533-554. (*sm1*)
8. Anderson, E.G. and R.A. Emerson. 1931. Am. Nat. 65:253-257. (*ch1*)
9. Anderson, P.C. and M. Georgeson. 1989. Genome 31:994-999. (*als2, als1*)
10. Ashman, R.B. 1960. Genetics 45:19-34. (*mst1*)
11. Ashman, R.B. and A.J. Ullstrup. 1976. J. Hered. 67:220-222. (*les3*)
12. Baba et al. 1991. Biochem. Biophys. Res. Commun. 181:87-94. (*bre1*)
13. Bachmann, M.D., et al. 1973. J. Ultrastruct. Res. 45:384-406. (*l11*)
14. Badu-Apraku, B., et al. 1987. Plant Breed. 98:194-199. (*cgl1*)
15. Baker, A. and C.J. Leaver. 1985. Nucl. Ac. Res. 13:5357-5867. (*ant1*)
16. Barkan, A., et al. 1993. MNL 67:41-42. (*pet1, cps2, psb1, psa1, psb2, psa2, psa3, psa4, pet2, pet3, pet4, pet5, crp1, crp2, cps1, cgx1, cgx2, tha1, tha2, hcf7*)
17. Bathgate, B., et al. 1989. Eur. J. Biochem. 183:303-310. (*ant2*)
18. Baum, J.A. and J.G. Scandalios. 1982. J. Hered. 73:95-100. (*sod1, sod3, sod4*)
19. Beadle, G.W. 1929. Am. Nat. 63:189-192. (*ys1*)
20. Beadle, G.W. 1931. Cornell Univ. Agric. Exp. Stn. Memoir 135:1-12. (*po1*)
21. Beadle, G.W. 1932. Cytologia 3:142-155. (*va1*)
22. Beadle, G.W. 1932. Genetics 17:413-431. (*ms10, ms11, ms12, ms13, ms14, ms5, ms7, ms8, ms9, ra1*)
23. Beadle, G.W. 1932. Ztschr. ind. Abst. Vererbungsl. 63:195-217. (*st1*)
24. Beadle, G.W. and B. McClintock. 1928. Science 68:433. (*as1*)
25. Beckett, J.B. 1971. Crop Sci. 11:724-727. (*cms-C*)
26. Beckett, J.B. and M.G. Neuffer. 1973. MNL 47:147. (*v21*)
27. Beckman, L., et al. 1964. Science 146:1174-1175. (*cat1*)
28. Bellmann, R. and W. Werr. 1992. EMBO J. 11:3367-3374. (*hox1, hox2*)
29. Benner, M.S., et al. 1989. Theor. Appl. Genet. 78:761-767. (*zpr10(22), zps10(22)*)
30. Berlani, R.E., et al. 1988. Plant Molec. Biol. 11:173-182. (*ars1, ars2, ars3*)
31. Bianchi, G. 1987. Gazz. Chim. Ital. 117:707-716. (*gl11, gl2*)
32. Bird, R.M. and M.G. Neuffer. 1985. MNL 59:42. (*sdw1*)
33. Bird, R.M., and M.G. Neuffer. 1985. p. 818-822 in M. Freeling (ed.) Plant Genetics, Alan R. Liss, New York. (*hsf1, rld1*)
34. Bockholt, A.J. and J.D. Smith. 1989. MNL 63:56. (*wrp1*)
35. Brewbaker, J.L. 1974. Proc. Annu. Corn Sorghum Res. Conf. 29:118-133. (*mv1*)
36. Brewbaker, J.L. and Y. Hasegawa. 1974. MNL 48:35-37. (*px8, px9*)
37. Brink, R.A. 1933. J. Hered. 24:325-326. (*lg2*)
38. Brink, R.A. 1935. J. Hered. 26:249-251. (*pm1*)
39. Brink, R.A. and P.H. Senn. 1931. J. Hered. 22:155-161. (*rg1*)
40. Brink, R.A. and R.A. Nilan. 1952. Genetics 37:519-544. (*Mp*)
41. Brinkmann, H., et al. 1987. J. Mol. Evol. 26:320-328. (*gpa1, gpc1*)
42. Brown, W.L. and D.N. Duvick. 1958. MNL 32:120-121. (*wsp*)
43. Bryan, A.A. and J.E. Sass. 1941. J. Hered. 32:342-346. (*kn1*)
44. Buchert, J.G. 1961. Proc. Natl. Acad. Sci. 47:1436-1440. (*rf3*)
45. Bureau, T.E. and S.R. Wessler. 1992. Plant Cell 4:1283-1294. (*Tourist*)
46. Burnham, C.R. 1936. J. Am. Soc. Agron. 28:968-975. (*ga2*)
47. Burnham, C.R. 1947. MNL 21:36. (*bm4*)
48. Burnham, C.R. 1958. MNL 32:93. (*eg1*)
49. Burnham, C.R. 1961. MNL 35:87. (*tb1*)
50. Burnham, C.R. and R.A. Brink. 1932. J. Am. Soc. Agron. 24:960-963. (*bm2*)
51. Burr, B. and F.A. Burr. 1989. Plant Phys. 94:11-17. (*aco1, dia1*)
52. Burr, B., et al. 1982. J. Mol. Biol. 154:33-49. (*zpb36*)
53. Burr, B., et al. 1988. Genetics 118:519-526. (*ssu1, tua1*)
54. Burr, B., et al. 1991. MNL 65:105-110. (*als1, als2, mgs2, nabp1, php1, rab30, trAc*)
55. Campbell, W.H. 1992. Plant Phys. 99:693-699. (*nnr1, nnr3*)
56. Campos, N., et al. 1989. Nucl. Ac. Res. 17:1573-1588. (*srp1*)
57. Cannon, R.E. and J. G. Scandalios. 1987. Isozymes Curr. Top. Biol. Med. Res. 14:73-81. (*sod2*)
58. Cannon, R.E., et al. 1987. Proc. Natl. Acad. Sci. USA 84:179-183. (*sod2*)
59. Ceballos, H. and J.A. Deutsch. 1992. Phytopathology 82:505-512. (*tsc1*)
60. Chang, M.T. and M.G. Neuffer. 1987. J. Hered. 78:163-170. (*Mr*)
61. Chang, S-H. and J.L. Brewbaker. 1976. MNL 50:31-32. (*aph1*)
62. Chao, S.E. 1993. Personal Communication. (*atp1, me3, mdh4*)
63. Chao, S.E. and J.G. Scandalios. 1969. Biochem. Genet. 3:537-547. (*amy2*)
64. Chao, S.E. and J.G. Scandalios. 1971. Genetics 69:47-61. (*amy1*)
65. Chaubet, N., et al. 1986. Plant Molec. Biol. 6:253-263. (*his4*)
66. Chaubet, N., et al. 1989. Molec. Gen. Genet. 219:404-412.

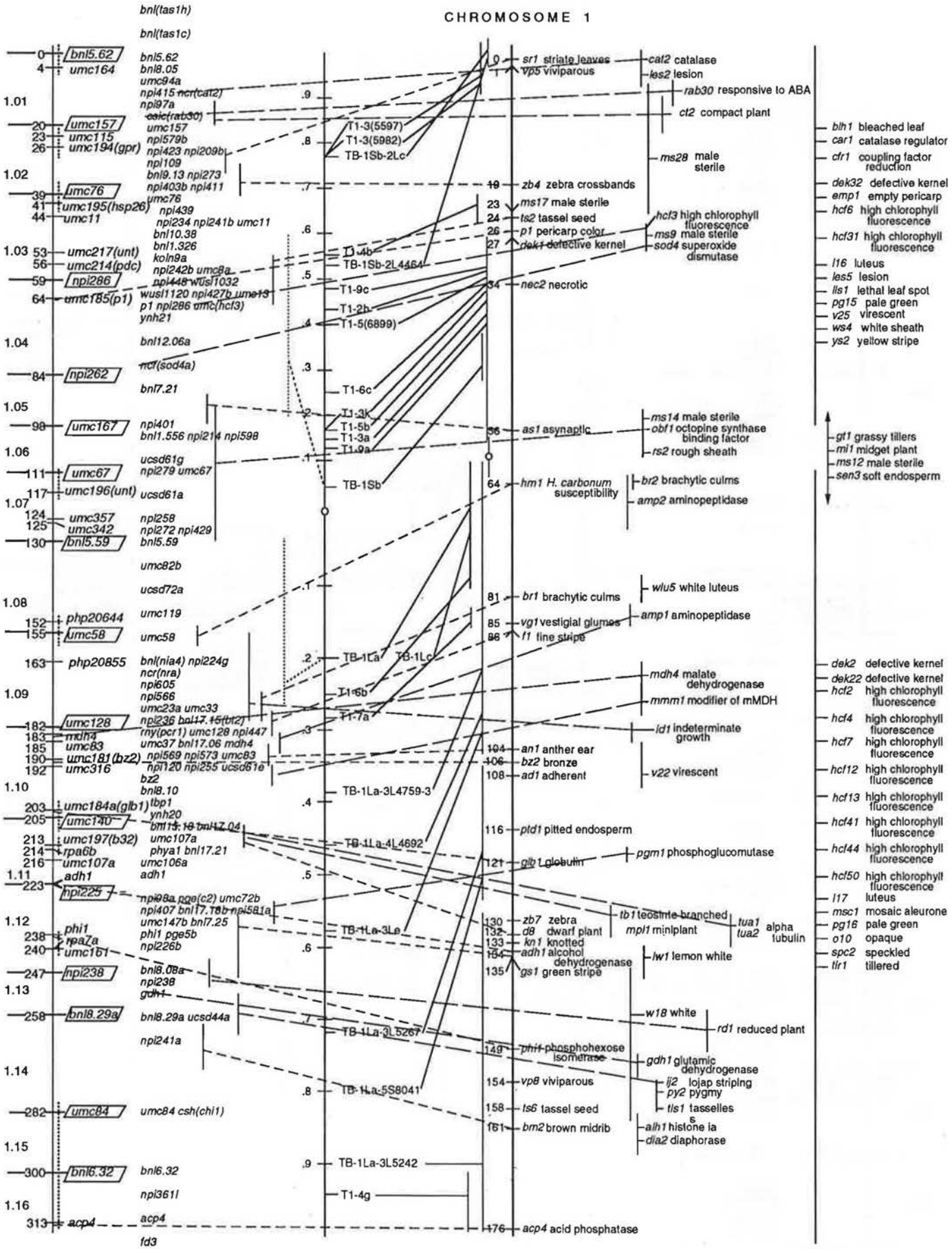
(*his3, his4*)
67. Chen, K. and I. Rubenstein. 1991. Gene 107:205-212. (*ubf9*)
68. Chourey, P.S. and C. Mouli. 1975. Genetics 77:s11. (*ce1*)
69. Christensen, A.H., et al. 1992. Plant Molec. Biol. 18:675-689. (*ubi1, ubi2*)
70. Clark, F.J. 1940. Am. J. Bot. 27:547-559. (*dv1*)
71. Clark, J.K. and W.F. Sheridan. 1986. J. Hered. 77:83-92. (*dek22, dek23*)
72. Close, T.J., et al. 1989. Plant Molec. Biol. 13:95-108. (*rab17*)
73. Coe, E.H. 1983. Maydica 28:151-168. (*NCS2, NCS3*)
74. Coe, E.H. 1987. MNL 61:47. (*anl1, v30*)
75. Coe, E.H. and J.B. Beckett. 1987. MNL 61:46-47. (*baf1*)
76. Coe, E.H., et al. 1981. J. Hered. 72:318-320. (*whp1*)
77. Collazo, P., et al. 1992. Plant Molec. Biol. 20:857-867. (*omt1*)
78. Collins, G.N. 1909. USDA, Plant Indus. Bur. Bull. 161:1-30. (*wx1*)
79. Collins, G.N. 1917. J. Agric. Res. 9:383-395. (*tu1*)
80. Collins, G.N. 1917. Proc. Natl. Acad. Sci. 3:345-349. (*tu1*)
81. Collins, G.N. and J.H. Kempton. 1920. J. Hered. 11:3-6. (*li1*)
82. Cook, W.B. and C.D. Miles. 1988. MNL 62:50. (*hcf113*)
83. Cook, W.B. and C.D. Miles. 1989. MNL63:65-66. (*hcf103, hcf104, hcf108, hcf111*)
84. Cook, W.B. 1992. Nucl. Ac. Res. 20:359-364. (*nbp1*)
85. Cooper, P., et al. 1990. Genetics 126:461-467. (*mct1*)
86. Correns, C. 1901. Bibliotheca Bot. 53:1-161. (*su1, y1*)
87. Couture, R.M., et al. 1971. Phys. Plant Pathol. 1:515-521. (*bx1*)
88. Cox, E.L. and D.B. Dickinson. 1971. Biochem. Genet. 5:15-25. (*l12, w14, w15*)
89. de la Roche, I.A., et al. 1971. Crop Sci. 11:856-859. (*ln1*)
90. Demerec, M. 1921. J. Hered. 12:406-407. (*zb1*)
91. Demerec, M. 1924. Cornell Univ. Agric. Exp. Stn. Memoir 84. (*v1, v3, v4, v5*)
92. Demerec, M. 1926. Am. Nat. 60:172-176. (*d3, v8, w11*)
93. Demerec, M. 1926. J. Hered. 17:301-306. (*pb1, pb4*)
94. Dempsey, E. 1971. MNL 45:58. (*K3L*)
95. Dempsey, E. 1985. p. 311-316 in M. Freeling (eds.) Plant Genetics, Alan R. Liss, New York. (*Ac2*)
96. Didierjean, L. 1992. Plant Molec. Biol. 18:847-849. (*grp1*)
97. Diedrick, T.J., et al. 1990. Theor. Appl. Genet. 79:209-215. (*ask1, ask2*)
98. Doebley, J., et al. 1992. MNL 66:95. (*tga1*)
99. Doerschug, E.B. 1973. Theor. Appl. Genet. 43:182-189. (*Dt4, Dt5*)
100. Dollinger, E.J. 1984. MNL 58:209-210. (*ora2*)
101. Dollinger, E.J. 1985. Crop Sci. 25:819-821. (*lty1, lty2, ora3, zpg1*)
102. Dollinger, E.J. 1987. MNL 61:103. (*rDt, sft1*)
103. Dooner, H.K. and J.L. Kermicle. 1971. Genetics 67:427-436. (*P, S*)
104. Dooner, H.K. and J.L. Kermicle. 1976. Genetics 82:309-322. (*lc1*)
105. Doyle, G. G. 1992. MNL 66:38-39. (*vsi1*)
106. Doyle, G.G. 1978. MNL 52:77. (*agt1*)
107. Duvick, D.N. 1965. Adv. Genet. 13:1-56. (*rf2*)
108. East, E.M. and H.K. Hayes. 1911. Conn. Agric. Exp. Stn. Bull. 167. (*c1, pr1, r1*)
109. Echt, C., et al. 1987. Molec. Gen. Genet. 208:230-234. (*cfr1*)
111. El-Metainy, A.Y. and A.A. Omar. 1981. Biochem. Genet. 19:635-640. (*acp1, acp2*)
112. Emerson, R.A. 1911. Nebr. Agric. Exp. Stn. Ann. Rep. 24:59-90. (*p1*)
113. Emerson, R.A. 1912. Am. Breeders Assoc. Ann. Rep. 8:385-399. (*an1, d1, g1, lg1, w1*)
114. Emerson, R.A. 1912. Nebr. Agric. Exp. Stn. Ann. Rep. 25:81-88. (*lg1, w1*)
115. Emerson, R.A. 1912. Nebr. Agric. Exp. Stn. Ann. Rep. 25:89-105. (*g1, j1, v2*)
116. Emerson, R.A. 1918. Cornell Univ. Agric. Exp. Stn. Memoir 16. (*a1*)
117. Emerson, R.A. 1920. J. Hered. 11:65-76. (*ts1, ts2*)
118. Emerson, R.A. 1921. Am. J. Bot. 8:411-424. (*gs1*)
119. Emerson, R.A. 1921. Cornell Univ. Agric. Exp. Stn. Memoir 39. (*b1, pl1*)
120. Emerson, R.A. 1921. J. Hered. 12:267-270. (*cr1*)
121. Emerson, R.A. 1932. Science 75:566. (*ms17*)
122. Emerson, R.A. 1932. Sixth Int. Congress Genet. Proc. 1:141-152. (*ts5*)
123. Emerson, R.A. and S.H. Emerson. 1922. Genetics 7:203-236. (*an1*)
124. Emerson, R.A., et al. 1935. Cornell Univ. Agric. Exp. Stn. Memoir 180. (*bm3, bt2, du1, fl2, gl5, gl6, gl7, gl8, gl9, gs2, j2, l6, mi1, nl1, o1, o2, ra2, sr1, zb3*)
125. Everett, H.L. 1949. Proc. Natl. Acad. Sci. 35:628-634. (*cl1, dm1*)
126. Eyster, W.H. 1926. Science 64:22. (*bm1, yg1*)
127. Eyster, W.H. 1929. Ztschr. ind. Abst. Vererbungsl. 49:105-130. (*ar1*)
128. Eyster, W.H. 1931. Genetics 16:574-590. (*vp1, vp2*)
129. Eyster, W.H. 1931. J. Hered. 22:99-102. (*ms2, ms3*)
130. Eyster, W.H. 1933. Am. Nat. 67:75. (*oy1*)
131. Eyster, W.H. 1934. Bibliographia Genetica. 11:187-392. (*da1, l7, ms2, ms20, ms3, su2*)
132. Ferguson, J.E., et al. 1978. J. Hered. 69:377-380. (*se1*)
133. Fleenor, D.E., et al. 1990. Nucl. Ac. Res. 18:6725-6725. (*Mu8*)
134. Fowler, J. and M. Freeling. 1991. MNL 65:30-31. (*lg4*)
135. Fraser, A.C. 1924. J. Hered. 15:119-123. (*in1*)
136. Fraser, A.C. 1933. J. Hered. 24:41-46 . (
137. Freeling, M. and S. Hake. 1985. Genetics 111:617-634. (*kn2*)
138. Freeling, M. and J.C. Woodman. 1978. MNL 52:9-10. (*cdh1*)
139. Friedemann, P. and P.A. Peterson. 1982. Mol. Gen. Genet. 187:19-29. (*ruq, Uq1*)
140. Galbiati, M and G. Gavazzi. 1992. MNL 66:80. (*des17*)
141. Galinat, W. C. 1990. MNL 64:120-121. (*tpe1*)
142. Galinat, W.C. 1969. Mass. Agric. Exp. Stn. Bull. 577:1-19. (*sg1*)
143. Galinat, W.C. 1971. MNL 45:98-99. (*is1*)
144. Galinat, W.C. 1975. MNL 49:100-102. (*ph1, ri1*)
145. Galinat, W.C. and P.C. Mangelsdorf. 1957. MNL 31:67. (*pn1*)
146. Galinat, W.C., et al. 1978. MNL 52:58. (*bu1*)
147. Gavazzi, G., et al. 1975. Theor. Appl. Genet. 46:339-346. (*rip1, pro1*)
148. Gavazzi, G., et al. 1986. p. 91-103 in G.M. Reddy and E.H. Coe (eds.) Gene Structure and Function in Higher Plants, Oxford-IBH, New Delhi. (*sn1*)
149. Gelinas, D.A., et al. 1966. Am. J. Bot. 53:615. (*clt1*)
150. Gernert, W.B. 1912. Am. Nat. 46:616-622. (*ra1*)
151. Giesbrecht, J. 1965. J. Hered. 56:118, 130. (*zn2*)
152. Glackin, CA; Grula, JW. 1990. Proc. Natl. Acad. Sci. USA 87:3004-3008. (*pdk1*)
153. Glover, D.V. 1968. MNL 42:151. (*ct2, sr3*)
154. Glover, D.V. 1970. Crop Sci. 10:611-612. (*rd2*)
155. Golubovskaya, I.N. 1989. Advan. Genet. 26:149-192. (*dsy3, dsy4*)
156. Golubovskaya, I.N. 1979. Int. Rev. Cytol. 58:247-290. (*mei1, ms43*)
157. Golubovskaya, I.N. and A.S. Mashnenkov. 1976. Genetika 12:7-14. (*dsy1*)
158. Golubovskaya, I.N. and A.S. Mashnenkov. 1977. Genetika 13:1910-1921. (*pam1*)
159. Golubovskaya, I.N. and D.V. Sitnikova. 1980. Genetika 16:656-666. (*mei1, ms28, ms43*)
160. Golubovskaya, I.N. and N.B. Khristolyubova. 1985. p. 723-738 in M. Freeling (ed.) Plant Genetics, Alan R. Liss, New York. (*dsy2, pam2*)

161. Golubovskaya, I.N., et al. 1975. Genetika. 11:11-17. (*afd1*)
162. Gonella, J.A. and P.A. Peterson. 1975. MNL 49:71-73. (*ga10*)
163. Gonella, J.A. and P.A. Peterson. 1977. Genetics 85:629-645. (*Fcu, rcu*)
164. Goodman, M.M. and C.W. Stuber. 1982. MNL 56:125. (*gdh2*)
165. Goodman, M.M. and C.W. Stuber. 1983. Maydica 28:169-188. (*acp1, acp2*)
166. Goodman, M.M., et al. 1980. Genetics 96:697-710. (*got2, got3, idh1, idh2, me1, pgd1, pgd2, pgm1, pgm2, phi1*)
167. Gowri, G. and W.H. Campbell. 1988. Plant Phys. 90:792-798. (*nnr1*)
168. Gracen, V.E., et al. 1979. Proc. Annu. Corn Sorghum Res. Conf. 34:76-91. (*rf4*)
169. Graham, G.I., et al. 1993. MNL 67:102. (*o8*)
170. Grana, X. 1992. J. Biol. Chem. 267:12797-12803. (*pmg1*)
171. Greenblatt, I.M. and R. A. Brink. 1954. J. Hered. 45:47-50. (*c2*)
172. Grogan, C.O., et al. 1963. Crop Sci. 3:451. (*ats1*)
173. Gupta, M., et al. 1982. Molec. Gen. Genet. 188:266-271. (*Cin*)
174. Hake, S., et al. 1985. J. Biol. Chem. 260:5050-5054. (*ald1*)
175. Hamill, D.E., and J.L. Brewbaker. 1969. Physiol. Plant. 22:945-958. (*px1*)
176. Hamilton, D.A., et al. 1989. Sex. Plant Reprod. 2:208-212. (*mgs1*)
177. Harberd, N., and M. Freeling. 1989. Genetics 121:827-838. (*mpl1*)
178. Harpster, M.H., et al. 1984. Plant Molec. Biol. 3:59-71. (*v31*)
179. Harris, J.W. 1968. Genetics 60:186-187. (*e4*)
180. Hayes, H.K. 1932. J. Hered. 23:415-419. (*zb4*)
181. Hayes, H.K. and E.M. East. 1915. Conn. Agric. Exp. Stn. Bull. 188:1-31. (*fl1*)
182. Hayes, H.K. and H.E. Brewbaker. 1928. Am. Nat. 62:228-235. (*gl2, gl3, sl1*)
183. Hayes, H.K. and M.S. Chang. 1938. MNL 12. (*gl11, zb6*)
184. Hofmeyr, J.D.J. 1930. Unpub. thesis, Cornell Univ. (*ba1, ba2*)
185. Hoisington, D.A. 1985. MNL 60:50-51. (*les4, les5, les6, les7, les8, les9*)
186. Hoisington, D.A. 1987. MNL 61:48-49. (*les10*)
187. Hoisington, D.A. and M. G. Neuffer. 1983. MNL 57:159-160. (*nec4*)
188. Hooker, A.L. 1963. Crop Sci. 3:381-383. (*ht1*)
189. Hooker, A.L. 1977. Crop Sci. 17:132-135. (*ht2*)
190. Hooker, A.L. 1981. MNL 55:87-88. (*ht3*)
191. Horovitz, S. 1948. MNL 22:42. (*zn1*)
192. Hu, W., et al. 1993. MNL 67:94. (*Zeon1*)
193. Huang, J and F. Struck. 1991. Proc. Natl. Acad. Sci. USA 88:10716-10720. (*isp1*)
194. Huelsen, W.A. and M.C. Gillis. 1929. Ill. Agric. Exp. Stn. Bull. 320. (*pi1, pi2*)
195. Hussey, P.J., et al. 1990. Plant Molec. Biol. 15:957-972. (*tub1, tub2*)
196. Hutchison, C.B. 1921. J. Hered. 12:76-83. (*sh1*)
197. Hutchison, C.B. 1922. Cornell Univ. Agric. Exp. Stn. Memoir 60:1419-1473. (*na1*)
198. Huynh, Q.K. 1992. J. Biol. Chem. 267:6635-6640. (*cta1, ctb1*)
199. James, M.G., et al. 1992. MNL 66:6. (*dsc1, ren2*)
200. Jenkins, M.T. 1924. J. Hered. 15:467-472. (*ij1*)
201. Jenkins, M.T. 1926. Am. Nat. 60:484-488. (*g2*)
202. Jenkins, M.T. 1927. Genetics 12:492-518. (*yg2*)
203. Jenkins, M.T. 1930. J. Hered. 21:79-80. (*rt1*)
204. Jenkins, M.T. 1932. J. Agric. Res. 44:495-502. (*a2*)
205. Jenkins, M.T. 1947. MNL 21:33. (*y8*)
206. Jenkins, M.T. and F. Gerhardt. 1931. Iowa Agric. Exp. Stn. Res. Bull. 138:121-151. (*la1*)
207. Jenkins, M.T. and M.A. Bell. 1930. Genetics 15:253-282. (*l3, l4*)
208. Joachim, G. and C.R. Burnham. 1953. MNL 27:66. (*sr2*)
209. Johal, G., et al. 1993. MNL 67:53. (*Hbr*)
210. Johns, M.A., et al. 1985. EMBO J. 4:1093-1102. (*Bs-1*)
211. Jones, D.F. 1925. J. Hered. 16:339-341. (*sk1*)
212. Jones, D.F. 1951. Proc. Natl. Acad. Sci. 37:408-410. (*rf1*)
213. Jones, D.F. 1954. Proc. IX Int. Genet. Cong. 1225-1237. (*cms-S, cms-T*)
214. Jones, D.F. and P.C. Mangelsdorf. 1925. Anat. Rec. 31:351. (*ga1*)
215. Jose-Estanyol, M. 1992. Plant Cell 4:413-423. (*hyp1*)
216. Josephson, L.M. 1955. Empire J. Exp. Agric. 23(89):1-10. (*cms-S, cms-T, S*)
217. Josephson, L.M., et al. 1978. Proc. Annu. Corn Sorghum Res. Conf. 33:48-59. (*rf5, rf6*)
218. Jupe, E.R. and E. Zimmer. 1990. Plant Molec. Biol. 14:333-347. (*rDNA5.8S, rDNA18S, rDNA25S*)
219. Kahler, A.L. 1983. J. Hered. 74:239-246. (*acp2, acp4*)
220. Kang, M.S. 1981. MNL 55:26. (*btn1*)
221. Kawamura, T., et al. 1992. J Biochem 112:147-154. (*pep4*)
223. Kelley, P.M. 1989. Plant Molec. Biol. 13:213-222. (*pdc1*)
224. Kelley, P.M. and M. Freeling. 1984. J. Biol. Chem. 259:14180-14183. (*ald1*)
225. Kempton, J.H. 1920. J. Hered. 11:111-115. (*br1*)
226. Kempton, J.H. 1920. J. Hered. 11:317-322. (*ad1*)
227. Kempton, J.H. 1921. Am. Nat. 56:461-464. (*ws1, ws2*)
228. Kempton, J.H. 1921. USDA Bull. 925:1-28. (*br1*)
229. Kempton, J.H. 1934. J. Hered. 25:29-32. (*bd1*)
230. Kermicle, J.L. 1969. Science 166:1422-1424. (*ig1*)
231. Kermicle, J.L. and J.D. Axtell. 1981. Maydica 26:185-197. (*isr1*)
232. Khadzhinov, M.I. 1937. Bull. Appl. Bot. Gen. Plant Breed. Ser. II. 7:247-258. (*rs1, rs2*)
233. Kramer, H.H. 1957. MNL 31:120. (*rgd1*)
234. Kriz, A.L. 1989. Biochem. Genet. 27:239-251. (*glb1, glb2*)
235. Kuc, J., et al. 1968. Phytochemistry 7:1435-1436. (*bm3*)
236. Kulkarni, C.G. 1927. Mich. Acad. Sci. Arts and Letters Papers. 6:253-273. (*wc1*)
237. Kvakan, P. 1924. Cornell Univ. Agric. Exp. Stn. Memoir 83:1-22. (*bn1, gl1*)
238. Lai, Y-K. and J.G. Scandalios. 1980. Devel. Genet. 1:311-324. (*adr1*)
239. Lal, S.K., et al. 1991. Plant Molec. Biol. 16:787-795. (*eno1*)
240. Lambert, R.J. and G.F. Sprague. 1987. MNL 61:96. (*mg1*)
241. Langham, D.G. 1940. Genetics 25:88-107. (*pd1, tr1*)
242. Langham, D.G. 1940. MNL 14:21. (*bk2*)
243. Larkin, J.C., et al. 1989. Genes Devel. 3:500-509. (*mch1, mch2*)
244. Lauer, M., et al. 1990. New Biologist 2:179-186. (*NCS6*)
245. Laughnan, J. R. 1949. Proc. Natl. Acad. Sci. 35:167-178. (*alpha, beta*)
246. Lemke-Keyes, C. A. and M. M. Sachs. 1989. J. Hered. 80:316-319. (*atn1*)
247. Leng, E.R. and L.F. Bauman. 1955. Agron. J. 47:189-191. (*ms21, sks1*)
248. Leng, E.R. and M.L. Vineyard. 1951. MNL 25:31-32. (*br2*)
249. Leto, K.J. 1982. p. 317-325 in W.F. Sheridan (ed.) Plant Mol. Biol. Assoc., Charlottesville, Virginia. (*hcf12, hcf13, hcf15, hcf19, hcf23, hcf26, hcf34, hcf38, hcf41, hcf46, hcf6*)
250. Li, H.W. 1931. J. Hered. 22:14-16. (*bv1*)
251. Li, H.W. 1937. J. Hered. 24:279-281. (*na1*)
252. Lindstrom, E.W. 1917. Am. Nat. 51:225-237. (*l1*)
253. Lindstrom, E.W. 1918. Cornell Univ. Agric. Exp. Stn. Memoir 13:1-68. (*l1, w1*)
254. Lindstrom, E.W. 1921. Genetics 6:91-110. (*f1*)
255. Lindstrom, E.W. 1924. Genetics 9:305-326. (*w2, w3*)
256. Lindstrom, E.W. 1925. J. Hered. 16:135-140. (*tp1*)
257. Lindstrom, E.W. 1935. Iowa St. Coll. J. Sci. 9:451-459. (*a3, og1*)
258. Lock, R.H. 1906. Roy. Bot. Gard. Annals 3:95-184. (*p1*)
259. Longley, A.E. 1937. J. Agric. Res. 54:835-862. (*K10*)

260. Lorenzoni, C., et al. 1974. MNL 48:19-20. (*cp1*)
261. Lowe, J. and O.E. Nelson, Jr. 1946. Genetics 31:525-533. (*mn1*)
262. Lysikov, V.N., et al. 1984. Sov. Genet. 20:72-80. (*cg2*)
263. Macdonald, T. and J.L. Brewbaker. 1972. J. Hered. 63:11-14. (*px2, px3, px4, px5, px6, px7, ta1*)
264. Macdonald, T. and J.L. Brewbaker. 1974. J. Hered. 65:37-42. (*e10, e5(l), e5(ll), e6, e7, e8, e9*)
265. Mains, E.B. 1926. J. Hered. 17:313-325. (*rp1*)
266. Mains, E.B. 1931. J. Agric. Res. 43:419-430. (*rp1*)
267. Mains, E.B. 1949. J. Hered. 40:21-24. (*sh2*)
268. Mangelsdorf, P.C. 1926. Conn. Agric. Exp. Stn. Bull. 279:509-614. (*bt1*)
269. Mangelsdorf, P.C. 1947. Genetics 32:448-458. (*du1*)
270. Marsan, P.A et al. 1992. MNL 66:20-21. (*rip1*)
271. Martienssen, R.A., et al. 1989. EMBO J. 8:1633-1639. (*hcf106*)
272. Martinez, P., et al. 1989. J. Mol. Biol. 208:551-565. (*gpc1*)
273. Mascia, P.N. 1978. Molec. Gen. Genet. 161:237-244. (*l13, oro1, oro2, orom1*)
274. Mascia, P.N. and D.S. Robertson. 1980. J. Hered. 71:19-24. (*nec1*)
275. Mashnenkov, A.S. and M.I. Khadjinov. 1979. Proc. IX Eucarpia Corn and Sorghum Sect. :447-450. (*sup1*)
276. Matthews, D.L., et al. 1974. J. Agric. Sci. 82:433-435. (*te1*)
277. Matz, E.C., et al. 1991. MNL 65:104-105. (*rd3*)
278. McCarty, D.P., et al. 1986. Proc. Natl. Acad. Sci. 83:9099-9103. (*css1*)
279. McClintock, B. 1934. Z. Zellforsch. Mikrosk. Anat. 21:294-328. (*NOR*)
280. McClintock, B. 1944. Genetics 29:478-502. (*pyd1, wd1*)
281. McClintock, B. 1950. Proc. Natl. Acad. Sci. 36:344-355. (*Ac, Ds*)
282. McClintock, B. 1956. Brookhaven Symp. Biol. 8:58-74. (*Spm*)
283. McClintock, B. 1957. Carnegie Inst. Wash. Yrbk. 56:393-401. (*Mod*)
284. McMullen, M.D., and R. Louie. 1989. Mol. Plant-Microbe Interact. 2:309-314. (*mdm1*)
285. Melville, J.C. and J.G. Scandalios, J.G. 1972. Biochem. Genet. 7:15-31. (*enp1*)
286. Micu, V. 1981. Genetical Studies of Maize. Shtiintsa, Kishinev, Mold. SSR. (*bs1, tl, dib1*)
287. Micu, V. 1980. MNL 54:63-64. (*dib1*)
288. Micu, V. and S.I. Mustyatsa. 1978. Genetika 14:365-368. (*dep1*)
289. Micu, V. 1992. MNL 66:86. (*dib1*)
290. Miles, C.D. 1989. MNL 63:66-67. (*lbl1*)
291. Miles, C.D. and D.J. Daniel. 1974. Plant Phys. 53:589-595. (*hcf1, hcf2, hcf3*)
292. Miles, C.D. 1982. p. 75-107 in M. Edelman, et al. (eds.) Methods in Chloroplast Molecular Biology, Elsevier, Amsterdam. (*hcf21, hcf4, hcf42, hcf44, hcf50*)
293. Miles, C.D., unpublished. (*hcf102*)
294. Miles, D., et al. 1986. p. 361-365 in K.E. Steinbeck, et al. (eds.) Molecular Biology of the Photosynthetic Apparatus, Cold Spring Harbor Laboratory, New York. (*hcf101, hcf18, hcf28, hcf31, hcf316, hcf323, hcf36, hcf408, hcf47, hcf48, hcf5*)
295. Miles, F.C. 1915. J. Genetics 4:193-214. (*gs1*)
296. Misra, P.S., et al. 1972. Science 176:1425-1427. (*o7*)
297. Modena, S.A. 1983. MNL 57:38. (*tpm1*)
298. Modena, S.A. 1984. MNL 58:211-212. (*pdf1*)
299. Modena, S.A. 1984. MNL 58:79-82. (*lcs1, lct1, lct2*)
300. Montoliu, L., et al. 1989. Plant Molec. Biol. 14:1-15. (*tua1, tua2*)
301. Montoliu, L., et al. 1990. Gene 94:201-207. (*tua3*)
302. Motto, M., et al. 1983. Maydica 28:25-39. (*w16, w17, wyg1*)
303. Mumm, W.J. 1929. Anat. Rec. 44:279. (*h1*)
304. Nelson, O.E. 1976. MNL 50:114. (*fl3*)
305. Nelson, O.E. 1981. MNL 55:68. (*lp1, o10, o11, o12, o13, o9*)
306. Nelson, O.E. and A.J. Ohlrogge. 1957. Science 125:1200. (*ct1, rd1*)
307. Nelson, O.E. and A.J. Ullstrup. 1964. J. Hered. 55:194-199. (*hm2*)
308. Nelson, O.E. and G.B. Clary. 1952. J. Hered. 43:205-210. (*dy1, lo2*)
309. Nelson, O.E. and S.N. Postlethwait. 1954. Am. J. Bot. 41:739-748. (*pt1*)
310. Nelson, O.E., et al. 1965. Science 150:1469-1470. (*fl2, o1, o2*)
311. Neuffer, M.G. 1973. MNL 47:150. (*nec3*)
312. Neuffer, M.G. 1988. MNL 62:53. (*lxm1*)
313. Neuffer, M.G. 1989. MNL 63:62. (*blh1, fbr1, gl22, sr4, w18, wi2, wlu5, wrp1, ws4*)
314. Neuffer, M.G. 1990. MNL 64:51. (*d9, gs4, rs4, sbd1, wi3*)
315. Neuffer, M.G. 1991. MNL 65:51-52. (*d9*)
316. Neuffer, M.G. 1992. MNL 66:39. (*dek33, les11, les12, les13, les14, les15, les16, les17, o14, sdw2*)
317. Neuffer, M.G. 1993. MNL 67:33. (*trn1*)
318. Neuffer, M.G. 1993. Personal Communication. (*rgh1, w19*)
319. Neuffer, M.G. and D. England. 1984. MNL 58:77-78. (*clt1*)
320. Neuffer, M.G. and J.B. Beckett. 1987. MNL 61:50. (*g6, gl19, gl21, grt1, gs3, ij2, l13, l16, l17, l18, l19, ms41, msc1, msc2, nec5, nec6, nec7, pg15, pg16, ppg1, py2, spc2, spc3, spt1, spt2, tlr1, v24, v*
321. Neuffer, M.G. and K.A. Sheridan. 1977. MNL 51:60. (*bif1, nl2, spc1, ysk1, zb8*)
322. Neuffer, M.G. and M.T. Chang. 1986. MNL 60:55. (*orp1, orp2*)
323. Neuffer, M.G. and O.H. Calvert. 1975. J. Hered. 66:265-270. (*les1, les2*)
324. Neuffer, M.G. and W.F. Sheridan. 1980. Genetics 95:929-944. (*dek1, dek10, dek11, dek12, dek13, dek14, dek15, dek2, dek3, dek4, dek5, dek6, dek7, dek8, dek9*)
325. Neuffer, M.G. and W.F. Sheridan. 1980. MNL 55:29-30. (*dek1, dek10, dek11, dek12, dek13, dek14, dek15, dek2, dek3, dek4, dek5, dek6, dek7, dek8, dek9*)
326. Neuffer, M.G., et al. 1968. The Mutants of Maize. Crop Sci. Soc. Amer., Madison, Wisconsin. (*cp2, ub1*)
327. Newhouse, K., et al. 1991. p. 139-150 in D.L. Shaner and S.L. O'Connor Imidazoline Herbicides, CRC Press, Boca Raton. (*als2, als1*)
328. Newton, K.J. and D. Schwartz. 1980. Genetics 95:425-442. (*mdh1, mdh2, mdh3, mdh4, mdh5, mmm1*)
329. Newton, K.J., et al. 1990. Plant Cell 2:107-113. (*NCS5*)
330. Nickerson, N.H. and E.E. Dale. 1955. Ann. Mo. Bot. Gard. 42:195-212. (*ra2, ts6*)
331. Nuffer, M.G. 1954. MNL 28:63-64. (*bz2*)
332. Nuffer, M.G. 1955. Science 121:399-400. (*Dt2, Dt3*)
333. Olive, M.R., et al. 1991. Nucl. Ac. Res. 19:7053-7060. (*gcb1*)
334. Ott, L. and J.G. Scandalios. 1978. Genetics 89:137-146. (*amp1, amp2, amp3, amp4*)
335. Paliy, A.F. and A.I. Rotar. 1979. Genetika 15:478-481. (*cfl2*)
336. Palmer, R.G. 1971. Chromosoma 35:233-246. (*am1*)
337. Pan, Y. B. and P. A. Peterson. 1991. Molec. Gen. Genet. 229:1661-174. (*Uq2, Uq3, Uq6, Uq4, Uq5*)
338. Pan, Y. B., and P. A. Peterson. 1990. MNL 64:8-9. (*ba3*)
339. Parker, W.B., et al. 1990. Proc. Natl. Acad. Sci. USA 87:7175-7179. (*acc1*)
340. Peacock, W.J., et al. 1981. Proc. Natl. Acad. Sci. USA 78:4490-4494. (*K*)
341. Perry, H.S. 1939. MNL 13:7. (*lg3*)
342. Perry, H.S. and G.F. Sprague. 1936. J. Am. Soc. Agron. 28:990-996. (*y3*)
343. Peschke, V.M. and M.M. Sachs. 1993. Molec. Gen. Genet. (in press). (*pdc1, pdc2, pdc3*)
344. Peterson, H. 1959. MNL 33:41. (*tp2*)
345. Peterson, P.A. 1960. Genetics 45:115-133. (*En*)

346. Pfund, J.H. and C.W. Crum. 1977. Agron. Abstr. p. 66. (*thc1*)
347. Phinney, B.O. 1956. Proc. Natl. Acad. Sci. 42:185-189. (*d8*)
348. Phipps, I.F. 1928. J. Hered. 19:399-404. (*ts4*)
349. Phipps, I.F. 1929. Cornell Univ. Agric. Exp. Stn. Memoir 125:1-63. (*al1, v12, v13, v16, v17, v18*)
350. Piovarci, A. 1982. MNL 56:157. (*bv2*)
351. Pla, M. 1991. Molec. Gen. Genet. 230:394-400. (*rab28*)
352. Plewa, M. 1979. MNL 53:93-96. (*loc1*)
353. Pogna, N.E., et al. 1982. MNL 56:153. (*ys2*)
354. Postlethwait, S.N. and O.E. Nelson, Jr. 1957. Am. J. Bot. 44:628-633. (*wi1*)
355. Pryor, A.J. 1974. Heredity 32:397-401. (*gdh1*)
356. Pryor, A.J. 1976. MNL 50:15-16. (*glu1*)
357. Pryor, T. and D. Schwartz. 1973. Genetics 75:75-92. (*cx1*)
358. Qin, T-C., et al. 1990. MNL 64:124. (*rf7*)
359. Ralston, E., et al. 1988. Genetics 119:185-197. (*lns1, lns2*)
360. Rashid, A. and P.A. Peterson. 1992. J Hered 83:130-134. (*cif1, cim1, cim2*)
361. Razafimahatratra, P., et al. 1991. Nucl. Ac. Res. 19:1491-1496. (*his1*)
362. Rhoades, M.M. 1935. Am. Nat. 69:74-75. (*Dt1*)
363. Rhoades, M.M. 1939. Genetics 24:62. (*ws3*)
364. Rhoades, M.M. 1948. MNL 22:9. (*ga7*)
365. Rhoades, M.M. 1951. Am. Nat. 85:105-110. (*pg11, pg12*)
366. Rhoades, M.M. 1952. Am. Nat. 86:105-106. (*bz1*)
367. Rhoades, M.M. 1956. MNL 30:38-42. (*am1, el1*)
368. Rhoades, M.M. and E. Dempsey. 1954. MNL 28:58. (*gl17*)
369. Rhoades, M.M. and E. Dempsey. 1982. MNL 56:21-22. (*Mrh, Mut, rMrh, rMut*)
370. Robertson, D.S. 1952. Proc. Natl. Acad. Sci. 38:580-583. (*vp5*)
371. Robertson, D.S. 1955. Genetics 40:745-760. (*vp8, vp9*)
372. Robertson, D.S. 1961. Genetics 46:649-662. (*y10*)
373. Robertson, D.S. 1967. MNL 41:94. (*o5*)
374. Robertson, D.S. 1973. MNL 47:82. (*l10*)
375. Robertson, D.S. 1974. MNL 48:70. (*yd2*)
376. Robertson, D.S. 1975. J. Hered. 66:127-130. (*y9*)
377. Robertson, D.S. 1978. Mutat. Res. 51:21-28. (*Mu*)
378. Robertson, D.S. 1981. MNL 55:115. (*l15*)
379. Robertson, D.S. 1984. MNL 58:18. (*brn1*)
380. Rochester, D.E., et al. 1986. EMBO J. 5:451-458. (*hsp1*)
381. Rothermel, B.A. and T. Nelson. 1989. J. Biol. Chem. 264:19587-19592. (*me3*)
382. Russell, D.A., and M.M. Sachs. 1989. Plant Cell 1:793-803. (*gpc1, gpc2, gpc3*)
383. Russell, D.A. and M.M. Sachs. 1991. Molec. Gen. Genet. 229:219-228. (*gpc3, gpc4*)
384. Sachan, J.K. and K.R. Sarkar. 1978. MNL 52:119-120. (*rgo1*)
386. Salamini, F. 1980. Cold Spr. Harb. Symp. 45:467-476. (*Bg, rBg*)
387. Salamini, F., et al. 1983. Theor. Appl. Genet. 65:123-128. (*mc1*)
388. Saxena, K.M.S. and A.L. Hooker. 1968. Proc. Natl. Acad. Sci. 61:1300-1305. (*rp5*)
389. Scandalios, J.G., et al. 1972. Arch. Biochem. Biophys. 153:695-705. (*cat2*)
390. Scandalios, J.G., et al. 1975. Biochem. Genet. 13:759-769. (*got1*)
391. Scandalios, J.G., et al. 1980. Mol. Gen. Genet. 179:33-41. (*cat3*)
392. Scandalios, J.G., et al. 1980. Proc. Natl. Acad. Sci. 77:5360-5364. (*car1*)
393. Scanlon, M., et al. 1992. MNL 66:8. (*dsc1, ptd1, ptd2, ren1, ren2, emp1, emp2*)
394. Scanlon, M. 1992. Personal Communication. (*ren1, ren3*)
395. Schiefelbein, J.W., et al. 1985. Proc. Natl. Acad. Sci. 82:4783-4787. (*dSpm*)
396. Schnable, P.S. and P.A. Peterson. 1986. Maydica 31:59-82. (*Cy, rcy*)
397. Schwartz, D. 1951. Genetics 36:676-696. (*ms21, sks1*)
398. Schwartz, D. 1951. MNL 25:30. (*ga8*)
399. Schwartz, D. 1960. Proc. Natl. Acad. Sci. USA 46:1210-1215. (*e1*)
400. Schwartz, D. 1964. Proc. Natl. Acad. Sci. USA 51:602-605. (*e3*)
401. Schwartz, D. 1965. Proc. XI Int. Genet. Cong. 2:131-135. (*e2*)
402. Schwartz, D. 1966. Proc. Natl. Acad. Sci. USA 56:1431-1436. (*adh2*)
403. Schwartz, D. 1979. Molec. Gen. Genet. 174:233-240. (*glb1, mep1*)
404. Schwartz, D. and T. Endo. 1966. Genetics 53:709-715. (*adh1*)
405. Shaver, D. 1983. Proc. Annu. Corn Sorghum Res. Conf. 38:161-180. (*lfy1*)
406. Shaver, D.L. 1967. J. Hered. 58:270-273. (*gt1, pe1*)
407. Sheen, J. and L. Bogorad. 1986. Proc. Natl. Acad. Sci. USA 83:7811-7815. (*lhcb1, lhcb2*)
408. Sheridan, W. F. 1988. Ann. Rev. Genet. 22:353-385. (*dek31, fae1, tru1*)
409. Sheridan, W.F., et al. 1986. MNL 60:64. (*dek22, dek23, dek24, dek25, dek26, dek27, dek28, dek29, dek30*)
410. Sheridan, W.F., et al. 1984. MNL 58:98-99. (*dek16, dek17, dek18, dek19, dek20*)
411. Shortess, D.K. and R.P. Amby. 1979. Maydica 24:215-221. (*pg13*)
412. Shortess, D.K., et al. 1968. Genetics 58:227-235. (*lu1*)
413. Shumway, L.K. and L.F. Bauman. 1967. Genetics 55:33-38. (*NCS1*)
414. Simcox, K. and J.L. Bennetzen. 1993. MNL 67:118-119. (*htn1*)
415. Singh, K., et al. 1990. Plant Cell 2:891-903. (*obf1, obf2*)
416. Singleton, W.R. 1946. J. Hered. 37:61-64. (*id1*)
417. Singleton, W.R. 1951. Am. Nat. 85:81-96. (*cg1*)
418. Singleton, W.R. 1959. MNL 33:3. (*br3*)
419. Singleton, W.R. and D.F. Jones. 1930. J. Hered. 21:266-268. (*ms1*)
420. Smith, D.R. and A.L. Hooker. 1973. Crop Sci. 13:330-331. (*rhm1*)
421. Smith, J.D. and M.G. Neuffer. 1992. MNL 66:34. (*vp10*)
422. Smith, R.D. and J.C. Walker. 1991. Plant Phys. 97:677-683. (*prh1*)
423. Soave, C., et al. 1978. Theor. Appl. Genet. 52:263-267. (*zp*)
424. Soave, C., et al. 1981. Genetics 97:363-377. (*os1, zp*)
425. Souciet, G. 1992. Plant Sci 81:215-225. (*acpt1*)
426. Sprague, G.F. 1932. U.S. Tech. Bull. 292:1-43. (*ps1, sy1*)
426a. Sprague, G. F. 1938. MNL 12:2-3. (*gl11*)
427. Sprague, G.F. 1939. J. Hered. 30:143-145. (*vg1*)
428. Sprague, G.F. 1984. MNL 58:197. (*Dt6*)
429. Sprague, G.F. 1987. MNL 61:97. (*gl20, gl5, y11, y12*)
430. Sprague, G.F. 1990. MNL 64:110. (*gl23, gl24, sh5*)
431. Sprague, G.F., et al. 1965. MNL 39:164. (*wt1*)
432. Sprague, G.F., unpublished. 1935. (*gl4, gl5*)
433. Sprague, G.F. 1992. Maydica 37:107-110. (*gl13*)
434. Stadler, L.J. 1940. MNL 14:26. (*et1*)
435. Stadler, L.J. and M.G. Nuffer. 1953. Science 117:471-472. (*P*)
436. Stadler, L.J. and M.H. Emmerling. 1956. Genetics 41:124-137. (*P, S*)
437. Stadler, L.J., and H. Roman. 1948. Genetics 33:273-303. (*w19*)
438. Steffensen, D.M. and E.B. Patterson. 1979. Genetics 123s:123. (*rDNA5S*)
439. Stiefel, V., et al. 1990. Plant Cell 2:785-793. (*hrg1*)
440. Stierwalt, T.R. and P.L. Crane. 1974. MNL 48:139. (*sen1, sen2, sen3, sen4, sen5, sen6*)
441. Stinard, P.S. 1992. MNL 66:4-5. (*su3*)
442. Stinard, P.S. 1991. MNL 65:16-17. (*mn3, cr4*)
443. Stinard, P.S. and D.S. Robertson. 1987. MNL 61:7-9. (*dap1*)
444. Stinard, P.S., et al. 1993. MNL 67:9. (*et2*)

445. Stinson, J.R., et al. 1987. Plant Phys. 83:442-447. (*mgs1*)
446. Stout, J.T. and R.L. Phillips. 1973. Proc. Natl. Acad. Sci. 70:3043-3047. (*alh1, clh1*)
447. Stroman, G.N. 1924. Genetics 9:493-512. (*zb2*)
448. Stroup, D. 1970. J. Hered. 61:139-141. (*cm1*)
449. Styles, E.D., et al. 1987. MNL 61:100. (*ufo1*)
450. Sullivan, T., et al. 1989. Molec. Gen. Genet. 215:431-440. (*lhcb2*)
451. Suttle, A.D. 1924. Unpub. thesis, Cornell Univ. (*d2, d5, py1*)
452. Talbert, L.E., et al. 1989. J. Mol. Evol. 29:28-39. (*Mu4, Mu5*)
453. Tavcar, A. 1932. Jugoslav. Akad. Znanosti i Umjetnosti Prestampo. 244:74-93. (*hs1*)
454. Teas, H.J. and A.N. Teas. 1953. J.Hered. 44:156-158. (*bt2*)
455. Teas, H.J. and E.G. Anderson. 1951. Proc. Natl. Acad. Sci. 37:645-649. (*bf1*)
456. Tillman, U., et al. 1989. EMBO J. 8:2463-2467. (*abp1*)
457. Trimnell, M.R., and M.C. Albertsen. 1993. MNL 67:51. (*tls1*)
458. Tsai, C. and O.E. Nelson. 1969. Genetics 61:813-821. (*sh4*)
459. Tulpule, S.H. 1954. Am. J. Bot. 41:294-301. (*lw1, lw2, lw3, lw4*)
460. Ullstrup, A.J. 1965. Phytopathology 55:425-428. (*rpp9*)
461. Ullstrup, A.J. and A.F. Troyer. 1968. Phytopathology 57:1282-1283. (*lls1*)
462. Ullstrup, A.J. and A.M. Brunson. 1947. J. Am. Soc. Agron. 39:606-609. (*hm1*)
463. Vahrusheva, E.I. 1975. MNL 49:95-96. (*cto1*)
464. Vidakovic, M. 1988. Maydica 33:51-64. (*rf5, rf6*)
465. Villemur, R., et al. 1992. J. Mol. Biol. 227:81-96. (*tua4, tua5, tua6*)
466. Vineyard, M.L. and R.P. Bear. 1952. MNL 26:5. (*ae1*)
467. Walsh, T.A., et al. 1991. J. Biol. Chem. 266:23422-23427. (*rip1*)
468. Walters, D.A., et al. 1992. Plant Molec. Biol. 18:189-200. (*hpt1*)
469. Watson, N.R., et al. 1992. Biochem. Genet. 30:371-383. (*alt1, alt2, alt3*)
470. Wen, L. 1992. Plant Molec. Biol. 18:813-814. (*hfi1*)
471. Wendel, J.F., et al. 1986. MNL 60:109. (*adk1*)
472. Wendel, J.F., et al. 1986. Theor. Appl. Genet. 72:178-185. (*hex1, hex2*)
473. Wendel, J.F., et al. 1989. J. Hered. 80:218-228. (*tpi1, tpi2, tpi3, tpi4, tpi5*)
474. Wendel, J.F., et al. 1985. MNL 59:87-88. (*aco1, aco2, aco3, aco4, dia1, dia2, sad1*)
475. Wentz, J.B. 1926. J. Hered. 17:327-329. (*bt1*)
476. West, D.P. and M.C. Albertsen. 1985. MNL 85:87. (*ms22, ms23, ms24*)
477. Weydemann, U., et al. 1988. MNL 62:48. (*Mpi1*)
478. Wilkinson, D.R. and A.L. Hooker. 1968. Phytopathology 58:605-608. (*rp3, rp4, rp6*)
479. Wilson, C.M. 1991. MNL 65:91. (*zpl1, zpl2a, zpl2b, zpl3a*)
480. Worrell, A.C., et al. 1991. Plant Cell 3:1121-1130. (*sps1*)
481. Wright, J.E. 1961. MNL 35:111. (*ys3*)
482. Wright, S. and T. Helentjaris. 1988. MNL 62:104. (*lhcb1, lhcb2*)
483. Wright, A., et al. 1992. Plant Cell 4:711-719. (*orp1*)
484. Zaitlin, D., et al. 1992. MNL 66:70-71. (*rDNA5S*)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umc11
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p1 npi286 umc(hcf3)
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bnl7.21
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npi401
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adh1
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rpa7a
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TB-1La-3L5267
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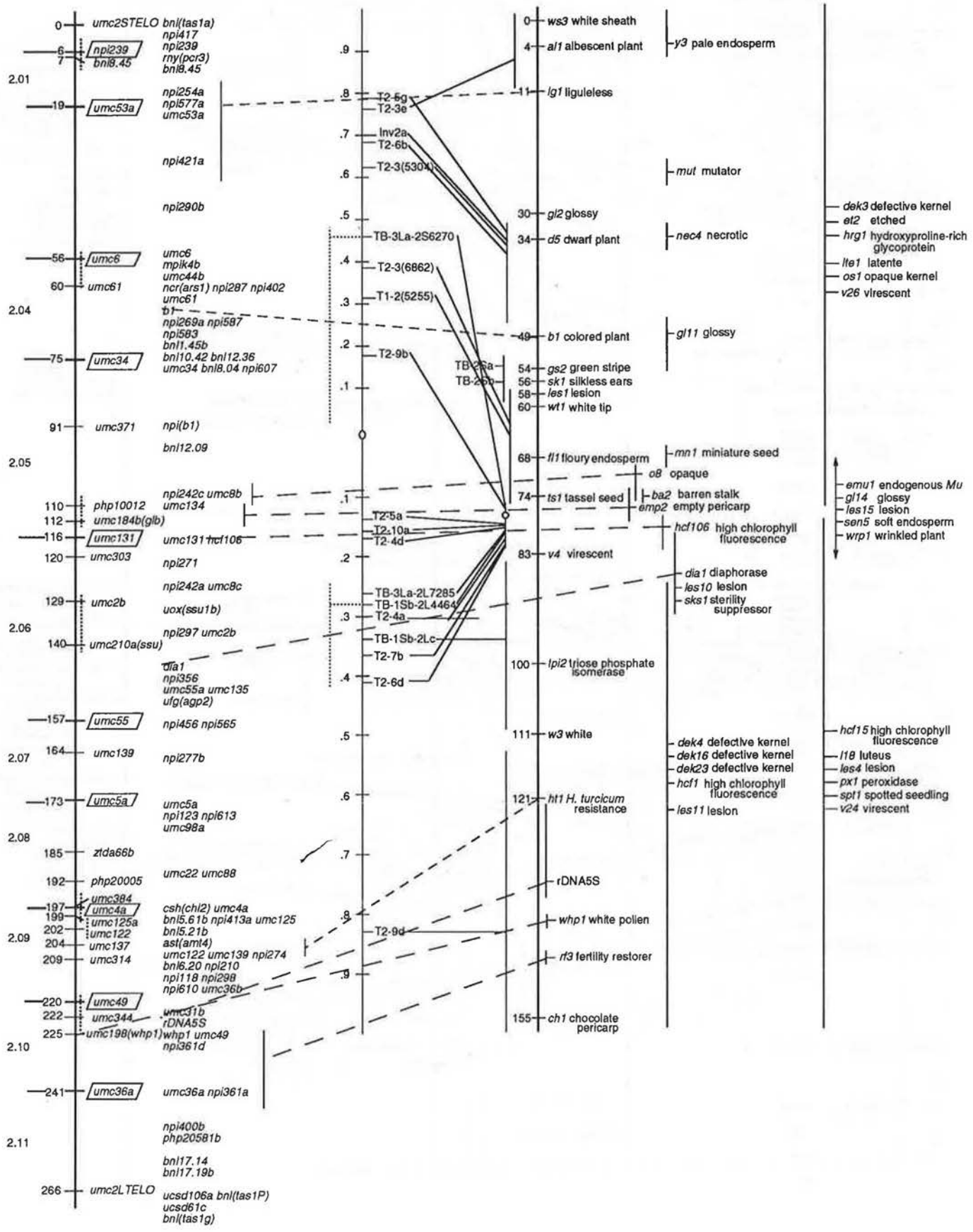
TB-1La-3L5242
T1-4g
sr1 striate leaves
vp5 viviparous
zb4 zebra crossbands
ms17 male sterile
ts2 tassel seed
p1 pericarp color
dek1 defective kernel
nec2 necrotic
as1 asynaptic
hm1 H. carbonum susceptibility
br1 brachytic culms
vg1 vestigial glumes
f1 fine stripe
an1 anther ear
bz2 bronze
ad1 adherent
pld1 pitted endosperm
glb1 globulin
zb7 zebra
d8 dwarf plant
kn1 knotted
adh1 alcohol dehydrogenase
gs1 green stripe
phi1 phosphohexose isomerase
vp8 viviparous
ts6 tassel seed
bm2 brown midrib
acp4 acid phosphatase
cat2 catalase
les2 lesion
rab30 responsive to ABA
ct2 compact plant
ms28 male sterile
hcf3 high chlorophyll fluorescence
ms9 male sterile
sod4 superoxide dismutase
ms14 male sterile
obf1 octopine synthase binding factor
rs2 rough sheath
br2 brachytic culms
amp2 aminopeptidase
wlu5 white luteus
amp1 aminopeptidase
mdh4 malate dehydrogenase
mmm1 modifier of mMDH
id1 indeterminate growth
v22 virescent
pgm1 phosphoglucomutase
tb1 teosinte branched
mpl1 miniplant
tua1 tua2 alpha tubulin
lw1 lemon white
w18 white
rd1 reduced plant
gdh1 glutamic dehydrogenase
lj2 lojap striping
py2 pygmy
tls1 tasselles
alh1 histone 1a
dia2 diaphorase
blh1 bleached leaf
car1 catalase regulator
cfr1 coupling factor reduction
dek32 defective kernel
emp1 empty pericarp
hcf6 high chlorophyll fluorescence
hcf31 high chlorophyll fluorescence
l16 luteus
les5 lesion
lls1 lethal leaf spot
pg15 pale green
v25 virescent
ws4 white sheath
ys2 yellow stripe
gt1 grassy tillers
mi1 midget plant
ms12 male sterile
sen3 soft endosperm
dek2 defective kernel
dek22 defective kernel
hcf2 high chlorophyll fluorescence
hcf4 high chlorophyll fluorescence
hcf7 high chlorophyll fluorescence
hcf12 high chlorophyll fluorescence
hcf13 high chlorophyll fluorescence
hcf41 high chlorophyll fluorescence
hcf44 high chlorophyll fluorescence
hcf50 high chlorophyll fluorescence
l17 luteus
msc1 mosaic aleurone
pg16 pale green
o10 opaque
spc2 speckled
tlr1 tillered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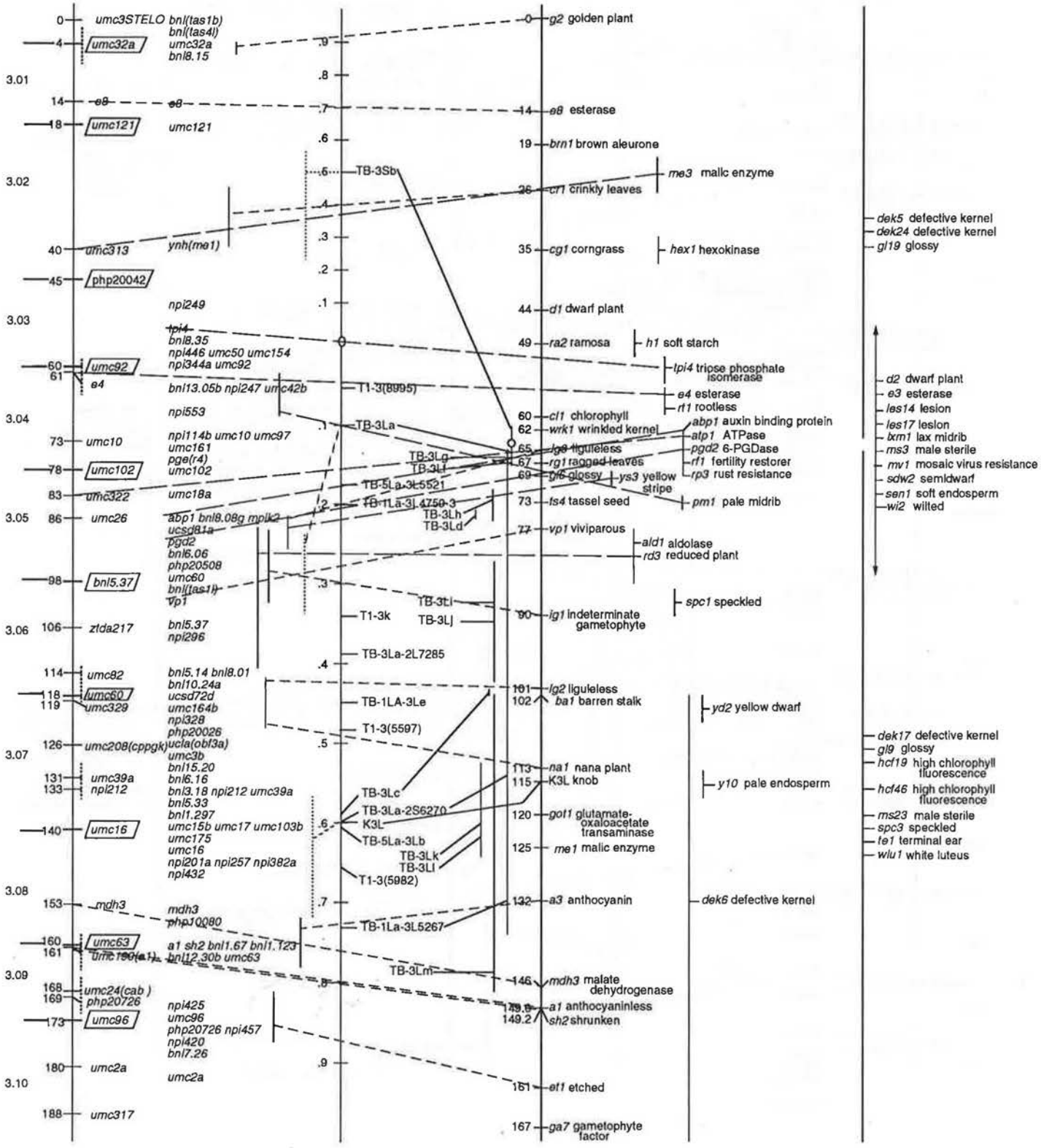
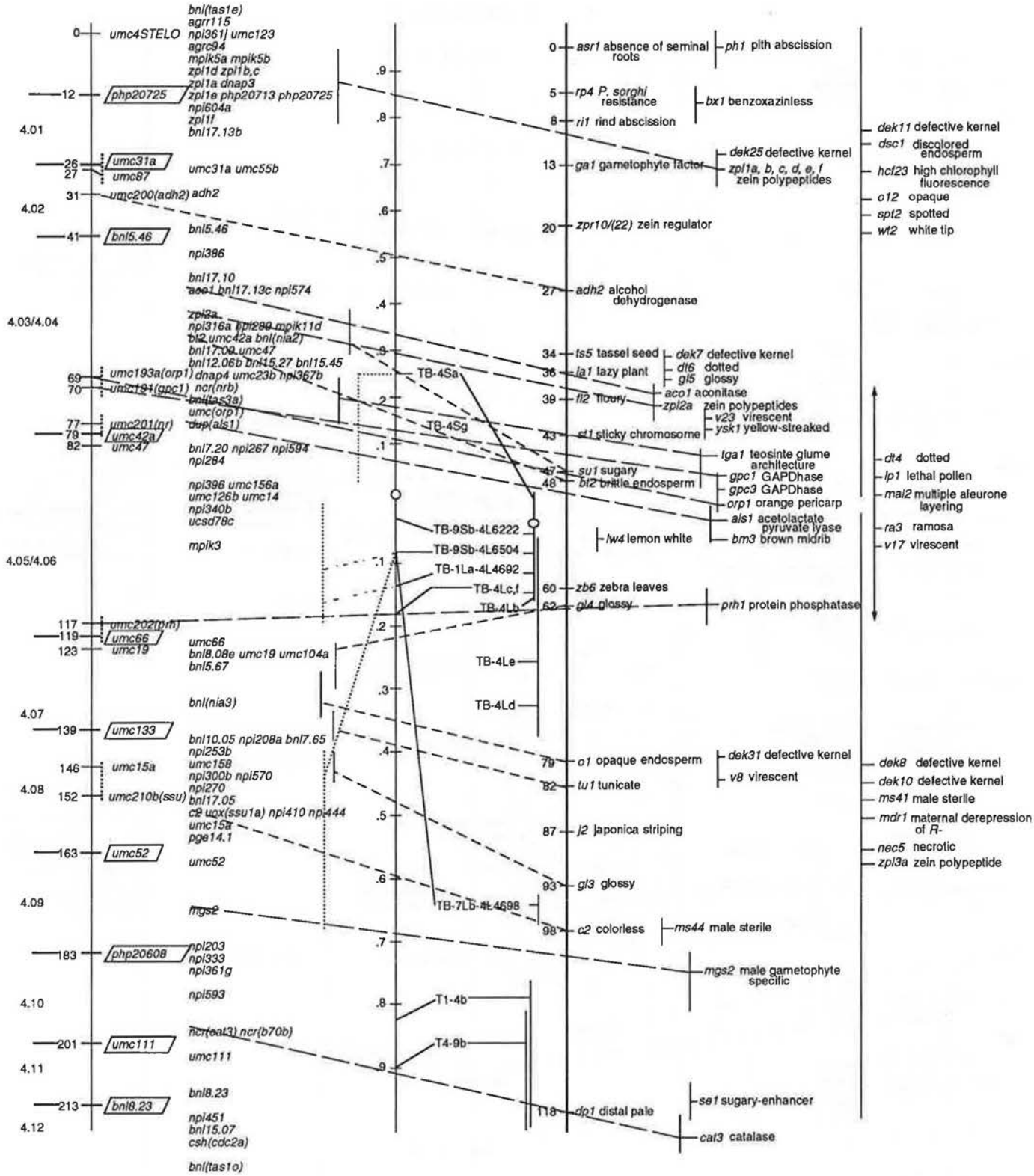
.7 Inv2a
T2-6b
.6 T2-3(5304)
.5
TB-3La-2S6270
.4 T2-3(6862)
.3 T1-2(5255)
.2 T2-9b
.1
.1
T2-5a
T2-10a
T2-4d
.2
TB-3La-2L7285
TB-1Sb-2L4464
.3 T2-4a
TB-1Sb-2Lc
T2-7b
.4 T2-6d
.5
.6
.7
.8 T2-9d
.9
0 ws3 white sheath
4 al1 albescent plant
11 lg1 liguleless
30 gl2 glossy
34 d5 dwarf plant
49 b1 colored plant
54 gs2 green stripe
56 sk1 silkless ears
58 les1 lesion
60 wt1 white tip
68 fl1 floury endosperm
74 ts1 tassel seed
83 v4 virescent
100 lpi2 triose phosphate isomerase
111 w3 white
121 ht1 H. turcicum resistance
155 ch1 chocolate pericarp
rDNA5S
whp1 white pollen
rf3 fertility restorer
y3 pale endosperm
mut mutator
nec4 necrotic
gl11 glossy
mn1 miniature seed
o8 opaque
ba2 barren stalk
emp2 empty pericarp
hcf106 high chlorophyll fluorescence
dia1 diaphorase
les10 lesion
sks1 sterility suppressor
dek4 defective kernel
dek16 defective kernel
dek23 defective kernel
hcf1 high chlorophyll fluorescence
les11 lesion
dek3 defective kernel
et2 etched
hrg1 hydroxyproline-rich glycoprotein
lte1 latente
os1 opaque kernel
v26 virescent
emu1 endogenous Mu
gl14 glossy
les15 lesion
sen5 soft endosperm
wrp1 wrinkled plant
hcf15 high chlorophyll fluorescence
l18 luteus
les4 lesion
px1 peroxidase
spt1 spotted seedling
v24 virescent

CHROMOSOME 3




bnl(tas1e)
agrr115
0 umc4STELO npi361j umc123
agrc94
mpik5a mpik5b
zpl1d zpl1b,c
zpl1a dnap3
12 php20725 zpl1e php20713 php20725
npi604a
4.01 zpl1f
bnl17.13b
26 umc31a
27 umc87 umc31a umc55b
31 umc200(adh2) adh2
4.02
41 bnl5.46 bnl5.46
npi386
bnl17.10
aco1 bnl17.13c npi574
4.03/4.04 zpl2a
npi316a bpi289 mpik11d
bl2 umc42a bnl(nia2)
bnl17.09 umc47
bnl12.06b bnl15.27 bnl15.45
69 umc193a(orp1) dnap4 umc23b npi367b
70 umc191(gpc1) ncr(nrb)
bnl(tas3a)
umc(orp1)
77 umc201(nr) dup(als1)
79 umc42a
82 umc47 bnl7.20 npi267 npi594
npi284
npi396 umc156a
umc126b umc14
npi340b
ucsd78c
mpik3
4.05/4.06
117 umc202(prh)
119 umc66
123 umc19 umc66
bnl8.08e umc19 umc104a
bnl5.67
bnl(nia3)
4.07
139 umc133
bnl10.05 npi208a bnl7.65
npi253b
146 umc15a umc158
npi300b npi570
4.08 npi270
152 umc210b(ssu) bnl17.05
c2 uox(ssu1a) npi410 npi444
umc15a
pge14.1
163 umc52 umc52
4.09
mgs2
183 php20608 npi203
npi333
npi361g
4.10 npi593
ncr(eat3) ncr(b70b)
201 umc111 umc111
4.11
bnl8.23
213 bnl8.23
npi451
4.12 bnl15.07
csh(cdc2a)
bnl(tas1o)
.9
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.6
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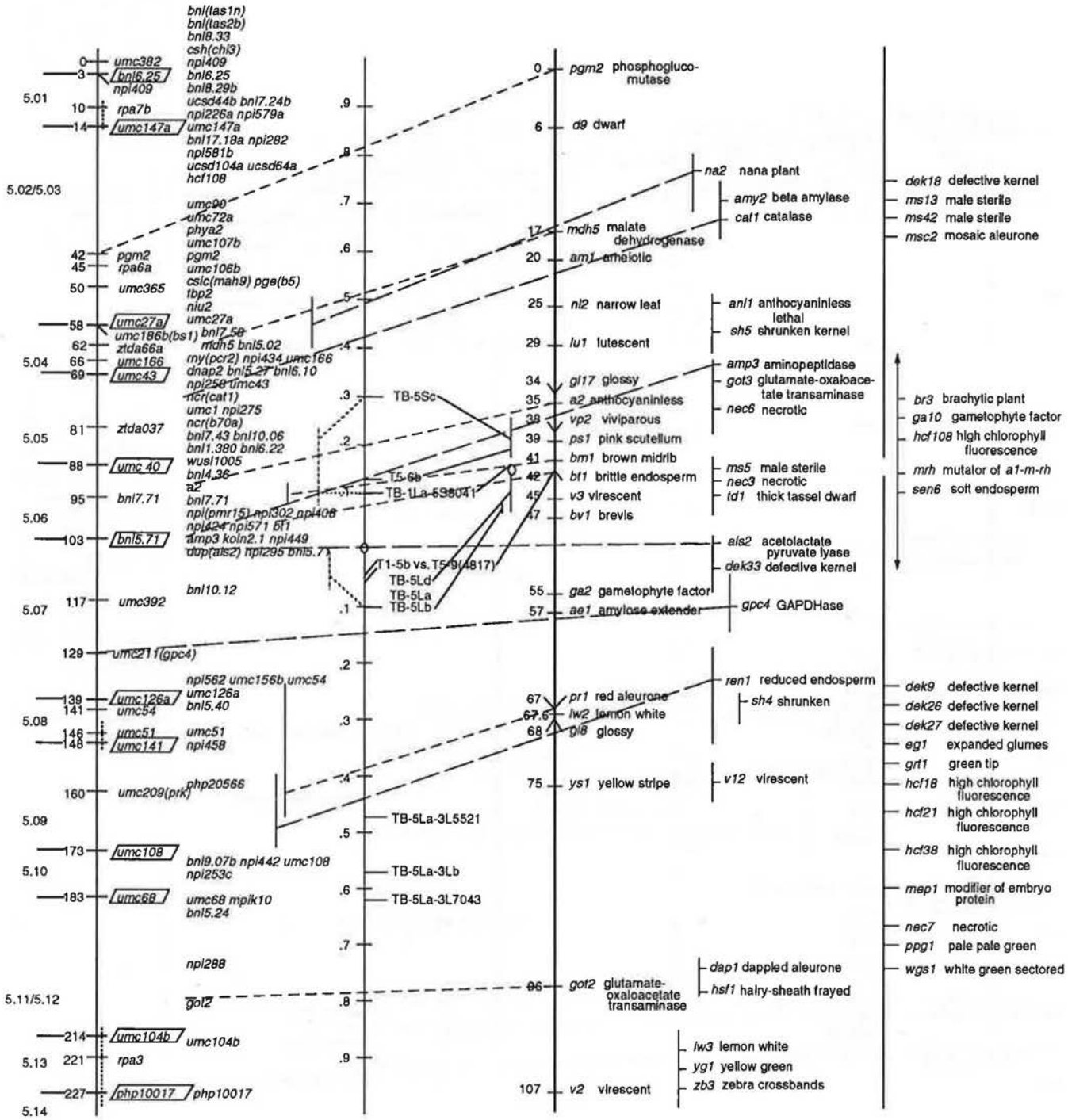
.4
.3
.2
.1
TB-4Sa
TB-4Sg
TB-9Sb-4L6222
TB-9Sb-4L6504
TB-1La-4L4692
TB-4Lc,f
TB-4Lb
TB-4Le
TB-4Ld
TB-7Lb-4L4698
T1-4b
T4-9b
0 asr1 absence of seminal roots
ph1 plth abscission
5 rp4 P. sorghi resistance
bx1 benzoxazinless
8 ri1 rind abscission
dek11 defective kernel
dsc1 discolored endosperm
13 ga1 gametophyte factor
dek25 defective kernel
zpl1a, b, c, d, e, f zein polypeptides
hcf23 high chlorophyll fluorescence
o12 opaque
spt2 spotted
wt2 white tip
20 zpr10/(22) zein regulator
27 adh2 alcohol dehydrogenase
34 ts5 tassel seed
dek7 defective kernel
36 la1 lazy plant
dl6 dotted
gl5 glossy
aco1 aconitase
38 fl2 floury
zpl2a zein polypeptides
v23 virescent
ysk1 yellow-streaked
43 st1 sticky chromosome
tga1 teosinte glume architecture
dt4 dotted
lp1 lethal pollen
47 su1 sugary
gpc1 GAPDhase
48 bt2 brittle endosperm
gpc3 GAPDhase
mal2 multiple aleurone layering
orp1 orange pericarp
als1 acetolactate pyruvate lyase
ra3 ramosa
lw4 lemon white
bm3 brown midrib
v17 virescent
60 zb6 zebra leaves
62 gl4 glossy
prh1 protein phosphatase
79 o1 opaque endosperm
dek31 defective kernel
v8 virescent
dek8 defective kernel
dek10 defective kernel
82 tu1 tunicate
ms41 male sterile
mdr1 maternal derepression of R-
87 j2 japonica striping
nec5 necrotic
zpl3a zein polypeptide
93 gl3 glossy
98 c2 colorless
ms44 male sterile
mgs2 male gametophyte specific
se1 sugary-enhancer
118 dp1 distal pale
cat3 catalase


bnl(tas1n)
bnl(tas2b)
bnl8.33
csh(chi3)
0 umc382
3 bnl6.25
npi409
5.01
10 rpa7b
14 umc147a
bnl6.25
bnl8.29b
ucsd44b bnl7.24b
npi226a npi579a
umc147a
bnl17.18a npi282
npi581b
ucsd104a ucsd64a
hcf108
5.02/5.03
umc90
umc72a
phya2
umc107b
42 pgm2
45 rpa6a
pgm2
umc106b
50 umc365
cslc(mah9) pge(b5)
tbp2
niu2
58 umc27a
umc186b(bs1)
umc27a
bnl7.58
62 ztda66a
mdh5 bnl5.02
5.04
66 umc166
69 umc43
rny(pcr2) npi434 umc166
dnap2 bnl5.27 bnl6.10
npi258 umc43
ncr(cat1)
umc1 npi275
81 ztda037
ncr(b70a)
5.05
bnl7.43 bnl10.06
bnl1.380 bnl6.22
88 umc 40
wusl1005
bnl4.36
a2
95 bnl7.71
bnl7.71
npi(pmr15) npi302 npi408
5.06
npi424 npi571 bf1
103 bnl5.71
amp3 koln2.1 npi449
dup(als2) npi295 bnl5.7
bnl10.12
1.17 umc392
5.07
129 umc211(gpc4)
npi562 umc156b umc54
139 umc126a
umc126a
141 umc54
bnl5.40
5.08
146 umc51
148 umc141
umc51
npi458
160 umc209(prk)
php20566
5.09
173 umc108
bnl9.07b npi442 umc108
5.10
npi253c
183 umc68
umc68 mpik10
bnl5.24
npi288
5.11/5.12
got2
214 umc104b
umc104b
5.13 221 rpa3
227 php10017
php10017
5.14
.9
.8
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.5
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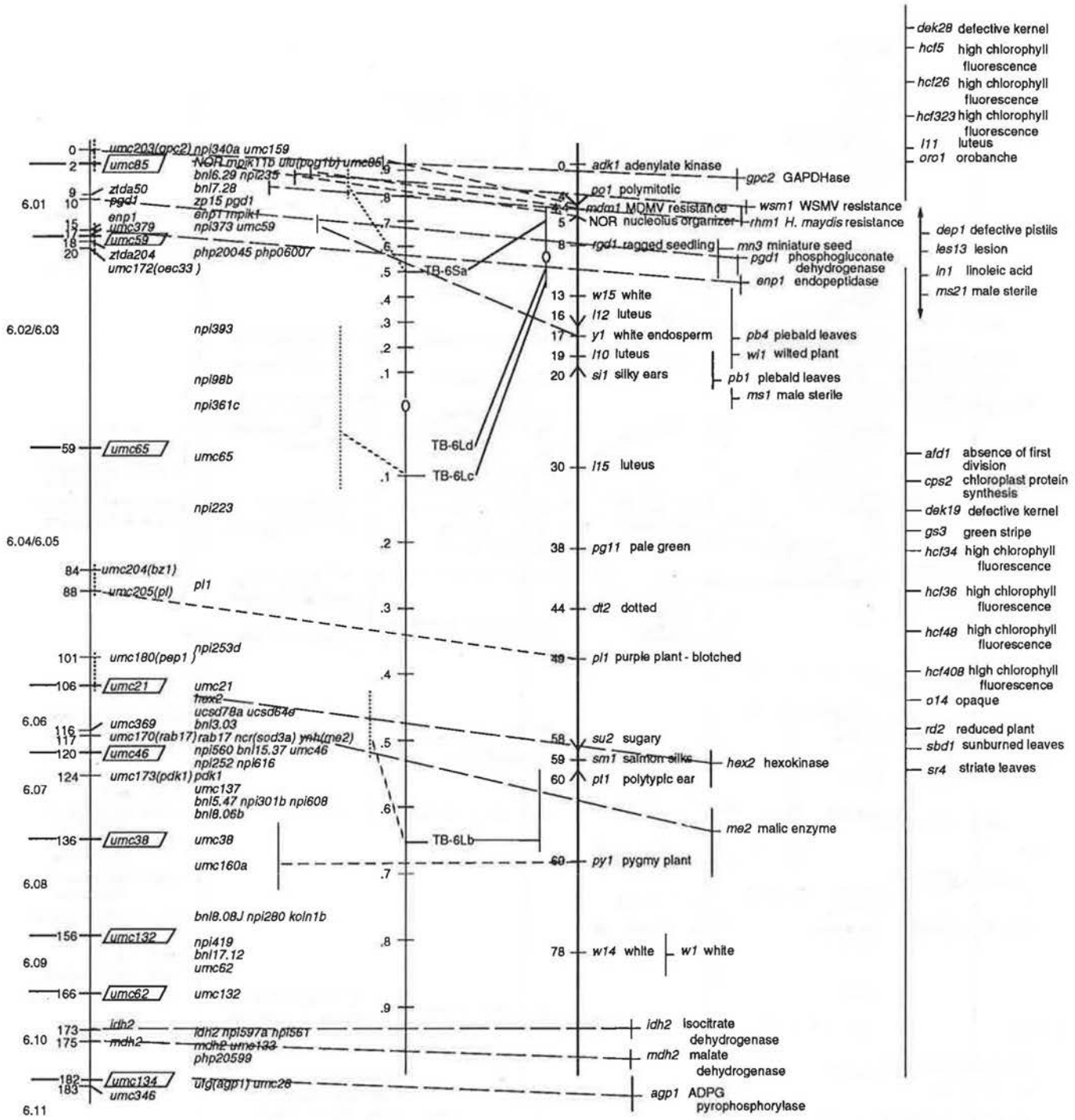
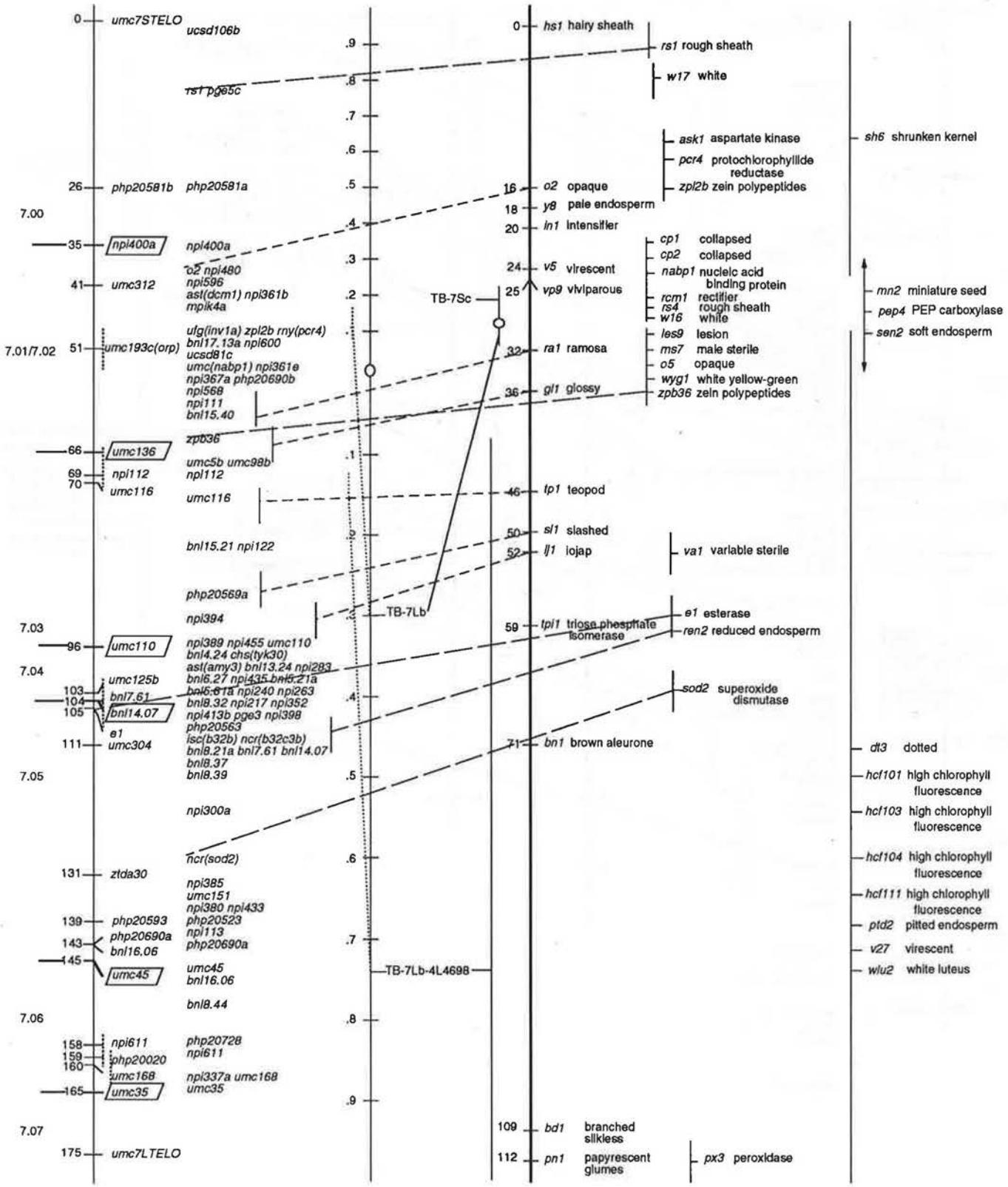
.9
TB-5Sc
T5-6b
TB-1La-6S8041
T1-5b vs. T5-9(4817)
TB-5Ld
TB-5La
TB-5Lb
TB-5La-3L5521
TB-5La-3Lb
TB-5La-3L7043
0 pgm2 phosphogluco-mutase
6 d9 dwarf
na2 nana plant
amy2 beta amylase
cat1 catalase
17 mdh5 malate dehydrogenase
20 am1 ameiotic
25 nl2 narrow leaf
anl1 anthocyaninless lethal
sh5 shrunken kernel
29 lu1 lutescent
amp3 aminopeptidase
got3 glutamate-oxaloace-tate transaminase
34 gl17 glossy
35 a2 anthocyaninless
nec6 necrotic
38 vp2 viviparous
39 ps1 pink scutellum
41 bm1 brown midrib
42 bf1 brittle endosperm
ms5 male sterile
nec3 necrotic
45 v3 virescent
td1 thick tassel dwarf
47 bv1 brevis
als2 acetolactate pyruvate lyase
dek33 defective kernel
55 ga2 gametophyte factor
57 ae1 amylose extender
gpc4 GAPDHase
ren1 reduced endosperm
67 pr1 red aleurone
sh4 shrunken
67.6 lw2 lemon white
68 gl8 glossy
75 ys1 yellow stripe
v12 virescent
dap1 dappled aleurone
86 got2 glutamate-oxaloacetate transaminase
hsf1 hairy-sheath frayed
lw3 lemon white
yg1 yellow green
107 v2 virescent
zb3 zebra crossbands
dek18 defective kernel
ms13 male sterile
ms42 male sterile
msc2 mosaic aleurone
br3 brachytic plant
ga10 gametophyte factor
hcf108 high chlorophyll fluorescence
mrh mutator of a1-m-rh
sen6 soft endosperm
dek9 defective kernel
dek26 defective kernel
dek27 defective kernel
eg1 expanded glumes
grt1 green tip
hcf18 high chlorophyll fluorescence
hcf21 high chlorophyll fluorescence
hcf38 high chlorophyll fluorescence
mep1 modifier of embryo protein
nec7 necrotic
ppg1 pale pale green
wgs1 white green sectored


umc203(gpc2) npi340a umc159
umc85
NOR mpik11b ufg(pgg1b) umc85
bnl6.29 npi235
ztda50
bnl7.28
pgd1
zp15 pgd1
enp1
enp1 mpik1
umc379
npi373 umc59
umc59
ztda204
php20045 php06007
umc172(oec33)
6.01
6.02/6.03
npi393
npi98b
npi361c
umc65
umc65
npi223
6.04/6.05
umc204(bz1)
umc205(pl)
pl1
umc180(pep1)
npi253d
umc21
umc21
hex2
ucsd78a ucsd64e
6.06
umc369
bnl3.03
umc170(rab17)
rab17 ncr(sod3a) mth(me2)
umc46
npi560 bnl15.37 umc46
umc173(pdk1)
npi252 npi616
pdk1
6.07
umc137
bnl5.47 npi301b npi608
bnl8.06b
umc38
umc38
umc160a
6.08
bnl8.08J npi280 koln1b
umc132
npi419
bnl17.12
umc62
6.09
umc62
umc132
idh2
idh2 npi597a npi561
6.10
mdh2
mdh2 ume133
php20599
umc134
ufg(agp1) umc28
umc346
6.11
TB-6Sa
TB-6Ld
TB-6Lc
TB-6Lb
adk1 adenylate kinase
gpc2 GAPDHase
po1 polymitotic
mdm1 MDMV resistance
wsm1 WSMV resistance
NOR nucleolus organizer
rhm1 H. maydis resistance
rgd1 ragged seedling
mn3 miniature seed
pgd1 phosphogluconate dehydrogenase
enp1 endopeptidase
w15 white
l12 luteus
y1 white endosperm
pb4 plebald leaves
l10 luteus
wi1 wilted plant
si1 silky ears
pb1 plebald leaves
ms1 male sterile
l15 luteus
pg11 pale green
dt2 dotted
pl1 purple plant - blotched
su2 sugary
sm1 salmon silks
hex2 hexokinase
pl1 polytypic ear
me2 malic enzyme
py1 pygmy plant
w14 white
w1 white
idh2 isocitrate dehydrogenase
mdh2 malate dehydrogenase
agp1 ADPG pyrophosphorylase
dek28 defective kernel
hcf5 high chlorophyll fluorescence
hcf26 high chlorophyll fluorescence
hcf323 high chlorophyll fluorescence
l11 luteus
oro1 orobanche
dep1 defective pistils
les13 lesion
ln1 linoleic acid
ms21 male sterile
afd1 absence of first division
cps2 chloroplast protein synthesis
dek19 defective kernel
gs3 green stripe
hcf34 high chlorophyll fluorescence
hcf36 high chlorophyll fluorescence
hcf48 high chlorophyll fluorescence
hcf408 high chlorophyll fluorescence
o14 opaque
rd2 reduced plant
sbd1 sunburned leaves
sr4 striate leaves


0 umc7STELO
ucsd106b
rs1 pge5c
26 php20581b
php20581a
7.00
35 npi400a
npi400a
o2 npi480
41 umc312
npi596
ast(dcm1) npi361b
mpik4a
ufg(inv1a) zpl2b rny(pcr4)
7.01/7.02 51 umc193c(orp)
bnl17.13a npi600
ucsd81c
umc(nabp1) npi361e
npi367a php20690b
npi568
npi111
bnl15.40
zpb36
66 umc136
umc5b umc98b
69 npi112
npi112
70 umc116
umc116
bnl15.21 npi122
php20569a
7.03
npi394
96 umc110
npi389 npi455 umc110
bnl4.24 chs(tyk30)
7.04
ast(amy3) bnl13.24 npi283
umc125b
bnl6.27 npi435 bnl5.21a
103 bnl7.61
bnl6.61a npi240 npi263
104 bnl8.32 npi217 npi352
105 bnl14.07
npi413b pge3 npi398
e1
php20563
111 umc304
isc(b32b) ncr(b32c3b)
bnl8.21a bnl7.61 bnl14.07
bnl8.37
7.05
bnl8.39
npi300a
ncr(sod2)
131 ztda30
npi385
umc151
npi380 npi433
139 php20593
php20523
143 php20690a
npi113
bnl16.06
php20690a
145 umc45
umc45
bnl16.06
bnl8.44
7.06
158 npi611
php20728
159 php20020
npi611
160 umc168
npi337a umc168
165 umc35
umc35
7.07
175 umc7LTELO
.9
.8
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.6
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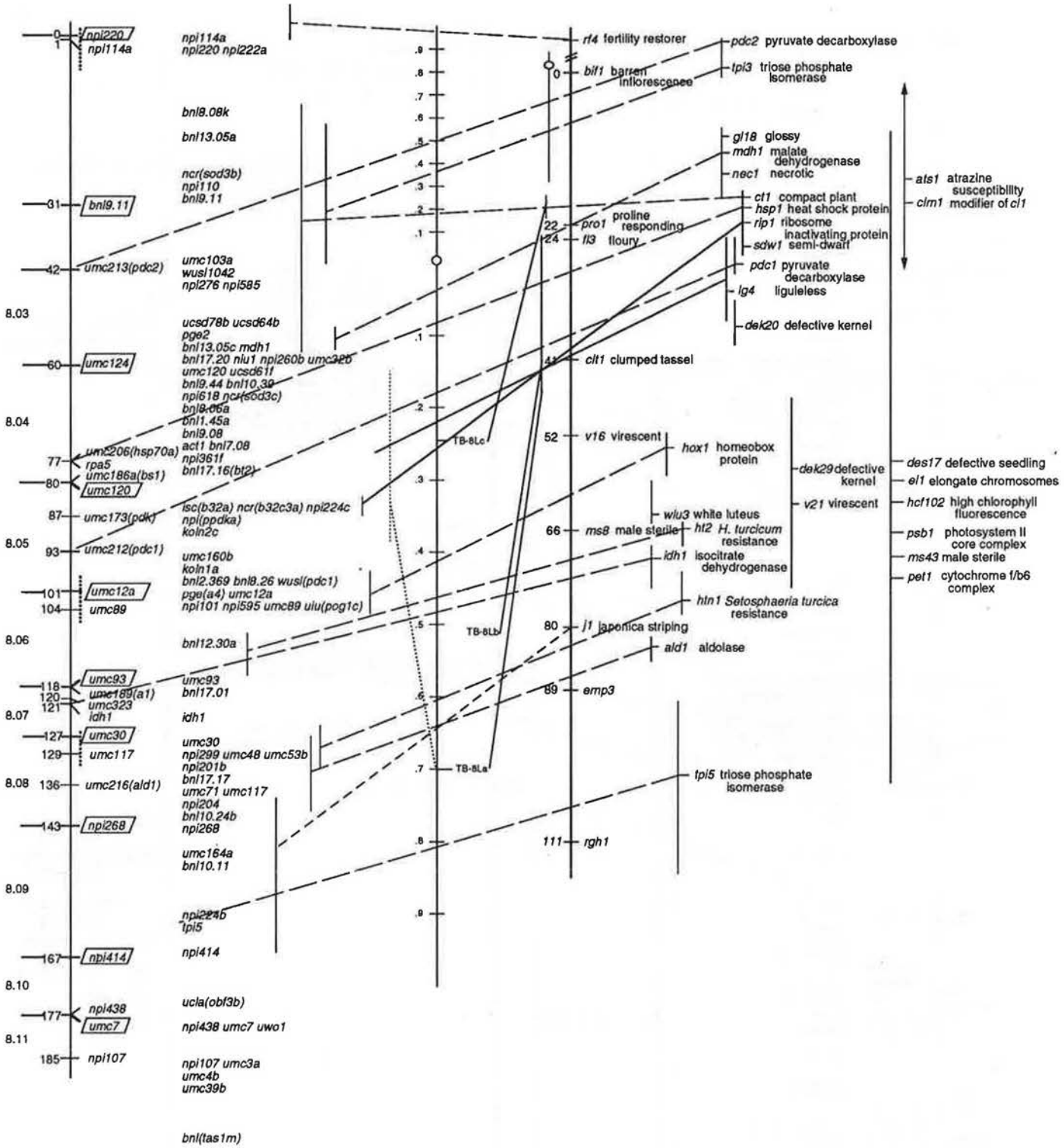
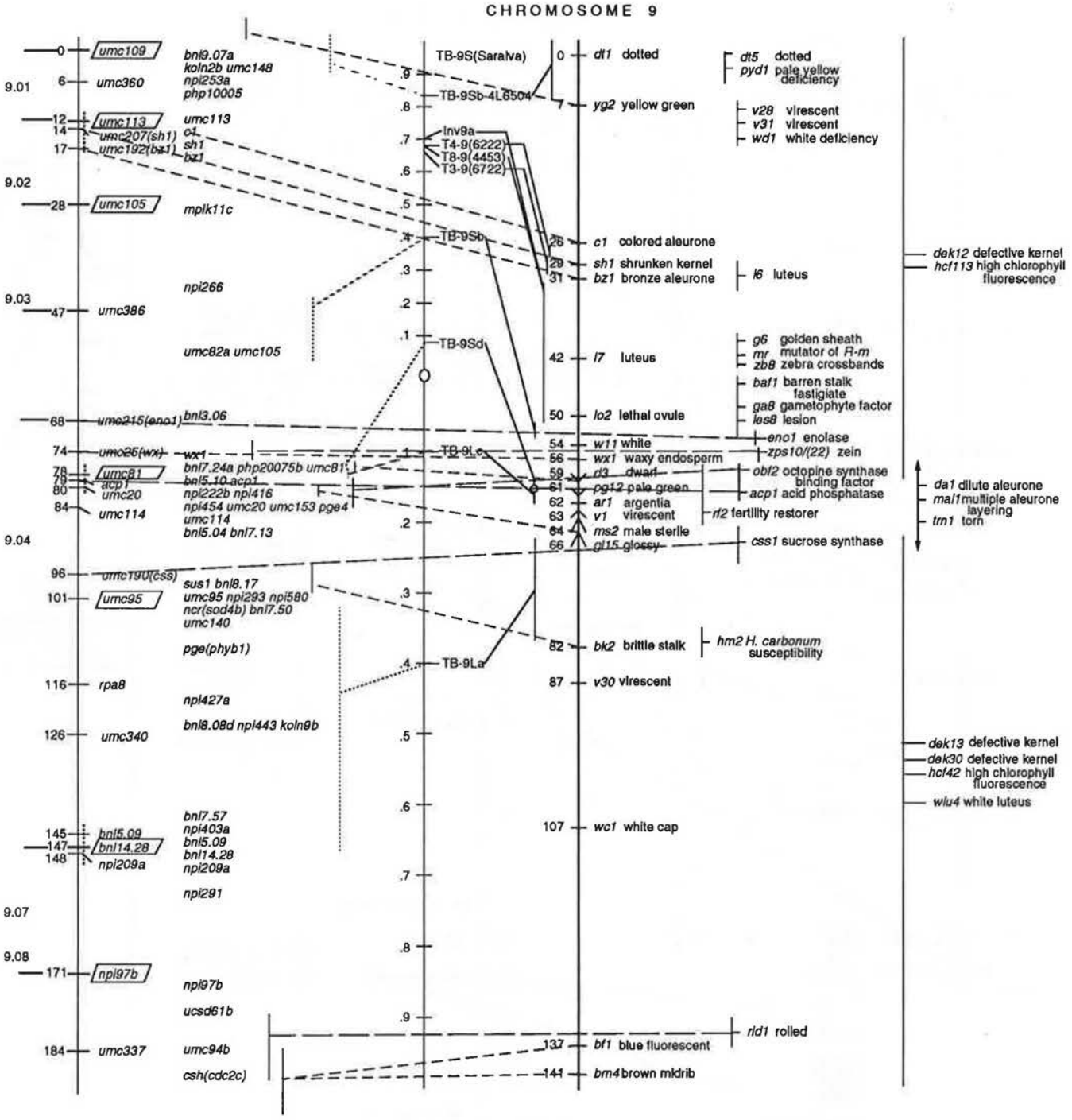
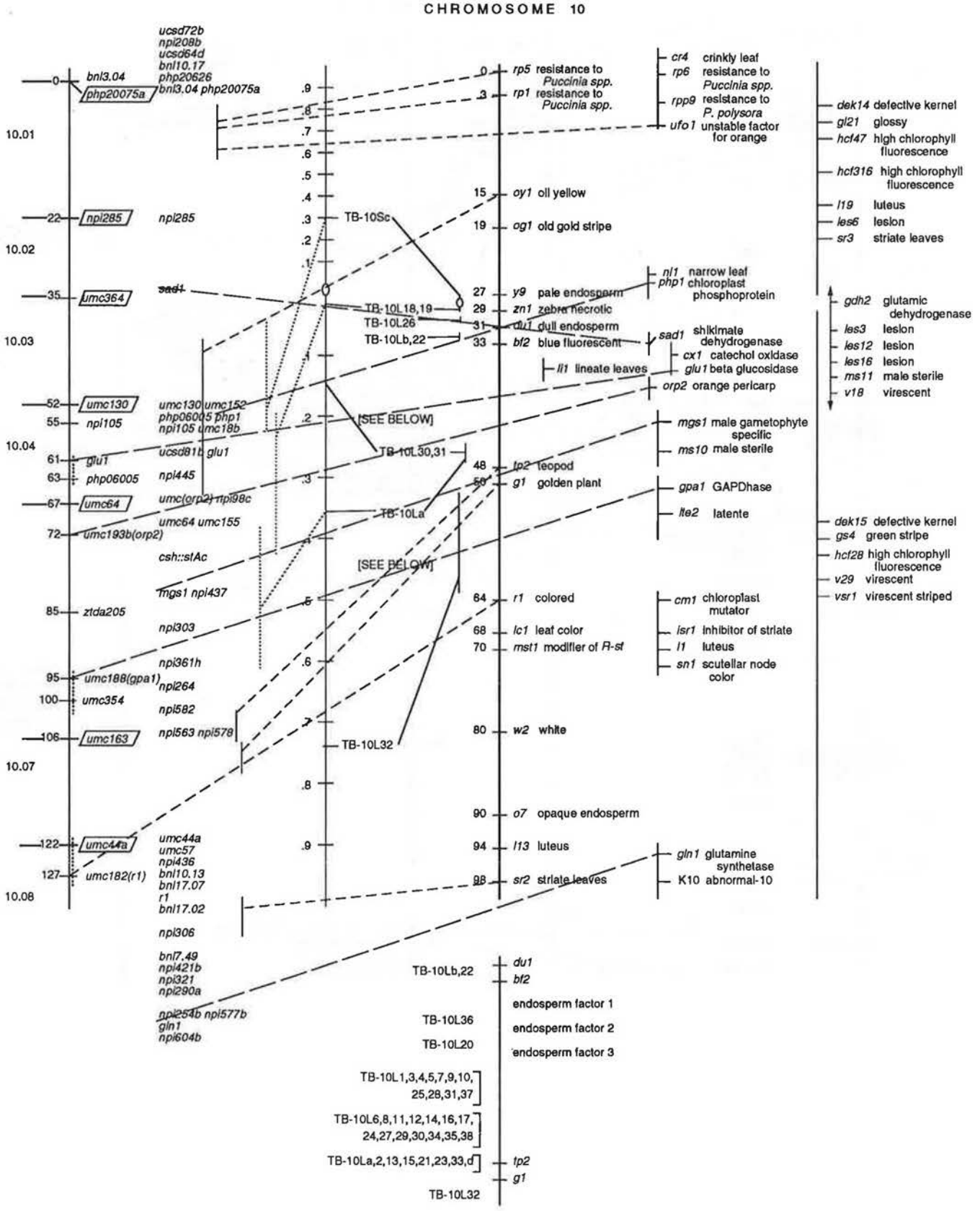
.4
.3
.2
.1
.0
.1
.2
.3
.4
.5
.6
.7
.8
.9
TB-7Sc
TB-7Lb
TB-7Lb-4L4698
0 hs1 hairy sheath
rs1 rough sheath
w17 white
ask1 aspartate kinase
pcr4 protochlorophyllide reductase
16 o2 opaque
zpl2b zein polypeptides
18 y8 pale endosperm
20 in1 intensifier
cp1 collapsed
cp2 collapsed
24 v5 virescent
nabp1 nucleic acid binding protein
25 vp9 viviparous
rcm1 rectifier
rs4 rough sheath
w16 white
les9 lesion
32 ra1 ramosa
ms7 male sterile
o5 opaque
wyg1 white yellow-green
36 gl1 glossy
zpb36 zein polypeptides
46 tp1 teopod
50 sl1 slashed
52 lj1 iojap
va1 variable sterile
e1 esterase
59 tpi1 triosa phosphate isomerase
ren2 reduced endosperm
sod2 superoxide dismutase
71 bn1 brown aleurone
109 bd1 branched silkless
112 pn1 papyrescent glumes
px3 peroxidase
sh6 shrunken kernel
mn2 miniature seed
pep4 PEP carboxylase
sen2 soft endosperm
dt3 dotted
hcf101 high chlorophyll fluorescence
hcf103 high chlorophyll fluorescence
hcf104 high chlorophyll fluorescence
hcf111 high chlorophyll fluorescence
ptd2 pitted endosperm
v27 virescent
wlu2 white luteus


0 npi220
1 npi114a
31 bnl9.11
42 umc213(pdc2)
60 umc124
77 umc206(hsp70a)
rpa5
80 umc186a(bs1)
umc120
87 umc173(pdk)
93 umc212(pdc1)
101 umc12a
104 umc89
118 umc93
120 umc189(a1)
121 umc323
127 umc30
129 umc117
136 umc216(ald1)
143 npi268
167 npi414
177 npi438
umc7
185 npi107
8.03
8.04
8.05
8.06
8.07
8.08
8.09
8.10
8.11
npi114a
npi220 npi222a
bnl8.08k
bnl13.05a
ncr(sod3b)
npi110
bnl9.11
umc103a
wusl1042
npi276 npi585
ucsd78b ucsd64b
pge2
bnl13.05c mdh1
bnl17.20 niu1 npi260b umc32b
umc120 ucsd61f
bnl9.44 bnl10.39
npi618 ncr(sod3c)
bnl8.06a
bnl1.45a
bnl9.08
act1 bnl7.08
npi361f
bnl17.16(bt2)
isc(b32a) ncr(b32c3a) npi224c
npi(ppdka)
koln2c
umc160b
koln1a
bnl2.369 bnl8.26 wusl(pdc1)
pge(a4) umc12a
npi101 npi595 umc89 ulu(pog1c)
bnl12.30a
umc93
bnl17.01
umc323
idh1
umc30
npi299 umc48 umc53b
npi201b
bnl17.17
umc71 umc117
npi204
bnl10.24b
npi268
umc164a
bnl10.11
npi224b
tpi5
npi414
ucla(obf3b)
npi438 umc7 uwo1
npi107 umc3a
umc4b
umc39b
bnl(tas1m)
.9
.8
.7
.6
.5
.4
.3
.2
.1
TB-8Lc
TB-8Lb
TB-8La
rf4 fertility restorer
0 bif1 barren inflorescence
22 pro1 proline responding
24 fl3 floury
41 clt1 clumped tassel
52 v16 virescent
66 ms8 male sterile
80 j1 japonica striping
89 emp3
111 rgh1
pdc2 pyruvate decarboxylase
tpi3 triose phosphate isomerase
gl18 glossy
mdh1 malate dehydrogenase
nec1 necrotic
ct1 compact plant
hsp1 heat shock protein
rip1 ribosome inactivating protein
sdw1 semi-dwarf
pdc1 pyruvate decarboxylase
lg4 liguleless
dek20 defective kernel
hox1 homeobox protein
wlu3 white luteus
ht2 H. turcicum resistance
idh1 isocitrate dehydrogenase
htn1 Setosphaeria turcica resistance
ald1 aldolase
tpi5 triose phosphate isomerase
dek29 defective kernel
v21 virescent
als1 atrazine susceptibility
clm1 modifier of cl1
des17 defective seedling
el1 elongate chromosomes
hcf102 high chlorophyll fluorescence
psb1 photosystem II core complex
ms43 male sterile
pet1 cytochrome f/b6 complex


CHROMOSOME 9
umc109
bnl9.07a
koln2b umc148
9.01
6 umc360
npi253a
php10005
12 umc113
14 umc207(sh1)
17 umc192(bz1)
umc113
c1
sh1
bz1
9.02
28 umc105
mpik11c
TB-9S(Saralva)
TB-9Sb-4L6504
Inv9a
T4-9(6222)
T8-9(4453)
T3-9(6722)
TB-9Sb
TB-9Sd
TB-9Le
TB-9La
0 dt1 dotted
7 yg2 yellow green
26 c1 colored aleurone
29 sh1 shrunken kernel
31 bz1 bronze aleurone
42 l7 luteus
50 lo2 lethal ovule
54 w11 white
56 wx1 waxy endosperm
59 d3 dwarf
61 pg12 pale green
62 ar1 argentia
63 v1 virescent
64 ms2 male sterile
66 gl15 glossy
82 bk2 brittle stalk
87 v30 virescent
107 wc1 white cap
137 bf1 blue fluorescent
141 bm4 brown midrib
dt5 dotted
pyd1 pale yellow deficiency
v28 virescent
v31 virescent
wd1 white deficiency
l6 luteus
g6 golden sheath
mr mutator of R-m
zb8 zebra crossbands
baf1 barren stalk fastigiate
ga8 gametophyte factor
les8 lesion
eno1 enolase
zps10/(22) zein
obf2 octopine synthase binding factor
acp1 acid phosphatase
rf2 fertility restorer
css1 sucrose synthase
hm2 H. carbonum susceptibility
rld1 rolled
dek12 defective kernel
hcf113 high chlorophyll fluorescence
da1 dilute aleurone
mal1 multiple aleurone layering
tm1 torn
dek13 defective kernel
dek30 defective kernel
hcf42 high chlorophyll fluorescence
wlu4 white luteus
9.03
47 umc386
npi266
umc82a umc105
68 umc215(eno1)
bnl3.06
74 umc25(wx)
wx1
78 umc81
79 acp1
80 umc20
84 umc114
bnl7.24a php20075b umc81
bnl5.10 acp1
npi222b npi416
npi454 umc20 umc153 pge4
umc114
bnl5.04 bnl7.13
9.04
96 umc190(css)
sus1 bnl8.17
101 umc95
umc95 npi293 npi580
ncr(sod4b) bnl7.50
umc140
pge(phyb1)
116 rpa8
npi427a
126 umc340
bnl8.08d npi443 koln9b
bnl7.57
145 bnl5.09
147 bnl14.28
148 npi209a
npi403a
bnl5.09
bnl14.28
npi209a
npi291
9.07
9.08
171 npi97b
npi97b
ucsd61b
184 umc337
umc94b
csh(cdc2c)


CHROMOSOME 10
ucsd72b
npi208b
ucsd64d
bnl10.17
php20626
bnl3.04 php20075a
bnl3.04
php20075a
10.01
npi285
10.02
umc364
10.03
umc130
npi105
10.04
glu1
php06005
umc64
umc193b(orp2)
ztda205
umc188(gpa1)
umc354
umc163
10.07
umc44a
umc182(r1)
10.08
umc130 umc152
php06005 php1
npi105 umc18b
ucsd81b glu1
npi445
umc(orp2) npi98c
umc64 umc155
csh::stAc
mgs1 npi437
npi303
npi361h
npi264
npi582
npi563 npi578
umc44a
umc57
npi436
bnl10.13
bnl17.07
r1
bnl17.02
npi306
bnl7.49
npi421b
npi321
npi290a
npi254b npi577b
gln1
npi604b
TB-10Sc
TB-10L18,19
TB-10L26
TB-10Lb,22
[SEE BELOW]
TB-10L30,31
TB-10La
[SEE BELOW]
TB-10L32
rp5 resistance to Puccinia spp.
rp1 resistance to Puccinia spp.
oy1 oil yellow
og1 old gold stripe
y9 pale endosperm
zn1 zebra necrotic
du1 dull endosperm
bf2 blue fluorescent
li1 lineate leaves
orp2 orange pericarp
tp2 teopod
g1 golden plant
r1 colored
lc1 leaf color
mst1 modifier of R-st
w2 white
o7 opaque endosperm
l13 luteus
sr2 striate leaves
cr4 crinkly leaf
rp6 resistance to Puccinia spp.
rpp9 resistance to P. polysora
ufo1 unstable factor for orange
nl1 narrow leaf
php1 chloroplast phosphoprotein
sad1 shikimate dehydrogenase
cx1 catechol oxidase
glu1 beta glucosidase
mgs1 male gametophyte specific
ms10 male sterile
gpa1 GAPDhase
lte2 latente
cm1 chloroplast mutator
isr1 inhibitor of striate
l1 luteus
sn1 scutellar node color
gln1 glutamine synthetase
K10 abnormal-10
dek14 defective kernel
gl21 glossy
hcf47 high chlorophyll fluorescence
hcf316 high chlorophyll fluorescence
l19 luteus
les6 lesion
sr3 striate leaves
gdh2 glutamic dehydrogenase
les3 lesion
les12 lesion
les16 lesion
ms11 male sterile
v18 virescent
dek15 defective kernel
gs4 green stripe
hcf28 high chlorophyll fluorescence
v29 virescent
vsr1 virescent striped
TB-10Lb,22
du1
bf2
endosperm factor 1
TB-10L36
endosperm factor 2
TB-10L20
endosperm factor 3
TB-10L1,3,4,5,7,9,10, 25,28,31,37
TB-10L6,8,11,12,14,16,17, 24,27,29,30,34,35,38
TB-10La,2,13,15,21,23,33,d
tp2
g1
TB-10L32

GENETIC MAP OF THE *ZEA MAYS* PLASTID CHROMOSOME

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The complete sequences of a number of 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.

See the 1987-1992 News Letters for descriptions of other sequenced genes: **MNL 62**:148; **MNL 63**:155; **MNL 64** :164; **MNL 65**:164; and **MNL 66**:160.

References:

Kangasjärvi, J., A. McCullough and B.S. Gengenbach. 1991. Nucleotide sequence and transcription of maize plastid genome *Bam*HI fragment 14 containing ORF170. **Plant Mol. Biol. 17:** 513-515.

Kanakari, S., G. Timmler, K. von Knoblauch and A.R. Subramanian. 1992. Nucleotide sequence, map position and transcript pattern of the intron-containing gene for maize chloroplast ribosomal protein S16. **Plant Mol. Biol. 18**: 419-422.

Maier, R.M. , B. Hoch, P. Zeltz and H. Kössel. 1992. Internal editing of the maize chloroplast *ndh*A transcript restores codons for conserved amino acids. **Plant Cell 4**: 609-616.

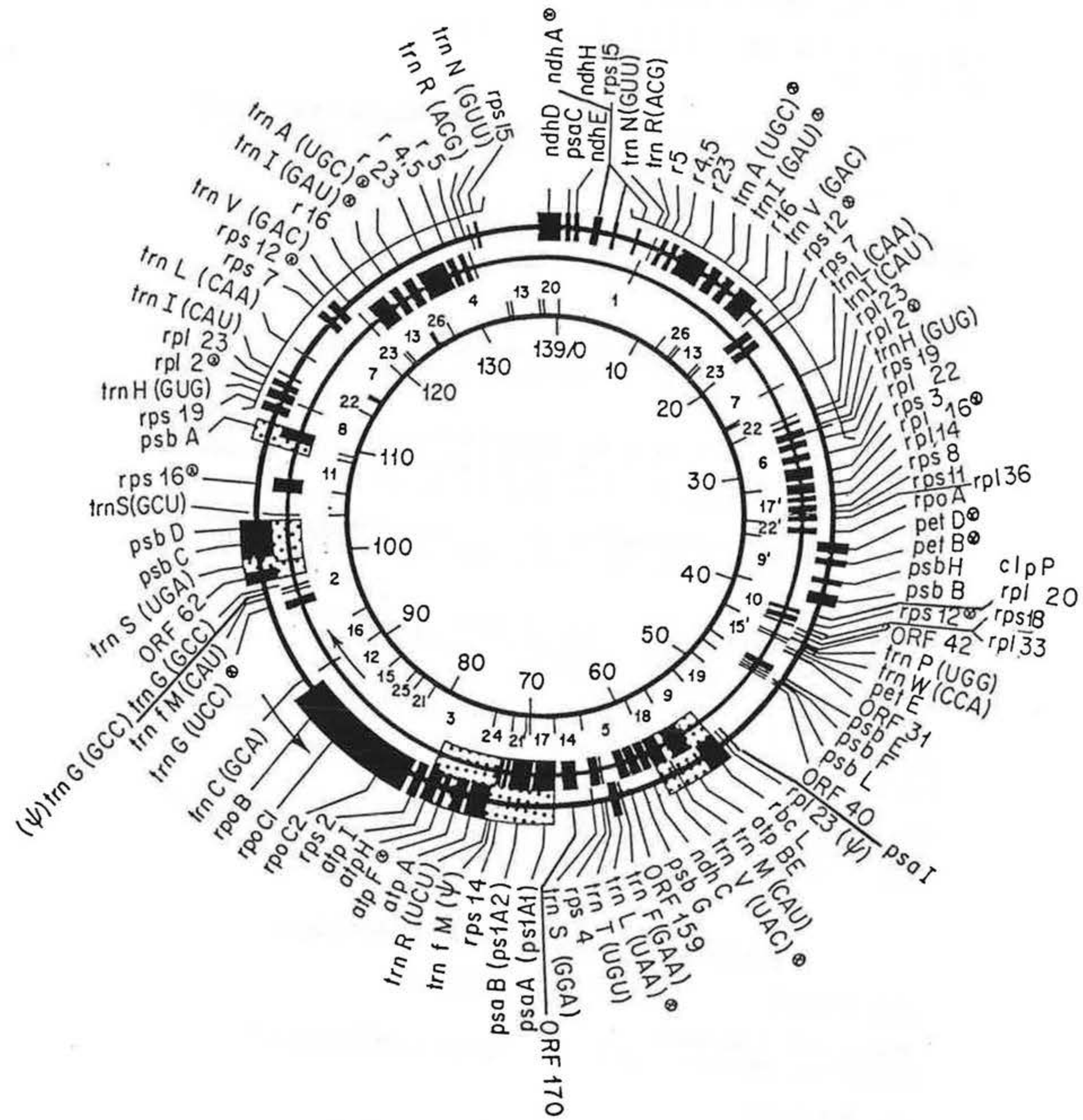
Rodermel, S.R. 1993. Nucleotide sequence of a maize chloroplast DNA fragment containing an inversion breakpoint, *trn*G (GCC), *trn*G (UCC), *trn*fM, and a *trn*G pseudogene. **Nucleic Acids Res. 20**: 5844.

Rodermel, S.R. and L. Bogorad. 1992. Nucleotide sequence of the photoregulated maize chloroplast *psa*I gene, encoding a 4.0-kilodalton component of photosystem I. **Plant Physiol. 98**: 406-407.

Weglöhner, W. and A.R. Subramanian. 1992. Nucleotide sequence of a region of maize chloroplast DNA containing the 3' end of *clp*P, exon 1 of *rps*12 and *rpl*20 and their cotranscription. **Plant Mol. Biol. 18**: 415-418.

RECENTLY-REPORTED MAIZE PLASTID GENES

Gene Product	Gene	Reference
Photosystem I proteins:		
4.0 kD hydrophobic core	*psa*I	Rodermel and Bogorad, 1992
70S Ribosomal Proteins:		
S16	*rps*16	Kanakari *et al.*, 1992
S12	*rps*12	Weglöhner and Subramanian, 1992
L20	*rpl*20	Weglöhner and Subramanian, 1992
tRNAs:		
Glycine	*trn*G (GCC)	Rodermel, 1993
Glycine	*trn*G (UCC)	Rodermel, 1993
Methionine (initiator)	*trnfM*	Rodermel, 1993
Glycine (pseudogene)	y*trn*G	Rodermel, 1993
NADH dehydrogenase subunit proteins:		
Homolog of the ND1 subunit of the mammalian mitochondrial NADH-ubiquinone reductase	*ndh*A	Maier *et al.*, 1992
Other:		
Proteolytic subunit of an ATP-dependent protease	*clp*P	Weglöhner and Subramanian, 1992


ndhD
ndhA
psaC
ndhH
ndhE
rps15
trn N(GUU)
trn R(ACG)
r5
r4.5
r23
trn A (UGC)
trn I (GAU)
r16
trn V (GAC)
rps12
rps 7
trn L (CAA)
trn I (CAU)
rpl 23
rpl 2
trn H (GUG)
rps 19
rpl 22
rps 3
rpl 16
rpl 14
rps 8
rps 11
rpl 36
rpo A
pet D
pet B
psb H
clp P
psb B
rpl 20
rps 12
rps 18
ORF 42
rpl 33
trn P (UGG)
trn W (CCA)
pet E
ORF 31
psb E
psb F
psb L
ORF 40
rpl 23 (ψ)
psa I
rbc L
atp BE
trn M (CAU)
trn V (UAC)
ndh C
psb G
ORF 159
trn F (GAA)
trn L (UAA)
trn T (UGU)
rps 4
trn S (GGA)
ORF 170
psa A (ps1A1)
psa B (ps1A2)
rps 14
trn f M (ψ)
trn R (UCU)
atp A
atp F
atp H
atp I
rps 2
rpo C2
rpo C1
rpo B
trn C (GCA)
(ψ) trn G (GCC)
trn G (UCC)
trn f M (CAU)
trn G (GCC)
ORF 62
trn S (UGA)
psb C
psb D
trnS(GCU)
rps 16
psb A
rps 15
trn N (GUU)
trn R (ACG)
r 5
r 4.5
r 23
trn I (GAU)
trn A (UGC)
r 16
trn V (GAC)
rps 12
rps 7
trn L (CAA)
trn I (CAU)
rpl 23
rpl 2
trn H (GUG)
rps 19
139/0
10
20
30
40
50
60
70
80
90
100
110
120
130

VI. Maizedb: MAIZE GENOME DATABASE

One of the key developments in the Plant Genome Initiative is the design and implementation of a database and network system for genetic data, analysis of data, and linked access to sequences, clones, biosynthetic pathways, and the like, across species boundaries. In addition to its grants through the Competitive Grants Program of Cooperative States Research Service, the Initiative supports database development through the Agricultural Research Service; both are branches of the U.S. Dept. of Agriculture. A Plant Genome Database is being derived by "Prototype Developers" working first on maize, soybean, wheat, forest trees, and *Arabidopsis*, soon to be followed up with other species. The structure will be inclusive of higher plant data and is to be focused at the National Agricultural Library (NAL). The Newsletter "Probe", available from Plant Genome Data and Information Center, USDA - National Agricultural Library, 10301 Baltimore Blvd. Room 1402, Beltsville, MD 20705-2351, offers coverage of current issues, descriptions of developing programs, and updates on plant genome activities. Expectation is that full, on-line access to plant genome databases will be available by the latter half of 1993 in their first implementations. The Maize Genome Database is a developing prototype in this network. This is the second report on development and progress of Maizedb; the first report was presented in MNL 66:162-163.

The Working Group (Ed Coe, Mary Berlyn, Stan Letovsky, Mary Polacco, Marty Sachs, Denis Hancock) has been enhanced by the addition during the past year of Pat Byrne and Georgia Davis (Georgia Yerk). Version 1 of the relational database, having served its purpose, is being replaced by Version 2 at the time of this writing. The Gene List and Stock List this year were derived by output from Maizedb, and the contents of Zealand 93 are being incorporated into the database itself.

The Nomenclature and Standards Committee, chaired by Oliver Nelson, has completed revisions for nomenclature and standards, which are reproduced after this section of the Newsletter.

A demonstration of the Maizedb Prototype was given in the Maize Genetics Conference at Asilomar in March, 1992, in the Plant Genome Symposium in San Diego in the fall of 1992, and in the Maize Genetics Conference at Pheasant Run, in March, 1993. Version 2 development was greatly aided by comments and responses from the 1992 and 1993 demonstrations, and on experience with the implementation before and after the demos.

The content of Maizedb can now be accessed via Gopher, as described on the following page. This is a simple and convenient means by which to look up information in the database, but is in an experimental phase and should be expected to change, hopefully for the better, without fanfare. Gopher is itself a developing tool, from which not all performances have an obvious explanation. The Maizedb Group asks not only your patience as refinements are developing, but your corrections, suggestions and ideas as they arise from using the database.

Copies of a guide that was distributed at the 1993 Maize Genetics Conference, "HOW YOU CAN HELP BY REPORTING INFORMATION THAT WILL FACILITATE INCORPORATION INTO THE MAIZE DATABASE", accompanied by the Nomenclature Standard, are available on request to Ed Coe or any of the other members of the Maizedb working group.

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ACCESS TO MAIZE GENOME DATA IS AVAILABLE THROUGH GOPHER

The Maize Genome Database, which has been under development since January of 1991, is available for information retrieval through a convenient software tool called Gopher. The Gopher protocol was developed at the University of Minnesota, and through a network of information servers and links, provides access to hundreds of locations where data are made available.

To obtain such information, you need to have some kind of connection, either direct or indirect, to the Internet. This can be through a direct connection of your computer to your institutional backbone, which then is connected to the Internet, or it can be through a modem that connects to a computer that is, in turn, connected to the Internet.

If you are directly connected to the Internet you will need to obtain a Gopher client program. If you are not directly connected to the Internet, but have a modem, you will need to dial into a machine that is connected. The best results will be obtained if you can emulate a vt100 terminal. These Gopher client packages are all free, as is the server software, should you choose to set up an information server of your own.

For Unix machines, have your system administrator (or a friendly hacker) obtain and compile a gopher client. This can be obtained through anonymous ftp from teosinte.agron.missouri.edu and is found in the /pub/gopher_stuff directory. The latest versions may always be obtained by anonymous ftp from boombox.micro.umn.edu.

For Macintosh computers, the University of Minnesota has developed an excellent application called TurboGopher. It can also be obtained from boombox.micro.umn.edu.

For MSDOS-compatible computers, client software is available from boombox.micro.umn.edu, but we have not tested it, so we are unable to make a recommendation.

For those unfamiliar with anonymous ftp, you type "ftp teosinte.agron.missouri.edu" at the command line (on a networked Unix machine), or use the appropriate Macintosh utility (Fetch or Xferit). Log in using the user name "anonymous" and give your email address as a password. Type "cd /pub/gopher_stuff" and press the return key. Type "dir <return>" and you will see a list of filenames. Type "binary <return>" and then "get <filename> <return>". Within several seconds the file should be residing on your local hard drive.

Once you have installed the software, it is a simple matter to direct it to a gopher server. For Unix, type "gopher teosinte.agron.missouri.edu 70" on the command line, and in a second or two the main menu from the MaizeDB server will appear. For Macintoshes, simply double-click the TurboGopher icon. From there, things should be self-explanatory. When you see an information file, please take the time to read it, as it has important information and helps for you. Navigation through the menus can be accomplished through arrow keys and the return key. Both the Unix and Macintosh versions have help facilities.

A special note for Macintosh users: TurboGopher, as distributed, will go to the University of Minnesota gopher by default. You can change that by going into the Setup menu and selecting 'Configure TurboGopher...". Just set the Internet host to "teosinte.agron.missouri.edu" or "128.206.11.1" if you don't have name service, and you will connect to the MaizeDB gopher by default. There are two lines for gopher hosts, and TurboGopher will alternately call those two hosts. If you desire, make the two lines the same, and you will always get the one you want.

Your system administrator can modify both the Unix and Macintosh versions so they automatically point to the MaizeDB gopher; otherwise they will probably point to the University of Minnesota gopher. The MaizeDB gopher has links to other plant genome gophers as well as to the Gopher at U of M.

The MaizeDB gopher is available to all interested researchers and there are no charges for its use. Our sole condition is that if you see something that needs correction or improvement, you will let us know. Keep in mind that Gopher is a relatively new protocol, and improvements and bug fixes are constantly being made. In addition, we are constantly improving the way we present information, so things may appear to be different each time you log on.

The maize genetic information that is presented in the MaizeDB Gopher is extracted every day from a large relational database, and thus is up-to-date. We do not present all permutations of the data, but if there are data you would like to see presented in a particular way, please let us know.

At present, Gopher allows text searches of the portions of the Maize Genome Database documenting sites, mutations, stocks, maps, and people. For example, one could search for the site "ht1" or the gene product "chitinase" under Sites; the inbred line "W22" under Stocks; or researchers named "Smith" under People. Gopher will respond with a list of "documents" containing the specified text, which can then be viewed one by one.

For those who require more advanced access to the Maize Genome Database, it continues to be under development, and we expect that it will be available for direct browsing during the last half of calendar year 1993. This will require more sophisticated and possibly expensive software (e.g. X-Windows server, query generators, etc.) to access the database efficiently. It will also require us to add you as a user on our system. A few test locations are already in place.

Contacts:

Denis Hancock (dhancock@teosinte.agron.missouri.edu) or Ed Coe (ed@teosinte.agron.missouri.edu)

Faxed information and queries may be sent to (314) 874-4063. If you are unable to use the addresses as given, teosinte.agron.missouri.edu is also known as 128.206.11.1.

A STANDARD FOR MAIZE GENETICS NOMENCLATURE

PREAMBLE: We wish to have a system that is consistent, compatible with the historical background of maize genetics (insofar as these two goals can be reconciled), is easily understood by plant geneticists working with other species, and forms the basis for the importation of maize data into a general plant genetics data base so that the basic knowledge concerning maize genes is available to researchers with other species and *vice versa*. We believe that this goal is best implemented by the researchers in each species having their own working vocabulary, while the identification of genes that catalyze the same functions in all species should rely on entry into a relational data base of the genes' function as an E.C. number (2.4.1.13), trivial name (sucrose synthase), and systematic name (UDPglucose:D-fructose 2-glucosyltransferase). The situation can be less completely categorized for genes whose products are transcription factors, structural proteins, storage proteins, etc.

If one accepts the premise outlined above that the common ground between species need not reside in the working vocabulary of geneticists using any species as a model system but in the manner in which their data are expressed in the data base, then the previously adopted names for maize genes can be retained. It will not be necessary to rename the genes previously named on the basis of the mutant phenotype produced as soon as the function of the nonmutant alleles becomes known, but we should proceed to define more precisely words or terms whose meanings need clarification and to decide how we wish to deal with the new information becoming available.

1. DEFINITIONS: The words "locus" and "gene" should not be treated as synonymous. A locus can be defined as "a chromosomal site of variable size at or within which is located a gene, a restriction site, a knob, a breakpoint, an insertion, or other distinguishable feature". This necessitates specifying whether we mean a gene locus or an RFLP locus, *etc.* We can then define a plant gene as "a DNA sequence of which a segment is regularly or conditionally transcribed at some time in either or both generations of the plant. The DNA is understood to include not only the exons and introns of the structural gene but the *cis* 5' and 3' regions in which a sequence change can affect gene expression". This treats the gene as a functionally defined entity that is not circumscribed by the transcribed region or other fixed limits.

2. ANONYMOUS TRANSCRIPTS: For most of the history of genetics, the existence of a gene was recognized when a mutation occurred, and the gene was then named by a word/term that was descriptive of the mutant phenotype. That will continue to be the practice except with isozyme markers, for which the designation will be the enzyme in question, or the instances in which the biochemical lesion responsible for the mutant phenotype is identified before the locus is reported. The loci of these genes have then been placed on chromosome maps in relation to other mapped loci. However, we now have the possibility of recognizing genes in which no mutation has occurred through the construction of cDNA libraries. These anonymous cDNAs are often used as probes in RFLP mapping. When such a probe hybridizes to a single band, it is clear that the RFLP loci circumscribe the transcriptional unit that encodes the message represented by the cDNA, and these RFLP loci with other RFLP loci can be used as the basis for mapping the gene. Mapping a locus in this fashion is encouraged as a means of obtaining maximum coverage of the genome. As long as the locus retains an anonymous status (unknown function or no mutant phenotype), the symbol for the locus should be assigned according to the convention used for RFLP loci (as *umc148*, see Section 8) but with the letters *gfu* in parentheses after the RFLP designation to make it clear that this is the location of a *gene, function unknown*; further information about the probe and its derivation is best provided in tabular or data base form rather than in the symbol itself.

A gene name identifying function for a locus detected with a cloned sequence should be given only when there is unambiguous evidence that this is the site by which that function is encoded. Particular caution should be taken in identifying genes (and their function) from several RFLPs hybridizing to a gene-specific probe from another organism. Until a sequence has been shown to encode the function in question, the gene designation should be that of an RFLP locus (see Section 8).

3. STANDARD NOMENCLATURE AND SYMBOLS: The names and symbols that have been used for maize genes should be retained. The name and symbol of a gene locus should be represented with lower-case, italic characters (*defective kernel12, dek12*). Note that no hyphen separates the gene name from a numerical suffix, which is a change from previous usage. We use a hyphen in the case of mutant alleles (or a + in the case of nonmutant alleles) to separate the allele designation from a suffix specifying the particular allele (see Section 5). We advocate strongly that all genes identified in the future be given a three letter symbol.

4. LOCI WITH THE SAME GENE NAME: Where we have more than one nonallelic mutant with the same gene name, the earlier recommendation was that the first one to receive that name should not have a numerical suffix but the second has 2 as a suffix. Thus we have *shrunken (sh), shrunken2 (sh2)*, and *shrunken4 (sh4)* mutants. Geneticists outside the maize community are apt to misinterpret this convention. We recommend that we be consistent and write *shrunken1* or *sh1* and advocate that even if a new locus is identified and given a unique name, it be designated as 1. This has the definite advantage in maintaining data bases and indices that no retrospective correction would be necessary if a second gene locus receives the same designation.

5. ALLELIC DESIGNATIONS: Where a mutant allele is recessive, it should be designated by an italicized symbol (lower case) as *dek12*, which is the same as the symbol of the locus. Since it is unlikely that any two mutant or nonmutant alleles in a highly polymorphic species such as maize have identical sequences, maize geneticists are encouraged to specify the particular allele with which they are working (see in this Section, Alleles of Independent Mutational Origin and Designation of Nonmutant Alleles). The symbol for dominant, nonmutant (i.e., conditioning a normal phenotype) alleles will be the same italicized three letter symbol as the mutant alleles but with the first letter capitalized (*Dek12*). The symbol of the gene product should not be italicized and should be written with all letters capitalized (e.g., ADH1). The name of the gene product (alcohol dehydrogenase) should neither be capitalized nor italicized.

When the mutant alleles of a gene are dominant, the first letter of the mutant symbol is capitalized. The nonmutant symbol has all the letters lower case. For example, the *corn grass1* (*cg1*) gene locus has several dominant mutant (*Cg1*) alleles as well as nonmutant (*cg1*) alleles. Potential confusion would be reduced if a nonmutant allele were symbolized as *cg1+W22*, where + indicates that this is a nonmutant allele and W22 the inbred from which his particular allele was derived. The reference mutant allele is designated as *Cg1-R* or *-1*.

Codominant alleles such as isozymes where the variants are functional and distinguished from each other by electrophoretic mobility, should be designated by symbols with the first letter capitalized and identified by allelic specifications as *Pgm2+5* or *Pgm2+7*.
The gene loci encoding transcription factors (e.g.: *b, r*) represent a special case since several functional, naturally occurring variants exist at each locus that condition the intense pigmentation of a different tissue or tissues than those pigmented by the most common functional allele. We suggest that these variants should have a + between the locus designation and the allelic specification. For example, we would then have *B+Bar*, and *B+Peru* as contrasted to *b-W23*, which makes no visible pigment, and *b-weak*, which weakly pigments a few tissues but not most.

It is not possible to anticipate all the instances in which one might be in doubt as to whether a particular allelic specification should be preceded by a + or a -. These instances will usually arise when a researcher is making an intensive study of the allelic variation (natural and induced) at a locus, and that person is in the best position to make the assignment. Another possibility is to refer the question to the proposed Nomenclature Clearing House (see section 11).

ALLELES OF INDEPENDENT MUTATIONAL ORIGIN: The unambiguous designation of mutant alleles that have arisen as independent mutational events is increasingly important. It is generally understood that a gene symbol followed by a hyphen plus a letter or number(s) specifies a particular recessive allele at that gene locus. We have referred to the mutation by which the gene was identified as the reference allele; e.g. *bz1-Ref* or *bz1-R*. It is equally appropriate to refer to that allele as *bz1-1*. The mutations in any gene that were identified subsequently have been categorized in various idiosyncratic ways. Alleles that have arisen by independent mutational events have been designated by letters, numbers, a letter plus numbers, the name of the inbred in which the mutation occurred, and sometimes all of these applied to a group of alleles at a gene locus. While all of these designations served the purpose of indicating that these alleles had independent mutational origins, there is a clear advantage to greater standardization. As in the 1973 Nomenclature Standard, it is recommended that new alleles be identified by a laboratory number that might indicate the year of isolation as *sh2-6801*. This has the definite advantage that two laboratories are unlikely to designate two new mutations of the same gene by the same number. Also recommended is the convention of referring to a new mutation of a given phenotype by a provisional designation as *bt*-lab number* until it is ascertained whether the mutant is a new allele of a known gene or identifies a previously unidentified gene. In the first instance, the proper gene symbol (*bt1* or *sh2*) replaces *bt**, but the *lab number* is retained (e.g., *bt1-8711*). In the second instance (a previously unidentified locus), a new gene name and symbol would be selected, and this mutant would become the reference allele (-R or -1).

When mutant alleles are referred to in the generic sense without specification of their origin, a hyphen without further designation (e.g., *bz1-, dek12-*) is desirable to make it clear that one is referring to an allele or alleles, not the gene locus.

DESIGNATION OF NONMUTANT ALLELES: Since it is now apparent that in a species as polymorphic as maize, nonmutant alleles from different sources are apt to have a number of sequence differences one from the other, and these differences can be reflected in gene action (nonmutant isoalleles), it is desirable to specify the nonmutant allele being investigated or used as a control. Incorporating the name of the inbred as part of the allelic designation, *Bz1+W22*, is an appropriate method of doing this. However, mutant alleles should not be designated by the inbred in which they arose (e.g., *bz1-W22*) to avoid confusion with the progenitor allele. Also, there may eventually be numerous mutant alleles of a particular gene isolated in that inbred if a researcher uses that inbred in a mutagenesis experiment. A particular nonmutant allele may be found in an exotic race or other accession that is not an inbred. A unique designator (e.g., a PI number or Bolivia #) should be part of the allelic designation. A counterpart to the note in the section above about using a hyphen with no further designation following unspecified recessive alleles is to use a + for nonmutant alleles (e.g., the *Sh2+* alleles).

RFLPs AND RAPDs AS ALLELES: The presence or absence of a restriction site or a primer-amplifiable sequence at a particular locus represent Mendelian alternatives. They fall under the broadest definition of an allele, and it is appropriate to refer to these alternatives as alleles as has already been done in some reports.

6. NAMING DELETIONS: When it is clear that a mutation results from a deletion that has removed all or part of two gene loci, it would be appropriate to indicate this in the following manner. For *an1-6923*, this would be *def(an1..bz2)-6923*, and for *sh-bz-X2*, *def(bz1..sh1)-X2*. When molecular evidence indicates that a deletion has removed all of the structural portion of a gene as is true of *wx1-C34*, it should be indicated in the same manner; i.e., *def(wx1)-C34*.

7. MUTATIONS RESULTING FROM TRANSPOSABLE ELEMENT INSERTIONS: There is one further point concerning allelic specification. Maize in particular has many mutable alleles resulting from the insertion of a transposable element. These have been designated by the mutant symbol, a hyphen, a lower case "m", and an isolation number; e.g., *wx-m1*. When the transposable element insertion [*Ac, Ds, Spm(En), dSpm(I), Mu1..MuX*, etc.] is known, it is suggested that this be indicated by a double colon following the allele as *wx-m1::Ds1*. Since a maize stock may have more than one transposable element family active at the same time, firm genetic and/or molecular evidence is necessary to ascribe mutability to a particular transposable element family. Further, mutable alleles generate both stable nonmutant and stable mutant alleles when the transposable element excises from the gene locus. Since the mutant derivatives are certain to differ in sequence from the nonmutant progenitor allele around the site of the transposable element insertion and the nonmutant derivatives are very likely to differ at that site, researchers should be certain to indicate the origin of such alleles in their reports. One means of doing this is to indicate such an origin by an apostrophe following the locus symbol as *Bz1'+7801* or *bz1'-8905*. The specifics of its origin including the transposable element involved could then be included in the text and entered in the Maize Genome Data Base.
Since transpositions of a transposable element from a site within a gene often insert in locations where they have no phenotypic effect but can be useful markers, it is desirable to have a standard to refer to such insertions. Designate them as RFLP's would be designated (see Section 8), but follow the institutional symbol and number with a double colon and the symbol of the transposable element (e.g., *dnap2094::Ac*).

8. NAMING RFLPs AND RAPDS: In naming RFLPs and RAPDs, use a lower case three or four letter code designating the originating university or company followed by a laboratory number (no space between the code and the number). When the probe used is a cDNA or a

subclone of a gene, the gene symbol should be added in parentheses after the RFLP locus designation, as *umc000(a1)*. Since a probe not infrequently recognizes RFLPs on two or more chromosomes, these should be designated by the same institutional code, number, and probe followed immediately by A, or B, or C. In so far as possible, the locus with the strongest hybridization should be designated A and the more weakly hybridizing loci be designated B, C etc. in descending order of signal strength.

9. CHROMOSOME REARRANGEMENTS: The conventions for dealing with chromosomal rearrangements are well established and adequate for the purpose. To designate particular reciprocal translocations as T1-2a or T1-9(4995) etc. with the breakpoints noted parenthetically or in a table of supporting information is explicit and sufficient. Additional information (the fact that the translocation stock is homozygous for *wx1*) can be incorporated by prefacing the translocation number with the gene symbol as the Co-op does in its stock lists (e.g., *wx1* T1-9c). Translocations with B chromosomes have designations that indicate the arm of the A chromosome involved (L or S) as well as a lower case letter distinguishing that translocation from any others involving that particular chromosome arm, as TB-5Sc. The cytological breakpoint in the A chromosome as well as the loci uncovered when the TB translocation is used as a male parent can be noted in the text or in a table of supplementary information. The designations for inversions (e.g., Inv9b again with the breakpoints, 9S.05-L.87, listed in a supporting table) are succinct and convey the necessary information.

10. ORGANELLAR GENES: For chloroplast and mitochondrial genes, we accept for the present the proposals already in place. For chloroplast genes, this is Hallick and Bottomley, 1983. Plant Mol. Biol. Rep. 1(4): 38-43. For mitochondrial genes, this is Lonsdale and Leaver, 1988. *Ibid.* 6:14-21. For brevity's sake, these are not summarized here.

11. CLEARING HOUSE FOR NOMENCLATURE: We also believe that it is desirable to initiate a clearing house for maize nomenclature so that a researcher wishing to name a recently identified gene can ascertain almost immediately that no one has used the proposed designation and symbol. This clearing house can, in principle, function through the maize genome data base, which will be refereed by a cooperator. The same facility could be used to insure that allelic designations are not duplicated or to answer questions concerning nomenclature.

Submitted February 1, 1993 by the Nomenclature Subcommittee.

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VIII. RECENT MAIZE PUBLICATIONS

1. Abe, M; Abe, K; Kuroda, M; Arai, S, 1992. Corn kernel cysteine proteinase inhibitor as a novel cystatin superfamily member of plant origin - molecular cloning and expression studies. Eur J Biochem 209:933-937.
2. Abe, S; You, W; Davies, E, 1991. Protein bodies in corn endosperm are enclosed by and enmeshed in F-actin. Protoplasma 165:139-149.
3. Acevedo, A; Scandalios, JG, 1991. Catalase and superoxide dismutase gene expression and distribution during stem development in maize. Dev Genetics 12:423-430.
4. Adriaanse, FG; Human, JJ, 1992. A nitrogen-response comparison between semiprolific and non-prolific maize hybrids with regard to grain yield and plant nitrogen concentrations. Field Crop Res 30:53-61.
5. Aeschbacher, RA; Benfey, PN, 1992. Genes that regulate plant development. Plant Sci 83:115-126.
6. Afele, JC; Kannenberg, LW; Keats, R; Sohota, S; Swanson, EB, 1992. Increased induction of microspore embryos following manipulation of donor plant environment and culture temperature in corn (*Zea mays* L.). Plant Cell Tissue Organ Cult 28:87-90.
7. Agostino, A; Jeffrey, P; Hatch, MD, 1992. Amino acid sequence and molecular weight of native NADP malate dehydrogenase from the C4 plant *Zea mays*. Plant Physiol 98:1506-1510.
8. Ajala, SO, 1992. Combining ability for maturity and agronomic traits in some tropical maize (*Zea mays* L.) populations. Trop Agr 69:29-34.
9. Ajmone-Marsan, P; Livini, C; Messmer, MM; Melchinger, AE; Motto, M, 1992. Cluster analysis of RFLP data from related maize inbred lines of the BSSS and LSC heterotic groups and comparison with pedigree data. Euphytica 60:139-148.
10. Allard, RW, 1992. Predictive methods for germplasm identification. Pp. 119-146 in Plant Breeding in the 1990s. H. T. Stalker and J. P. Murphy, ed., Wallingford, UK: CAB Internatl.
11. Allen, RL; Lonsdale, DM, 1992. Sequence analysis of 3 members of the maize polygalacturonase gene family expressed during pollen development. Plant Mol Biol 20:343-345.
12. Altmann, T; Schmidt, R; Willmitzer, L, 1992. Establishment of a gene tagging system in *Arabidopsis thaliana* based on the maize transposable element *Ac*. Theor Appl Genet 84:371-383.
13. Amancio, S; Lopes, A; Santos, H; Rodrigues, M; Teixeira, A, 1991. Behaviour of inbred and hybrid maize lines in the presence of aluminium as evaluated by some physiological and biochemical parameters. Devel Plant Soil Sci 45:795-803.
14. Amoore, D, 1992. Barbara McClintock. The Times, September 5, 1992.
15. Anaya, AL; Hernandez-Bautista, BE; Jimenez-Estrada, M; Velascoibarra, L, 1992. Phenylacetic acid as a phytotoxic compound of corn pollen. J Chem Ecol 18:897-905.
16. Andre, C; Levy, A; Walbot, V, 1992. Small repeated sequences and the structure of plant mitochondrial genomes. Trends Genet 8:128-132.
17. Anonymous, 1992. Barbara McClintock. Agron News October 1992.
18. Anonymous, 1992. Barbara McClintock. Spectra: Newsl Carnegie Inst Wash November 1992:6-7.
19. Anonymous, 1992. Mcclintock,Barbara - obituary. Scientist 6:23.
20. Armstrong, CL; Romero-Severson, J; Hodges, TK, 1992. Improved tissue culture response of an elite maize inbred through backcross breeding, and identification of chromosomal regions important for regeneration by RFLP analysis. Theor Appl Genet 84:755-762.
21. Arnason, JT; Gale, J; deBeyssac, BC; Sen, A; Miller, SS; Philogene, BJR; Lambert, JDH; Fulcher, RG; Serratos, A; Mihm, J, 1992. Role of phenolics in resistance of maize grain to the stored grain insects, *Prostephanus truncatus* (Horn) and *Sitophilus zeamais* (Motsch). J Stored Prod Res 28:119-126.
22. Athma, P; Grotewold, E; Peterson, T, 1992. Insertional mutagenesis of the maize *P*-gene by intragenic transposition of *Ac*. Genetics 131:199-209.
23. Aung, LH; Fouse, DC; Harris, CM, 1992. Effect of postharvest desiccation at high temperature on soluble sugar changes of 2 supersweet sweet corn cultivars. J Hort Sci 67:745-750.
24. Avalkina, NA; Golubovskaya, IN; Peremislova, EE, 1992. *Mei* genes and female meiosis in maize. Genetika SSSR 28:130-141.
25. Azevedo, RA; Blackwell, RD; Smith, RJ; Lea, PJ, 1992. 3 aspartate kinase isoenzymes from maize. Phytochemistry 31:3725-3730.
26. Azevedo, RA; Smith, RJ; Lea, PJ, 1992. Aspartate kinase regulation in maize - evidence for co-purification of threonine-sensitive aspartate kinase and homoserine dehydrogenase. Phytochemistry 31:3731-3734.
27. Azevedo, RA; Smith, RJ; Lea, PJ, 1992. Aspartate kinase regulation in maize - regulation by calcium and calmodulin. Phytochemistry 31:3735-3737.
28. Aziz, A; Saleem, M; Hidayat-ur-Rehman; Muhammad, F, 1992. Genetic variability for yield and disease resistance in full and short season varieties of maize. Sarhad J Agric 8:195-198.
29. Baba, T; Kimura, K; Mizuno, K; Etoh, H; Ishida, Y; Shida, O; Arai, Y, 1991. Sequence conservation of the catalytic regions of amylolytic enzymes in maize branching enzyme I. Biochem Biophys Res Commun 181:87-94.
30. Bachireddy, VR; Payne, R; Chin, KL; Kang, MS, 1992. Conventional selection versus methods that use genotype X environment interaction in sweet corn trials. Hortscience 27:436-438.
31. Bacic, G; Safranj, A; Ratkovic, S, 1991. A differential scanning calorimetry (DSC) and differential thermogravimetric (DTG) study of seed embryo of normal and opaque-2 maize. J Genet Breed 45:297-300.
32. Baggett, JR; Tibbs, J, 1992. Relationship between starchy versus sugary endosperm and head smut susceptibility of corn seedlings. Hortscience 27:438-440.
33. Bailey-Serres, J; Nguyen, MT, 1992. Purification and characterization of cytosolic 6-phosphogluconate dehydrogenase isozymes from maize. Plant Physiol 100:1580-1583.
34. Bailey-Serres, J; Tom, J; Freeling, M, 1992. Expression and distribution of cytosolic 6-phosphogluconate dehydrogenase isozymes in maize. Biochem Genet 30:233-246.
35. Baluska, F; Kubica, S, 1992. Relationships between the content of basic nuclear proteins, chromatin structure, rDNA transcription and cell size in different tissues of the maize root apex. J Exp Bot 43:991-996.
36. Baluska, F; Parker, JS; Barlow, PW, 1992. Specific patterns of cortical and endoplasmic microtubules associated with cell growth and tissue differentiation in roots of maize (*Zea mays* L.). J Cell Sci 103:191-200.

37. Bancroft, I; Bhatt, AM; Sjodin, C; Scofield, S; Jones, JDG; Dean, C, 1992. Development of an efficient 2-element transposon tagging system in *Arabidopsis thaliana*. Mol Gen Genet 233:449-461.
38. Bansal, KC; Viret, JF; Haley, J; Khan, BM; Schantz, R; Bogorad, L, 1992. Transient expression from *cab-m1* and *rbcS-m3* promoter sequences is different in mesophyll and bundle sheath cells in maize leaves. Proc Natl Acad Sci USA 89:3654-3658.
39. Barabas, Z, 1992. A full life - Pap, A., father of European hybrid maize - lived 95 years. Cereal Res Commun 20:155-157.
40. Baran, G; Echt, C; Bureau, T; Wessler, S, 1992. Molecular analysis of the maize *wx-B3* allele indicates that precise excision of the transposable *Ac* element is rare. Genetics 130:377-384.
41. Barent, RL; Elthon, TE, 1992. Two-dimensional gels: an easy method for large quantities of proteins. Plant Mol Biol Rep 10:338-344.
42. Barrow, MR, 1992. Development of maize hybrids resistant to maize streak virus. Crop Prot 11:267-271.
43. Bashan, B; Levy, Y, 1992. Differential response of sweet corn cultivars to phytotoxic water-soluble compounds from culture filtrates of *Exserohilum turcicum*. Plant Dis 76:451-454.
44. Bass, HW; Webster, C; Obrian, GR; Roberts, JKM; Boston, RS, 1992. A maize ribosome-inactivating protein is controlled by the transcriptional activator opaque-2. Plant Cell 4:225-234.
45. Bassiri, A; Irish, EE; Poethig, RS, 1992. Heterochronic effects of teopod-2 on the growth and photosensitivity of the maize shoot. Plant Cell 4:497-504.
46. Beavis, WD; Grant, D; Albertsen, M; Fincher, R, 1991. Quantitative trait loci for plant height in 4 maize populations and their associations with qualitative genetic loci. Theor Appl Genet 83:141-145.
47. Becker, D; Lutticke, R; Li, MG; Starlinger, P, 1992. Control of excision frequency of maize transposable element *Ds* in Petunia protoplasts. Proc Natl Acad Sci USA 89:5552-5556.
48. Becker, TW; Templeman, TS; Viret, JF; Bogorad, L, 1992. The *cab-m7* gene - a light-inducible, mesophyll-specific gene of maize. Plant Mol Biol 20:49-60.
49. Bedinger, P, 1992. The remarkable biology of pollen. Plant Cell 4:879-887.
50. Beilinson, VA; Reverdatto, SV; Efimov, VA, 1992. Directed mutagenesis of genes of some plant proteins. Bioorg Khim 18:920-923.
51. Bellmann, R; Werr, W, 1992. Zmhox1a, the product of a novel maize homeobox gene, interacts with the shrunken 26-bp feedback control element. EMBO J 11:3367-3374.
52. Belzile, F; Yoder, JI, 1992. Pattern of somatic transposition in a high copy *Ac* tomato line. Plant J 2:173-179.
53. Bennetzen, JL; Hulbert, SH, 1992. Extramarital sex amongst the beets - organization, instability and evolution of plant disease resistance genes. Plant Mol Biol 20:575-578.
54. Bennetzen, JL; Jones, JDG, 1992. Approaches and progress in the molecular cloning of plant disease resistance genes. Pp. 99-124 in Genetic Engineering, Vol 14. J. K. Setlow, ed., New York: Plenum.
55. Bentolila, S; Hardy, T; Guitton, C; Freyssinet, G, 1992. Comparative genetic analyses of F2 plants and anther culture derived plants of maize. Genome 35:575-582.
56. Benz, BF; Iltis, HH, 1990. Studies in archaeological maize. I. The "wild" maize from San Marcos cave reexamined. Amer Antiquity 55:500-511.
57. Benz, BF; Iltis, HH, 1992. Evolution of female sexuality in the maize ear (*Zea mays* L. subsp. *mays* Gramineae). Econ Bot 46:212-222.
58. Bergquist, RR; Thompson, DL, 1992. Corn grain density characterized by two specific gravity techniques. Crop Sci 32:1287-1290.
59. Bernardo, R, 1992. Relationship between single-cross performance and molecular marker heterozygosity. Theor Appl Genet 83:628-634.
60. Bernardo, R, 1992. Retention of genetically superior lines during early-generation testcrossing of maize. Crop Sci 32:933-937.
61. Bernardo, R; Bourrier, M; Olivier, JL, 1992. Generation means analysis of resistance to head smut in maize. Agronomie 12:303-306.
62. Beuselinck, PR; Steiner, JJ, 1992. A proposed framework for identifying, quantifying, and utilizing plant germplasm resources. Field Crop Res 29:261-272.
63. Binelli, G; Gianfranceschi, L; Pe, ME; Taramino, G; Busso, C; Stenhouse, J; Ottaviano, E, 1992. Similarity of maize and sorghum genomes as revealed by maize RFLP probes. Theor Appl Genet 84:10-16.
64. Bing, JW; Guthrie, WD; Dicke, FF, 1992. Genetics of resistance in maize to the corn leaf aphid (Homoptera, aphididae). J Econ Entomol 85:1476-1479.
65. Birch, RG; Franks, T, 1991. Development and optimisation of microprojectile systems for plant genetic transformation. Aust J Plant Physiol 18:453-469.
66. Blumler, MA, 1992. Independent inventionism and recent genetic evidence on plant domestication. Econ Bot 46:98-111.
67. Bockholt, AJ; Rooney, L, 1992. QPM hybrids for the USA. Pp. 111-121 in Quality Protein Maize. E. T. Mertz, ed., St. Paul: Amer Assoc Cereal Chem.
68. Bodeau, JP; Walbot, V, 1992. Regulated transcription of the maize bronze-2 promoter in electroporated protoplasts requires the *C1* and *R* gene products. Mol Gen Genet 233:379-387.
69. Bogdanov, Y, 1992. To the 90th anniversary of McClintock, B. Genetika 28:6.
70. Bolanos, J; Edmeades, GO, 1991. Value of selection for osmotic potential in tropical maize. Agron J 83:948-956.
71. Booy, G; Krens, FA; Bino, RJ, 1992. Analysis of pollen-tube growth in cultured maize silks. Sex Plant Reprod 5:227-231.
72. Boppenmaier, J; Melchinger, AE; Brunklaus-Jung, E; Geiger, HH; Herrmann, RG, 1992. Genetic diversity for RFLPs in European maize inbreds. 1. Relation to performance of flint x dent crosses for forage traits. Crop Sci 32:895-902.
73. Borlaug, NE, 1992. Potential role of quality protein maize in sub-Saharan Africa. Pp. 79-97 in Quality Protein Maize. E. T. Mertz, ed., St. Paul: Amer Assoc Cereal Chem.
74. Borrero, JC; Pandey, S; Ceballos, H, 1992. Performance and stability of tropical maize hybrids developed from lines with different levels of inbreeding. Maydica 37:251-258.
75. Bossinger, G; Maddaloni, M; Motto, M; Salamini, F, 1992. Formation and cell lineage patterns of the shoot apex of maize. Plant J 2:311-320.
76. Bourdu, R, 1991. From germination to post-emergence - a series of integrated processes with constraints. Pp. 17-18 in Physiologie et Production du Mais. D. Picard, ed., Paris: INRA.
77. Bourdu, R; Brousser, A, 1991. Cytological changes during germination and the subsequent seedling growth of maize. Pp. 19-28 in Physiologie et Production du Mais. D. Picard, ed., Paris: INRA.

78. Boyat, A; Kaan, F; Panouille, A, 1991. Adaptation of 4 corn varieties to plant population density. Pp. 335-344 in Physiologie et Production du Mais. D. Picard, ed., Paris: INRA.
79. Boyer, JS, 1992. Mechanisms for obtaining water use efficiency and drought resistance. Pp. 181-200 in Plant Breeding in the 1990s. H. T. Stalker and J. P. Murphy, ed., Wallingford, UK: CAB Internatl.
80. Brenner, D; Decarvalho, FIF; Federizzi, LC, 1991. Estimates of combining ability for grain yield in maize (*Zea mays* L.) populations. Rev Bras Genet 14:1001-1009.
81. Brettell, RIS; Dennis, ES, 1991. Reactivation of a silent *Ac* following tissue culture is associated with heritable alterations in its methylation pattern. Mol Gen Genet 229:365-372.
82. Brewster, VA; Carson, ML; Wicks, ZW, 1992. Mapping components of partial resistance to northern leaf blight of maize using reciprocal translocations. Phytopathology 82:225-229.
83. Briggs, SP, 1992. Identification and isolation of agronomically important genes from plants. Pp. 373-387 in Plant Breeding in the 1990s. H. T. Stalker and J. P. Murphy, ed., Wallingford, UK: CAB Internatl.
84. Brochetto-Braga, MR; Leite, A; Arruda, P, 1992. Partial purification and characterization of lysine-ketoglutarate reductase in normal and opaque-2 maize endosperms. Plant Physiol 98:1139-1147.
85. Brosch, G; Georgieva, EI; Lopez-Rodas, G; Lindner, H; Loidl, P, 1992. Specificity of *Zea mays* histone deacetylase is regulated by phosphorylation. J Biol Chem 267:20561-20564.
86. Broun, P; Ganal, MW; Tanksley, SD, 1992. Telomeric arrays display high levels of heritable polymorphism among closely related plant varieties. Proc Natl Acad Sci USA 89:1354-1357.
87. Brown, J; Sundaresan, V, 1992. Genetic study of the loss and restoration of Mutator transposon activity in maize - evidence against dominant-negative regulator associated with loss of activity. Genetics 130:889-898.
88. Brown, WL; Robinson, HF, 1992. The status, evolutionary significance and history of Eastern Cherokee maize. Maydica 37:29-39.
89. Bureau, TE; Wessler, SR, 1992. Tourist - a large family of small inverted repeat elements frequently associated with maize genes. Plant Cell 4:1283-1294.
90. Burr, B; Burr, FA; Matz, EC; Romero-Severson, J, 1992. Pinning down loose ends - mapping telomeres and factors affecting their length. Plant Cell 4:953-960.
91. Caldwell, EEO; Peterson, PA, 1992. The *Ac* and *Uq* transposable element systems in maize - interactions among components. Genetics 131:723-731.
92. Callahan, FE; Davis, FM; Williams, WP, 1992. Steady-state polypeptide profiles of whorl tissue from Lepidoptera-resistant and susceptible corn inbred lines. Crop Sci 32:1203-1207.
93. Camara-Hernandez, J; Gambino, S, 1991. Early ontogenetic development of the pistillate inflorescence in a diploid perennial teosinte (*Zea diploperennis*, Poaceae). Bot J Linn Soc 107:211-216.
94. Campbell, WH, 1990. Higher plant nitrate reductase: Effects of light and nitrate on gene expression and enzyme activity. Bol Acad Nac Cienc 59:55-70.
95. Campbell, WH, 1992. Expression in *Escherichia coli* of cytochrome C reductase activity from a maize NADH nitrate reductase complementary DNA. Plant Physiol 99:693-699.
96. Campenot, MK; Zhang, G; Cutler, AJ; Cass, DD, 1992. *Zea mays* embryo sacs in culture. I. Plant regeneration from 1 day after pollination embryos. Amer J Bot 79:1368-1373.
97. Cantone, FA; Dunkle, LD, 1991. Reversible effects of inhibitory diffusates from maize inoculated with *Cochliobolus carbonum*. Physiol Molec Plant Pathol 39:111-122.
98. Cao, YW; Anderova, M; Crawford, NM; Schroeder, JI, 1992. Expression of an outward-rectifying potassium channel from maize messenger RNA and complementary RNA in *Xenopus* oocytes. Plant Cell 4:961-969.
99. Carlson, WR; Roseman, RR, 1992. A new property of the maize B-chromosome. Genetics 131:211-223.
100. Carpenter, JE; Wiseman, BR, 1992. Effects of inherited sterility and insect resistant dent-corn silks on *Helicoverpa zea* (Lepidoptera, noctuidae) development. J Entomol Sci 27:413-420.
101. Casacuberta, JM; Raventos, D; Puigdomenech, P; Segundo, BS, 1992. Expression of the gene encoding the PR-like protein PRms in germinating maize embryos. Mol Gen Genet 234:97-104.
102. Ceballos, H; Deutsch, JA, 1992. Inheritance of resistance to tar spot complex in maize. Phytopathology 82:505-512.
103. Chang, MT; Neuffer, MG, 1992. Position of the vegetative and sperm cells of germinating pollen grain in maize (*Zea mays* L.). Maydica 37:233-243.
104. Chang, TT, 1992. Availability of plant germplasm for use in crop improvement. Pp. 17-35 in Plant Breeding in the 1990s. H. T. Stalker and J. P. Murphy, ed., Wallingford, UK: CAB Internatl.
105. Chapman, SR; Bahls, S, 1992. Patent laws and plant science. J Nat Resour Life Sci Educ 21:149-152.
106. Charlesworth, D; Charlesworth, B, 1992. The effects of selection in the gametophyte stage on mutational load. Evolution 46:703-720.
107. Chasan, R, 1991. Filling tomorrow's shopping basket - practical applications of plant molecular biology. Plant Cell 3:1141-1142.
108. Chasan, R, 1992. Endosperm - food for thought. Plant Cell 4:235-236.
109. Chasan, R, 1992. A feast of maize genetics. Plant Cell 4:507-512.
110. Chasan, R, 1992. In the realm of the homeodomain. Plant Cell 4:237-240.
111. Chasan, R, 1992. Maize telomeres - the end of the line. Plant Cell 4:865-867.
112. Chasan, R, 1992. Signal sensations. Plant Cell 4:1171-1173.
113. Chaubal, R; Reger, BJ, 1990. Demonstration of high calcium levels in synergid cells of angiosperm ovaries. Pp. 196-197 in Current Topics in Plant Physiology, Vol. 4: Calcium in Plant Growth and Development. R. T. Leonard and P. K. Hepler, ed., Rockville, MD: Amer Soc Plant Physiol.
114. Chaubet, N; Philipps, G; Gigot, C; Guitton, C; Bouvet, N; Freyssinet, G; Schneerman, M; Weber, DF, 1992. Subfamilies of histone-H3 and histone-H4 genes are located on most, possibly all of the chromosomes in maize. Theor Appl Genet 84:555-559.
115. Chaves, LJ; Filho, JBD, 1992. Plot size for progeny selection in maize (*Zea mays* L.). Theor Appl Genet 84:963-970.
116. Chen, J; Greenblatt, IM; Dellaporta, SL, 1992. Molecular analysis of *Ac* transposition and DNA replication. Genetics 130:665-676.

117. Chen, K; Rubenstein, I, 1991. Characterization of the structure and transcription of an ubiquitin fusion gene from maize. Gene 107:205-212.
118. Cherney, JH; Cherney, DJR; Akin, DE; Axtell, JD, 1991. Potential of brown-midrib, low-lignin mutants for improving forage quality. Pp. 157-198 in Advances in Agronomy, Vol 46. D. L. Sparks, ed., San Diego: Academic Press Inc.
119. Chopra, S; Del-Favero, J; Dolferus, R; Jacobs, M, 1992. Sucrose synthase of *Arabidopsis* - genomic cloning and sequence characterization. Plant Mol Biol 18:131-134.
120. Christensen, AH; Sharrock, RA; Quail, PH, 1992. Maize polyubiquitin genes - structure, thermal perturbation of expression and transcript splicing, and promoter activity following transfer to protoplasts by electroporation. Plant Mol Biol 18:675-689.
121. Chu, B; Xin, Z; Li, PH; Carter, JV, 1992. Depolymerization of cortical microtubules is not a primary cause of chilling injury in corn (*Zea mays* L. cv. Black Mexican Sweet) suspension culture cells. Plant Cell Environ 15:307-312.
122. Coello, P; Rodriguez, R; Garcia, E; Vazquez-Ramos, JM, 1992. A DNA polymerase from maize axes: its purification and possible role. Plant Mol Biol 20:1159-1168.
123. Coghlan, A, 1992. Toxic gene could rid maize of boring pest. New Sci 135:21.
124. Collazo, P; Montoliu, L; Puigdomenech, P; Rigau, J, 1992. Structure and expression of the lignin O-methyltransferase gene from *Zea mays* L. Plant Mol Biol 20:857-867.
125. Comeau, A; Nadeau, P; Plourde, A; Simard, A; Maes, O; Kelly, S; Harper, L; Lettre, J; Landry, B; St. Pierre, C-A, 1992. Media for the in ovulo culture of proembryos of wheat and wheat-derived interspecific hybrids or haploids. Plant Sci 81:117-126.
126. Compton, ME; Benton, CM; Gray, DJ; Songstad, DD, 1992. Plant recovery from maize somatic embryos subjected to controlled relative humidity dehydration. In Vitro Cell Dev Biol-Plant 28P:197-201.
127. Consonni, G; Viotti, A; Dellaporta, SL; Tonelli, C, 1992. cDNA nucleotide sequence of *Sn*, a regulatory gene in maize. Nucleic Acids Res 20:373.
128. Cook, WB; Miles, D, 1992. Nuclear mutations affecting plastoquinone accumulation in maize. Photosynth Res 31:99-111.
129. Cook, WB; Walker, JC, 1992. Identification of a maize nucleic acid-binding protein (NBP) belonging to a family of nuclear-encoded chloroplast proteins. Nucleic Acids Res 20:359-364.
130. Coplin, DL; Frederick, RD; Majerczak, DR, 1992. New pathogenicity loci in *Erwinia stewartii* identified by random Tn5 mutagenesis and molecular cloning. Mol Plant Microbe Interaction 5:266-268.
131. Cottingham, CK; Hatzios, KK, 1992. Basis of differential tolerance of 2 corn hybrids (*Zea mays*) to metolachlor. Weed Sci 40:359-363.
132. Cowen, NM; Johnson, CD; Armstrong, K; Miller, M; Woosley, A; Pescitelli, S; Skokut, M; Belmar, S; Petolino, JF, 1992. Mapping genes conditioning in vitro androgenesis in maize using RFLP analysis. Theor Appl Genet 84:720-724.
133. Crafts-Brandner, SJ; Poneleit, CG, 1992. Selection for seed growth characteristics - effect on leaf senescence in maize. Crop Sci 32:127-131.
134. Cramer, GR, 1992. Kinetics of maize leaf elongation. 2. Responses of a Na-excluding cultivar and a Na-including cultivar to varying Na/Ca salinities. J Exp Bot 43:857-864.
135. Cramer, MM; Kannenberg, LW, 1992. 5 years of HOPE - the hierarchical open-ended corn breeding system. Crop Sci 32:1163-1171.
136. Curtis, CA; Doyle, GG, 1992. Production of aneuploid and diploid eggs by meiotic mutants of maize. J Hered 83:335-341.
137. Daniel, L, 1992. The *se* (Sugary enhancer) gene enhancing the saccharose content of sweet corn. Novenytermeles 41:167-175.
138. Darrah, LL; Lundquist, R; Bockholt, AJ; West, DR; Poneleit, CG; Wassom, CE; Barry, BD; Crane, PL; Zehr, BE; Rooney, LW; Colbert, TR; Elmore, RW; Martin, PR, 1991. White Food Corn 1991 Performance Test. Columbia, MO: Mo Agr Exp Stn.
139. Darvasi, A; Weller, JI, 1992. On the use of the moments method of estimation to obtain approximate maximum likelihood estimates of linkage between a genetic marker and a quantitative locus. Heredity 68:43-46.
140. Das, OP; Ward, K; Ray, S; Messing, J, 1991. Sequence variation between alleles reveals two types of copy correction at the 27-kDa zein locus of maize. Genomics 11:849-856.
141. Dean, C; Sjodin, C; Page, T; Jones, J; Lister, C, 1992. Behaviour of the maize transposable element *Ac* in *Arabidopsis thaliana*. Plant J 2:69-81.
142. deBarros, EG; Takasaki, K; Kirleis, AW; Larkins, BA, 1991. Nucleotide sequence of a cDNA clone encoding gamma-kafirin protein from Sorghum bicolor. Plant Physiol 97:1606-1607.
143. Debnath, SC; Khan, MF, 1991. Genotypic variation, covariation, and path coefficient analysis in maize. Pak J Sci Ind Res 34:391-394.
144. Decarvahlo, HWL; Magnavaca, R; Leal, MDD, 1992. Corn cultivars production stability in Sergipe, Brazil. Pesquisa Agr Brasil 27:1073-1082.
145. Dembinski, E; Rafalski, A; Wisniewska, I, 1991. Effect of long-term selection for high and low protein content on the metabolism of amino acids and carbohydrates in maize kernel. Plant Physiol Biochem 29:549-557.
146. deMiranda, JB, 1992. Exotic germplasms introduced in a Brazilian maize breeding program. Rev Bras Genet 15:631-642.
147. Denney, JO, 1992. Xenia includes metaxenia. Hortscience 27:722-728.
148. Derieux, M, 1991. Crop establishment - a decisive phase for determining the number of grains. Pp. 103-104 in Physiologie et Production du Mais. D. Picard, ed., Paris: INRA.
149. deSouza, AP; Jubier, MF; Delcher, E; Lancelin, D; Lejeune, B, 1991. A trans-splicing model for the expression of the tripartite nad5 gene in wheat and maize mitochondria. Plant Cell 3:1363-1378.
150. deVetten, NC; Lu, GH; Ferl, RJ, 1992. A maize protein associated with the G-Box binding complex has homology to brain regulatory proteins. Plant Cell 4:1295-1307.
151. Dewald, CL; Taliaferro, CM; Dunfield, PC, 1992. Registration of 4 fertile triploid germplasm lines of eastern gamagrass. Crop Sci 32:504.
152. Dewey, RE; Timothy, DH; Levings, CS, 1991. Chimeric mitochondrial genes expressed in the C-male-sterile cytoplasm of maize. Curr Genetics 20:475-482.
153. Didierjean, L; Frendo, P; Burkard, G, 1992. Stress responses in maize - sequence analysis of cDNAs encoding glycine-rich proteins. Plant Mol Biol 18:847-849.
154. Ding, QX; He, QF; Sai, JQ; Sun, YM; Xie, YJ; Mi, JJ, 1992. Identification of an effective vector constructed for maize transformation and study of PEG-mediated transformation method. Sci China Ser B 35:1187-1195.

155. Dobrowolska, G; Boldyreff, B; Issinger, OG, 1991. Cloning and sequencing of the casein kinase-2 alpha-subunit from *Zea mays*. Biochim Biophys Acta 1129:139-140.
156. Doebley, J, 1992. Mapping the genes that made maize. Trends Genet 8:302-307.
157. Dogan, Z, 1990. Investigation of content of carotenoid in BC maize hybrids from different maturity groups. Poljopr Znan Smotra 55:19-36.
158. Dolezelova, M; Dolezel, J; Nesticky, M, 1992. Relationship of embryogenic competence in maize (*Zea mays* L.) leaves to mitotic activity, cell cycle and nuclear DNA content. Plant Cell Tissue Organ Cult 31:215-221.
159. Dolfini, SF; Landoni, M; Tonelli, C; Bernard, L; Viotti, A, 1992. Spatial regulation in the expression of structural and regulatory storage-protein genes in *Zea mays* endosperm. Dev Genet 13:264-276.
160. Dolgykh, YI; Larina, SN; Bolonkina, YV, 1992. Analysis of somaclonal variability of inbred maize line A188. Genetika SSSR 28:74-82.
161. Dombrink-Kurtzman, MA; Wilson, CM, 1992. Characterization of proteins in hard and soft endosperm of quality protein maize. Pp. 155-173 in Quality Protein Maize. E. T. Mertz, ed., St. Paul: Amer Assoc Cereal Chem.
162. Doolittle, WE; Frederick, CD, 1991. Phytoliths as indicators of prehistoric maize (*Zea mays* subsp mays, Poaceae) cultivation. Plant Syst Evol 177:175-184.
163. Dooner, HK; Robbins, TP; Jorgensen, RA, 1992. Genetic and developmental control of anthocyanin biosynthesis. Annu Rev Genet 25:173-200.
164. Dorsey-Redding, C; Hurburgh, CR; Johnson, LA; Fox, SR, 1991. Relationships among maize quality factors. Cereal Chem 68:602-605.
165. Dow, DA; Mascarenhas, JP, 1991. Optimization of conditions for in situ hybridization and determination of the relative number of ribosomes in the cells of the mature embryo sac of maize. Sex Plant Reprod 4:244-249.
166. Dow, DA; Mascarenhas, JP, 1991. Synthesis and accumulation of ribosomes in individual cells of the female gametophyte of maize during its development. Sex Plant Reprod 4:250-253.
167. Downing, T; Kenny, G; Harrison, P, 1991. Heat and maize. New Sci 132:58.
168. Draganic, M; Boric, B, 1991. Inheritance of resistance to root rot *Fusarium solani* mart sacc. in maize. Zast Bilja 42:33-38.
169. Draganic, M; Boric, B, 1991. Survey of studies on maize resistance to stalk and ear rot pathogens in Yugoslavia. Zast Bilja 42:173-182.
170. Dronamraju, KR, 1991. Profiles in genetics - Beadle,George Wells and the origins of the gene-enzyme concept. J Hered 82:443-446.
171. Dronavalli, S; Kang, MS, 1992. General and specific combining ability for seed quality traits in maize with *Lfy* gene. Plant Var Seeds 5:53-59.
172. Dudley, JW, 1992. Theory for identification of marker locus-QTL associations in population by line crosses. Theor Appl Genet 85:101-104.
173. Dudley, JW; Lambert, RJ, 1992. 90-Generations of selection for oil and protein in maize. Maydica 37:81-87.
174. Dudley, JW; Maroof, MAS; Rufener, GK, 1992. Molecular marker information and selection of parents in corn breeding programs. Crop Sci 32:301-304.
175. Duvick, D, 1991. Technology for the 1990s - rate and potential limitations of technology generation. Pp. 39-55 in Agricultural Issues in the 1990s.
176. Duvick, DN, 1992. Genetic contributions to advances in yield of United States maize. Maydica 37:69-79.
177. Duvick, JP; Rood, T; Rao, AG; Marshak, DR, 1992. Purification and characterization of a novel antimicrobial peptide from maize (*Zea mays* L.) kernels. J Biol Chem 267:18814-18820.
178. Dwyer, LM; Stewart, DW; Hamilton, RI; Houwing, L, 1992. Ear position and vertical distribution of leaf area in corn. Agron J 84:430-438.
179. Ebong, UU; Wahua, TAT, 1991. Effects of plant population densities on the phenotypic correlations among the yield components of maize (*Zea mays* L.). Discov Innovat 3:111-114.
180. Echeverria, RG, 1991. Impact of research and seed trade on maize productivity. Pp. 365-396 in Agricultural Research Policy.
181. Edwards, KJ; Thompson, H; Edwards, D; deSaizieu, A; Sparks, C; Thompson, JA; Greenland, AJ; Eyers, M; Schuch, W, 1992. Construction and characterisation of a yeast artificial chromosome library containing 3 haploid maize genome equivalents. Plant Mol Biol 19:299-308.
182. Edwards, M, 1992. Use of molecular markers in the evaluation and introgression of genetic diversity for quantitative traits. Field Crop Res 29:241-260.
183. Edwards, MD; Helentjaris, T; Wright, S; Stuber, CW, 1992. Molecular-marker-facilitated investigations of quantitative trait loci in maize. 4. Analysis based on genome saturation with isozyme and restriction fragment length polymorphism markers. Theor Appl Genet 83:765-774.
184. Ellis, JG; Finnegan, EJ; Lawrence, GJ, 1992. Developing a transposon tagging system to isolate rust-resistance genes from flax. Theor Appl Genet 85:46-54.
185. Ellis, RH; Summerfield, RJ; Edmeades, GO; Roberts, EH, 1992. Photoperiod, leaf number, and interval from tassel initiation to emergence in diverse cultivars of maize. Crop Sci 32:398-403.
186. Ellis, RH; Summerfield, RJ; Edmeades, GO; Roberts, EH, 1992. Photoperiod, temperature, and the interval from sowing to tassel initiation in diverse cultivars of maize. Crop Sci 32:1225-1232.
187. Elsayed, H; Kirkwood, RC, 1992. Effects of NaCl salinity and hydrogel polymer treatments on viability, germination and solute contents in maize (*Zea mays*) pollen. Phyton-Ann Rei Bot Austria 32:143-157.
188. Emons, AMC; Kieft, H, 1991. Histological comparison of single somatic embryos of maize from suspension culture with somatic embryos attached to callus cells. Plant Cell Rep 10:485-488.
189. Erdelska, O; Vidovencova, Z, 1992. Cleavage polyembryony in maize. Sex Plant Reprod 5:224-226.
190. Ervandyan, SG; Galukyan, MG, 1991. Waxy maize mutants as test objects in assessing the radiomodifying effect of bioregulators. Radiobiologiya 31:394-399.
191. Esen, A, 1991. Reaction of anti-alpha-zein sera with synthetic peptides derived from selected regions of alpha-zein polypeptides. J Cereal Sci 14:257-266.
192. Esen, A, 1992. Purification and partial characterization of maize (*Zea mays* L.) β-glucosidase. Plant Physiol 98:174-182.
193. Esen, A; Stetler, DA, 1992. Immunocytochemical localization of delta-zein in the protein bodies of maize endosperm cells. Amer J Bot 79:243-248.
194. Eskridge, KM; Mumm, RF, 1992. Choosing plant cultivars based on the probability of outperforming a check. Theor Appl Genet 84:494-500.
195. Eta-Ndu, JT; Openshaw, SJ, 1992. Selection criteria for grain yield and moisture in maize yield trials. Crop Sci 32:332-335.

196. Eyherabide, GH; Hallauer, AR, 1991. Reciprocal full-sib recurrent selection in maize. 2. Contributions of additive, dominance, and genetic drift effects. Crop Sci 31:1442-1448.
197. Falconer, DS, 1992. Early selection experiments. Annu Rev Genet 26:1-14.
198. Fang, LY; Gross, PR; Chen, CH; Lillis, M, 1992. Sequence of 2 acetohydroxyacid synthase genes from *Zea mays*. Plant Mol Biol 18:1185-1187.
199. Fantin, GM; Sawazaki, E; Barros, BC, 1991. Evaluation of popcorn genotypes to disease resistance and popcorn quality. Summa Phytopathol 17:90-99.
200. Fauron, CMR; Casper, M; Gesteland, R; Albertsen, M, 1992. A multi-recombination model for the mtDNA rearrangements seen in maize cmsT regenerated plants. Plant J 2:949-958.
201. Fedenko, VS, 1991. Variability of the chemical composition of the grain of different maize genotypes. Khim Prir Soedin 441-442.
202. Fedenko, VS; Struzhko, VS, 1991. Absorption spectra of carotenoids from maize grains with different genotypes. Khim Prir Soedin 858-859.
203. Fedoroff, N, 1992. McClintock, Barbara - the geneticist, the genius, the woman. Cell 71:181-182.
204. Fedoroff, N; Botstein, D, 1992. The Dynamic Genome: Barbara McClintock's Ideas in the Century of Genetics. Cold Spring Harbor: Cold Spring Harbor Lab Press.
205. Fedoroff, NV, 1991. Maize transposable elements. Perspect Biol Med 35:2-19.
206. Feldmar, S; Kunze, R, 1991. The ORFa protein, the putative transposase of maize transposable element *Ac*, has a basic DNA binding domain. EMBO J 10:4003-4010.
207. Feldwisch, J; Zettl, R; Hesse, F; Schell, J; Palme, K, 1992. An auxin-binding protein is localized to the plasma membrane of maize coleoptile cells - identification by photoaffinity labeling and purification of a 23-kDa polypeptide. Proc Natl Acad Sci USA 89:475-479.
208. Felker, FC, 1992. Participation of cob tissue in the uptake of medium components by maize kernels cultured in vitro. J Plant Physiol 139:647-652.
209. Fennell, A; Hauptmann, R, 1992. Electroporation and PEG delivery of DNA into maize microspores. Plant Cell Rep 11:567-570.
210. Ferl, RJ; Paul, AL, 1992. Chemical detection of Z-DNA within the maize *Adh1* promoter. Plant Mol Biol 18:1181-1184.
211. Fincham, JRS, 1992. The dynamic genome - McClintock,Barbara ideas in the century of genetics, by N. Fedoroff, D. Botstein. Nature 358:631-632.
212. Finer, JJ; Vain, P; Jones, MW; McMullen, MD, 1992. Development of the particle inflow gun for DNA delivery to plant cells. Plant Cell Rep 11:323-328.
213. Fink, GR, 1992. McClintock,Barbara (1902-1992) - obituary. Nature 359:272.
214. Fitzmaurice, WP; Lehman, LJ; Nguyen, LV; Thompson, WF; Wernsman, EA; Conkling, MA, 1992. Development and characterization of a generalized gene tagging system for higher plants using an engineered maize transposon *Ac*. Plant Mol Biol 20:177-198.
215. Fleury, A, 1991. Methodology of yield analysis in maize. Pp. 279-290 in Physiologie et Production du Mais. D. Picard, ed., Paris: INRA.
216. Forlani, G; Nielsen, E; Landi, P; Tuberosa, R, 1991. Chlorsulfuron tolerance and acetolactate synthase activity in corn (*Zea mays* L.) inbred lines. Weed Sci 39:553-557.
217. Forlani, G; Nielsen, E; Racchi, ML, 1992. A glyphosate-resistant 5-enol-pyruvyl-shikimate-3-phosphate synthase confers tolerance to a maize cell line. Plant Sci 85:9-15.
218. Fosket, DE; Morejohn, LC, 1992. Structural and functional organization of tubulin. Annu Rev Plant Physiol Plant Mol Biol 43:201-240.
219. Fox, PC; Vasil, V; Vasil, IK; Gurley, WB, 1992. Multiple ocs-like elements required for efficient transcription of the mannopine synthase gene of T-DNA in maize protoplasts. Plant Mol Biol 20:219-233.
220. Frascaroli, E; Landi, P; Sari Gorla, M; Ottaviano, E, 1992. Variability of pollen and plant responses to glyphosate in maize. J Genet Breed 46:49-56.
221. Freeling, M, 1992. Barbara McClintock and 20th century biology (book review). Trends Genet 8:463.
222. Freeling, M, 1992. A conceptual framework for maize leaf development. Dev Biol 153:44-58.
223. Freeling, M; Bertrand-Garcia, R; Sinha, N, 1992. Maize mutants and variants altering developmental time and their heterochronic interactions. Bioessays 14:227-236.
224. Frendo, P; Didierjean, L; Passelegue, E; Burkard, G, 1992. Abiotic stresses induce a thaumatin-like protein in maize - cDNA isolation and sequence analysis. Plant Sci 85:61-69.
225. Freppon, JT; St. Martin, SK; Pratt, RC; Henderlong, PR, 1992. Selection for low ear moisture in corn using a hand-held meter. Crop Sci 32:1062-1064.
226. Frick, H; Goffner, D, 1992. Potassium flux and anthesis in the tassel of maize (*Zea mays* L.). Maydica 37:133-136.
227. Fritz, VA; Hebel, JB; Borowski, AM, 1991. Sweet corn genotypes versus ethephon in relation to yield components. Agron J 83:991-995.
228. Furini, A; Jewell, DC, 1991. Somatic embryogenesis and plant regeneration of *Tripsacum dactyloides* L. Euphytica 55:111-115.
229. Fussell, B, 1992. The Story of Corn. New York: Alfred A. Knopf.
230. Gaborjanyi, R; Hoang, DN; Kovacs, G, 1992. Resistance of maize inbred lines and sorghum species to potyviruses found in Hungary. Cereal Res Commun 20:131-137.
231. Gaillard, A; Matthys-Rochon, E; Dumas, C, 1992. Selection of microspore derived embryogenic structures in maize related to transformation potential by microinjection. Bot Acta 105:313-318.
232. Gakuru, S; Lefebvre, C, 1991. Acid phosphatase - screening Zairian *Zea mays* varieties for aluminium tolerance. Cereal Res Commun 19:477-481.
233. Galatis, B; Apostolakos, P, 1991. Microtubule organization and morphogenesis of stomata in caffeine-affected seedlings of *Zea mays*. Protoplasma 165:11-26.
234. Galinat, WC, 1992. Corn, Columbus, and culture. Perspect Biol Med 36:1-12.
235. Galinat, WC, 1992. Evolution of corn. Adv Agron 47:203-231.
236. Galinat, WC, 1992. Maize: gift from America's first peoples. Pp. 47-60 in Chilies to Chocolate. N. Foster and L. S. Cordell, ed., Tucson: Univ Arizona Press.
237. Gallais, A, 1992. Why develop synthetic varieties? Agronomie 12:601-609.

238. Gama, EEGE; Guimaraes, PED; Magnavaca, R; Parentoni, SN; Pacheco, CAP, 1992. General and specific combining ability in 7 Latin America maize populations. Pesquisa Agr Brasil 27:1167-1172.
239. Gardiner-Garden, M; Frommer, M, 1992. Significant CpG-rich regions in angiosperm genes. J Mol Evol 34:231-245.
240. Geetha, KB; Lending, CR; Lopes, MA; Wallace, JC; Larkins, BA, 1991. Opaque-2 modifiers increase gamma-zein synthesis and alter its spatial distribution in maize endosperm. Plant Cell 3:1207-1219.
241. Geiger, HH; Seitz, G; Melchinger, AE; Schmidt, GA, 1992. Genotypic correlations in forage maize. 1. Relationships among yield and quality traits in hybrids. Maydica 37:95-99.
242. Geng, R-S; Miao, S-H, 1991. Selection and characterization of maize variants resistant to S-2 aminoethyl-l-cysteine and 5 methyltryptophan. Acta Bot Sin 33:863-869.
243. Gevers, HO; Lake, JK, 1992. Development of modified opaque-2 maize in South Africa. Pp. 49-78 in Quality Protein Maize. E. T. Mertz, ed., St. Paul: Amer Assoc Cereal Chem.
244. Gierl, A; Saedler, H, 1992. Plant-transposable elements and gene tagging. Plant Mol Biol 19:39-49.
245. Giovinazzo, G; Manzocchi, LA; Bianchi, MW; Coraggio, I; Viotti, A, 1992. Functional analysis of the regulatory region of a zein gene in transiently transformed protoplasts. Plant Mol Biol 19:257-263.
246. Glover, DV, 1992. Corn protein - genetics, breeding, and value in foods and feeds. Pp. 9-26 in Quality Protein Maize. E. T. Mertz, ed., St. Paul: Amer Assoc Cereal Chem.
247. Godfrey, LD; Holtzer, TO; Spomer, SM; Norman, JM, 1991. European corn borer (Lepidoptera, Pyralidae) tunneling and drought stress - effects on corn yield. J Econ Entomol 84:1850-1860.
248. Goff, SA; Cone, KC; Chandler, VL, 1992. Functional analysis of the transcriptional activator encoded by the maize-*B* gene - evidence for a direct functional interaction between 2 classes of regulatory proteins. Gene Develop 6:864-875.
249. Golubovskaya, IN; Grebennikova, ZK; Avalkina, NA, 1992. The new allele of maize *Mei*-gene ameiotic-1 (*am1*) and the problem of genic control of the initiation of meiosis in higher plants. Genetika SSSR 28:137-146.
250. Good, AG; Paetkau, DH, 1992. Identification and characterization of a hypoxically induced maize lactate dehydrogenase gene. Plant Mol Biol 19:693-697.
251. Goodrich, J; Carpenter, R; Coen, ES, 1992. A common gene regulates pigmentation pattern in diverse plant species. Cell 68:955-964.
252. Gordon-Kamm, WJ; Spencer, TM; O'Brien, JV; Start, WG; Daines, RJ; Adams, TR; Mangano, ML; Chambers, SA; Zachwieja, SJ, et al., 1991. Transformation of maize using microprojectile bombardment - an update and perspective. In Vitro Cell Dev Biol Plant 27P:21-27.
253. Gorman, DP; Kang, MS; Cleveland, T; Hutchinson, RL, 1992. Field aflatoxin production by *Aspergillus flavus* and *Aspergillus parasiticus* on maize kernels. Euphytica 61:187-191.
254. Gouesnard, B; Gallais, A, 1992. Genetic variance component estimation in a nested mating design with positive assortative mating, and application to maize. Crop Sci 32:1127-1131.
255. Govedarica, M, 1991. A specific relationship between *Azotobacter chroococcum* strains and some corn hybrids. Mikrobiologija 28:45-56.
256. Gowri, G; Kenis, JD; Ingemarsson, B; Redinbaugh, MG; Campbell, WH, 1992. Nitrate reductase transcript is expressed in the primary response of maize to environmental nitrate. Plant Mol Biol 18:55-64.
257. Grana, X; Delecea, L; Elmaghrabi, MR; Urena, JM; Caellas, C; Carreras, J; Puigdomenech, P; Pilkis, SJ; Climent, F, 1992. Cloning and sequencing of a cDNA encoding 2,3-bisphosphoglycerate-independent phosphoglycerate mutase from maize - possible relationship to the alkaline phosphatase family. J Biol Chem 267:12797-12803.
258. Grandbastien, MA, 1992. Retroelements in higher plants. Trends Genet 8:103-108.
259. Grasser, KD; Haass, MM; Griess, EA; Feix, G, 1992. Detection of the chromosomal HMGa protein in crude maize extracts. J Plant Physiol 140:634-636.
260. Gray, MW; Hanic-Joyce, PJ; Covello, PS, 1992. Transcription, processing and editing in plant mitochondria. Annu Rev Plant Physiol 43:145-175.
261. Grebennikova, ZC; Golubovskaya, IN, 1991. Arrest of chromosome synapsis and meiosis in maize *mei*-mutant *pra1*. Tsitologiya SSSR 33:20.
262. Grevelding, C; Becker, D; Kunze, R; VonMenges, A; Fantes, V; Schell, J; Masterson, R, 1992. High rates of *Ac*/*Ds* germinal transposition in *Arabidopsis* suitable for gene isolation by insertional mutagenesis. Proc Natl Acad Sci USA 89:6085-6089.
263. Grotewold, E; Athma, P; Peterson, T, 1991. A possible hot spot for *Ac* insertion in the maize *P*-gene. Mol Gen Genet 230:329-331.
264. Groth, JV; Pataky, JK; Gingera, GR, 1992. Virulence in eastern north american populations of Puccinia sorghi to rp resistance genes in corn. Plant Dis 76:1140-1144.
265. Guei, RG; Wassom, CE, 1992. Inheritance of some drought adaptive traits in maize. 1. Interrelationships between yield, flowering, and ears per plant. Maydica 37:157-164.
266. Gueldner, RC; Snook, ME; Widstrom, NW; Wiseman, BR, 1992. TLC screen for maysin, chlorogenic acid, and other possible resistance factors to the fall armyworm and the corn earworm in *Zea mays*. J Agr Food Chem 40:1211-1213.
267. Guerbette, F; Quintin-Colona, F; Grosbois, M; Yamada, M; Sossountzov, L; Kader, J-C, 1990. Immunochemical studies on lipid transfer proteins. Pp. 275-277 in Plant Lipid Biochemistry, Structure and Utilization. P. J. Quinn and J. L. Harwood, ed., London: Portland Press.
268. Gupta, D; Abbott, AG, 1991. Higher plant mitochondrial DNA expression. 2. Influence of nuclear background on the transcription of a mitochondrial open reading frame, ORF25. Theor Appl Genet 82:723-728.
269. Gur'ev, BP; Kozubenko, LV; Luk'yanenko, LM; Virchenko, LI, 1991. Inheritance of consistency in modified opaque-2 of maize endosperm. Tsitol Genet 25:34-37.
270. Haass, MM; Feix, G, 1992. 2 different cDNAs encoding TFIID proteins of maize. FEBS Lett 301:294-298.
271. Hake, S, 1992. Unraveling the knots in plant development. Trends Genet 8:109-114.
272. Hake, S; Sinha, N, 1991. Genetic analysis of leaf development. Pp. 187-222 in Oxford Surveys of Plant Molecular and Cell Biology. B. J. Miflin, ed., Oxford: Oxford Univ Press.
273. Hallauer, AR, 1992. Use of genetic variation for breeding populations in cross-pollinated species. Pp. 37-67 in Plant Breeding in the 1990s. H. T. Stalker and J. P. Murphy, ed., Wallingford, UK: CAB Internatl.
274. Hamilton, DA; Roy, M; Rueda, J; Sindhu, RK; Sanford, J; Mascarenhas, JP, 1992. Dissection of a pollen-specific promoter from maize by transient transformation assays. Plant Mol Biol 18:211-218.

275. Han, CD; Coe, EH; Martienssen, RA, 1992. Molecular cloning and characterization of iojap (*ij*), a pattern striping gene of maize. EMBO J 11:4037-4046.
276. Hanson, MR, 1992. Plant mitochondrial mutations and male sterility. Annu Rev Genet 25:461-486.
277. Hanson, PM; Smith, L, 1992. Field evaluation of Kenyan open-pollinated maize populations for reaction to rust. Trop Agr 69:137-140.
278. Hase, T; Mizutani, S; Mukohata, Y, 1991. Expression of maize ferredoxin cDNA in *Escherichia coli* - comparison of photosynthetic and non-photosynthetic ferredoxin isoproteins and their chimeric molecule. Plant Physiol 97:1395-1401.
279. Hattori, T; Vasil, V; Rosenkrans, L; Hannah, LC; McCarty, DR; Vasil, IK, 1992. The viviparous-1 gene and abscisic acid activate the *C1* regulatory gene for anthocyanin biosynthesis during seed maturation in maize. Gene Develop 6:609-618.
280. Hayes, MK; Luethy, MH; Elthon, TE, 1991. Mitochondrial malate dehydrogenase from corn - purification of multiple forms. Plant Physiol 97:1381-1387.
281. Hebert, Y; Barriere, Y; Bertholeau, JC, 1992. Root lodging resistance in forage maize - genetic variability of root system and aerial part. Maydica 37:173-183.
282. Hebert, Y; Duparque, A; Pellerin, S, 1991. Root lodging - the genetic variability of the root system and the aerial part of the maize plant and its consequences in breeding. Pp. 215-226 in Physiologie et Production du Mais. D. Picard, ed., Paris: INRA.
283. Helentjaris, TG, 1992. RFLP analyses for manipulating agronomic traits in plants. Pp. 357-372 in Plant Breeding in the 1990s. H. T. Stalker and J. P. Murphy, ed., Wallingford, UK: CAB Internatl.
284. Heng, Z; Srinivasan, C; Sticklen, MB, 1992. In vitro morphogenesis of corn (*Zea mays* L.). 1. Differentiation of multiple shoot clumps and somatic embryos from shoot tips. Planta 187:483-489.
285. Heng, Z; Srinivasan, C; Sticklen, MB, 1992. In vitro morphogenesis of corn (*Zea mays* L.). 2. Differentiation of ear and tassel clusters from cultured shoot apices and immature inflorescences. Planta 187:490-497.
286. Hershberger, RJ; Warren, CA; Walbot, V, 1991. Mutator activity in maize correlates with the presence and expression of the *Mu*-transposable element *Mu9*. Proc Natl Acad Sci USA 88:10198-10202.
287. Honda, H; Hirai, A, 1992. The gene for the alpha-subunit of ATPase - a site of homologous recombination in plant mitochondrial DNA also functions in somatic hybrid cells. Theor Appl Genet 84:33-38.
288. Hood, EE; Hood, KR; Fritz, SE, 1991. Hydroxyproline-rich glycoproteins in cell walls of pericarp from maize. Plant Sci 79:13-22.
289. Hope, HJ; White, RP; Dwyer, LM; Maamari, R; Seguin, S; Hamilton, RI, 1992. Low temperature emergence potential of short season corn hybrids grown under controlled environment and plot conditions. Can J Plant Sci 72:83-91.
290. Hopf, N; Plesofskyvig, N; Brambl, R, 1992. The heat shock response of pollen and other tissues of maize. Plant Mol Biol 19:623-630.
291. Hormaza, JI; Herrero, M, 1992. Pollen selection. Theor Appl Genet 83:663-672.
292. Howarth, CJ, 1991. Molecular responses of plants to an increased incidence of heat shock. Plant Cell Environ 14:831-841.
293. Howe, GT; Strauss, SH; Goldfarb, B, 1991. Insertion of the maize transposable element *Ac* into poplar. Pp. 283-294 in NATO ASI Series, Series A - Life Sciences, Vol. 210. M. R. Ahuja, ed., New York: Plenum Press.
294. Hsu, SY; Peterson, PA, 1991. The upper and lower florets of spikelets in maize. J Genet Breed 45:215-222.
295. Hu, GS; Liang, GH; Wassom, CE, 1991. Chemical induction of apomictic seed formation in maize. Euphytica 56:97-105.
296. Huang, AHC, 1992. Oil bodies and oleosins in seeds. Annu Rev Plant Physiol Plant Mol Biol 43:177-200.
297. Huang, CJ; Zayas, JF, 1991. Aroma quality of corn germ protein flours determined by sensory and gas chromatographic profiles. J Food Qual 14:377-390.
298. Huang, J; Struck, F; Matzinger, DF; Levings, CS, 1991. Functional analysis in yeast of cDNA coding for the mitochondrial Rieske iron-sulfur protein of higher plants. Proc Natl Acad Sci USA 88:10716-10720.
299. Hudon, M; Pitblado, RE; Hamilton, RI; Bourgeois, G; Kuzir, S; Girgis, R, 1991. Response of maize inbred lines to 2 European corn borer (*Ostrinia nubilalis*) strains in Canada. Phytoprotection 72:69-76.
300. Hudspeth, RL; Grula, JW; Dai, Z; Edwards, GE; Ku, MSB, 1992. Expression of maize phosphoenolpyruvate carboxylase in transgenic tobacco - effects on biochemistry and physiology. Plant Physiol 98:458-464.
301. Hughes, MA; Dunn, MA; Pearce, RS; White, AJ; Zhang, L, 1992. An abscisic-acid-responsive, low temperature barley gene has homology with a maize phospholipid transfer protein. Plant Cell Environ 15:861-865.
302. Hunt, CW; Kezar, W; Vinande, R, 1992. Yield, chemical composition, and ruminal fermentability of corn whole plant, ear, and stover as affected by hybrid. J Prod Agric 5:286-290.
303. Hunter, T, 1991. Protein-serine kinase receptors? Curr Biol 1:15-16.
304. Huynh, QK; Borgmeyer, JR; Zobel, JF, 1992. Isolation and characterization of a 22-kDa protein with antifungal properties from maize seeds. Biochem Biophys Res Commun 182:1-5.
305. Huynh, QK; Hironaka, CM; Levine, EB; Smith, CE; Borgmeyer, JR; Shah, DM, 1992. Antifungal proteins from plants - purification, molecular cloning, and antifungal properties of chitinases from maize seed. J Biol Chem 267:6635-6640.
306. Igloi, GL; Kossel, H, 1992. The transcriptional apparatus of chloroplasts. Crit Rev Plant Sci 10:525-558.
307. Inagaki, MN; Alek, W; Tahir, M, 1991. A comparison of haploid production frequencies in barley crossed with maize and *Hordeum bulbosum* L. Cereal Res Commun 19:385-390.
308. Inagaki, MN; Tahir, M, 1992. Production of haploid wheat through intergeneric crosses. Hereditas 116:117-120.
309. Ingels, SC; Bennetzen, JL; Hulbert, SH; Qin, M; Ellingboe, AH, 1992. Mutator transposable elements that occur in clusters in the maize genome. J Hered 83:114-118.
310. Inouchi, N; Glover, DV; Sugimoto, Y; Fuwa, H, 1991. DSC characteristics of gelatinization of starches of single-mutants, double-mutants, and triple-mutants and their normal counterpart in the inbred Oh43 maize (*Zea mays* L.) background. Starch 43:468-472.
311. Inouchi, N; Glover, DV; Sugimoto, Y; Fuwa, H, 1991. DSC characteristics of retrograded starches of single-mutants, double-mutants, and triple-mutants and their normal counterpart in the inbred Oh43 maize (*Zea mays* L.) background. Starch 43:473-477.
312. Inoue, Y; Mochizuki, N; Koinuma, K; Kato, A, 1991. Development and characteristics of new maize parental line NA 7. Bull Natl Grassl Res Inst 43-52.
313. Ito, GM; Brewbaker, JL, 1991. Genetic analysis of pericarp thickness in progenies of 8 corn hybrids. J Amer Soc Hort Sci 116:1072-1077.

314. Ivanov, S, 1991. Combining ability and some genetic characters of inbred high lysine maize lines homozygous for the gene opaque-2. I. Grain yield. Genet Sel 24:327-334.
315. Ivanovic, D; Levic, J; Sataric, I, 1992. Inheritance of resistance to maize dwarf mosaic virus in some maize hybrids. Maydica 37:293-298.
316. Jablonka, E; Lachmann, M; Lamb, MJ, 1992. Evidence, mechanisms and models for the inheritance of acquired characters. J Theor Biol 158:245-268.
317. Jackson, LL; Dewald, CL; Bohlen, CC, 1992. A macromutation in *Tripsacum dactyloides* (Poaceae) - consequences for seed size, germination, and seedling establishment. Amer J Bot 79:1031-1038.
318. Jacobs, BC; Pearson, CJ, 1992. Pre-flowering growth and development of the inflorescences of maize. 1. Primordia production and apical dome volume. J Exp Bot 43:557-563.
319. Jacobs, BC; Pearson, CJ, 1992. Pre-flowering growth and development of the inflorescences of maize. 2. Accumulation and partitioning of dry matter and nitrogen by inflorescences. J Exp Bot 43:565-569.
320. Jansson, S; Pichersky, E; Bassi, R; Green, BR; Ikeuchi, M; Melis, A; Simpson, DJ; Spangfort, M; Staehelin, LA; Thornber, JP, 1992. A nomenclature for the genes encoding the chlorophyll a/b-binding proteins of higher plants. Plant Mol Biol Rep 10:242-253.
321. Jarchow, E; Grimsley, NH; Hohn, B, 1991. virF, the host-range-determining virulence gene of *Agrobacterium tumefaciens*, affects T-DNA transfer to *Zea mays*. Proc Natl Acad Sci USA 88:10426-10430.
322. Jingru, L; Yonglian, Z; Jilin, L, 1992. The study of the relativity between the plasmid-likes in mitochondria and unstable fertility of cms-S in maize. Acta Genet Sin 19:47-54.
323. Joanin, P; Gigot, C; Philipps, G, 1992. Nucleotide sequence and expression of 2 cDNA coding for 2 histone H2B variants of maize. Plant Mol Biol 20:581-588.
324. Joanny, K; Reddy, AR, 1992. Subcellular localization of anthocyanin pigments in red and bronze (*bz1*) aleurone cells of maize. Maydica 37:165-167.
325. Johal, GS; Briggs, SP, 1992. Reductase activity encoded by the *HM1* disease resistance gene in maize. Science 258:985-987.
326. John, I; Wang, HQ; Held, BM; Wurtele, ES; Colbert, JT, 1992. An messenger RNA that specifically accumulates in maize roots delineates a novel subset of developing cortical cells. Plant Mol Biol 20:821-831.
327. Jolliot, A; Vignols, F; Baillet, B; Arondel, V; Tchang, F; Kader, JC, 1990. Organ specificity of LTP mRNA distribution in maize seedlings. Pp. 468-470 in Plant Lipid Biochemistry, Structure and Utilization. P. J. Quinn and J. L. Harwood, ed., London: Portland Press.
328. Jones, AM; Prasad, PV, 1992. Auxin-binding proteins and their possible roles in auxin-mediated plant cell growth. Bioessays 14:43-48.
329. Jones, JDG; Bishop, G; Carroll, B; Dickinson, M; English, J; Harrison, K; Jones, D; Scofield, S; Thomas, CM, 1992. Prospects for establishing a tomato gene tagging system using the maize transposon activator (*Ac*). Proc Roy Soc Edinburgh Sect B 99:107-119.
331. Jordan, MO, 1992. Can rhizotrons be used for the study of corn (*Zea mays* L.) root ramification. Agronomie 12:3-14.
332. Jordan, MO; Picard, D; Trendel, R, 1992. Secondary ramification of corn (*Zea mays* L.) nodal roots - kinetics and structural data. Agronomie 12:15-30.
333. Jorgensen, JA; Weng, J; Ho, THD; Nguyen, HT, 1992. Genotype-specific heat shock proteins in 2 maize inbreds. Plant Cell Rep 11:576-580.
334. Jose-Estanyol, M; Ruiz-Avila, L; Puigdomenech, P, 1992. A maize embryo-specific gene encodes a proline-rich and hydrophobic protein. Plant Cell 4:413-423.
335. Joyce, CM; Villemur, R; Snustad, DP; Silflow, CD, 1992. Tubulin gene expression in maize (*Zea mays* L.) - change in isotype expression along the developmental axis of seedling root. J Mol Biol 227:97-107.
336. Jurgens, G, 1992. Genes to greens - embryonic pattern formation in plants. Science 256:487-488.
337. Kaeppler, HF; Somers, DA; Rines, HW; Cockburn, AF, 1992. Silicon carbide fiber-mediated stable transformation of plant cells. Theor Appl Genet 84:560-566.
338. Kahmann, R, 1992. Barbara McClintock - a personal view. Trends Genet 8:407.
339. Kaleikau, EK; Andre, CP; Walbot, V, 1992. Structure and expression of the rice mitochondrial apocytochrome-b gene (*cob-1*) and pseudogene (*cob-2*). Curr Genetics 22:463-470.
340. Kamara, A; Pressolska, P, 1992. Performance of heterosis and degrees of dominance of some characters in the single interlineal hybrid Knezha BP-556 and the modified single hybrid Knezha BMP-556. III. Number of ears per plant and length of ears. Genet Sel 25:328-334.
341. Kamikoga, ATM; Salgado, CL; Balmer, E, 1991. Reactions of different populations of popcorn (*Zea mays*) to *Helminthosporium turcicum*. Summa Phytopathol 17:100-104.
342. Kanakari, S; Timmler, G; Vonknoblauch, K; Subramanian, AR, 1992. Nucleotide sequence, map position and transcript pattern of the intron-containing gene for maize chloroplast ribosomal protein S16. Plant Mol Biol 18:419-422.
343. Katagiri, F; Chua, NH, 1992. Plant transcription factors - present knowledge and future challenges. Trends Genet 8:22-27.
344. Kawamura, T; Shigesada, K; Toh, H; Okumura, S; Yanagisawa, S; Izui, K, 1992. Molecular evolution of phosphoenolpyruvate carboxylase for C-4 photosynthesis in maize - comparison of its cDNA sequence with a newly isolated cDNA encoding an isozyme involved in the anaplerotic function. J Biochem Tokyo 112:147-154.
345. Keller, J; Lim, E; James, DW; Dooner, HK, 1992. Germinal and somatic activity of the maize element activator (*Ac*) in *Arabidopsis*. Genetics 131:449-459.
346. Ketcham, D, 1992. About Long Island - A very private legend. The New York Times, July 26, 1992.
347. Khush, GS; Brar, DS, 1991. Genetics of resistance to insects in crop plants. Adv Agron 45:223-274.
348. Khush, RS; Yoder, JI, 1992. Independent transposition of multiple *Ac* elements in the same transgenic tomato cell. Plant Mol Biol 18:1065-1072.
349. Kiel, C; Stamp, P, 1992. Internal root anatomy of maize seedlings (*Zea mays* L.) as influenced by temperature and genotype. Ann Bot 70:125-128.
350. Kindiger, B; Beckett, JB, 1992. Popcorn germplasm as a parental source for maize x *Tripsacum dactyloides* hybridization. Maydica 37:245-249.
351. Kindiger, B; Hamann, S, 1992. B-A chromosome mediated 3:1 disjunction in heterozygous reciprocal translocations of maize. Genome 35:714-718.

352. Kiss, HG; Evans, ML; Johnson, JD, 1991. Cytoplasmic calcium levels in protoplasts from the cap and elongation zone of maize roots. Protoplasma 163:181-188.
353. Klein, TM; Arentzen, R; Lewis, PA; Fitzpatrickmcelligott, S, 1992. Transformation of microbes, plants and animals by particle bombardment. Biotechnology 10:286-291.
354. Kloeckener-Gruissem, B; Vogel, JM; Freeling, M, 1992. The TATA box promoter region of maize *Adh1* affects its organ-specific expression. EMBO J 11:157-166.
355. Knight, ME; Ray, JA; Schuch, W, 1992. Isolation of a gene from maize encoding a chlorophyll a/b-binding protein. Plant Mol Biol 19:533-536.
356. Koch, KE; Nolte, KD; Duke, ER; McCarty, DR; Avigne, WT, 1992. Sugar levels modulate differential expression of maize sucrose synthase genes. Plant Cell 4:59-69.
357. Kolata, G, 1992. Dr. Barbara McClintock, 90, gene research pioneer dies. The New York Times, September 4, 1992.
358. Kong, F; Zhang, S; Han, L; Su, S; Hu, X; Zheng, C, 1991. Genetic potential and its utilization of opaque-2 maize in north China. Sci Agric Sin 24:11-19.
359. Korth, KL; Kaspi, CI; Siedow, JN; Levings, CS, 1991. *URF13*, a maize mitochondrial pore-forming protein, is oligomeric and has a mixed orientation in *Escherichia coli* plasma membranes. Proc Natl Acad Sci USA 88:10865-10869.
360. Kostandi, SF, 1992. Smut incidence and yield losses of corn cultivars under different plant population densities. J Agron Crop Sci 168:201-207.
361. Koternyak, VV; Karaivanov, GP, 1991. Instable mutation at the opaque-2 locus in maize. Genetika 27:1814-1819.
362. Koutsos, T; Koutsika-Sotiriou, M; Fasoulas, AC, 1992. The impact of genotype x soil texture interaction on the efficiency of selection for yield in maize (*Zea mays* L.). Euphytica 61:61-65.
363. Kovacs, G; Hoang, NG; Gaborjanyi, R, 1991. Resistance of inbred maize lines and sorghum species to Hungarian maize pathogenic potyviruses. Novenytermeles 40:289-294.
364. Kovacs, G; Kovacs, M; Barnabas, B, 1992. Studying the effect of genotype and induction nutrient medium in maize anther culture. Novenytermeles 41:193-200.
365. Kovacs, J; Csapo, B; Paldi, E; Szigeti, Z, 1992. Chilling resistance of maize (*Zea mays* L.) inbred lines in early phase of development as characterized by fluorescence induction parameters. Novenytermeles 41:97-103.
366. Kovacs-Schneider, M; Mathegaspar, G; Mathe, P; Kovacs, G, 1991. Quality improvement of maize grain by modifying the rate of endosperm germ ratio. Novenytermeles 40:295-302.
367. Kovalskaya, VS; Kadura, SN; Savenkova, TN; Vanyushin, BF, 1992. Chromatin organization and genome methylation in inbred lines and heterosis hybrids of maize. Proc. Izv Akad Nauk SSSR Ser Biol 59-65.
368. Kowles, RV; McMullen, MD; Yerk, G; Phillips, RL; Kraemer, S; Srienc, F, 1992. Endosperm mitotic activity and endoreduplication in maize affected by defective kernel mutations. Genome 35:68-77.
369. Kowles, RV; Yerk, GL; Srienc, F; Phillips, RL, 1992. Maize endosperm tissue as an endoreduplication system. Pp. 65-88 in Genetic Engineering, Vol 14. J. K. Setlow, ed., New York: Plenum.
370. Kruleva, MM; Korol, AB; Dankov, TG; Skorpan, VG; Preygel, IA, 1992. The effect of genotype x cytoplasm interaction on meiotic behaviour of maize chromosomes. Genome 35:653-658.
371. Kubica, S; Baluska, F; Hauskrecht, M, 1991. Elemental growth rate and rRNA transcript maturation exhibit the same pattern in individual tissues of the maize root apex. Ann Bot 68:387-391.
372. Kuehnle, AR; Earle, ED, 1992. Evaluation of in vitro selection regimes for a mitochondrial trait (methomyl resistance) in cms-T maize. Plant Cell Tissue Organ Cult 28:129-137.
373. Kumar, H, 1992. Oviposition, larval arrest and establishment of *Chilo partellus* (Lepidoptera, pyralidae) on maize genotypes during anthesis. Bull Entomol Res 82:355-360.
374. Kumar, H; Saxena, KN, 1992. Resistance in certain maize cultivars to 1st-instar and 3rd-instar *Chilo partellus* larvae. Entomol Exp Appl 65:75-80.
375. Lambert, R, 1992. Keen on maize. New Sci 134:53.
376. Lamkey, KR, 1992. 50 Years of recurrent selection in the Iowa Stiff Stalk Synthetic maize population. Maydica 37:19-28.
377. Lanahan, MB; Ho, THD; Rogers, SW; Rogers, JC, 1992. A gibberellin response complex in cereal alpha-amylase gene promoters. plant cell 4:203-211.
378. Langridge, P; Brettschneider, R; Lazzeri, P; Lorz, H, 1992. Transformation of cereals via Agrobacterium and the pollen pathway - a critical assessment. Plant J 2:631-638.
379. Lapitan, NLV, 1992. Organization and evolution of higher plant nuclear genomes. Genome 35:171-181.
380. Larkins, BA; Lopes, MA; Lending, CR, 1992. A genetic, biochemical and ultrastructural analysis of modified opaque-2 maize. Pp. 174-204 in Quality Protein Maize. E. T. Mertz, ed., St. Paul: Amer Assoc Cereal Chem.
381. Lauriere, C; Doyen, C; Thevenot, C; Daussant, J, 1992. Beta-amylases in cereals - a study of the maize beta-amylase system. Plant Physiol 100:887-893.
382. Lavergne, D; Nato, A; Dupuis, JM; Pean, M; Chagvardieff, P, 1992. Evidence for the expression of morphological and biochemical characteristics of C-3-photosynthesis in chlorophyllous callus cultures of *Zea mays*. Physiol Plant 84:292-300.
383. Lazarus, CM; Napier, RM; Yu, L-X; Lynas, C; Venis, MA, 1991. Auxin-binding protein antibodies and genes. Pp. 129-148 in Molecular Biology of Plant Development. G. I. Jenkins and W. Schuch, ed., Cambridge: Company of Biologists.
384. Lee, JS; Eum, JS; Moon, HY; Sim, WS, 1992. Cloning of the *rbcl* gene from maize chloroplast. Korean J Bot 35:165-171.
385. Leite, A; Yunes, JA; Turcinelli, SR; Arruda, P, 1992. Cloning and characterization of a cDNA encoding a sulfur-rich coixin. Plant Mol Biol 18:171-174.
386. Leon, P; O'Brien-Vedder, C; Walbot, V, 1992. Expression of ORF1 of the linear 2.3 kb plasmid of maize mitochondria - product localization and similarities to the 130 kDa protein encoded by the S2 episome. Curr Genetics 22:61-67.
387. Leonardi, A; Damerval, C; DeVienne, D, 1991. Heterosis for genetic expression during development. Pp. 175-182 in Physiologie et Production du Mais. D. Picard, ed., Paris: INRA.

388. Leu, JY; Sun, YH; Lai, YK; Chen, J, 1992. A maize cryptic *Ac*-homologous sequence derived from an Activator transposable element does not transpose. Mol Gen Genet 233:411-418.
389. Levings, CS; Siedow, JN, 1992. Molecular basis of disease susceptibility in the Texas cytoplasm of maize. Plant Mol Biol 19:135-147.
390. Levy, Y; Pataky, JK, 1992. Epidemiology of northern leaf blight on sweet corn. Phytoparasitica 20:53-66.
391. Li, HM; Sullivan, TD; Keegstra, K, 1992. Information for targeting to the chloroplastic inner envelope membrane is contained in the mature region of the maize *Bt1*-encoded protein. J Biol Chem 267:18999-19004.
392. Li, W; Song, T, 1991. Estimates of genetic parameters for 13 quantitative traits in a recombined high oil maize F2 population of IHO C80 x Alexho C23. Acta Agron Sin 17:470-475.
393. Li, W; Zhu, Q, 1992. A preliminary study of the callus formation and organogenesis and their ploidy of immature endosperm from maize. Acta Genet Sin 19:67-70.
394. Lima, M; Furlani, PR; deMiranda, JB, 1992. Divergent selection for aluminum tolerance in a maize (*Zea mays* L.) population. Maydica 37:123-132.
395. Liu, CN; Rubenstein, I, 1992. Genomic organization of an alpha-zein gene cluster in maize. Mol Gen Genet 231:304-312.
396. Liu, CN; Rubenstein, I, 1992. Molecular characterization of 2 types of 22 kilodalton alpha-zein genes in a gene cluster in maize. Mol Gen Genet 234:244-253.
397. Liu, K; Su, H; Cui, Y; Ma, C; Chen, W; Li, D, 1991. Reaction of different male-sterile cytoplasm subgroups of the C group maize to the infection of *Bipolaris maydis* race C. Sci Agric Sin 24:58-60.
398. Liu, Z; He, Q; Xie, Y; Dai, J; Mi, J, 1992. Construction and identification of vectors containing maize plasmid-like DNA S-1 and reporter genes. Acta Genet Sin 19:61-66.
399. Livini, C; Ajmone-Marsan, P; Melchinger, AE; Messmer, MM; Motto, M, 1992. Genetic diversity of maize inbred lines within and among heterotic groups revealed by RFLPs. Theor Appl Genet 84:17-25.
400. Livini, C; Pirovano, L; Brandolini, A; Lorenzoni, C; Motto, M, 1992. Evaluation of 3 cycles of recurrent selection for yield in an opaque-2 variety of maize. Maydica 37:89-93.
401. Lloyd, AM; Walbot, V; Davis, RW, 1992. *Arabidopsis* and *Nicotiana* anthocyanin production activated by maize regulator-*R* and regulator-*C1*. Science 258:1773-1775.
402. Lobreaux, S; Massenet, O; Briat, JF, 1992. Iron induces ferritin synthesis in maize plantlets. Plant Mol Biol 19:563-575.
403. Long, A; Benz, BF; Donahue, DJ; Jull, AJT; Toolin, LJ, 1989. First direct AMS dates on early maize from Tehuacan Mexico. Radiocarbon 31:1035-1040.
404. Long, DM; Oaks, A; Rothstein, SJ, 1992. Regulation of maize root nitrate reductase messenger RNA levels. Physiol Plant 85:561-566.
405. Lopes, MA; Larkins, BA, 1991. Gamma-zein content is related to endosperm modification in quality protein maize. Crop Sci 31:1655-1662.
406. Lorens, GF; Blechl, AE; Anderson, OD; Greene, FC, 1991. Wheat storage protein gene promoter activity in maize endosperm protoplasts. Pp. 666-671 in Gluten Proteins 1990.
407. Lorgeou, J, 1991. References for grain varieties. Pp. 291-306 in Physiologie et Production du Mais. D. Picard, ed., Paris: INRA.
408. Louis-Alexandre, A; Mestres, C; Faure, J, 1991. Measurement of endosperm vitreousness of corn - a quantitative method and its application to African cultivars. Cereal Chem 68:614-617.
409. Lowe, B; Mathern, J; Hake, S, 1992. Active mutator elements suppress the knotted phenotype and increase recombination at the *Kn1-O* tandem duplication. Genetics 132:813-822.
410. Lu, BW; Hanson, MR, 1992. A single nuclear gene specifies the abundance and extent of RNA editing of a plant mitochondrial transcript. Nucleic Acids Res 20:5699-5703.
411. Lu, GG; Campbell, W; Lindqvist, Y; Schneider, G, 1992. Crystallization and preliminary crystallographic studies of the FAD domain of corn NADH - nitrate reductase. J Mol Biol 224:277-279.
412. Lu, GH; DeLisle, AJ; deVetten, NC; Ferl, RJ, 1992. Brain proteins in plants - an *Arabidopsis* homolog to neurotransmitter pathway activators is part of a DNA binding complex. Proc Natl Acad Sci USA 89:11490-11494.
413. Lu, GH; Ferl, RJ, 1992. Site-Specific oligodeoxynucleotide binding to maize *Adh1* gene promoter represses *Adh1-GUS* gene expression in vivo. Plant Mol Biol 19:715-723.
414. Lu, WP; Kirkham, MB, 1991. Genotypic tolerance to metals as indicated by ethylene production. Pp. 605-616 in Proc. Internatl Conf on Metals in Soils, Waters, Plants and Animals, Orlando, FL.
415. Lu, WP; Kirkham, MB; Wassom, CE, 1991. Water and osmotic potentials of ethephon-treated maize genotypes varying in drought resistance. Biol Plant 33:354-357.
416. Luan, S; Bogorad, L, 1992. A rice cab gene promoter contains separate cis-acting elements that regulate expression in dicot and monocot plants. Plant Cell 4:971-981.
417. Ludevid, MD; Freire, MA; Gomez, J; Burd, CG; Albericio, F; Giralt, E; Dreyfuss, G; Pages, M, 1992. RNA binding characteristics of a 16 kDa glycine-rich protein from maize. Plant J 2:999-1003.
418. Luehrsen, KR; Walbot, V, 1992. Insertion of non-intron sequence into maize introns interferes with splicing. Nucleic Acids Res 20:5181-5187.
419. Lukina, LA; Yudin, BF, 1991. Parthogenetic polyhaploids in progenies of the 58-chromosomal maize x *Tripsacum* hybrid. Genet Manip Plants 7:81-87.
420. Luo, G; Hepburn, AG; Widholm, JM, 1992. Preparation of plant DNA for PCR analysis: a fast, general and reliable procedure. Plant Mol Biol Rep 10:319-323.
421. Lusardi, MC; Locatelli, F; Stadler, J; Lupotto, E, 1991. In vitro characterization of salt-selected maize genotypes. J Genet Breed 45:285-291.
422. Lyakh, VA; Soroka, AI, 1992. Influence of pollen storage in tassel on the quality of pollen grains and structure of resulting populations. Maydica 37:299-303.
423. Lyznik, LA; McGee, JD; Tung, PY; Bennetzen, JL; Hodges, TK, 1991. Homologous recombination between plasmid DNA molecules in maize protoplasts. Mol Gen Genet 230:209-218.
424. Ma, H; Wassom, CE; Liang, GH, 1991. Direct generation of maize haploids via anther culture. Cytologia 56:103-106.
425. MacDonald, MV, 1992. Donor plant growth factors affecting anther culture of maize and sweetcorn (*Zea mays*). Ann Bot 70:357-363.

426. MacRae, AF; Clegg, MT, 1992. Evolution of *Ac* and *Ds1* elements in select grasses (Poaceae). Genetica 86:55-66.
427. Maddaloni, M; Barbieri, L; Lohmer, S; Motto, M; Salamini, F; Thompson, R, 1991. Characterization of an endosperm-specific developmentally regulated protein synthesis inhibitor from maize seeds. J Genet Breed 45:377-380.
428. Magnavaca, R, 1992. Quality protein maize development in Brazil. Pp. 98-110 in Quality Protein Maize. E. T. Mertz, ed., St. Paul: Amer Assoc Cereal Chem.
429. Maguire, MP, 1992. The evolution of meiosis. J Theor Biol 154:43-55.
430. Maguire, MP; Paredes, AM; Riess, RW, 1991. The desynaptic mutant of maize as a combined defect of synaptonemal complex and chiasma maintenance. Genome 34:879-887.
431. Mahajan, V; Khehra, AS; Apal, SS, 1991. Stability analysis in maize (*Zea mays* L.) over diverse seasons. Crop Res (Hisar) 4:264-267.
432. Mahajan, V; Khehra, AS; Pal, SS; Gupta, AS, 1992. Combining ability of plant height and leaves gained per day in maize. Crop Res 5:391-394.
433. Mahajan, V; Patel, CS, 1992. Cold tolerance at vegetative stage in maize in valley lands of Meghalaya. Crop Res 5:333-338.
434. Mahajan, V; Patel, CS; Gupta, AS, 1992. Stability analysis over varying plant densities in maize. Crop Rs 5:294-297.
435. Maier, RM; Hoch, B; Zeltz, P; Kossel, H, 1992. Internal editing of the maize chloroplast *ndhA* transcript restores codons for conserved amino acids. Plant Cell 4:609-616.
436. Mariat, D; Vergnaud, G, 1992. Detection of polymorphic loci in complex genomes with synthetic tandem repeats. Genomics 12:454-458.
437. Marivet, J; Frendo, P; Burkard, G, 1992. Effects of abiotic stresses on cyclophilin gene expression in maize and bean and sequence analysis of bean cyclophilin cDNA. Plant Sci 84:171-178.
438. Marques, CAD; Osuna, JA, 1991. Selection among endogamic S-1 progenies for corn earworm resistance in 2 opaque-2 maize populations. Pesquisa Agr Brasil 26:1859-1864.
439. Marquez-Sanchez, F, 1992. Inbreeding and yield prediction in synthetic maize cultivars made with parental lines. 1. Basic methods. Crop Sci 32:345-349.
440. Marquez-Sanchez, F, 1992. Backcross theory for maize. 2. Additive genetic variance and response to selection. Maydica 37:225-229.
441. Marquez-Sanchez, F, 1992. On the yield prediction of composite varieties of maize. Maydica 37:271-274.
442. Marshall, LC; Somers, DA; Dotray, PD; Gengenbach, BG; Wyse, DL; Gronwald, JW, 1992. Allelic mutations in acetyl-coenzyme a carboxylase confer herbicide tolerance in maize. Theor Appl Genet 83:435-442.
443. Martin, C; Jones, J, 1992. Plant genetics flourishes. Trends Genet 8:368-370.
444. Martinez-Barajas, E; Villanueva-Verduzco, C; Molina-Galan, J; Loza-Tavera, H; Sanchez-de-Jimenez, E, 1992. Relation of rubisco to maize grain yield improvement effect of water restriction. Crop Sci 32:718-722.
445. Mather, DE; Kannenberg, LW, 1992. Genetic simulation of ratio-based recurrent selection. Maydica 37:169-172.
446. McCarty, DR, 1992. The role of *VP-I* in regulation of seed maturation in maize. Biochem Soc Trans 20:89-92.
447. McCullough, AJ; Kangasjarvi, J; Gengenbach, BG; Jones, RJ, 1992. Plastid DNA in developing maize endosperm - genome structure, methylation, and transcript accumulation patterns. Plant Physiol 100:958-964.
448. McElroy, D; Blowers, AD; Jenes, B; Wu, R, 1991. Construction of expression vectors based on the rice actin 1 (act1) 5' region for use in monocot transformation. Mol Gen Genet 231:150-160.
449. McKendree, WL; Ferl, RJ, 1992. Functional elements of the *Arabidopsis Adh* promoter include the G-box. Plant Mol Biol 19:859-862.
450. McMichael, RW, Jr.; Lagarias, JC, 1990. Polycation-stimulated phytochrome phosphorylation: is phytochrome a protein kinase? Curr Top Plant Biochem Physiol 9:259-270.
451. McMurphy, LM; Rayburn, AL, 1992. Chromosomal and cell size analysis of cold tolerant maize. Theor Appl Genet 84:798-802.
452. Medford, JI, 1992. Vegetative apical meristems. Plant Cell 4:1029-1039.
453. Meeley, RB; Johal, GS; Briggs, SP; Walton, JD, 1992. A biochemical phenotype for a disease resistance gene of maize. Plant Cell 4:71-77.
454. Meeley, RB; Walton, JD, 1991. Enzymatic detoxification of Hc-toxin the host-selective cyclic peptide from *Cochliobolus carbonum*. Plant Physiol 97:1080-1086.
455. Mehta, H; Sarkar, KR, 1992. Heterosis for leaf photosynthesis, grain yield and yield components in maize. Euphytica 61:161-168.
456. Mehta, H; Sarkar, KR; Sharma, SK, 1992. Genetic analysis of photosynthesis and productivity in corn. Theor Appl Genet 84:242-255.
457. Melchinger, AE; Boppenmaier, J; Dhillon, BS; Pollmer, WG; Herrmann, RG, 1992. Genetic diversity for RFLPs in European maize inbreds. 2. Relation to performance of hybrids within versus between heterotic groups for forage traits. Theor Appl Genet 84:672-681.
458. Mertz, ET, 1992. Discovery of high lysine high tryptophan cereals. Pp. 1-8 in Quality Protein Maize. E. T. Mertz, ed., St. Paul: Amer Assoc Cereal Chem.
459. Messing, J; Fisher, H, 1991. Maternal effect on high methionine levels in hybrid corn. J Biotechnol 21:229-237.
460. Messmer, MM; Melchinger, AE; Boppenmaier, J; Herrmann, RG; Brunklaus-Jung, E, 1992. RFLP analyses of early-maturing European maize germplasm. I. Genetic diversity among flint and dent inbreds. Theor Appl Genet 83:1003-1012.
461. Messmer, MM; Melchinger, AE; Lee, M; Woodman, WL; Lee, EA; Lamkey, KR, 1991. Genetic diversity among progenitors and elite lines from the Iowa Stiff Stalk Synthetic (BSSS) maize population - comparison of allozyme and RFLP data. Theor Appl Genet 83:97-107.
462. Meyer, P; Linn, F; Heidmann, I; Meyer, H; Niedenhof, I; Saedler, H, 1992. Endogenous and environmental factors influence 35S promoter methylation of a maize *A1* gene construct in transgenic petunia and its colour phenotype. Mol Gen Genet 231:345-352.
463. Miao, S-H; He, L-M; Xiao, L, 1992. Fertility, inheritance and amino acid analysis of lysine plus threonine-resistant mutant progenies of maize. Acta Bot Sin 34:90-95.
464. Miernyk, JA, 1992. Purification and characterization of the major acid phosphatase isozyme secreted by maize endosperm cultures. Phytochemistry 31:2613-2616.
465. Miernyk, JA; Duck, NB; Shatters, RG; Folk, WR, 1992. The 70-kilodalton heat shock cognate can act as a molecular chaperone during the membrane translocation of a plant secretory protein precursor. Plant Cell 4:821-829.
466. Mihailovic, N; Jelic, G; Filipovic, R; Djurdjevic, M; Dzeletovic, Z, 1992. Effect of nitrogen form on maize response to drought stress. Plant Soil 144:191-197.
467. Mikus, M; Bobak, M; Lux, A, 1992. Structure of protein bodies and elemental composition of phytin from dry germ of maize (*Zea mays* L.). Bot Acta 105:26-33.

468. Milas, S, 1990. Relationship between parameters of stability and correlation coefficients for yield and yield components for some maize and wheat genotypes. Poljopr Znan Smotra 55:245-278.
469. Miller, ME; Chourey, PS, 1992. The maize invertase-deficient miniature-1 seed mutation is associated with aberrant pedicel and endosperm development. Plant Cell 4:297-305.
470. Ming, D, 1991. The application of the method of weighted least squares on the basis of lot means of generations to analyse the quantitative character of maize. Acta Genet Sin 18:513-519.
471. Mo, YY; Nagel, C; Taylor, LP, 1992. Biochemical complementation of chalcone synthase mutants defines a role for flavonols in functional pollen. Proc Natl Acad Sci USA 89:7213-7217.
472. Moffat, AS, 1992. Improving plant disease resistance. Science 257:482-483.
473. Molot, PM; Mas, P; Pivot, V; Bruneteau, M; Staron, T; Ferriere, H; Lemaire, JM, 1992. Measurement of electrolyte leakage to assess host susceptibility and elicitor-induced resistance: Study of 3 experimental models. Agronomie 12:105-113.
474. Moneger, F; Mandaron, P; Niogret, MF; Freyssinet, G; Mache, R, 1992. Expression of chloroplast and mitochondrial genes during microsporogenesis in maize. Plant Physiol 99:396-400.
475. Montero, LM; Filipski, J; Gil, P; Capel, J; Martinez-Zapater, JM; Salinas, J, 1992. The distribution of 5 methylcytosine in the nuclear genome of plants. Nucl Acid Res 20:3207-3210.
476. Montoliu, L; Rigau, J; Puigdomenech, P, 1992. Analysis by PCR of the number of homologous genomic sequences to alpha-tubulin in maize. Plant Sci 84:179-185.
477. Moore, I; Feldwisch, J; Campos, N; Zettl, R; Brzobohaty, B; Bako, L; Schell, J; Palme, K, 1992. Auxin-binding proteins of *Zea mays* identified by photoaffinity labelling. Biochem Soc Trans 20:70-73.
478. Moreno, MA; Chen, J; Greenblatt, I; Dellaporta, SL, 1992. Reconstitutional mutagenesis of the maize *P*-Gene by short-range *Ac* transpositions. Genetics 131:939-956.
479. Morizet, J; Tribol, A-M; Pollascek, M, 1991. Drought resistance of corn. Pp. 167-174 in Physiologie et Production du Mais. D. Picard, ed., Paris: INRA.
480. Morse, S; Wratten, SD; Edwards, PJ; Niemeyer, HM, 1991. Changes in the hydroxamic acid content of maize leaves with time and after artificial damage - implications for insect attack. Ann Appl Biol 119:239-249.
481. Motte, PM; Loppes, R; Menager, M; Deltour, R, 1991. Three-dimensional electron microscopy of ribosomal chromatin in two higher plants - a cytochemical, immunocytochemical and in situ hybridization approach. J Histochem Cytochem 39:1495-1506.
482. Muimba-Kankolongo, A; Bergstrom, GC, 1992. Wound predisposition of maize to anthracnose stalk rot as affected by internode position and inoculum concentration of *Colletotrichum graminicola*. Plant Dis 76:188-195.
483. Muise, RC; Hauswith, WW, 1992. Transcription in maize mitochondria - effects of tissue and mitochondrial genotype. Curr Genetics 22:235-242.
484. Mukuru, SZ, 1992. Breeding for grain mold resistance. Pp. 273-285 in Sorghum and Millets Diseases: A Second World Review. W. A. J. De Milliano, R. A. Frederiksen and G. D. Bengston, ed., Patancheru, Andhra Pradesh, India: ICRISAT.
485. Mulder, MM; Dolstra, O; Boon, JJ, 1991. Pyrolysis mass spectrometry as a scanning tool for plant breeders - a study of 2 public inbred lines of *Zea mays*. Pp. 291-307 in Production and Utilization of Lignocellulosics.
486. Munteanu, I; Munteanu, M, 1992. Pathogenesis, pathosystems and genetics of the host-pathogen interaction in plants. Part III. Genetics of pathosystems and genes for disease resistance in plants. Stud Univ Babes-Bolyai Biol 35:8-30.
487. Murray, MG; Pitas, JW; DeMars, SJ; Cramer, JH, 1992. Long-lived Southern blots using RNA probes. Plant Mol Biol Rep 10:173-177.
488. Myers, PN; Setter, TL; Madison, JT; Thompson, JF, 1992. Endosperm cell division in maize kernels cultured at 3 levels of water potential. Plant Physiol 99:1051-1056.
489. Napier, RM; Fowke, LC; Hawes, C; Lewis, M; Pelham, HRB, 1992. Immunological evidence that plants use both HDEL and KDEL for targeting proteins to the endoplasmic reticulum. J Cell Sci 102:261-271.
490. Napier, RM; Venis, MA, 1992. Epitope mapping reveals conserved regions of an auxin-binding protein. Biochem J 284:841-845.
491. Nash, J; Walbot, V, 1992. Bronze-2 gene expression and intron splicing patterns in cells and tissues of *Zea mays* L. Plant Physiol 100:464-471.
492. Nelson, T; Dengler, NG, 1992. Photosynthetic tissue differentiation in C4 plants. Int J Plant Sci 153:S93-S105.
493. Nelson, T; Langdale, JA, 1992. Developmental genetics of C-4 photosynthesis. Annu Rev Plant Physiol 43:25-47.
494. Neucere, JN, 1992. Electrophoretic analysis of cationic proteins extracted from aflatoxin-resistant susceptible varieties of corn. J Agr Food Chem 40:1422-1424.
495. Newhouse, K; Singh, B; Shaner, D; Stidham, M, 1991. Mutations in corn (*Zea mays* L.) conferring resistance to imidazolinone herbicides. Theor Appl Genet 83:65-70.
496. Newhouse, K; Wang, T; Anderson, P, 1991. Imidazolinone-tolerant crops. Pp. 139-150 in Imidazolinone Herbicides. D. L. Shaner and S. L. O'Connor, ed., Boca Raton: CRC Press.
497. Ngong-Nassah, EN; Carson, ML; Wicks, ZW, 1992. Inheritance of resistance to leaf freckles and wilt caused by *Clavibacter michiganense* subsp. *nebraskense* in early maturing maize inbred lines. Phytopathology 82:142-146.
498. Nicholas, G, 1991. Corny problem. New Sci 132:57.
499. Nordquist, PT; Hergert, GW; Skates, BA; Compton, WA; Markwell, JP, 1992. Phenotypic expression of different maize hybrid genotypes grown on saline-sodic soil. J Plant Nutr 15:2137-2144.
500. Oard, JH; Dronavalli, S, 1992. Rapid isolation of rice and maize DNA for analysis by random-primer PCR. Plant Mol Biol Rep 10:236-241.
501. Ober, ES; Setter, TL, 1992. Water deficit induces abscisic acid accumulation in endosperm of maize viviparous mutants. Plant Physiol 98:353-356.
502. Oishi, MY; Bewley, JD, 1992. Premature drying, fluridone-treatment, and embryo isolation during development of maize kernels (*Zea mays* L.) induce germination, but the protein synthetic responses are different - potential regulation of germination and protein synthesis by abscisic acid. J Exp Bot 43:759-767.
503. Okagaki, RJ; Sullivan, TD; Schiefelbein, JW; Nelson, OE, 1992. Alternative 3' splice acceptor sites modulate enzymic activity in derivative alleles of the maize bronze1-mutable-13 allele. Plant Cell 4:1453-1462.

504. Olive, MR; Peacock, WJ; Dennis, ES, 1991. The anaerobic responsive element contains two GC-rich sequences essential for binding a nuclear protein and hypoxic activation of the maize *Adh1* promoter. Nucleic Acids Res 19:7053-7060.
505. Orman, BA; Lawrance, GD; Downes, PM; Phillips, DS; Ripberger, CJ, 1991. Assessment of maize inbred genetic purity by isozyme electrophoresis. Seed Sci Technol 19:527-535.
506. Ott, T; Nelsen-Salz, B; Doring, HP, 1992. PCR-aided genomic sequencing of 5' subterminal sequences of the maize transposable element activator (*Ac*) in transgenic tobacco plants. Plant J 2:705-711.
507. Paiva, E; Vasconcelos, MJV; Parentoni, SN; Gama, EEGE; Magnavaca, R, 1992. Selection of high quality protein sweet corn progenies through electrophoresis techniques. Pesquisa Agr Brasil 27:1213-1218.
508. Palaversic, B, 1992. Study of maize inbred lines and hybrids for resistance to stalk rot and breakage under natural and induced stress conditions. Poljopr Znan Smotra 57:53-77.
509. Palaversic, B; Brekalo, J; Rojc, M, 1991. Comparison of maize resistance to stalk rot under conditions of artificial infection with *Colletotrichum graminicola* ces G. W. Wils. and *Fusarium graminearum* Schwabe. Zast Bilja 42:39-50.
510. Palme, K; Diefenthal, T; Vingron, M; Sander, C; Schell, J, 1992. Molecular cloning and structural analysis of genes from *Zea mays* (L.) coding for members of the ras-related *ypt* gene family. Proc Natl Acad Sci USA 89:787-791.
511. Pan, Y-B; Peterson, PA, 1991. Newly activated germinal *Uq* elements in maize are clustered on one linkage group independently of the standard *Uq* element. Mol Gen Genet 229:161-174.
512. Panaccione, DG; Scott-Craig, JS; Pocard, JA; Walton, JD, 1992. A cyclic peptide synthetase gene required for pathogenicity of the fungus *Cochliobolus carbonum* on maize. Proc Natl Acad Sci USA 89:6590-6594.
513. Pareddy, DR; Petolino, JF, 1992. Maturation of maize pollen in vitro. Plant Cell Rep 11:535-539.
514. Parera, CA; Cantliffe, DJ, 1991. Improved germination and modified imbibition of shrunken-2 sweet corn by seed disinfection and solid matrix priming. J Amer Soc Hort Sci 116:942-945.
515. Parry, AD; Horgan, R, 1992. Abscisic acid biosynthesis in roots. 1. The identification of potential abscisic acid precursors, and other carotenoids. Planta 187:185-191.
516. Pastori, GM; Trippi, VS, 1992. Oxidative stress induces high rate of glutathione reductase synthesis in a drought-resistant maize strain. Plant Cell Physiol 33:957-961.
517. Pasztor, K; Borsos, O, 1990. Heritability and chemical composition of inbred maize (*Zea mays* L.) x teosinte (*Zea mays* ssp. *mexicana* Schrader-Iltis) hybrid. Novenytermeles 39:193-213.
518. Pataky, JK, 1992. Relationship between yield of sweet corn and northern leaf blight caused by *Exserohilum turcicum*. Phytopathology 82:370-375.
519. Paterson, AH; Tanksley, SD; Sorrells, ME, 1991. DNA markers in plant improvement. Pp. 39-90 in Advances in Agronomy, Vol 46. D. L. Sparks, ed., San Diego: Academic Press Inc.
520. Paulis, JW; Bietz, JA; Felker, FC; Nelsen, TC, 1992. Evaluating quality protein maize genotypes by reversed-phase high-performance liquid chromatography. Pp. 122-140 in Quality Protein Maize. E. T. Mertz, ed., St. Paul: Amer Assoc Cereal Chem.
521. Payne, GA, 1992. Aflatoxin in maize. Crit Rev Plant Sci 10:423-440.
522. Pejic, I; Kozumplik, V, 1990. Possibility of using local maize genotypes for development of breeding populations. Poljopr Znan Smotra 55:307-314.
523. Pellerin, S; Tardieu, F, 1991. Genetic variability and the effect of growth conditions on the number of nodal roots of maize. Pp. 123-132 in Physiologie et Production du Mais. D. Picard, ed., Paris: INRA.
524. Peplinski, AJ; Paulsen, MR; Bouzaher, A, 1992. Physical, chemical, and dry-milling properties of corn of varying density and breakage susceptibility. Cereal Chem 69:397-400.
525. Pereira de Souza, A; Jubier, M-F; Lejeune, B, 1992. The higher plant NAD5 mitochondrial gene - a conserved discontinuous transcription pattern. Curr Genet 22:75-82.
526. Petersen, WL; Sulc, S; Armstrong, CL, 1992. Effect of nurse cultures on the production of macro-calli and fertile plants from maize embryogenic suspension culture protoplasts. Plant Cell Rep 10:591-594.
527. Peterson, PA, 1992. Quantitative inheritance in the era of molecular biology. Maydica 37:7-18.
528. Pfarr, DG; Lamkey, KR, 1992. Comparison of methods for identifying populations for genetic improvement of maize hybrids. Crop Sci 32:670-677.
529. Pfarr, DG; Lamkey, KR, 1992. Evaluation of theory for identifying populations for genetic improvement of maize hybrids. Crop Sci 32:663-669.
530. Phillips, RL; Plunkett, DJ; Kaeppler, SM, 1992. Novel approaches to the induction of genetic variation and plant breeding implications. Pp. 389-408 in Plant Breeding in the 1990s. H. T. Stalker and J. P. Murphy, ed., Wallingford, UK: CAB Internatl.
531. Pisabarro, AG; Martin, WF; Peterson, PA; Saedler, H; Gierl, A, 1991. Molecular analysis of the ubiquitous (*Uq*) transposable element system of *Zea mays*. Mol Gen Genet 230:201-208.
532. Pla, M; Gomez, J; Goday, A; Pages, M, 1991. Regulation of the abscisic acid-responsive gene *rab28* in maize viviparous mutants. Mol Gen Genet 230:394-400.
533. Plana, M; Itarte, E; Eritja, R; Goday, A; Pages, M; Martinez, MC, 1991. Phosphorylation of maize RAB-17 protein by casein kinase-2. J Biol Chem 266:22510-22514.
534. Plewa, MJ, 1991. The biochemical basis of the activation of promutagens by plant cell systems. Pp. 287-296 in Plants for Toxicity Assessment. J. W. Gorsuch et al., ed., Philadelphia: Amer Soc Testing Materials.
535. Plotnikov, VK; Bakaldina, NB; Efimov, VA, 1991. Stability of corn zein messenger RNAs under conditions of normal and high temperatures. Sov Plant Physiol-Engl Tr 38:712-719.
536. Plotnikov, VK; Bakaldina, NB; Efimov, VA, 1991. Stability of zein mRNA in maize at normal and high temperatures. Fiziol Rast 38:981-990.
537. Pollacsek, M, 1992. Management of the *ig* gene for haploid induction in maize. Agronomie 12:247-251.
538. Pollak, LM; Torrescardona, S; Sotomayor-Rios, A, 1991. Evaluation of heterotic patterns among Caribbean and tropical X temperate maize populations. Crop Sci 31:1480-1483.

539. Prasad, SK; Singh, TP, 1990. Genetic diversity under different environments in maize (*Zea mays* L.). Indian J Genet Plant Breed 50:407-411.
540. Prasad, SK; Singh, TP, 1991. Phenotypic stability of some Indian and exotic genotypes of maize. Crop Improv 18:41-45.
541. Prasad, TK; Stewart, CR, 1992. cDNA clones encoding *Arabidopsis thaliana* and *Zea mays* mitochondrial chaperonin-HSP60 and gene expression during seed germination and heat shock. Plant Mol Biol 18:873-885.
542. Prasad, TVR; Krishnamurthy, K; Kailasam, C, 1992. Allometric relationships of growth components in maize (*Zea mays* L.) cultivars. J Agron Crop Sci 168:181-185.
543. Prasad, TVR; Krishnamurthy, K; Kailasam, C, 1992. Functional crop and cob growth models of maize (*Zea mays* L.) cultivars. J Agron Crop Sci 168:208-212.
544. Prasad, TVR; Krishnamurthy, K; Shivashankar, K, 1990. Canopy and growth differences in maize genotypes in relation to plant densities and nitrogen levels. Mysore J Agric Sci 24:437-444.
545. Premachandran, MN; Sarkar, KR, 1992. On the development of a new set of primary trisomics in an Indian maize. Indian J Genet 52:7-10.
546. Pring, DR; Mullen, JA; Kempken, F, 1992. Conserved sequence blocks 5' to start codons of plant mitochondrial genes. Plant Mol Biol 19:313-317.
547. Quarrie, SA, 1991. Implications of genetic differences in ABA accumulation for crop production. Pp. 227-244 in Abscisic Acid: Physiology and Biochemistry. W. J. Davies and H. G. Jones, ed., Oxford: Bios Scientific Publ.
548. Quayle, T; Feix, G, 1992. Functional analysis of the - 300 region of maize zein genes. Mol Gen Genet 231:369-374.
549. Radicella, JP; Brown, D; Tolar, LA; Chandler, VL, 1992. Allelic diversity of the maize-*B* regulatory gene - different leader and promoter sequences of 2 B-alleles determine distinct tissue specificities of anthocyanin production. Gene Develop 6:2152-2164.
550. Raina, AK; Kingan, TG; Mattoo, AK, 1992. Chemical signals from host plant and sexual behavior in a moth. Science 255:592-594.
551. Rakha, FA; Abou-Youssef, AY; Omar, AA; El-Bendary, AA; El-Fouly, MM, 1992. Genetic nature of phosphorus accumulation in maize. J Plant Nutr 15:501-512.
552. Ramesh, MS; Patil, SJ; Goud, JV; Patil, SS, 1990. Correlation, combining ability, and heterosis studies on husk number and shank length in maize. Indian J Genet Plant Breed 50:338-341.
553. Rapp, WD; Stern, DB, 1992. A conserved 11 nucleotide sequence contains an essential promoter element of the maize mitochondrial *atp1* gene. EMBO J 11:1065-1073.
554. Rashid, A; Peterson, PA, 1992. The RSS system of unidirectional cross-incompatibility in maize. 1. Genetics. J Hered 83:130-134.
555. Rashid, A; Peterson, PA, 1992. The RSS system of unidirectional cross-incompatibility in maize. 2. Cytology. Genome 35:560-564.
556. Rayburn, AL; Auger, JA; McMurphy, LM, 1992. Estimating percentage constitutive heterochromatin by flow cytometry. Exp Cell Res 198:175-178.
557. Raz, R; Jose, M; Moya, A; Martinez-Izquierdo, JA; Puigdomenech, P, 1992. Different mechanisms generating sequence variability are revealed indistinct regions of the hydroxyproline-rich glycoprotein gene from maize and related species. Mol Gen Genet 233:252-259.
558. Reddy, VK; Reddy, SM, 1992. Cyclopiazonic acid production by *Penicillium griseofulvum* in relation to different cultivars of maize. World J Microbiol Biotechnol 8:208-209.
559. Reid, LM; Arnason, JT; Nozzolillo, C; Hamilton, RI, 1991. Laboratory and field resistance to the European corn borer in maize germplasm. Crop Sci 31:1496-1502.
560. Reid, LM; Mather, DE; Arnason, JT; Hamilton, RI; Bolton, AT, 1992. Changes in phenolic constituents of maize silk infected with *Fusarium graminearum*. Can J Bot 70:1697-1702.
561. Reid, LM; Mather, DE; Hamilton, RI; Bolton, AT, 1992. Diallel analysis of resistance in maize to *Fusarium graminearum* infection via the silk. Can J Plant Sci 72:915-923.
562. Reid, LM; Mather, DE; Hamilton, RI; Bolton, AT, 1992. Genotypic differences in the resistance of maize silk to *Fusarium graminearum*. Can J Plant Pathol 14:211-214.
563. Riggin, TM; Isenhour, DJ; Espelie, KE, 1992. Effect on *Cotesia marginiventris* (Hymenoptera, Braconidae) when rearing host fall armyworm (Lepidoptera, Noctuidae) on meridic diet containing foliage from resistant or susceptible corn genotypes. Environ Entomol 21:214-219.
564. Riggin, TM; Wiseman, BR; Isenhour, DJ; Espelie, KE, 1992. Incidence of fall armyworm (Lepidoptera, noctuidae) parasitoids on resistant and susceptible corn genotypes. Environ Entomol 21:888-895.
565. Ristic, Z; Cass, DD, 1991. Morphological characteristics of leaf epidermal cells in lines of maize that differ in endogenous levels of abscisic acid and drought resistance. Bot Gaz 152:439-445.
566. Ristic, Z; Cass, DD, 1992. Chloroplast structure after water and high-temperature stress in 2 lines of maize that differ in endogenous levels of abscisic acid. Int J Plant Sci 153:186-196.
567. Ristic, Z; Gifford, DJ; Cass, DD, 1991. Heat shock proteins in 2 lines of *Zea mays* L that differ in drought and heat resistance. Plant Physiol 97:1430-1434.
568. Ristic, Z; Gifford, DJ; Cass, DD, 1992. Dehydration, damage to the plasma membrane and thylakoids, and heat-shock proteins in lines of maize differing in endogenous levels of abscisic acid and drought resistance. J Plant Physiol 139:467-473.
569. Rizzi, E; Balconi, C; Ajmone-Marsan, P; Salamini, F; Thompson, R; Motto, M, 1992. Growth requirements of the *o6* mutant and its relationship with the gene encoding the rip protein b-32 of maize endosperm. Maydica 37:275-281.
570. Roberts, JKM; Hooks, MA; Miaullis, AP; Edwards, S; Webster, C, 1992. Contribution of malate and amino acid metabolism to cytoplasmic pH regulation in hypoxic maize root tips studied using nuclear magnetic resonance spectroscopy. Plant Physiol 98:480-487.
571. Robertson, DS; Stinard, PS, 1992. Genetic regulation of somatic mutability of 2 *Mu*-induced *a1* mutants of maize. Theor Appl Genet 84:225-236.
572. Rocheford, TR; Kennell, JC; Pring, DR, 1992. Genetic analysis of nuclear control of *t-urf13/orf221* transcription in t cytoplasm maize. Theor Appl Genet 84:891-898.
573. Rodermel, SR, 1992. Nucleotide sequence of a maize chloroplast DNA fragment containing an inversion breakpoint, *trnG (GCC)*, *trnG (UCC)*, *trnfM*, and a *trnG* pseudogene. Nucleic Acids Res 20:5844.
574. Rodermel, SR; Bogorad, L, 1992. Nucleotide sequence of the photoregulated maize chloroplast *psaI* gene, encoding a 4.0-kilodalton component of photosystem-I. Plant Physiol 98:406-407.

575. Roeckel, P; Dupuis, I; Matthys-Rochon, E; Chaboud, A, 1991. Isolation of viable sperm cells from corn (*Zea mays*) pollen grains. Pp. 88-96 in Laboratory Guide for Cellular and Molecular Plant Biology. I. Negrutiu and G. B. Ghartichhetri, ed., Basel, Switzerland: Birkhauser Verlag.
576. Rogers, JC; Rogers, SW, 1992. Definition and functional implications of gibberellin and abscisic acid cis-acting hormone response complexes. Plant Cell 4:1443-1451.
577. Rohe, M; Schrunder, J; Tudzynski, P; Meinhardt, F, 1992. Phylogenetic relationships of linear, protein-primed replicating genomes. Curr Genet 21:173-176.
578. Rollo, F; Venanzi, FM; Amici, A, 1991. Nucleic acids in mummified plant seeds - biochemistry and molecular genetics of pre-Columbian maize. Genet Res 58:193-201.
579. Rommens, CMT; Rudenko, GN; Dijkwel, PP; vanHaaren, MJJ; Ouwerkerk, PBF; Blok, KM; Nijkamp, HJJ; Hille, J, 1992. Characterization of the *Ac*/*Ds* behaviour in transgenic tomato plants using plasmid rescue. Plant Mol Biol 20:61-70.
580. Rommens, CMT; Vanhaaren, MJJ; Buchel, AS; Mol, JNM; Vantunen, AJ; Nijkamp, HJJ; Hille, J, 1992. Transactivation of *Ds* by *Ac*-transposase gene fusions in tobacco. Mol Gen Genet 231:433-441.
581. Rozin, E, 1992. A new world of food (reprinted from Blue Corn and Chocolate, 1992). Food Technol 46:60-61.
582. Rozman, L, 1991. Inheritance of some maize (*Zea mays* L.) stalk and leaf. Poljopr Znan Smotra 56:251-264.
583. Rubino, DB; Davis, DW, 1991. Maintenance of tropical isozyme alleles during random mating in a semiexotic maize composite. J Hered 82:423-425.
584. Ruget, F, 1991. Climatic dependence of female floret number in maize. Pp. 161-166 in Physiologie et Production du Mais. D. Picard, ed., Paris: INRA.
585. Ruiz-Avila, L; Burgess, SR; Stiefel, V; Ludevid, MD; Puigdomenech, P, 1992. Accumulation of cell wall hydroxyproline-rich glycoprotein mRNA is an early event in maize embryo cell differentiation. Proc Natl Acad Sci USA 89:2414-2418.
586. Russell, DA; Sachs, MM, 1992. Protein synthesis in maize during anaerobic and heat stress. Plant Physiol 99:615-620.
587. Russell, JR; Irlbeck, NA; Hallauer, AR; Buxton, DR, 1992. Nutritive value and ensiling characteristics of maize herbage as influenced by agronomic factors. Anim Feed Sci Technol 38:11-24.
588. Russell, SH; Hoopes, JL; Odell, JT, 1992. Directed excision of a transgene from the plant genome. Mol Gen Genet 234:49-59.
589. Russell, WA, 1991. Genetic improvement of maize yields. Pp. 245-298 in Advances in Agronomy, Vol 46. D. L. Sparks, ed., San Diego: Academic Press Inc.
590. Russell, WA; Blackburn, DJ; Lamkey, KR, 1992. Evaluation of a modified reciprocal recurrent selection procedure for maize improvement. Maydica 37:61-67.
591. Sachs, MM, 1991. Molecular response to anoxic stress in maize. Pp. 129-139 in Plant Life Under Oxygen Deprivation. M. B. Jackson, D. D. Davies and H. Lambers, ed., The Hague: Academic Publishing.
592. Sakakibara, H; Kawabata, S; Takahashi, H; Hase, T; Sugiyama, T, 1992. Molecular cloning of the family of glutamine synthetase genes from maize - expression of genes for glutamine synthetase and ferredoxin-dependent glutamate synthase in photosynthetic and non-photosynthetic tissues. Plant Cell Physiol 33:49-58.
593. Sanchez, GJJ; Goodman, MM, 1992. Relationships among the Mexican races of maize. Econ Bot 46:72-85.
594. Sanchez, JJ; Goodman, MM, 1992. Relationships among Mexican and some North American and South American races of maize. Maydica 37:41-51.
595. Sanchez-Nieto, S; Rodriguez-Sotres, R; Gonzalez-Romo, P; Bernal-Lugo, I; Gavilanes-Ruiz, M, 1992. Tonoplast and plasma membrane ATPases from maize lines of high or low vigour. Seed Sci Res 2:105-111.
596. Santos, MS; Filho Miranda, JB, 1992. Genetic potential of two Brazilian races of maize (*Zea mays* L.) for breeding purposes. J Genet Breed 46:83-90.
597. Sanwo, MM; Demason, DA, 1992. Characteristics of alpha-amylase during germination of 2 high-sugar sweet corn cultivars of *Zea mays* L. Plant Physiol 99:1184-1192.
598. Saraiva, LS, 1992. Analysis of the delayed knob replication hypothesis induced by B chromosomes in maize. Rev Bras Genet 15:623-630.
599. Sari-Gorla, M; Pe, ME; Mulcahy, DL; Ottaviano, E, 1992. Genetic dissection of pollen competitive ability in maize. Heredity 69:423-430.
600. Sarkar, KR; Mehta, H; Sharma, SK, 1991. Genetic analysis of the structure and function of the photosynthetic apparatus and its efficiency - a review. Indian J Genet 51:1-27.
601. Sauvaire, D; Sanou, J, 1989. IRAT 296 - an example of temperate germplasm introgression into tropical plant material in maize. Agron Trop 44:197-202.
602. Savenkova, TN; Koval'skaya, VS; Vanyushin, BF, 1991. Kinetics of DNA reassociation in inbred strains and heterosis hybrids of maize. Biol Nauki 16-21.
603. Saville, BJ; Leong, SA, 1992. The molecular biology of pathogenesis in *Ustilago maydis*. Pp. 139-162 in Genetic Engineering, Vol 14. J. K. Setlow, ed., New York: Plenum.
604. Scandalios, JG, 1992. Regulation of the antioxidant defense genes *Cat* and *Sod* of maize. Pp. 117-152 in Current Communications in Cell and Molecular Biology, Vol. 5. J. G. Scandalios, ed., Plainview, NY: Cold Spring Harbor Lab Press.
605. Schaffner, AR; Sheen, J, 1992. Maize C4 photosynthesis involves differential regulation of phosphoenolpyruvate carboxylase genes. Plant J 2:221-232.
606. Schlappi, M; Hohn, B, 1992. Competence of immature maize embryos for Agrobacterium-mediated gene transfer. Plant Cell 4:7-16.
607. Schmidt, RJ; Ketudat, M; Aukerman, MJ; Hoschek, G, 1992. Opaque-2 is a transcriptional activator that recognizes a specific target site in 22-kD-zein genes. Plant Cell 4:689-700.
608. Schmitz, G; Theres, K, 1992. Structural and functional analysis of the *Bz2* locus of *Zea mays* - characterization of overlapping transcripts. Mol Gen Genet 233:269-277.
609. Schneider, G; Jensen, E; Spray, CR; Phinney, BO, 1992. Hydrolysis and reconjugation of gibberellin-A(20) glucosyl ester by seedlings of *Zea mays* L. Proc Natl Acad Sci USA 89:8045-8048.
610. Schwartz, D, 1993. Marcus M. Rhoades (1903-1991). Genetics 133:1-3.
611. Scofield, SR; Harrison, K; Nurrish, SJ; Jones, JDG, 1992. Promoter fusions to the activator transposase gene cause distinct patterns of dissociation excision in tobacco cotyledons. Plant Cell 4:573-582.

612. Scott, GE; Zummo, N, 1992. Registration of MP420 germplasm line of maize. Crop Sci 32:1296.
613. Seitz, G; Geiger, HH; Schmidt, GA; Melchinger, AE, 1992. Genotypic correlations in forage maize. 2. Relationship between inbred line and testcross performance. Maydica 37:101-105.
614. Selmani, A; Wassom, CE, 1991. Effect of mild drought on chlorophyll fluorescence and morphological traits in young maize seedlings. Trans Kans Acad Sci 94:85-94.
615. Sen, A; Miller, SS; Arnason, JT; Fulcher, RG, 1991. Quantitative determination by high performance liquid chromatography and microspectrofluorimetry of phenolic acids in maize grain. Phytochem Analysis 2:225-229.
616. Shaanker, RU; Ganeshaiah, KN, 1991. Gene wars. Curr Sci 61:440-441.
617. Shapiro, JA, 1992. McClintock,Barbara 1902-1992. Bioessays 14:791-792.
618. Sharma, BD; Hore, DK, 1991. Stability performance of maize (*Zea mays*) germplasm lines in acidic and phosphorus-deficient soils. Indian J Agr Sci 61:904-908.
619. Sharma, JK; Bhalla, SK, 1991. Path analysis on some drought tolerant lines of maize. Crop Improv 18:32-36.
620. Shattuck-Eidens, DM; Bell, RN; Mitchell, JT; McWhorter, VC, 1991. Rapid detection of maize DNA sequence variation. Genet Anal-Tech Appl 8:240-245.
621. Shaw, JR; Hannah, LC, 1992. Genomic nucleotide sequence of a wild-type shrunken-2 allele of *Zea mays*. Plant Physiol 98:1214-1216.
622. Shckorbatov, YG, 1991. Light-induced pH changes in water containing submerged *Zea mays* (L.) leaves in connection with heterosis. Stud Biophys 140:171-177.
623. Shen, WH; Das, S; Hohn, B, 1992. Mechanism of *Ds1* excision from the genome of maize streak virus. Mol Gen Genet 233:388-394.
624. Shen, WH; Hohn, B, 1992. Excision of a transposable element from a viral vector introduced into maize plants by agroinfection. Plant J 2:35-42.
625. Shopova, K; Khristov, K, 1991. Physiological and genetic studies of the maize mutant line Xm-532. Genet Sel 24:321-326.
626. Shtemenko, NI; Vinnichenko, AN; Mazhara, SV, 1991. Mutation and amino acid composition of the vegetation organs of maize. Dokl Vses Ordena Lenina Ordena Trud Krasnogo Znameni Akad S-Kh Nauk Im V I Lenina 20-26.
627. Shukla, TP, 1992. Trends in zein research and utilization - food technology. Cereal Food World 37:225.
628. Sibale, EM; Darrah, LL; Zuber, MS, 1992. Comparison of 2 rind penetrometers for measurement of stalk strength in maize. Maydica 37:111-114.
629. Sibale, EM; Darrah, LL; Zuber, MS; Sleper, DA, 1992. Correlated responses to selection for stalk crushing strength in 2 maize populations. Maydica 37:115-122.
630. Singh, M; Brown, GG, 1991. Suppression of cytoplasmic male sterility by nuclear genes alters expression of a novel mitochondrial gene region. Plant Cell 3:1349-1362.
631. Singh, RP; Sehgal, KL; Bakhshi, AK, 1991. Screening for popping quality in popcorn. J Food Sci Technol-Mysore 28:319-320.
632. Singh, SN; Singh, KN; Singh, HG, 1991. Genetic studies for alkaline resistance in varietal crosses of maize (*Zea mays* L.). Narendra Deva J Agric Res 6:97-102.
633. Sinibaldi, RM; Mettler, IJ, 1992. Intron splicing and intron-mediated enhanced expression in monocots. Pp. 229-257 in Progress in Nucleic Acid Research and Molecular Biology, Vol. 42. W. E. Cohn and K. Moldave, ed., San Diego: Academic Press.
634. Smiryaev, AV; Tarutina, LA, 1991. Regression models of variability of quantitative characters of F1 maize hybrids. Tsitol Genet 25:23-30.
635. Smith, JSC; Smith, OS; Wright, S; Wall, SJ; Walton, M, 1992. Diversity of United-States hybrid maize germplasm as revealed by restriction fragment length polymorphisms. Crop Sci 32:598-604.
636. Smith, LG; Greene, B; Veit, B; Hake, S, 1992. A dominant mutation in the maize homeobox gene, knotted-1, causes its ectopic expression in leaf cells with altered fates. Development 116:21.
637. Smith, LG; Hake, S, 1992. The initiation and determination of leaves. Plant Cell 4:1017-1027.
638. Smith, OS; Smith, JSC, 1992. Measurement of genetic diversity among maize hybrids - a comparison of isozymic, RFLP, pedigree, and heterosis data. Maydica 37:53-60.
639. Smith, RD; Walker, JC, 1991. Isolation and expression of a maize type 1 protein phosphatase. Plant Physiol 97:677-683.
640. Smith-White, BJ; Preiss, J, 1992. Comparison of proteins of ADP-glucose pyrophosphorylase from diverse sources. J Mol Evol 34:449-464.
641. Songstad, DD; Petersen, WL; Armstrong, CL, 1992. Establishment of friable embryogenic (Type-II) callus from immature tassels of *Zea mays* (Poaceae). Amer J Bot 79:761-764.
642. Souciet, G; Weil, JH, 1992. Molecular cloning and sequencing of a cDNA encoding the maize (*Zea mays* L.) acyl carrier protein (ACP) - comparison to other ACP genes and ACPs. Plant Sci 81:215-225.
643. South, JB; Morrison, WR; Nelson, OE, 1991. A relationship between the amylose and lipid contents of starches from various mutants for amylose content in maize. J Cereal Sci 14:267-278.
644. Southworth, D; Pfahler, P, 1992. The effects of genotype and ploidy level on pollen surface sculpturing in maize (*Zea mays* L.). Amer J Bot 79:1418-1422.
645. Spaner, D; Mather, DE; Hamilton, RI, 1992. Agronomic potential of northern temperate zone quality protein maize germplasm in eastern Canada. Plant Var Seeds 5:113-121.
646. Spencer, TM; O'Brien, JV; Start, WG; Adams, TR; Gordon-Kamm, WJ; Lemaux, PG, 1992. Segregation of transgenes in maize. Plant Mol Biol 18:201-210.
647. Sprague, GF, 1992. A complementation anomaly involving certain glossy mutants of maize. Maydica 37:107-110.
648. Sprague, GF; Lamkey, KR, 1992. Arnel R. Hallauer - an appreciation. Maydica 37:1-6.
649. Srinivas, T; Bhashyam, MK; Chand, N; Bhattacharya, S; Murthy, SS; Narasimha, HV, 1991. Relationship of cob characters with grain morphology in maize (*Zea mays*, Poaceae). Econ Bot 45:503-510.
650. Staiger, CJ; Cande, WZ, 1992. Ameiotic, a gene that controls meiotic chromosome and cytoskeletal behavior in maize. Dev Biol 154:226-230.
651. Stall, WM; Bewick, TA, 1992. Sweet corn cultivars respond differentially to the herbicide nicosulfuron. Hortscience 27:131-133.
652. Stamp, P; Kiel, C, 1992. Root morphology of maize and its relationship to root lodging. J Agron Crop Sci 168:113-118.
653. Stamp, P; Kiel, C, 1992. Seedling traits of maize as indicators of root lodging. Agronomie 12:157-162.

654. Stapleton, AE; Bedinger, PA, 1992. Immature maize spikelets develop and produce pollen in culture. Plant Cell Rep 11:248-252.
655. Stidham, MA; Singh, BK, 1991. Imidazolinone-acetohydroxyacid synthase interactions. Pp. 71-135 in Imidazolinone Herbicides. D. L. Shaner and S. L. O'Connor, ed., Boca Raton: CRC Press.
656. Stochaj, U; Silver, PA, 1992. A conserved phosphoprotein that specifically binds nuclear localization sequences is involved in nuclear import. J Cell Biol 117:473-482.
657. Stoilov, LM; Mirkova, V; Zlatanova, J; Djondjurov, L, 1992. Matrix-associated DNA from maize is enriched in repetitive sequences. Plant Cell Rep 11:355-358.
659. Stooksbury, D, 1991. Is climate change already giving us greater maize yields? New Scientist 132:48.
660. Stuber, CW; Lincoln, SE; Wolff, DW; Helentjaris, T; Lander, ES, 1992. Identification of genetic factors contributing to heterosis in a hybrid from 2 elite maize inbred lines using molecular markers. Genetics 132:823-839.
661. Stucker, DS; Hallauer, AR, 1992. Genetic variability as affected by selection in Iowa Stiff Stalk Synthetic maize. J Hered 83:410-418.
662. Sudupak, MA; Bennetzen, JL; Hulbert, SH, 1993. Unequal exchange and meiotic instability of disease-resistance genes in the *Rp1* region of maize. Genetics 133:119-125.
663. Sugiharto, B; Sugiyama, T, 1992. Effects of nitrate and ammonium on gene expression of phosphoenolpyruvate carboxylase and nitrogen metabolism in maize leaf tissue during recovery from nitrogen stress. Plant Physiol 98:1403-1408.
664. Sugiura, M, 1992. The chloroplast genome. Plant Mol Biol 19:149-168.
665. Sullivan, TD; Strelow, LI; Illingworth, CA; Phillips, RL; Nelson, OE, 1991. Analysis of maize brittle-1 alleles and a defective Suppressor-Mutator-induced mutable allele. Plant Cell 3:1337-1348.
666. Sun, Y; Liang, GH, 1991. Pollen surface sculpture of wheat, rice, maize and five sorghum species. Cytologia 56:659-664.
667. Suzuki, Y; Yamane, H; Spray, CR; Gaskin, P; MacMillan, J; Phinney, BO, 1992. Metabolism of *ent*-kaurene to gibberellin A12-aldehyde in young shoots of normal maize. Plant Physiol 98:602-610.
668. Swinburne, J; Balcells, L; Scofield, SR; Jones, JDG; Coupland, G, 1992. Elevated levels of activator transposase mRNA are associated with high frequencies of dissociation excision in *Arabidopsis*. Plant Cell 4:583-595.
669. Symonds, N, 1992. Geneticist's late harvest. The Guardian, September 5, 1992.
670. Tagu, D; Walker, N; Ruiz-Avila, L; Burgess, S; Martinez-Izquierdo, JA; Leguay, JJ; Netter, P; Puigdomenech, P, 1992. Regulation of the maize HRGP gene expression by ethylene and wounding - messenger RNA accumulation and qualitative expression analysis of the promoter by microprojectile bombardment. Plant Mol Biol 20:529-538.
671. Targon, MLN; Ottoboni, LMM; Leite, A; Ludevid, D; Puigdomenech, P; Arruda, P, 1992. Synthesis and deposition of coixin in seeds of *Coix lacryma jobi*. Plant Sci 83:169-180.
673. Tecsi, L; Margoczi, K; Maroti, I, 1991. Characterization of bean and maize genotypes by photosynthetic parameters during the start of illumination. Acta Agron Hung 40:69-78.
674. Thimann, KV; Galinat, WC, 1991. Paul Christoph Mangelsdorf (July 20, 1989-July 22, 1989). Proc Amer Phil Soc 135:469-472.
675. Thomann, EB; Sollinger, J; White, C; Rivin, CJ, 1992. Accumulation of group-3 late embryogenesis abundant proteins in *Zea mays* embryos - roles of abscisic acid and the viviparous-1 gene product. Plant Physiol 99:607-614.
676. Thomas, PA; Felker, FC; Crawford, CG, 1992. Sugar uptake and metabolism in the developing endosperm of tassel-seed tunicate (*Ts5 tu*) maize. Plant Physiol 99:1540-1545.
677. Thompson, GA; Siemieniak, DR; Sieu, LC; Slightom, JL; Larkins, BA, 1992. Sequence analysis of linked maize 22-kDa alpha-zein genes. Plant Mol Biol 18:827-833.
678. Thompson-Coffe, C; Findlay, TAS; Wagner, BA; Orr, AR, 1992. Changes in protein complement accompany early organogenesis in the maize ear. Int J Plant Sci 153:31-39.
679. Timmermans, MCP; Das, OP; Messing, J, 1992. Trans replication and high copy numbers of wheat dwarf virus vectors in maize cells. Nucleic Acids Res 20:4047-4054.
680. Timofeeva, LP; Golubovskaya, IN, 1991. A new type of desynaptic gene in maize revealed by the microspreading method of synaptonemal complexes. Tsitologiya SSSR 33:3-8.
681. Timofeeva, LP; Grebennikova, ZK; Gaft, MS; Golubovskaya, IN, 1991. Ultrastructural features of synaptonemal complexes of maize in norm. Tsitologiya SSSR 33:12.
682. Tito, CM; Poggio, L; Naranjo, CA, 1991. Cytogenetic studies in the genus *Zea*. 3. DNA content and heterochromatin in species and hybrids. Theor Appl Genet 83:58-64.
683. Tobias, RB; Boyer, CD; Shannon, JC, 1992. Alterations in carbohydrate intermediates in the endosperm of starch-deficient maize (*Zea mays* L.) genotypes. Plant Physiol 99:146-152.
684. Tobias, RB; Boyer, CD; Shannon, JC, 1992. Enzymes catalyzing the reversible conversion of fructose-6-phosphate and fructose-1,6-bisphosphate in maize (*Zea mays* L.) kernels. Plant Physiol 99:140-145.
685. Toki, S; Takamatsu, S; Nojiri, C; Ooba, S; Anzai, H; Iwata, M; Christensen, AH; Quail, PH; Uchimiya, H, 1992. Expression of a maize ubiquitin gene promoter-*bar* chimeric gene in transgenic rice plants. Plant Physiol 100:1503-1507.
686. Tollenaar, M; Aguilera, A, 1992. Radiation use efficiency of an old and a new maize hybrid. Agron J 84:536-541.
687. Tollenaar, M; Dwyer, LM; Stewart, DW, 1992. Ear and kernel formation in maize hybrids representing 3 decades of grain yield improvement in Ontario. Crop Sci 32:432-438.
688. Tollenaar, M; Mihajlovic, M, 1991. Bromoxynil tolerance during the seedling phase is associated with genetic grain yield improvement in maize. Can J Plant Sci 71:1021-1027.
689. Tranel, PJ; Dekker, J, 1992. Inheritance of clomazone tolerance in maize seedlings. Maydica 37:137-142.
690. Tsai, CL; Huber, DM; Warren, HL; Tsai, CY, 1991. Effects of cross-pollination on dry matter accumulation, nutrient partitioning and grain yield of maize hybrids grown under different levels of N-fertility. J Sci Food Agr 57:163-174.
691. Tsai, CY; Huber, DM; Warren, HL; Lyznik, A, 1991. Nitrogen uptake and redistribution during maturation of maize hybrids. J Sci Food Agr 57:175-187.
692. Tulsieram, L; Compton, WA; Morris, R; Thomas-Compton, M; Eskridge, K, 1992. Analysis of genetic recombination in maize populations using molecular markers. Theor Appl Genet 84:65-72.

693. Turlings, TCJ; Tumlinson, JH, 1992. Systemic release of chemical signals by herbivore-injured corn. Proc Natl Acad Sci USA 89:8399-8402.
694. Ueda, T; Waverczak, W; Ward, K; Sher, N; Ketudat, M; Schmidt, RJ; Messing, J, 1992. Mutations of the 22-kD and 27-kD zein promoters affect transactivation by the opaque-2 protein. Plant Cell 4:701-709.
695. Vaganov, EA; Ivanov, VB; Vysotskaya, LG, 1991. Histometric analysis of cell length variability in the corn root meristems. Tsitologiya SSSR 33:50-59.
696. Van Lijsebettens, M; Den Boer, B; Hernalsteens, J-P; Van Montagu, M, 1991. Insertional mutagenesis in *Arabidopsis thaliana*. Plant Sci 80:27-38.
697. Vanasch, MAJ; Rijkenberg, FHJ; Coutinho, TA, 1992. Phytotoxicity of fumonisin b1, moniliformin, and t-2 toxin to corn callus cultures. Phytopathology 82:1330-1332.
698. VanWert, SL; Saunders, JA, 1992. Reduction of nuclease activity released from germinating pollen under conditions used for pollen electrotransformation. Plant Sci 84:11-16.
699. Varagona, MJ; Purugganan, M; Wessler, SR, 1992. Alternative splicing induced by insertion of retrotransposons into the maize waxy gene. Plant Cell 4:811-820.
700. Varagona, MJ; Schmidt, RJ; Raikhel, NV, 1992. Nuclear localization signal(s) required for nuclear targeting of the maize regulatory protein opaque-2. Plant Cell 4:1213-1227.
701. Vasal, SK; Srinivasan, G; Beck, DL; Crossa, J; Pandey, S; DeLeon, C, 1992. Heterosis and combining ability of CIMMYT's tropical late white maize germplasm. Maydica 37:217-223.
702. Vasal, SK; Srinivasan, G; Crossa, J; Beck, DL, 1992. Heterosis and combining ability of CIMMTY's subtropical and temperate early-maturity maize germplasm. Crop Sci 32:884-890.
703. Vasal, SK; Srinivasan, G; Pandey, S; Cordova, HS; Han, GC; Gonzalez, F, 1992. Heterotic patterns of 92 white tropical CIMMYT maize lines. Maydica 37:259-270.
704. Vazquez-Ramos, JM; Jimenez, JR, 1990. Stimulation of DNA synthesis and DNA polymerase activity by benzyladenine during early germination of maize axes. Can J Bot 68:2590-2594.
705. Veena, T; Sigamani, N, 1991. Do objects in friezes of Somnathpur Temple (1268-AD) in south India represent maize ears. Curr Sci 61:395-397.
706. Veena, T; Sigamani, N, 1992. Of maize and models - reply. Curr Sci 62:711-712.
707. Venis, MA; Napier, RM, 1991. Auxin receptors - recent developments. Plant Growth Regul 10:329-340.
708. Venis, MA; Napier, RM; Barbier-Brygoo, H; Maurel, C; Perrot-Rechenmann, C; Guern, J, 1992. Antibodies to a peptide from the maize auxin-binding protein have auxin agonist activity. Proc Natl Acad Sci USA 89:7208-7212.
709. Verburg, JG; Smith, CE; Lisek, CA; Huynh, QK, 1992. Identification of an essential tyrosine residue in the catalytic site of a chitinase isolated from *Zea mays* that is selectively modified during inactivation with 1-ethyl-3-(3-dimethylaminopropyl)-carbodiimide. J Biol Chem 267:3886-3893.
710. Villegas, E; Vasal, SK; Bjarnason, M, 1992. Quality protein maize - what is it and how was it developed. Pp. 27-48 in Quality Protein Maize. E. T. Mertz, ed., St. Paul: Amer Assoc Cereal Chem.
711. Villemur, R; Joyce, CM; Haas, NA; Goddard, RH; Kopczak, SD; Hussey, PJ; Snustad, DP; Silflow, CD, 1992. alpha-tubulin gene family of maize (*Zea mays* L.) - evidence for 2 ancient alpha-tubulin genes in plants. J Mol Biol 227:81-96.
712. Voinikov, VK; Korytov, MV, 1991. Low-temperature induction of synthesis of cold shock proteins in seedlings of maize lines and hybrids. Tsitol Genet 25:64-69.
713. Vonallmen, JM; Rottmann, WH; Gengenbach, BG; Harvey, AJ; Lonsdale, DM, 1991. Transfer of methomyl and HmT-toxin sensitivity from T-cytoplasm maize to tobacco. Mol Gen Genet 229:405-412.
714. Vrbova, E; Duhova, V, 1991. Effect of sodium azide on mitotic activity in maize (*Zea mays* L.). Acta Fac Rerum Nat Univ Comenianae Genet 41-46.
715. Vulchinkov, S, 1992. Genotype-environment interaction in maize inbred lines and hybrids. III. Interrelation between the stability parameters of various characters in maize hybrids. Genet Sel 25:16-27.
716. Wagatsuma, T; Nakashima, T; Tawaraya, K, 1991. Identification of aluminium-tolerant protoplasts in the original root protoplast population from several plant species differing in aluminium tolerance. Devel Plant Soil Sci 45:789-793.
717. Walbot, V, 1992. Reactivation of mutator transposable elements of maize by ultraviolet light. Mol Gen Genet 234:353-360.
718. Walbot, V, 1992. Strategies for mutagenesis and gene cloning using transposon tagging and T-DNA insertional mutagenesis. Annu Rev Plant Physiol 43:49-82.
719. Walsh, NE; Charlesworth, D, 1992. Evolutionary interpretations of differences in pollen tube growth rates. Quart Rev Biol 67:19-37.
720. Walsh, TA; Morgan, AE; Hey, TD, 1991. Characterization and molecular cloning of a proenzyme form of a ribosome-inactivating protein from maize - novel mechanism of proenzyme activation by proteolytic removal of a 2.8 kilodalton internal peptide segment. J Biol Chem 266:23422-23427.
721. Walters, DA; Vetsch, CS; Potts, DE; Lundquist, RC, 1992. Transformation and inheritance of a hygromycin phosphotransferase gene in maize plants. Plant Mol Biol 18:189-200.
722. Wan, Y; McMurphy, LM; Rayburn, AL; Widholm, JM, 1992. Ploidy levels of plants regenerated from mixed ploidy maize callus cultures. In Vitro Cell Dev Biol-Plant 28P:87-89.
723. Wan, Y; Rocheford, TR; Widholm, JM, 1992. RFLP analysis to identify putative chromosomal regions involved in the anther culture response and callus formation of maize. Theor Appl Genet 85:360-365.
724. Wan, Y; Widholm, JM, 1992. Formation of multiple embryo-like structures from single microspores during maize anther culture. Plant Cell Rep 11:529-531.
725. Wang, H; Slater, GP; Fowke, LC; Saleem, M; Cutler, AJ, 1991. Comparison of cell wall regeneration on maize protoplasts isolated from leaf tissue and suspension cultured cells. In Vitro Cell Dev Biol Plant 27P:70-76.
726. Wang, H-X; Niu, Z-Q; Hu, Z-A, 1989. Electrophoretic study on seed proteins of maize hybrids and inbreds. Chin J Bot 1:139-144.
727. Wang, J; Barth, J; Abbott, AG, 1991. Higher plant mitochondrial DNA expression. 1. Variant expression of the plant mitochondrial open reading frame, ORF25, in B37N and B73N maize lines. Theor Appl Genet 82:765-770.

728. Wang, J-L; Sun, J-S; Lu, T-G; Fang, R; Cui, H-R; Cheng, S-Z; Yang, C, 1991. Fertilization and embryo development in wheat x maize crosses. Acta Bot Sin 33:674-679.
729. Wang, SM; Wu, SY; Chen, J, 1992. Purification and characterization of beta-amylase in maize kernel. Bot Bull Acad Sin 33:359-367.
730. Wang, YJ; White, P; Pollak, L, 1992. Thermal and gelling properties of maize mutants from the Oh43 inbred line. Cereal Chem 69:328-334.
731. Warner, RL; Kleinhofs, A, 1992. Genetics and molecular biology of nitrate metabolism in higher plants. Physiol Plant 85:245-252.
732. Watson, NR; Peschke, VM; Russell, DA; Sachs, MM, 1992. Analysis of L-alanine-2-oxoglutarate aminotransferase isozymes in maize. Biochem Genet 30:371-383.
733. Watve, MG, 1992. Of maize and models. Curr Sci 62:711.
734. Webster, C; Gaut, RL; Browning, KS; Ravel, JM; Roberts, JKM, 1991. Hypoxia enhances phosphorylation of eukaryotic initiation factor 4A in maize root tips. J Biol Chem 266:23341-23346.
735. Weerathaworn, P; Soldati, A; Stamp, P, 1992. Seedling root development of tropical maize cultivars at low water supply. Angew Bot 66:93-96.
736. Weglohner, W; Subramanian, AR, 1992. Nucleotide sequence of a region of maize chloroplast DNA containing the 3' end of *clpP*, exon 1 of *rps12* and *rpl20* and their cotranscription. Plant Mol Biol 18:415-418.
737. Weil, CF; Marillonnet, S; Burr, B; Wessler, SR, 1992. Changes in state of the *Wx-m5* allele of maize are due to intragenic transposition of *Ds*. Genetics 130:175-185.
738. Weiland, RT, 1992. Cross-pollination effects on maize (*Zea mays* L.) hybrid yields. Can J Plant Sci 72:27-33.
739. Wen, L; Huang, JK; Zen, KC; Johnson, BH; Muthukrishnan, S; Mackay, V; Manney, TR; Manney, M; Reeck, GR, 1992. Nucleotide sequence of a cDNA clone that encodes the maize inhibitor of trypsin and activated Hageman factor. Plant Mol Biol 18:813-814.
740. Wessler, SR, 1991. The maize transposable *Ds1* element is alternatively spliced from exon sequences. Mol Cell Biol 11:6192-6196.
741. West, DR; Kincer, DR; Graves, CR, 1992. Registration of T171 parental line of maize. Crop Sci 32:512.
742. Whitkus, R; Doebley, J; Lee, M, 1992. Comparative genome mapping of sorghum and maize. Genetics 132:1119-1130.
743. Widstrom, NW, 1992. Aflatoxin in developing maize - interactions among involved biota and pertinent econiche factors. Pp. 23-58 in Handbook of Applied Mycology, Vol 5.
744. Widstrom, NW; Bondari, K; McMillian, WW, 1992. Hybrid performance among maize populations selected for resistance to insects. Crop Sci 32:85-89.
745. Widstrom, NW; Williams, WP; Wiseman, BR; Davis, FM, 1992. Recurrent selection for resistance to leaf feeding by fall armyworm on maize. Crop Sci 32:1171-1174.
746. Wiesler, F; Horst, WJ, 1992. Differences between maize cultivars in yield formation, nitrogen uptake and associated depletion of soil nitrate. J Agron Crop Sci 168:226-237.
747. Wilkes, G, 1991. In situ conservation of agricultural systems. Pp. 86-101 in Biodiversity: Culture, Conservation, and Ecodevelopment. M. L. Oldfield and J. B. Alcorn, ed., Boulder: Westview Press.
748. Wilkes, G, 1992. Strategies for Sustaining Crop Germplasm Preservation, Enhancement, and Use. Washington: Consult Group Internatl Agric Res.
749. Williams, BA; Tsang, A, 1992. Nucleotide sequence of an abscisic acid-responsive, embryo-specific maize gene. Plant Physiol 100:1067-1068.
750. Williams, WP; Buckley, PM, 1992. Growth of fall armyworm (Lepidoptera, noctuidae) larvae on resistant and susceptible corn. J Econ Entomol 85:2039-2042.
751. Williams, WP; Windham, GL, 1992. Reaction of a diallel cross of maize to *Meloidogyne incognita* under field conditions. Field Crop Res 30:167-171.
752. Williamson, JD; Scandalios, JG, 1992. Differential response of maize catalases and superoxide dismutases to the photoactivated fungal toxin cercosporin. Plant J 2:351-358.
753. Williamson, JD; Scandalios, JG, 1992. Differential response of maize catalases to abscisic acid - *vp1* transcriptional activator is not required for abscisic acid-regulated *cat1* expression. Proc Natl Acad Sci USA 89:8842-8846.
754. Wilson, CM, 1992. Techniques for characterization of maize endosperm storage proteins and identification of nutritionally improved maize. Pp. 141-154 in Quality Protein Maize. E. T. Mertz, ed., St. Paul: Amer Assoc Cereal Chem.
755. Wilson, CM, 1992. Zein diversity in Reid, Lancaster, and Illinois chemical corn strains revealed by isoelectric focusing. Crop Sci 32:869-873.
756. Wilson, CM, 1992. Zeins in sweet corn (sugary-1). Cereal Chem 69:113-114.
757. Wilson, DO; Trawatha, SE, 1991. Physiological maturity and vigor in production of Florida Staysweet shrunken-2 sweet corn seed. Crop Sci 31:1640-1647.
758. Winning, BM; Sarah, CJ; Purdue, PE; Day, CD; Leaver, CJ, 1992. The adenine nucleotide translocator of higher plants is synthesized as a large precursor that is processed upon import into mitochondria. Plant J 2:763-773.
759. Wiseman, BR; Bondari, K, 1992. Genetics of antibiotic resistance in corn silks to the corn earworm (Lepidoptera, Noctuidae). J Econ Entomol 85:293-298.
760. Wiseman, BR; Isenhour, DJ, 1992. Relationship of planting dates and corn earworm developmental parameters and injury to selected maize entries. Maydica 37:149-156.
761. Wiseman, BR; Widstrom, NW, 1992. Resistance of corn populations to larvae of the corn earworm (Lepidoptera, Noctuidae). J Econ Entomol 85:601-605.
762. Wiseman, BR; Widstrom, NW, 1992. Resistance of dent corn inbreds to larvae of the corn earworm (Lepidoptera, Noctuidae). J Econ Entomol 85:289-292.
763. Wright, AD; Moehlenkamp, CA; Perrot, GH; Neuffer, MG; Cone, KC, 1992. The maize auxotrophic mutant orange pericarp is defective in duplicate genes for tryptophan synthase-beta. Plant Cell 4:711-719.
764. Wu, J; Tian, Z; Yu, X; Man, S, 1991. Studies on combining ability of selecting line transferred tropical germplasm in maize population. Acta Agron Sin 17:424-429.
765. Wurtzel, ET, 1992. Use of a *Ds* chromosome-breaking element to examine maize *Vp5* expression. J Hered 83:109-113.

766. Xie, YS; Arnason, JT; Philogene, BJR; Atkinson, J; Morand, P, 1992. Behavioral responses of Western corn rootworm larvae to naturally occurring and synthetic hydroxamic acids. J Chem Ecol 18:945-957.
767. Xin, ZG; Li, PH, 1992. Abscisic acid-induced chilling tolerance in maize suspension-cultured cells. Plant Physiol 99:707-711.
768. Xu, Y; Jia, J-F; Zheng, G-C, 1990. Interaction and transformation of cereal cells with phenolics-pretreated *Agrobacterium tumefaciens*. Chin J Bot 2:81-87.
769. Yadav, K; Prasad, V; Ahmad, N; Mandal, K, 1992. Response of maize genotypes to *Azospirillum lipoferum* in calcareous soils. J Indian Soc Soil Sci 40:195-197.
770. Yamada, M; Suenaga, K; Nakajima, K, 1992. Heterosis in plants starts immediately after fertilization. Pp. 426-428 in Angiosperm Pollen and Ovules. E. Ottaviano, ed., Berlin: Springer-Verlag.
771. Yanagisawa, S; Izui, K, 1992. MNF1, a leaf tissue-specific DNA-binding protein of maize, interacts with the cauliflower mosaic virus 35S promoter as well as the C(4) photosynthetic phosphoenolpyruvate carboxylase gene promoter. Plant Mol Biol 19:545-553.
772. Yanagisawa, S; Mori, Y; Kawamura, T; Izui, K, 1991. Mapping of non-methylated and methylated restriction sites in the promoter region of the maize gene for phosphoenolpyruvate carboxylase involved in C-4 photosynthesis. Agr Biol Chem Tokyo 55:2877-2879.
773. Yang, G; Wiseman, BR; Espelie, KE, 1992. Cuticular lipids from silks of 7 corn genotypes and their effect on development of corn earworm larvae [*Helicoverpa zea* (Boddie)]. J Agr Food Chem 40:1058-1061.
774. Yang, HY; Zhou, C, 1992. Experimental plant reproductive biology and reproductive cell manipulation in higher plants - now and the future. Amer J Bot 79:354-363.
775. Yun, SH; Matheson, NK, 1992. Structural changes during development in the amylose and amylopectin fractions (separated by precipitation with concanavalin-A) of starches from maize genotypes. Carbohydr Res 227:85-101.
776. Yunusa, IAM; Gworgwor, NA, 1991. Growth and yield of maize genotypes during dry seasons in northern Nigeria. Exp Agr 27:397-405.
777. Zanette, VA; Paterniani, E, 1992. The brachytic-2 gene effect in maize improved populations of reduced height plants. Pesquisa Agr Brasil 27:1173-1181.
778. Zargar, GH; Singh, RM, 1990. Genetic architecture of an improved version of a maize composite. I. Genetic analysis of grain yield, yield components and some agronomic traits. Indian J Genet Plant Breed 50:221-227.
779. Zehr, BE; Dudley, JW; Chojecki, J, 1992. Some practical considerations for using RFLP markers to aid in selection during inbreeding of maize. Theor Appl Genet 84:704-708.
780. Zehr, BE; Dudley, JW; Chojecki, J; Maroof, MAS; Mowers, RP, 1992. Use of RFLP markers to search for alleles in a maize population for improvement of an elite hybrid. Theor Appl Genet 83:903-911.
781. Zettl, R; Feldwisch, J; Boland, W; Schell, J; Palme, K, 1992. 5'-azido-[3,6-H-3(2)]-1-naphthylphthalamic acid, a photoactivatable probe for naphthylphthalamic acid receptor proteins from higher plants - identification of a 23-kDa protein from maize coleoptile plasma membranes. Proc Natl Acad Sci USA 89:480-484.
782. Zhang, C-L; Chen, G-C, 1991. Effects of mefluidide on membrane lipids and fatty acids of mitochondria isolated from corn leaves and its relation to chilling-resistance of corn seedling. Acta Phytophysiol Sin 17:8-14.
783. Zhang, G; Williams, CM; Campenot, MK; McGann, LE; Cass, DD, 1992. Improvement of longevity and viability of sperm cells isolated from pollen of *Zea mays* L. Plant Physiol 100:47-53.
784. Zhang, GC; Campenot, MK; McGann, LE; Cass, DD, 1992. Flow cytometric characteristics of sperm cells isolated from pollen of *Zea mays* L. Plant Physiol 99:54-59.
785. Zhang, W-Q; Tang, X-Z, 1991. Study on heat-sensitivity of esterase isozymes in maize inbreds. Acta Bot Sin 33:687-691.
786. Zhong, H; Srinivasan, C; Sticklen, MB, 1992. In vitro morphogenesis of corn (*Zea mays* L.). I. Differentiation of multiple shoot clumps and somatic embryos from shoot tips. Planta 187:483-489.
787. Zhong, H; Srinivasan, C; Sticklen, MB, 1992. In vitro morphogenesis of corn (*Zea mays* L.). II. Differentiation of ear and tassel clusters from cultured shoot apices and immature inflorescences. Planta 187:490-497.
788. Zhu, D; Scandalios, JG, 1992. Expression of the maize MnSOD (*Sod3*) gene in MnSOD-deficient yeast rescues the mutant yeast under oxidative stress. Genetics 131:803-809.
789. Zhu, X-Y; Negrutiu, I, 1991. Isolation and culture of protoplasts. Pp. 18-27 in Biomethods, Vol. 4. A Laboratory Guide for Cellular and Molecular Plant Biology. I. Negrutiu and G. G. Gharti-Chhetri, ed., Basel: Birkhaeuser Verlag.
790. Zummo, N; Scott, GE, 1992. Interaction of *Fusarium moniliforme* and *Aspergillus flavus* on kernel infection and aflatoxin contamination in maize ears. Plant Dis 76:771-773.

IX. SYMBOL INDEX

("r" refers to numbered references in the Recent Maize Publications section)

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a1-m(Au)pale 5
a1-m3 r91
a1-m16 r531
a1-m15719 5
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a1-Mum3 r571
a1-ruq r91 r531
a2 33 r163 r527 r718
a3 33 112 r527
ab r235
abp* r89 r383 r477 r489 r708
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abp1 r283 r328 r707
ABPHYL r272
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Ac2 r91
Ac11 3
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Acc1-h2 124 r442
Acc1-s1 124 r442
Acc1-s2 124 r442
Acc1-s3 124 r442
aco1 122
acp* 124 r464
acp1 124 r183 r283 r461 r505 r583 r599
act* r418
act1 124 r599
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ad2-2356A 33 124
adh1 32 106 120 121 r181 r210 r413 r423 r461 r505 r586 r591 r692
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adh1-3F1124r17 120
Adh1-Cm r89
Adh1-Fm r531
Adh1-S r418
adh2 24 r283 r591
adp* r283
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afd1 r24 r136
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agp2 7 121
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ba2 2
ba3 2
Bar 54 r646
BE-I* 125 r29
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bm1 33
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bm3 r118
bm4 33
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April, 1993

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